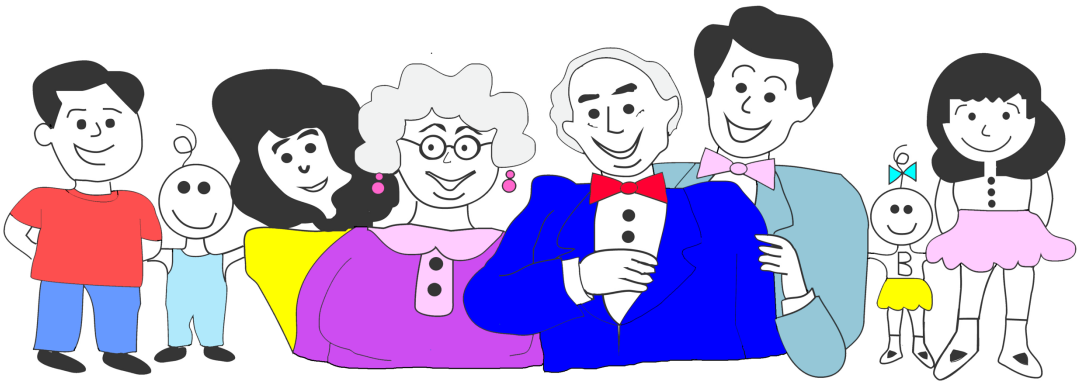
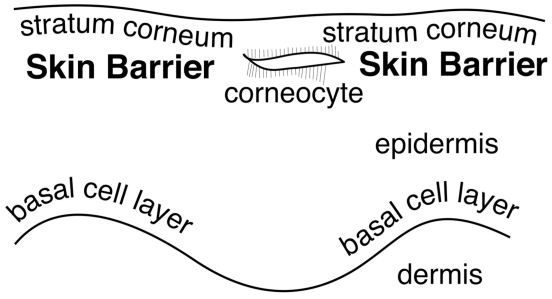


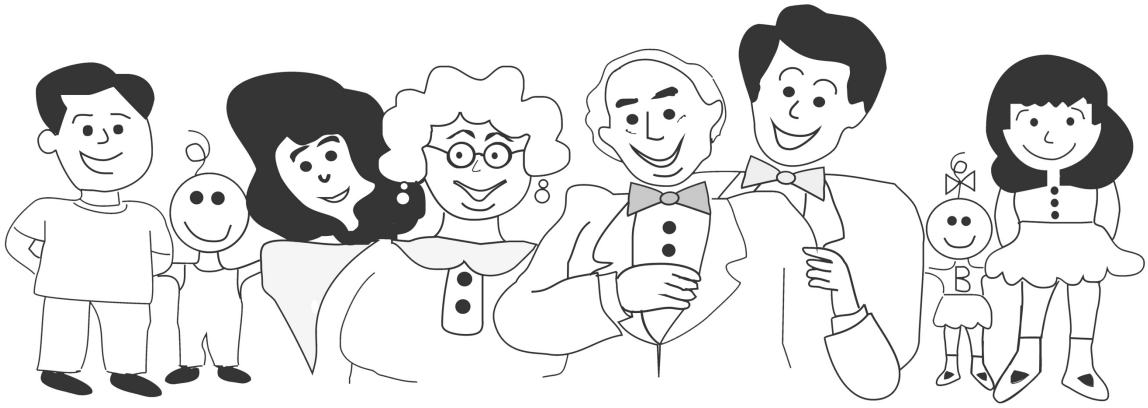
Dermatology Guidelines for the Primary Care* Resident: The Essentials

*Family Medicine
Internal Medicine
Pediatrics

by

Randy Jacobs, M.D.
Natalia Jacobs, M.D.





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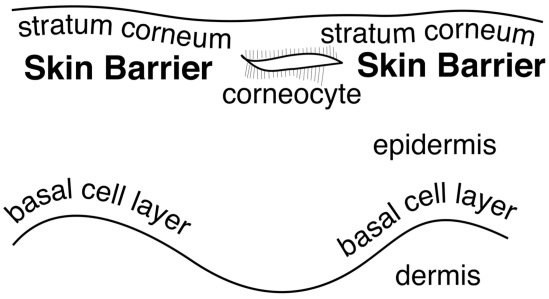
G. Alden Holmes, MD
Srita Chakka, MD (2020)
Chad Bowyer, BS
Jeffrie C. Jacobs



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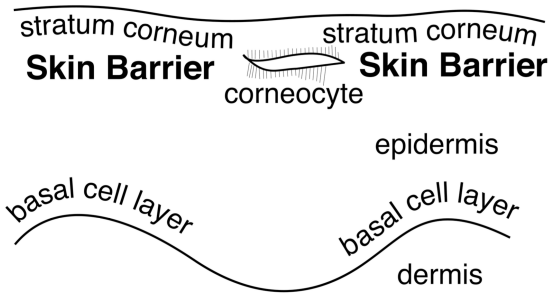

Dermatology Guidelines for the Primary Care Resident: The Essentials, is available as a free download. In the interest of quality resident physician education, this Guidelines notebook is 100% free and is not for sale. Please download and freely distribute as many copies as you wish. Dermatology Guidelines for the Primary Care Resident: The Essentials was written, not for profit, but purely to educate and guide primary care resident physicians in training as they complete their required one month dermatology rotation.

Natalia Jacobs, MD
Randy Jacobs, MD, FAAD

FREE DOWNLOAD

Go to:

http://www.randyjacobsmd.com/DermatologyGuidelines.pdf

Dermatology Guidelines for the Primary Care Resident: The Essentials

A greeting from the authors:

Hello Primary Care Residents (Family Medicine, Internal Medicine, Pediatrics) and Program Directors. Welcome to the Guidelines! Here are two important facts regarding dermatology in primary care medicine:

1. In a US study during a 2-year period, **36.5%** of patients who presented to their primary care physician had at least one skin problem. J Am Acad Dermatol. 2001 Aug; 45 (2): *250-5*
2. Primary care residents failed **50%** of the time to correctly *diagnose* non-melanoma skin cancer and malignant melanomas. *Arch Dermatol. 1996; 132: 1030-1038*

Residents and Directors, 36.5% of your primary care patients will present with a derm diagnosis. And, during residency, primary care residents are only required to complete a month-long dermatology rotation. One month is, simply, not enough, but it is all they get. Thus, in order to make that one month the best that it can be, we have written: **Dermatology Guidelines for the Primary Care Resident: The Essentials.**

Residents and Directors, what is the Guidelines notebook all about? The Guidelines is a **free** downloadable notebook style book that is like a flashlight in a dark cave. As you go through your primary care residency derm rotation, you can carry it with you at all times. The Guidelines will shine light on what you really need to understand in your brief one month dermatology rotation. Yes, derm is a deep abyss of more than 3,000 diagnoses. So, **what is most important** for the primary care resident to learn in one month? Answer: The Guidelines. Unlike big textbooks, the Guidelines is quick to the point to teach what **residents** need to know. So, don't get lost in the big dark cave, just learn Guidelines, and you will learn what you really need to know for 90% of your future primary care derm patients. Rather than confuse residents with piles of esoteric dermatology, we emphasize the basic diagnoses they will surely encounter in their everyday practice of primary care medicine. Thus, you can learn to diagnose and treat the basics, and, learn when to refer.

The Guidelines downloadable format allows you to insert your Derm Guidelines into a personalized 3-ring notebook. As you study, you can add your own notes, photos, and pages. You can photocopy articles and create your own unique derm notebook you can keep and use for your patients now, and in years to come.

The Guidelines notebook is not designed to be an authoritative referenced textbook. Rather, the Guidelines present derm concepts in a simple conversational style, so that residents can learn derm in a clear easy to understand way. The Guidelines notebook was also edited to be reader friendly for Physician Assistants.

In derm a picture is worth 1000 words. And, because thousands of high quality derm photos are already available on the internet for the residents to study, the Guidelines notebook has no photos. So, for helpful photos please refer to Visualdx.com or Derm101.com or similar photo based educational derm websites. Primary care residents, we wish you the **very best** from your one month clinical dermatology rotation!

Sincerely,
Natalia Jacobs, MD
Jacobs Natalia

PS, Residents, I am happy to personally help you with any derm questions you may have. Please feel free to email me. natashajacobsmd@gmail.com I understand what it's like caring for primary care patients as I am also a PCP. But, in Russia where I am from, I also learned extra dermatology- which is nice for any PCP to know. Now in the USA, my goal is to complete a primary care residency and to work as a PCP with derm as my extra interest. I am applying for the 2019 Match, so, if you know of any helpful residency programs for me to apply to, please send me an email. Thank you! ☺

Randy Jacobs, MD, FAAD
Randy J Jacobs, MD

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Dermatology Guidelines for the Primary Care Resident: The Essentials

Acknowledgements

Dermatology Guidelines for the Primary Care Resident: The Essentials required hours and hours of preparatory work. Special thanks go to our future dermatologists, University of California Riverside School of Medicine medical students: Calvin, Alden, & Srita. We are grateful for your contributions to the Dermatology Guidelines book to make it the best. You are each remarkable, inspiring, and a complete pleasure to work with. We wish you much happiness in medicine as you enter your dermatology residency programs.

Calvin Sung, MD (2019)

G. Alden Holmes, MD

Srita Chakka, MD (2020)

Special appreciation goes to University of Redlands pre-med student, Chad Bowyer. Chad, thank you for your conscientious time in proofreading the Dermatology Guidelines book. We wish you great success in medical school as you continue your studies.

Chad Bowyer, BS

A Word for Physician Assistants

In recent years, Physician Assistants have become increasingly important in providing mid-level care to dermatology patients. While the Dermatology Guidelines book was written for primary care physicians in training, we realize that most PA's are primary care based, and that PA students will also find the Dermatology Guidelines useful as they complete rotations in dermatology.

One point about medical educational books, in general, is that most books are written assuming that readers have basic medical science background at the physician level. However, as mid-level providers, PA's do not typically receive this med-school level of medical science education. With this in mind, Jeffrie C. Jacobs came on board with the Dermatology Guidelines and served as our manuscript editor. In editing, Jeffrie spent hours reviewing the Guidelines to assure that our Dermatology Guidelines, written for physicians in training, would also be reader friendly for PA's in training. Thus, a very special thank you to our manuscript editor, future Physician Assistant, Jeffrie C. Jacobs, pre-Physician Assistant and Applied Health Science student at Point Loma University.

Jeffrie C. Jacobs

Dermatology Guidelines for the Primary Care Resident: The Essentials

by
Randy Jacobs, MD, FAAD
Natalia Jacobs, MD

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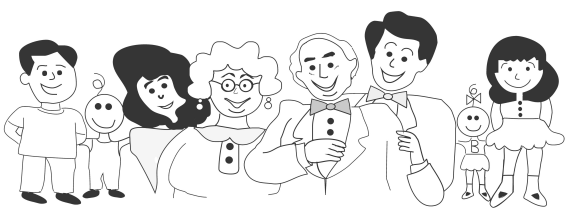


Dermatology Guidelines for the Primary Care Resident: The Essentials

Disclaimer

Dermatology Guidelines for the Primary Care Resident: The Essentials, is not an authoritative textbook. It is simply a dermatology notebook in which residents can add their own unique notes, photos, and pages to keep and use for their own patient care, now, and in years to come. The Dermatology Guidelines notebook was designed to assist residents in their learning of dermatology. If any diagnosis or treatment is in question, please refer to the following dermatology textbooks for more definitive answers.

Textbook References

1. **Fitzpatrick's Dermatology in General Medicine, Eighth Edition,**

by Lowell A Goldsmith , Stephen I. Katz , Barbara A. Gilchrest , Amy Paller , David J. Leffell , Klaus Wolff

2. **Dermatology: 4th Edition**

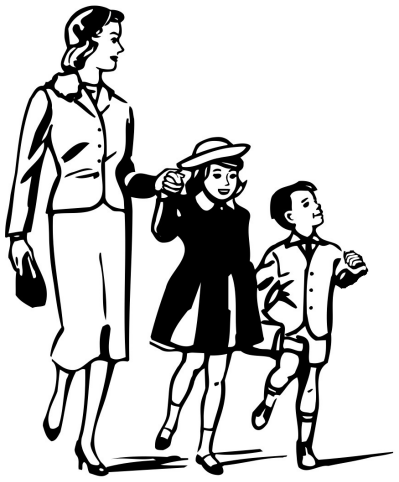
by Jean L. Bolognia MD , Julie V. Schaffer MD , Lorenzo Cerroni

3. **Andrews' Diseases of the Skin: Clinical Dermatology, 12th Edition**

by William D. James MD , Dirk Elston MD , Timothy Berger MD , Isaac Neuhaus MD

4. **Hurwitz Clinical Pediatric Dermatology: A Textbook of Skin Disorders of Childhood and Adolescence, 5th Edition**

by Amy S Paller , Anthony J. Mancini MD



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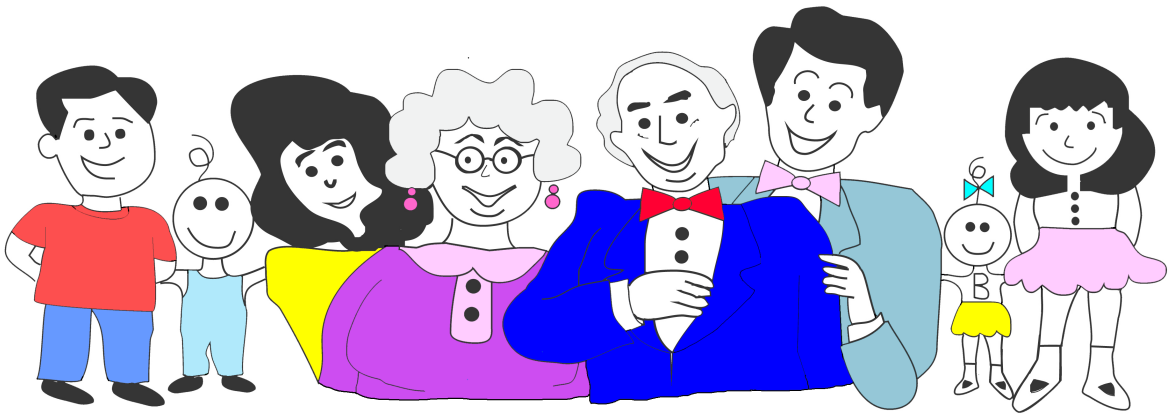
Questions? Comments? Suggestions? Are there any other topics you would like to see in the Guidelines?

Please contact the author, Natalia Jacobs, MD

Email: natashajacobsmd@gmail.com



Dry, Sensitive, Itchy, Inflammatory Skin Conditions Section 1

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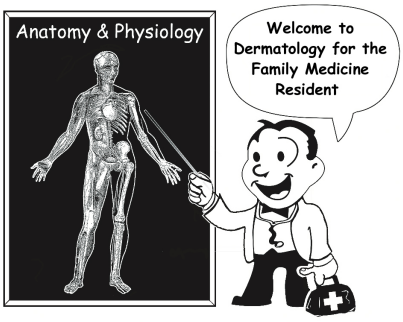
Skin Barrier Microanatomy & Physiology



UNDERSTANDING THE HUMAN SKIN BARRIER

"Get your facts first, and then you can distort them as much as you please." Mark Twain

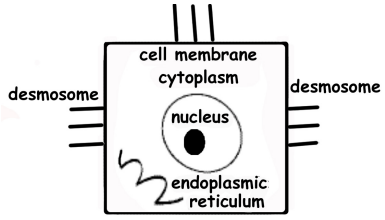
Not counting acne, if all of your primary care patients practiced #1) daily sun safety and #2) daily skin barrier moisturization, most of their skin problems would be gone. In this light, for the primary care physician, your most important goal should be to educate your patients on these two daily duties. With >2000 derm diagnoses, it's easy to get confused. But, for the primary care resident, let's set aside all of the esoteric and complicated diagnoses in dermatology, and let's focus on the most important. #1 sun safety. #2 skin barrier moisturization. Now, basic sun safety is straightforward; simply, block the sun and supplement with oral vitamin D and oral nicotinamide 500 mg po qd. On the other hand, skin barrier moisturization is not so simple.

In an ocean of new knowledge unfolded in the last 20 years, with sun safety as #1, healthy skin barrier moisturization has now become the #2 most important patient educational goal in dermatology. And, it would be an unfortunate omission for a primary care resident to not learn about the skin barrier during his or her time in resident education.

To better help you manage your future primary care dermatology patients, we have written this basic science section on normal skin barrier microanatomy & physiology. The more you understand the skin barrier, the more you will be able to help your primary care patients with their dry-sensitive eczematous skin problems.

The Basic Skin Cell

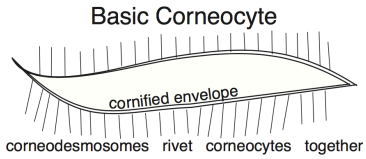
Here's a diagram of a generic basal skin cell:



A basal skin cell contains cytoplasm, a nucleus filled with DNA and an endoplasmic reticulum where proteins are made. All of this is packaged together within the cell membrane. On the outside are little hair like projections called desmosomes. Like tiny rivets, the desmosomes attach corneocyte cells together to promote adhesive strength.

Corneocyte

In medicine, the word "corn" refers to an area of hardened skin. A corneocyte (a hardened skin cell) is the specific skin cell that makes up your skin barrier. Skin cells begin as basal skin cells and eventually mature into hardened corneocyte skin cells. Study the following diagram. A corneocyte is a flattened cell with no nucleus and no cytoplasm. It is considered to be "dead" by some, but the corneocyte is not really "dead" as some may think. Though the nucleus is gone, certain important enzymatic reactions still take place inside the corneocyte. A corneocyte also functions osmotically. By "Osmotic" we are referring to water as it moves by osmosis into and out of a corneocyte skin cell.



On the inside, a corneocyte cell mainly contains:

1. Natural moisturizing factor (a water magnet).

2. Insoluble keratin proteins (for skin rigidity).

On the outside, a cornified envelope covers the entire corneocyte like a protein-fat coated brick. This resilient, water permeable, cornified

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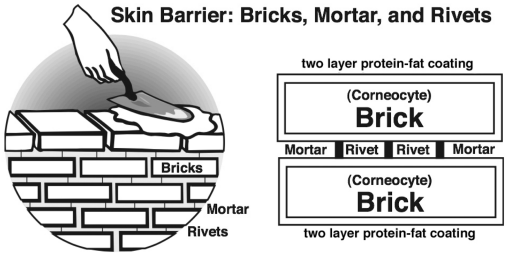
envelope is made of a layer of protein joined to a layer of fat. Countless corneocytes are embedded together in a waterproof lipid-fat matrix made of ceramides, cholesterol, and free fatty acids. Depending on the state of hydration, air temperature, and outside humidity, water can move into or out of the corneocytes.

Little spikes surrounding the corneocyte are called corneodesmosomes. These tiny spikes work like "rivets" to hold corneocytes together. In time, the corneodesmosomes will enzymatically dissolve, and the corneocytes will invisibly flake away like dust in the wind. This flaking process is your natural exfoliation.

Bricks, Mortar, and Rivets

You can visualize the skin barrier by picturing a wall of bricks. Each brick (the corneocyte) is protein-fat coated (the cornified envelope) and is surrounded with mortar (lipids). Spike-like "rivets" hold the bricks firmly together (corneodesmosomes). Envision this:



Now that you have photographed this brick, mortar, rivet image into your mind, let's give the picture a bit more clarity: Location. The brick, mortar, rivet skin barrier is located and works in the stratum corneum of the skin, also known as the cornified, or horny cell layer, situated in the outermost layers of the epidermis.

Anatomy: Location, Location, Location

If you look at a sample of skin under the microscope, you can see three distinct skin layers:

1. Epidermis, the outermost layer

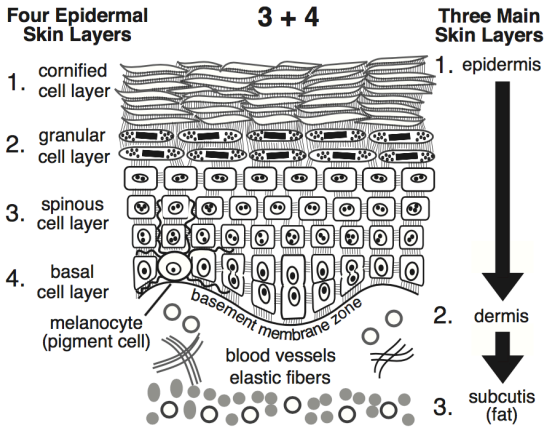
2. Dermis, the middle layer

3. Subcutis, the inner layer

Where is the Skin Barrier Located?

Your bricks-mortar-rivets skin barrier is located in the outer-most layers of the epidermis. There, you will find uncountable corneocytes in place, all interconnected like bricks-mortar-rivets to form your skin's protective barrier.

For a clearer understanding of the 3 + 4 layers, of the skin, please take a minute to review this diagram. So you can better explain skin problems to your primary care patients, it would be a good idea for you to memorize this diagram and to be able to draw a simple version of it.



Skin Layers Explained

Epidermis: The epidermis is the superficial layer and includes four layers situated above the basement membrane zone. Each layer features its own unique cell: 1. Basal, 2. Spinous, 3. Granular, and 4. Cornified. Pigment cells, called melanocytes, and immune cells, called Langerhans cells also reside in the epidermis.

Basement Membrane Zone: The basement membrane zone separates the dermis from the epidermis and keeps your epidermis attached to your dermis like "Velcro" adhesive. Without the basement membrane zone attaching the epidermis to the dermis, your skin would peel away like a peeled orange.

Dermis: The dermis is the middle layer and contains collagen and elastic tissue for flexible support. Lymph vessels, blood vessels, immune cells, fibroblast cells, blood cells, nerves, sweat glands, oil glands, and hair follicles also reside and work within the dermal layer of the skin.

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Subcutis: The subcutis is the deep layer that stores your fat energy, protects your inner body from heat and cold, and pads your body with supportive mechanical cushioning.

Physiology: Still Photo Becomes Motion Picture

So far, we've discussed skin microanatomy. Anatomy is like a photograph, while physiology is like a movie. Anatomy shows the basic parts; physiology explains how the parts move and work in time and space. Anatomy shows you the motor sitting in a car; physiology shows the motor running in the car. The next pages are devoted to physiology.

Step One, Step Two, Step Three...

Like a home movie, every working skin part is moving and changing in space and time. As skin barrier development can be a complicated topic, we will learn it in steps, frame by frame. Step one happens first, step two happens second, step three happens third, and so on. Please understand that everything is actually happening at the same time. We are only using individual "steps" for the sake of simplicity.

In the Embryo

Complete skin barrier formation occurs late in embryogenesis and is essential for terrestrial living. The human skin barrier, which protects the body from dehydration, mechanical trauma, and microbial insults, is not fully functional until about 32 weeks of gestation. Prior to that, it is still developing and is one very important factor affecting survival of preterm infants, especially those less than 24 weeks.

The human skin barrier is weakest at birth, but with exposure to the outside world, the barrier becomes stronger and more efficient. Abnormal skin development and barrier dysfunction are associated with critical hypothermia, perinatal dehydration, and systemic infections in premature infants. Interestingly, though high doses of retinoic acid result in teratogenicity, a small amount of retinoic acid is critical for timely periderm desquamation and formation of the cornified envelope. In retinoic acid deficiency we see loss of skin barrier function with disruption of lipid layers and a faulty cornified envelope.

Step One: What Happens at the Beginning?

To make the long story short, in term infants and beyond, skin barrier development begins with a simple basal cell and ends with a corneocyte. First, the basal cell divides to produce a spinous cell. As skin cells travel to the surface, they go through several internal and external changes. They finally mature into stratum corneum corneocyte cells via four developmental stages:

A. Basal cells at the bottom layer, divide to make ->

B. Spinous cells with spikes. These develop into ->

C. Granular cells with granules. These become ->

D. Cornified cells, AKA corneocytes or "bricks."

Lastly, the cornified cells form the "brick-mortar-rivet" skin permeability barrier. It takes at least 14 days for a basal cell to mature and migrate from the basal layer to the corneal layer.

Step Two: What Is the Intercellular Space?

The analogy of little bricks can help you to better understand skin microanatomy and how the anatomy relates to dry and sensitive skin problems in the primary care clinic.

Once again, referring to the bricks and mortar skin barrier model, the "mortar" is the space in between the corneocyte cells. This space is also referred to as the "intercellular space." The intercellular space is important for two reasons. First, this space is where corneocyte cells stick together via corneodesmosomes (rivets) like bricks in a wall, giving shear strength to your skin. Second, the intercellular space contains the bilayered lipids cholesterol, ceramide, and free fatty acids that stop unwanted agents, toxins, allergens, and infectants from entering your body, and prevent vital water from leaving your body. Recall the location of the skin barrier. Note the "fuzzy" corneocyte (brick) situated in the middle of the diagram:

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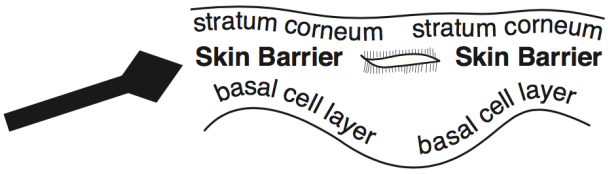
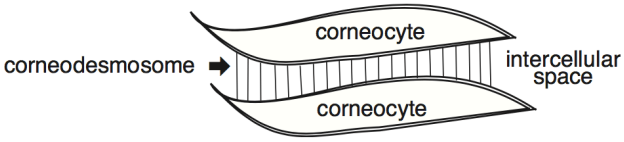
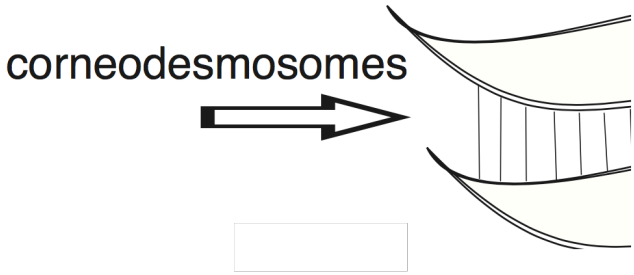


This next diagram shows the intercellular space area. This location is where the lipids are eventually deposited via lamellar granules and membrane fusion.



In the intercellular space, skin cells are joined together by special rivet like adhesive junctions called desmosomes. Microscopically, these are seen as the spines or spikes of the spinous layer. By the time the skin cells reach the cornified layer, these rivet like cell connectors still join corneocytes together. At this point, they are given the name “corneodesmosomes.” Here is a close-up view of the intercellular space.

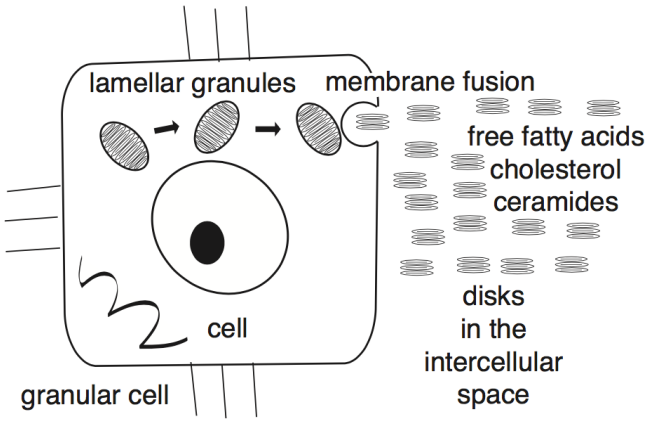
Note, the fine vertical lines represent tiny corneodesmosomes joining two corneocytes together within the intercellular space.



Step Three: Where Do the Lipids Come From?

Essentially, your three skin barrier lipids originate from the daily foods you eat. Your basic foods are first metabolized into lipid-fat precursors. Your skin barrier’s lipids then gather inside granular cells and are packaged as submicroscopic “disks” within tiny oval sacks called “lamellar” granules. Lamellar granules contain all the necessary lipid precursors and enzymes needed to form your skin barrier’s three intercellular lipids.

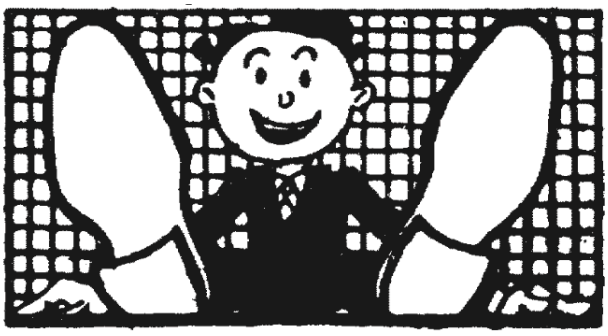
Just before a granular cell becomes a cornified cell, the lipid-fat disks are extruded outside of and in between cells, creating a waterproof lipid-fat-oil barrier made of 40% ceramides, 25% cholesterol, and 20% free fatty acids (FFA’s). The following diagram shows how the lipid disks are extruded by “membrane fusion” into the intercellular space, to be subsequently organized into bilayers:



Step Four:

How Are the Lipid-Fat Disks Organized?

After extrusion into the intercellular space, the lipid-filled disks travel to the lipid outer surface of the protein-lipid cornified envelope. The cornified envelope acts as an anchoring site. There, the disks arrange themselves into organized layers. In this way, the lipid-fat disks create your skin barrier by forming double layered rows called “bilayers.” Think of the structure as like a pair of shoes (bi = two) rather than one lonely shoe:



Think of the intercellular space as a big shoe rack and think of the lipids as zillions of little shoes all neatly organized in rows of two: The lipid bilayers:

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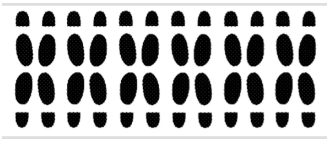
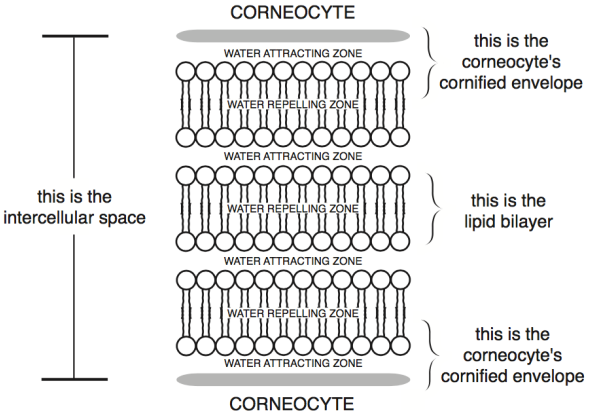


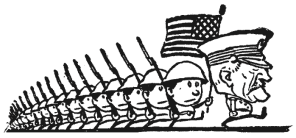
The lipid bilayers are made of innumerable molecules of ceramides, cholesterol, and free fatty acids, ceramides, cholesterol, and free fatty acids, ceramides, cholesterol, and free fatty acids, all arranged like these little shoes neatly stacked in bilayers.

Bilayers Are Better: A Practical Design

We don't mean to sound repetitious, but we are trying to make a memorable educational point. These zillions of ceramides, cholesterol, and free fatty acids are the same bilayered lipids that are lost when your patient uses too much soap. You can now understand that the lipids are not just haphazardly thrown together. They are neatly and purposefully arranged in bilayers so that your skin barrier can better do its barrier job. Keep these bilayers in mind when we talk about how soap depletes your skin barrier. Soap can actually strip away precious barrier lipids and cause disorganization of the lipid bilayers. This, in turn, causes a damaged dysfunctional skin barrier, which leads to your patient's dry and sensitive skin rash as well as the repetitive itch, scale, and scratch cycle. This next diagram shows the magnified intercellular space, revealing the lipid bilayers, sandwiched in between two corneocytes:



Compared to disorganized, haphazardly arranged lipids, the three lipid bilayers are better able to function as a true skin permeability barrier. As an analogy, picture a military battle: Organized rows of soldiers on a front battle line are better able to push back the enemy and hold off their fierce attacks. Ill-prepared and disorganized soldiers are ineffective and will lose the battle every time.

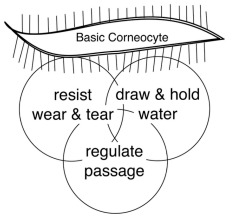


Think about your skin barrier as a military battle zone. The lipids are like little soldiers arranged in bilayers to better fight water loss. Also, because of its special bilayered structure, the lipid skin barrier can better resist the enemy (unwanted allergens) as they approach your body. Thus, soap is your biggest enemy! Soap and solvents can damage your bilayered lipid skin barrier and can potentiate a dry skin rash.

Soap is the Enemy!

Step Five: Corneocytes, How Do They Work?

For a healthy skin barrier, the corneocytes work in three important ways. First, corneocytes are able to withstand destructive shearing forces and wear and tear. Second, like a water magnet, corneocytes attract and retain water. Third, healthy corneocytes are able to physically prevent the passage of water, and passage of toxins, allergens, harmful infectants, and irritants. Thus, corneocytes maintain three skin barrier functions:



To carry out these three goals, corneocytes have the use of:

1. **A rigid keratin "cytoskeleton"**
2. **Natural moisturizing factor**

Dermatology Guidelines for the Primary Care Resident: The Essentials

Keratinization via Keratohyalin Granules

"Keratinization" refers to the process by which skin cells become "keratinized" and are thus hardened or "cornified." The biochemical process of keratinization begins with keratohyalin granules, which are small packages containing keratin filaments and a protein called profilaggrin. Granular cells are the first epidermal cells to form keratohyalin granules. Inside the keratohyalin granules, profilaggrin is modified to form filaggrin. As keratin undergoes disulfide bonding, histidine rich filaggrin works to hold keratin filaments together. The purpose is in its clever name, "Filament aggregating protein" or filaggrin Later in the story, filaggrin is additionally important as filaggrin is eventually hydrolyzed by water and enzymes to form a humectant called natural moisturizing factor. A "humectant" is any substance that acts as a water magnet to draw water and moisture unto itself. The natural moisturizing factor (NMF) is the skin's natural water magnet. Sodium PCA and glycerin are synthetic water magnets contained in certain skin moisturizers.

A Keratin "Skeleton" for Wear and Tear

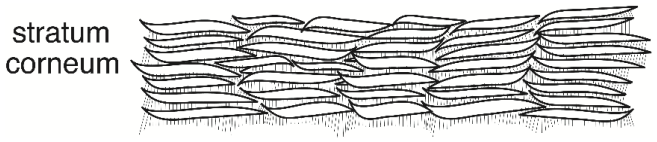
Innermost keratinocytes each undergo keratinization to finally form the outermost corneocytes filled with hard insoluble bonded keratin protein filaments. These keratin filaments are disulfide bonded together for wear and tear ability. Keratin gives skin its toughness, rigidity, and durability. It is the structural framing that supports the corneocyte. Keratin is also called the "cytoskeleton" of the epidermal skin cell and accounts for about 85% of the dry weight of a human corneocyte cell.

Filaggrin Proteins

Like intracellular glue, filaggrin proteins hold keratin filaments in place so that disulfide bonds can form. Disulfide chemical bonds lock hardened keratin proteins together to give your skin its toughness as well as wear and tear durability. Finally, after all the keratin filaments have bonded, the filaggrin breaks down by hydrolysis into natural moisturizing, a mixture of amino acids, PCA, urocanic acid, salts, sugars, lactic acid, and urea. Finally, the natural moisturizing factor remains inside of the corneocytes like a water magnet to draw and hold water within the stratum corneum.

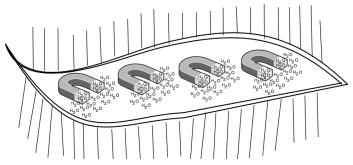
Natural Moisturizing Factor: Nature's Humectant

The natural moisturizing factor is located in the stratum corneum within the corneocytes. Natural moisturizing factor is derived from the hydrolytic breakdown of filaggrin proteins.



Interestingly, filaggrin breakdown into NMF requires water, this means that filaggrin breakdown is precisely controlled by climate and relative humidity. If the environmental air is humid or wet, filaggrin breakdown does not occur. If the outside air is excessively dry, filaggrin breakdown does occur. Thus, like a water regulating "aquastat," (similar to a thermostat) filaggrin breakdown and natural moisturizing factor levels are closely controlled and regulated so that your skin retains moisture when needed.

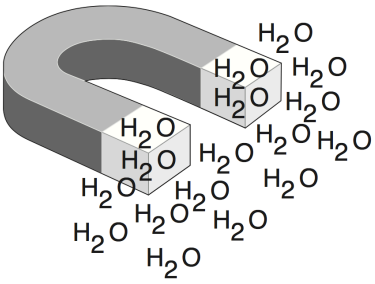
NMF: A Water Magnet



While 85% of the dry weight of the stratum corneum is keratin, 10% is natural moisturizing factor. Specifically, your natural moisturizing factor is made out of amino acids, sodium pyrrolidone carboxylic acid (PCA), lactate, and urea. NMF, natural moisturizing factor functions as a natural humectant to draw, attract, and hold water within your corneocytes. This next diagram shows a corneocyte filled with tiny NMF water magnets. Better moisturizers may also contain glycerin molecules, which, very much like PCA, enter skin cells to help retain water.

Higher quality personal skin care moisturizers may contain sodium pyrrolidone carboxylic acid

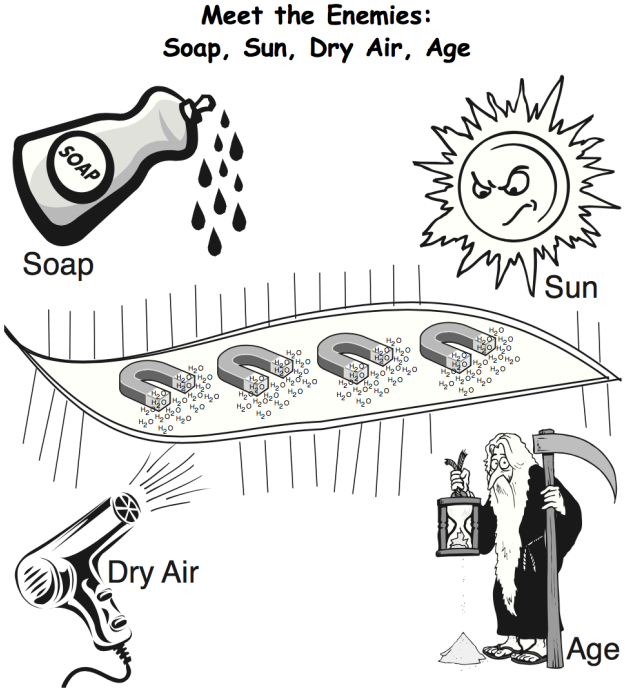
(PCA) as a physiologic natural humectant with exceptional water drawing ability. Glycerin is synthetic humectant. Both glycerin and sodium PCA are taken in by your granular cells and can help skin cells retain water. Humectant biomolecules are like water magnets. A "water magnet" draws and retains water to itself like a regular magnet attracts small pieces of metal.



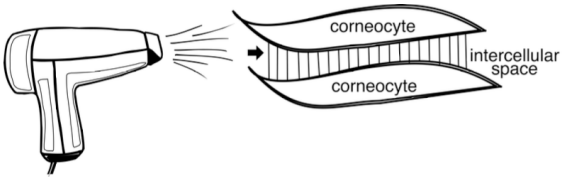
Desiccating factors can deplete corneocytes of their natural moisturizing factor. If natural moisturizing factor is lost, your skin becomes dry and loses its ability to attract and retain water. Thus, skin barrier moisturization is like a battle between forces of good and evil, moist and dry.

Four Enemies of Your NMF

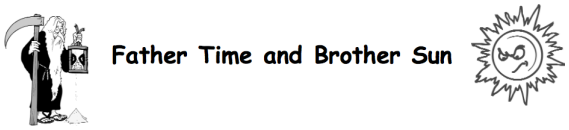
It is important to understand that corneocyte cells are under constant attack by four strong enemies: Soap, sun, dry air, and age. Before you know it, your skin is dry like a potato chip!



Dry Air Climate = Less Water in the Skin



Like a "thermostat" or "aquastat," changes in climate can result in either increased or decreased filaggrin breakdown. Experiments show that filaggrin breaks down when relative humidity reads between 70 to 95%. If it is below 70% and above 95%, nothing happens. Thus, a certain level of dry air can promote filaggrin breakdown and can increase your NMF. You see, filaggrin breakdown requires water for hydrolysis. When the relative humidity falls below 70%, there is not enough water to hydrolyze filaggrin, and you will see less filaggrin breakdown and less NMF. When relative humidity goes above 95% and the air is humid, less NMF is needed and filaggrin breakdown is also less.



Aged weather-beaten skin is often deficient in natural moisturizing factor. Pease note that both normal aging and ultraviolet sunlight can result in decreased profilaggrin. So, natural moisturizing factor decreases with both chronic sun exposure and aging. Just remember Brother Sun and Father Time the next time you see a sun damaged older patient with dry skin. This patient lacks profilaggrin and has windblown, weather-aged, dried skin- the result of poor sun safety and poor skin barrier moisturization.

Soap Causes the Loss of Two Components:

1. Lipids

2. NMF

Soap is a "double whammy" to dry skin. First, soap and detergents can damage the skin barrier so that cholesterol, ceramides, and free

fatty acids are lost. Second, in addition to lipid loss, a soapy skin barrier will always lose natural moisturizing factor. Remember, a damaged skin barrier is permeable & leaky. Thus, NMF will leak out of a disrupted skin barrier.

How Important is the NMF?

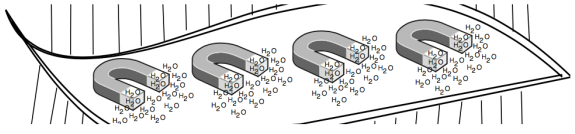
The natural moisturizing factor plays a considerable role in the water holding potential of the stratum corneum. However, it is short sighted to think that the natural moisturizing factor is the only important factor in your skin's water holding ability. There are other humectant type molecules.

When it comes to water holding capabilities, the intercellular lipids, especially ceramides, are also vital. We explain this because far too many skin care product companies over-emphasize the natural moisturizing factor as the most important element in skin care. The reality is, every humectant type molecule is important.

Healthy skin requires intact lipid bilayers with adequate cholesterol, ceramides, and free fatty acids, adequate natural moisturizing factor, and sometimes temporary barrier protection. This combination creates a skin barrier that works continuously to protect your patient.

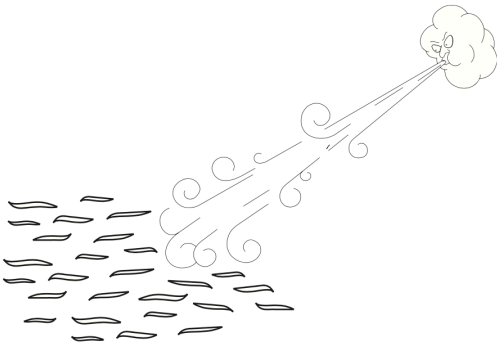
The Cornified Envelope

The NMF needs protection or it will leak out. The "protector" is the cornified envelope. By the time a skin cell reaches the stratum corneum, the corneocytes have developed a resilient outer layer called the "cornified envelope" to cover their outside surface. The cornified envelope is a two-layered, protein-lipid covering that is selectively permeable and provides the extra skin rigidity and strength needed for skin resistance to wear, tear, and disrupted skin barrier leakage.



The cornified envelope allows water to enter or leave the corneocyte, but prevents the natural moisturizing factor from leaking out from the cell. The envelope is likened to a "plastic" coating to cover the brick. The coating is formed by cross-link bonding of keratin, involucrin, envoplakin, and periplakin skin proteins. Transglutaminase enzymes and calcium assist in this process. Soap, the bitter enemy, can decrease NMF in corneocytes and may damage your skin barrier by lowering transglutaminase enzyme activity, thus inhibiting formation of the corneocyte's protecting cornified envelope.

Step Six: Dust in the Wind, The Final Stage



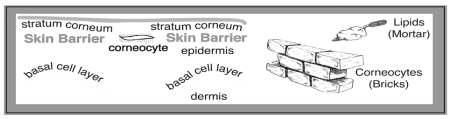
Corneocytes Have an Expiration Date

Corneocytes Will Eventually Turn Into "Dust"

In the end, stratum corneum chymotryptic enzymes break down the corneodesmosomes by hydrolytic action. This enzyme requires water and a slightly acidic pH, and thus, the importance of your body's "acid mantle." No longer bound by corneodesmosomes, the end-stage corneocytes are invisibly removed from your body.

Used corneocytes are lost in the air, to be replaced by new skin cells from below. The outdated corneocytes are now expired and will invisibly flake away like dust in the wind. So we can truthfully say, "Ashes to ashes, dust to dust, skin barrier moisturization is a must."

Skin Barrier in Healthy State



As long as the lipids are well organized and filaggrin is replenished, the skin barrier functions

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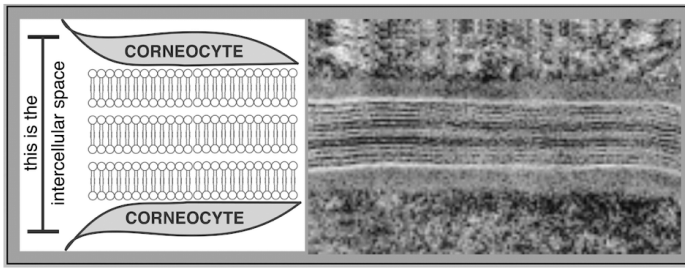
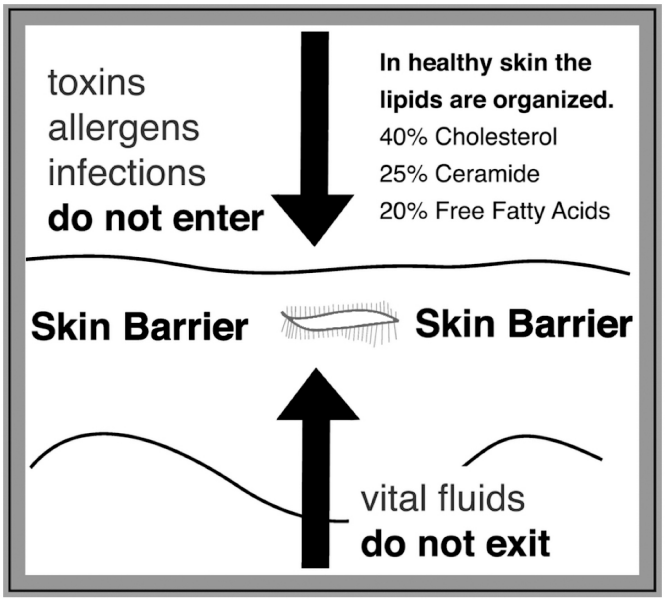
as it should and your skin can remain healthy, well moisturized, and free of inflammation and infection. Here is electron micrographic art showing healthy skin corneocytes and healthy bilayered skin barrier lipids.

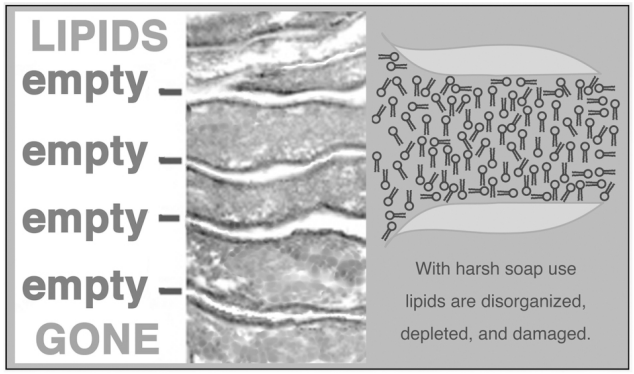


With a healthy skin barrier you have protection from the outside world. It is an entrance barrier. It is an exit barrier. Toxins, infectants, and allergens do not so easily enter. Water does not so easily escape. Your skin is well moisturized and everyone's happy!

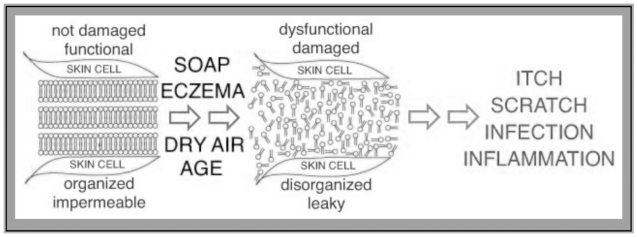


Skin Barrier In Damaged State

Skin barrier disruption usually begins with soap. It can be any soap, especially deodorant and gel soaps that contain harsh oil washing surfactants. Soap removes the lipids and NMF. You become squeaky clean. Without the lipids, the skin barrier is depleted, empty, damaged, and dysfunctional. It will leak to allow toxins, allergens, & infectants into the skin. Water exits. The patient feels dry & itchy. You see scaliness. Empty & depleted of lipids, the skin is dry.

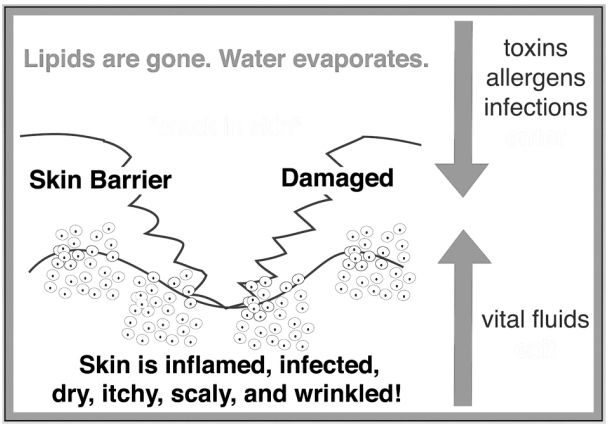


Cracks develop, lipids are disorganized, the skin appears xerotic wrinkled and weather beaten, and a dry skin rash ensues.



Though it is of the utmost importance for all of their eczema prone dry and sensitive patients, most primary care residents in this world have not studied the human skin barrier. As a primary care resident, it is important for you to understand what makes for a healthy skin barrier so that you can explain to your patients the woes of a disrupted skin barrier. Sometimes the patient lives so long with dry skin that he or she starts believing that it is normal for them to live with dry skin, but it is not normal.

As a primary care resident who understands the skin barrier, you can make a healthy skin difference in your patients' dry and itchy lives.



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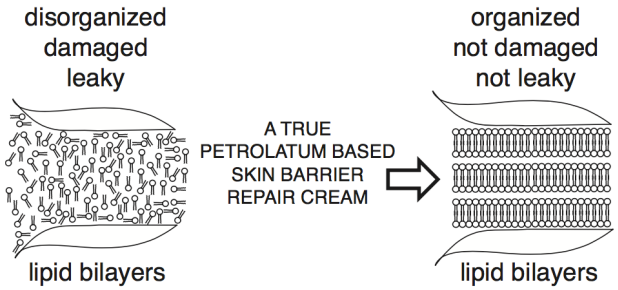
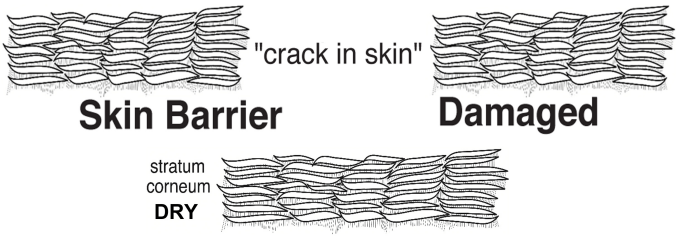
Soap makes everything worse.

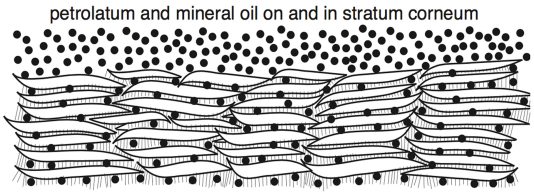
Now that you are aware, you can teach your patient how to care for their broken skin barrier and how make their skin barrier healthy again.



How To Fix a Dry Damaged Skin Barrier?



Add Vaseline!



Vaseline enters between your skin cells and allows your skin barrier to temporarily function again while your body heals itself.

As you apply pure Vaseline (generic petrolatum) on a regular basis, the petrolatum molecules will enter the intercellular spaces and will function as a temporary skin barrier. In time, the petrolatum will be replaced by true skin barrier lipids from the daily foods we eat. But, for those who want the best, there is a better way than petrolatum.

Cerave Ointment

In the diagram above, we show how Vaseline (generic petrolatum) percolates down into the intercellular spaces to restore skin barrier function. Yes, petrolatum is adequate for barrier repair; but, if you want to suggest the very best skin barrier product for your patient, tell your patient about Cerave ointment. Tell them to use Cerave Ointment in place of Vaseline. What makes Cerave Ointment better than Vaseline? Vaseline is good, but is made only of petrolatum and does not contain true lipids. On the other hand, Cerave Ointment is filled with physiologic lipids, and is in actuality, like “supercharged” petrolatum for skin barrier repair. So, for your patients who want something beyond Vaseline, Cerave Ointment is the most effective skin barrier product your patients can use.

Skin Barrier Microanatomy & Physiology

The skin barrier is a microscopic, often ignored, vital organ of your human skin that is structurally formed like bricks, mortar, and rivets.

Bricks- Corneocytes are protein-fat covered, epidermal skin cells, structured with a filaggin cytoskeleton, covered with a protective protein-lipid cornified envelope, and filled with natural moisturizing factor as filaggrin becomes NMF.

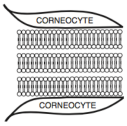
Mortar- Represents intercellular lipids organized into bilayers. For impermeability, lipids are structured like tiny “shoes” in a shoe rack and are neatly arranged into bilayer pairs.

Rivets- Represent corneodesmosomes and are structured like tiny “rivets” that protect the bilayer lipids and join all the corneocytes together.

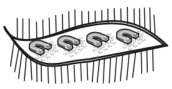
The Skin Barrier Provides “Aquastatic” Control

The skin barrier carefully balances skin water content via two bio-molecular mechanisms:

1. The lipid bilayers



2. The NMF water magnet



In a healthy state, the waterproof lipid bilayers made of cholesterol, ceramides, and free fatty acids prevent the escape of vital body water to

the outside world. At the same time, the lipid bilayers prevent the entrance of, unwanted allergens, infectants, and toxic agents.

Natural moisturizing factor acts like an “aquastat” controlled water magnet to draw and retain skin moisture against a dry outside environment. When relative humidity hits a certain dry level, internal NMF production is turned on. NMF draws moisture to your skin from both the water you drink (your inside water) and the water you bathe in (the outside water). This is the elegant way by which your skin barrier maintains the perfect moisture of your skin organ within an optimum daily water balance. Thus, your skin barrier is able to work as it should in all its daily functions.

Finally, a dry damaged skin barrier can be temporarily fixed by adding Vaseline or Cerave Ointment to dry skin. The Vaseline and Cerave Ointment molecules enter the dry intercellular spaces and function as a temporary skin barrier, while the body restores the intercellular spaces with true lipids from the everyday foods you eat.

Finally, as a primary care resident, you will be doing good for your patients, if you educate each patient about their skin barrier, and how to keep their skin barrier healthy and replenished.

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Dermatology Guidelines
for the Primary Care Resident:
The Essentials

Skin Barrier Care
Guidelines

UNDERSTANDING ECZEMA

When a patient walks into your primary care resident clinic with a fever of 104 F and comes to you for medical help, your #1 goal should be to carefully evaluate your patient and give the patient an accurate diagnosis. It's your #1 goal, because an accurate diagnosis means accurate treatment. But why is it, when a patient comes in with a dry scaly rash, some primary care residents will only quickly look, diagnose the itchy patient with "eczema," or "fungus," prescribe betamethasone dipropionate, and hope for the best? What about accuracy? This is the resident who never learned how to differentiate eczema from fungus.

A large percentage of patients in your primary care clinic, roughly one out of five, will have a skin problem. And, of these patients, eczematous type skin problems will be the most common. Accuracy is important. So, with your primary care patients in mind, we have devoted much of the Guidelines to caring for eczematous skin conditions.

If you go way back in time and trace the names given to modern diseases, many times the original name turns out to be just a visual description. If you trace the word "eczema," you will find that it comes from the Greek word *ekzein* meaning "to boil out or erupt with heat." (*ex* = out) + (*zein* = to boil).

As medical knowledge advanced, especially in the 1800's, physicians tried to make eczema into one specific diagnosis. But, now we know that eczema is not one exact diagnosis. Rather, "eczema," is a word that describes what you see. And, eczema is the name for an assemblage of related diagnoses that all manifest with dry, scaly, red, itchy, weepy, inflamed skin, and a disrupted skin barrier.

The Skin Barrier and Eczematous Skin

A medical condition deserves an anatomical explanation. If a patient turns yellow, a physician looks at the liver. If a patient has ischemic heart disease, a physician examines the coronary arteries. Similarly, if a patient has eczematous or dry-sensitive skin, a physician will review the skin barrier. The skin barrier is the specific anatomic body part in need of medical care. For a review of the skin barrier, please read Dermatology Guidelines: Skin Barrier Microanatomy & Physiology for the Primary Care Resident.

Primary care resident, please remember, the purpose of your skin is that it acts as a protective barrier between your inner body and the outside world, thus, minimizing water loss; while, at the same time, preventing entrance of outside infectants, allergens, irritants, & toxins.

When it comes to understanding eczema, there are several types and subtypes of eczema. They may all look alike in certain ways, and may look different in other ways, but all eczemas share one common weakness, and that is they each have a skin barrier that does not function well.

COMMON TYPES OF ECZEMA

Allergic Contact Eczema
Atopic Eczema
Dyshidrotic Eczema AKA Pompholyx
Eczema Herpeticum
Eyelid Eczema
Hand Eczema
Infective Eczematous Dermatitis
Neurodermatitis - Lichen Simplex Chronicus
Nummular Eczema AKA Discoid eczema
Primary Irritant Eczema
Seborrheic Dermatitis
Stasis Dermatitis
Xerotic Eczema

The Three Phases of Eczema

Eczema manifests with three stages, each with its own distinct features. The patient may

experience only the first stage, only the first and second, or the patient may go through all three stages. We call the three phases of eczema acute, subacute, and chronic. Acute eczema is stage one, starting with some redness, swelling, and itching. The redness quickly changes into blisters which, when they break, promptly begin oozing or weeping. The itching can become severe in stage one, but the patient must try hard not to scratch. Scratching only intensifies the itching, may inoculate infectants, and can worsen the patient's condition.

In phase two, subacute eczema, the blisters, weeping, and oozing lessen and crusts form. Redness, itching, and some marks from scratching, may be prominent. Phase three is called chronic eczema. The skin has suffered so much irritation that it begins to show thickening and lichenification, becoming tougher and pigmented. Often this toughened skin feels like leather, and it may actually crack and form deep painful grooves and fissures. Itching is still a persistent symptom. In every phase of eczema, the patient fights a constant battle with self-control: To scratch or not to scratch. The more the patient scratches, the more the skin is irritated, the more the eczema worsens, and the more likely it is to get infected.

HOW IS ECZEMA TREATED?

First of all, before proceeding with treatment, you will have to make sure that your patient has the correct diagnosis and actually has one of the eczema types listed above. When diagnosing eczema of any type, the patient is going to complain of itch plus symptoms from the three phases of eczema. For primary care residents just learning, eczema is most often confused with tinea corporis. Resident, please remember that tinea does not usually itch, while eczema almost always itches. To learn how to actually diagnose true eczema and differentiate eczema from similar conditions, you will have to spend time in the clinic with a good dermatologist. As part of your primary care residency, you are required to spend one clinical month learning with a dermatologist.

Dermatology Guidelines for the Primary Care Resident has no photos and is not designed to teach you with photos. Yes, you can look at photos, but nothing else is quite as educational as a clinical visit with an actual eczema patient and a good dermatologist. It may be a 14-month-old with atopic dermatitis, a young adult with poison oak contact dermatitis, a housewife with hand eczema, or an elderly gentleman with stasis dermatitis. It is much more meaningful when you interview and examine an actual eczema patient.

So, Dermatology Guidelines is designed to help you learn from actual patients as you follow your assigned dermatologist. Instead of learning from photos, your assigned dermatologist teacher will show you actual patients. He or she can explain fine diagnostic points with each live patient you encounter. For example, the dermatologist can show you how to differentiate each of the eczemas from the others. As you progress in your primary care residency education, please keep in mind that the Dermatology Guidelines are designed to teach you those daily dermatology learning points most useful for a primary care physician practicing in a busy primary care clinic.

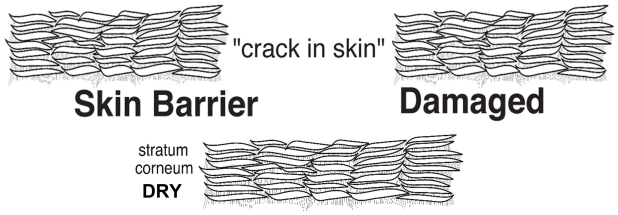
We have mentioned that each type of eczema has its own unique treatment plan, but, because each eczema type has a damaged skin barrier, the foundation for treatment for all types of eczema is always healthy skin barrier therapy. Regardless of eczema subtype, it is of utmost importance to restore a healthy skin barrier to each patient. Now, we will explain basic principles on how to restore the skin barrier. Afterwards, we will review eczema subtypes to explain fine points in the diagnosis and management of each.

Skin Barrier Restoration

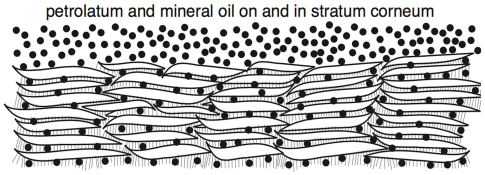
Skin barrier restoration is quite simple in concept: Just add oil. Though your actual skin barrier lipids are composed of ceramides, cholesterol, and free fatty acids, all you actually need in order to restore function to a damaged skin barrier is to apply heavy petrolatum ointment or cream to the damaged skin barrier area several times a day. The petrolatum molecules will percolate down into the stratum corneum to the intercellular spaces and function as a temporary skin barrier.

Dermatology Guidelines for the Primary Care Resident: The Essentials

How To Fix a Dry Damaged Skin Barrier?



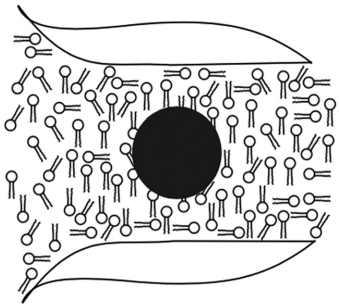
Add Vaseline!



Vaseline enters between your skin cells and allows your skin barrier to temporarily function again while your body heals itself.

As you apply pure petrolatum on a regular basis, the Vaseline molecules will enter the intercellular spaces and will function as a skin barrier. In time, the petrolatum will be replaced by ceramides, cholesterol, and free fatty acids- true skin barrier lipids from the foods you eat.

This drawing depicts a petrolatum molecule situated in a disrupted intercellular space sandwiched between two corneocytes. Notice the lipids are haphazardly disorganized and not arranged into healthy organized skin barrier lipid bilayers.



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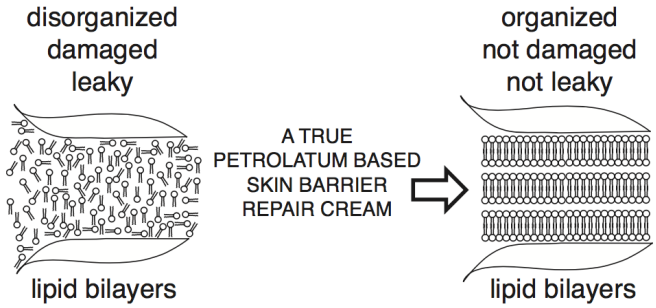
With time, patience, and regular application of petrolatum or heavy moisturizing cream, the lipid depleted, disorganized, damaged, dysfunctional, disrupted skin barrier is restored to a healthy, fully functional skin barrier replenished with lipids, natural moisturizing factor, and filaggrin. Conscientious skin barrier care combined with allergen avoidance is often just enough to remedy most patients with eczematous skin.



Skin Barrier Daily Care: The ABC's

Primary care residents, please teach your patients that If they neglect their skin, their skin will neglect them.

A sleep deprived child with dry and sensitive skin spends night after night awake, tossing and turning in bed. A forgotten grandmother with dry and sensitive skin waits endless days in her nursing home chair rubbing and scratching her arms and legs. They may lack the ability to ask for help. Though the skin organ is the largest organ of our human body, it is often the most neglected. We remember to comb our hair, brush our teeth, and apply deodorant, but we usually forget to take care of our dry skin though we see the dried flakes and feel the terrible itch.

Unfortunately, patients forget to take care of their daily skin care duties. To help your patients better remember how to care for their skin barrier, we have developed a helpful system called the ABC's of Dry and Sensitive Skin®.

Dermatology Guidelines for the Primary Care Resident: The Essentials

What is Dry Sensitive Eczematous Skin?

Dry sensitive skin is a problem for many people, especially people with eczematous tendencies, and, especially in cold weather when the air is dry and hot furnaces are turned on. Everybody gets dry skin from time to time, but people with eczema get it worse. When dry eczema prone skin is chronically abused with soap, dry air, allergic exposures, and inadequate moisturizing techniques, simple dry skin can finally deteriorate into a long-term itchy rash-like state that lingers, takes away the joy of living, and makes other skin problems worse than they would otherwise be.

If you look in the Webster's Dictionary, there is no definition for the term "sensitive skin," but those patients who have it understand what it is. Sensitive skin is skin that dries out ever so easily. Skin moisture is not retained. Sensitive skin may ignite like a lighted match, and any little allergic thing can set it off. People with sensitive skin are often bothered and made uncomfortable by itchy, tingly, or crawly skin. People with sensitive skin are those with a delicate fragile skin barrier. People with sensitive skin need the ABC's of skin care.

ABC Patient Education

Sometimes medical education is so complicated that students can grow up to become doctors who may have a difficult time explaining medical information to their patients on a patient level of understanding. The following explanation of the ABC's is meant to show you a patient friendly way how to explain a low allergy lifestyle plus healthy skin barrier care principles to your primary care patients. When reading, just assume that this info is an idea of what you could say to your patients in an easy to understand way, and how you could, in a somewhat simple and conversational way, educate your primary care patients about taking care of their own skin barrier care.

"A" Avoid

Our first ABC rule is: A- Avoid. "A" is simply this: Try to avoid touching or contacting anything that will react with sensitive skin. Don't let allergic things come in contact with your skin. In pro-boxing the very first rule is, "Don't get hit. "This rule also applies to people with dry and sensitive skin: "Don't get hit!" Don't touch! Do everything you can to avoid an allergic encounter. By following the ABC's, you can avoid potential allergens and *prevent* problems.

Foods? Drugs?

At first, for some unknown reason, most people with sensitive skin usually suspect a food or drug as the cause of their skin problem. Though foods and drugs can certainly play an allergic role, the most common allergic offenders will usually be things you physically touch rather than things you eat.

Allergic "Memory"

Patients often ask, "Doctor, how can I be allergic to my perfume? I have been using it for the last 10 years." Because the patient has used the perfume for 10 years, he or she has finally acquired a "memorized" perfume allergy. Fact: T memory cells can build allergic "memory" after repeated use. So, the more you use it, the more you are likely to develop contact allergic memory, and afterwards, an allergic reaction to that common item you have repeatedly used.

Dermatology Guidelines for the Primary Care Resident: The Essentials

A Loaded Gun

Allergic contact dermatitis is a Type IV delayed-type hypersensitivity reaction. In type IV reactions, the primary step is sensitization. On allergen presentation, cytokines are released and promote memory T cells. The memory T cells with antigen-specific receptors return to the site of exposure and recruit more inflammatory reactants. The result is a memorized allergic reaction due to repeated exposure.

You can explain to the patient: The first time you touch something allergenic, your immune system processes the item and puts it in a white blood cell memory bank. The next time you touch it, your immune system memorizes again. After many exposures, your allergic memory becomes like a loaded gun, ready to shoot! The more times you touch it, the stronger the allergy grows. Finally, you react with a full-blown allergic reaction. For example, you touch your cat or you apply your perfume and an allergic rash explodes! You itch, you scratch, you suffer.

Allergic memory explains why sensitive skin irritation such as redness, inflammation, and pruritus (itching) can all of a sudden occur after years of applying your favorite personal skin care products. Therefore, beware of anything you touch, even those favorite things that you have used for years. Surprise, familiar items can become allergic after years of use!

Once again, A-Avoid, think about anything that touches your skin, such as what you wear and where you sleep. Think about items you use on a daily basis, even common items you have used for many years.

Because you spend at least eight hours a day in your bed sheets, and the rest of your day in your clothing, your laundry can be a major factor in dry and sensitive skin. Laundry detergent can be highly allergenic with additives, chemicals, and perfumes. Fabric softeners and dryer sheets can cause you to scratch. Dry-cleaning agents can attack you with itch. Laundry can be your biggest allergic problem. Here is our advice. For laundry, use only pure baking soda. Do not use dryer sheets, softeners, or any additives. You may need to avoid dry-cleaning altogether.

Next, think about your pets. Sensitive people can be quite allergic to animals. Dogs, cats, birds, horses, pigs, hamsters, and other animals can induce itching. It may not be the pet itself, but something the pet has. Pets can harbor mites and fleas. Pets can go outside and bring in grass, weeds, pollen, poison oak, and other allergenic items into your home. Please understand that allergic people are at very high risk if they sleep with their pet. What troubles lurk in your pet's fluffy coat? Always use common sense and think "A- Avoid" before you sleep with your pet. Where has Fido been? Running in the bushes? Rolling on the lawn?

It is very difficult to find truly hypoallergenic personal care products. Certain personal skin care products can become a huge allergic problem. For women, make sure your makeup is an oil-free, low allergy type. Beware of perfumed hair conditioners and shampoos. DHS makes a low allergy shampoo called "DHS Clear Shampoo." Try DHS Clear. Look for hair spray and hair gel products that are fragrance free. If you must use hairspray or gel, use only small amounts and be careful not to get any directly on your skin. Some allergic patients will have to avoid hair spray or hair gel altogether. Other commonly overlooked allergens include toothpastes and mouthwashes. Also consider the allergenic potential of deodorants, especially the aluminum containing antiperspirant types. You can mix a little baking soda with water to use as a low allergy deodorant. For low allergy shaving, try using fragrance free Toleriane Gentle Hydrating Skin Cleanser. Remember, always use common sense and think "A- Avoid" first before touching any personal product.

Fragrance

Fragrances are the most common allergens contained in personal skin care products of all types. Preservatives are the second most common allergen. Most people think of fragrance as a cologne or perfume. However, when a fragrance allergy is suspected, the most common cause is a personal care product containing fragrance. Consumers beware. The term "fragrance free" does not always mean that the product is free of fragrance. An unscented item may contain masking fragrances to cover

the odor of other fragrances. Many patients have difficulty remembering that a large number of daily skin care products contain fragrance.

Room deodorizers, rug deodorizers, air fresheners, and household cleaning products can fill your home with allergic fragrances. Beware of household products. Another source of allergens can be leather cleaners, window cleaners, and air fresheners used for your car.

In fact, fragrance allergy is so commonly overlooked that perfume is an actual ingredient in several prescription medications made for the treatment of eczema. What? An eczema drug with fragrance? This shows you that even pharmaceutical companies can be oblivious to the hazards of fragrance. Think: "A- Avoid" and learn the importance of reading the ingredients.

Natural Products

Just because something is "natural" does not make it safe for sensitive people. Rattlesnake venom is a "natural" product." Poison oak is also a natural plant, but rattlesnake venom and poison oak are certainly not safe to use as skin care products. The same is true of aloe vera. Aloe vera is a wonderful plant for those who are not allergic to it. Like penicillin, if you are not allergic, aloe can work wonders. Topical vitamin E is another "natural" but sometimes allergenic agent; however, oral vitamin E is not allergic to the skin. Remember, just because something is "natural" doesn't always mean it is good for you.

A final suggestion to help A- Avoid: Do not touch everything with bare hands. Use gloves. Ruin gloves, not your hands.

"B" Bathe

Educate your patient: Our second ABC rule is: B- Bathe. When you bathe, there is a right way and a wrong way. Done right, a bath is the best way to give your dry thirsty skin barrier a big drink of water. Done wrong, a bath can actually steal water and leave your skin barrier drier. Learn how to bathe the healthy way.

The Right Way: Just Add Water.

You Can Bathe or Shower

Dry skin therapy is really quite simple. If your skin is dry, then add water. Pure water is the best moisturizer for dry skin. "B- Bathe to restore moisture," is a simple concept and is the easiest ABC to do. Just add water, and your skin moisture is restored. Adding water is so easy to do. You simply turn on the warm, not hot, water to the tub, get in and bathe. Pure water will rehydrate and revive your dry skin. **Now, after adding water, you must cover your skin to prevent the water from evaporating away.** The trickiest part of dry skin care is not restoring water. The difficult task is to protect and guard the water you've restored. We will review this concept more when we study "C- Cover to protect moisture."

Do It Right: To Bathe, Use Only Gentle Skin Cleansers

Bathe to restore moisture, and do not use harsh skin cleansers. Harsh cleansers like Dial Soap contain strong surfactants. Surfactants are detergent like chemicals that connect polar molecules like oil with non-polar molecules like water. Thus, surfactant detergents will remove oils and can wash away your skin barrier lipids. Surfactants range from gentle to harsh. The harsher the surfactant, the more it will wash away the lipids. Gentle skin cleansers contain weaker surfactants and when cleansing, will remove less skin barrier lipids. Harsh soaps like Zest will injure and disorganize your skin barrier

lipid bilayers and cause leakage of natural moisturizing factor. The net result of harsh soaps will be drier skin and a net loss of skin water. Thus, people with sensitive skin should avoid harsh gel cleansers, Ivory soap, and antibacterial type skin cleansers such as Irish Spring, Dial, Zest, Safeguard, Axe, and similar commonly used bath-time products.

"OK Doctor, so what do I cleanse with?" The answer: You need a truly gentle skin cleanser. Your special skin cleanser should gently cleanse away "dirty" sebum oils and not harm your "good" lipid oils. Your skin barrier lipids will be safe when you bathe with a truly gentle cleanser like Toleriane gentle skin cleanser OTC.

The Right Way: Use A Gentle Shampoo

What about your shampoo and conditioner? If you suffer with skin sensitivity, then your hair and scalp also need gentle care. For people with a dry and sensitive scalp, we suggest a low allergy, quaternium-15 free, fragrance-free, sulfate free, preferably, preservative free, gentle shampoo and conditioner. Truly low allergy shampoos and conditioners are almost impossible to find. We suggest DHS products. Ask your pharmacist specifically for DHS Clear Shampoo and DHS Hair Conditioner.

Is There A "Right" Diet for Dry Skin?

For convenience, many patients would like to treat their dry skin barrier with a pill, an injection, or food. But is there a magic pill or food? To answer this question, think about your skin barrier lipid bilayers. They are made of cholesterol, ceramide, and free fatty acids. Thus, a magic food, pill, or injection rich in cholesterol, free fatty acids, and ceramides should be good for dry skin. Although this is a nice idea, foods high in fat and cholesterol are bad for your heart. Omega 3 fish oil pills are popular and can help a little, but there is really no magic pill for a dry skin barrier. So, drink lots of water, bathe, cover your moisture and remember, "A bath is like a big drink of water for your thirsty skin barrier."

So, Drink Water.

In actuality, your skin draws moisture from your total internal body water supply. Thus, pure ingested water is the best oral cure for dry skin. The ingested water will travel to where it's most needed. So, drink a glass of water to replenish your lost skin water from the "inside out." But, just like you should moisturize after your bath, you must remember to keep your skin covered with cream to prevent your restored skin barrier water from evaporating away.

What Is the Right Way?

Here's the simple ABC principle to make each drop of bath water count. Think, "B- Bathe" to restore your skin barrier:

In your bath: Avoid harsh soaps and bath gels. You will always lose H2O with harsh soap. Do use a truly gentle skin cleanser to cleanse away the dirty oils and preserve lipids.

After your bath: Cover your body with cream to

preserve and protect your restored skin water. Cream will guard your skin barrier and will prevent your water from evaporation.

One final bathing suggestion: When you get out of the bath or shower, take a spray bottle filled with distilled water and spritz your entire self.

"C" Cover

Our third ABC rule is: C- Cover. If you buy a new car you can order a special clear coat sealant to protect the paint for the life of the car. What a concept. A one-time coating! If only this was true for our skin. Unfortunately, there is no one-time protective covering for your skin. The coating must be applied on a daily basis. What is the very best way to protect your skin moisture? On your body, apply a heavy cream or ointment, not a lotion. On your face, apply an oil free lotion.

Medical students are taught a derm saying, "If it is wet, then dry it. If it is dry, then wet it." This old saying is true, but we are going to attach a clarification to this age-old rule: If it's dry, then wet it, and cover it with cream.

"C– Cover to protect moisture" is a protective concept. Your final skin care goal is to protect the vital skin barrier moisture you have restored.

What should you use to moisturize your face? Apply an oil free / petrolatum free lotion like Toleriane Double Repair Face Moisturizer. What should you use to moisturize your body? Apply a heavy cream like Lipikar both available OTC.

I bathed you with water and anointed you with oil.
Ezekiel 16:9

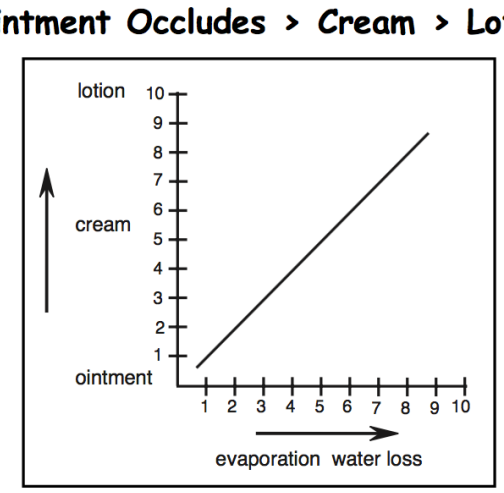
We often ask patient with dry skin, "How do you moisturize the skin of your body?" Many can't understand why their body skin is so dry. They reply, "I use intense care lotion every day." This explains their dry skin. A lotion is a thin liquid and tends to evaporate away in room air. Thus, despite what advertisements say, a lotion is not a very effective way to prevent water loss in the treatment of dry body skin.

In order for you to understand effective skin moisturization, you must first know the fundamental differences between a lotion, a cream, and an ointment. For your body, you should moisturize with cream or ointment, not lotion. A lotion is the thinnest skin moisturizer, an ointment is the thickest, and a cream is in between. For our purposes, in simple terms, a lotion is an oil suspension or oil mixture containing alcohol, water, or a water-soluble base. A pure ointment is greasy, like petrolatum. A pure ointment will not evaporate unless boiled, will not dry out, is not soluble in water, does not contain water or alcohol, is difficult to wash off, but can effectively prevent water evaporation in the treatment of dry skin. A pure ointment is messy and may or may not be cosmetically pleasing to use. On the other hand, a cream is less greasy, less messy, and is basically an ointment with water and/or an emulsifier mixed in to enhance cosmetic elegance. Like an ointment, a cream will also prevent water evaporation in dry skin care, but is much easier to use, and is, cosmetically more acceptable than an ointment.

Occlusion: The Seal

The action of sealing the skin to prevent skin barrier water loss is called "occlusion." Skin moisturizers exhibit a continuum of occlusive

ability from ointment to cream to lotion. Please study the following graph:



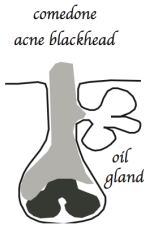
The above graph shows the amount of water that is lost over time when using an ointment, a cream, or a lotion.

Imagine wrapping your body with a roll of plastic food wrap. Your skin would be "occluded," meaning, locked ...sealed away from the outside world. Nothing gets in or out. We say that ointments are more "occlusive" and lotions are less "occlusive." Ointments are greasier, but are more occlusive (more sealing) and better at preventing water loss. Most lotions are not greasy or occlusive and do not prevent water loss as much as most people with dry and sensitive skin require. Pure mineral oil is like liquid petrolatum in that it also protects the skin barrier, but is greasy and very messy to use. A cream, on the other hand, is like an ointment, in that it seals in and protects restored water. A cream is less greasy, less messy, and easier to use than an ointment. Most importantly, a cream has adequate occlusive ability to seal and protect against evaporation. So, a cream is the body moisturizer of choice when protecting and repairing a damaged barrier. We suggest a cream called Lipikar available OTC.

One exception is your face. We do not suggest creams for the face, as creams can block pores and may cause acne. For faces, we advise a non-comedogenic, non-mineral oil, dimethicone or simethicone based, oil free lotion.

Cream for Your Body, Lotion for Your Face

A comedone is the medical name for an acne "blackhead." Comedogenicity is the ability for a skin product to block pores and oil glands, thus causing blackheads, whiteheads, and acne. Most creams and ointments are "comedogenic" and can cause acne. Thus, creams and ointments should not be used on the face of acne or blackhead prone patients for any long duration of time. To prevent acne and complexion problems, the face is best moisturized with an oil free, propylene glycol free, dimethicone based lotion. One good point: A lotion is usually adequate as a facial moisturizer, as the face usually makes enough of its own oil to keep your delicate facial skin barrier moisture in balance.



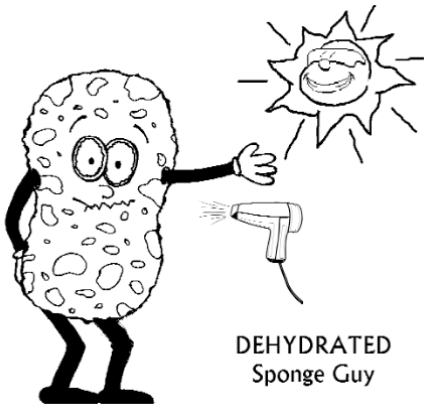
All faces beware! Many so called "non-comedogenic" lotions contain mineral oil and petrolatum based oily ingredients that can actually block your facial pores. Your face will be moisturized, but your blackheads, whiteheads, and pimples will grow. Thus, it is best to use a truly non-comedogenic pore blocking oil "PBO" free moisturizing lotion for your face. Look for a facial lotion that contains lipids, and is dimethicone or simethicone based, preservative free, and propylene glycol free. The right facial lotion should actually soak up sebum, and should leave your face feeling well moisturized without an oily shine. Thus, one ideal facial lotion should both add moisture and control unwanted oil.

Vapor Permeable Occlusion

Occlusion can be partial or complete. Partial occlusion is referred to as "vapor permeable occlusion" and allows selective passage of molecules into the skin. Complete occlusion is "vapor impermeable" so that nothing passes through. Petrolatum and mineral oil form a "vapor permeable" layer to allow passage of

necessary molecules such as drugs, lipids, and water. Complete occlusion occurs, for example, when the skin is fully covered with plastic wrap, so that water and other substances can't enter. It's interesting to note that complete vapor impermeable skin occlusion applied for an extended period of time shuts down lamellar body production in granulocytes. Passage stops, and skin barrier repair is inhibited. Note that petrolatum and mineral oil are only partially occlusive, vapor permeable, and do not shut down lamellar body production. Thus, passage of needed molecules and skin barrier repair continues with petrolatum based creams.

A Sponge Experiment



For visual impact, try this simple experiment at home on your bathroom counter. You will need:

a heavy moisturizing cream or ointment

a thin moisturizing lotion

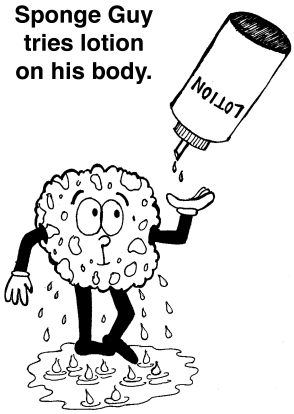
two small dry sponges

a cup of water

an electric fan to blow dry air

24 hours of time

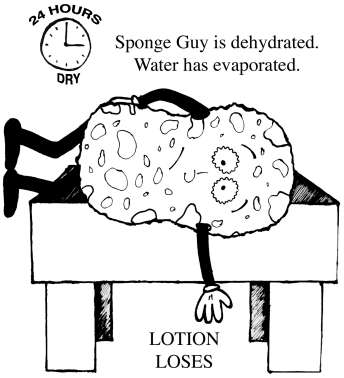
First, take one small dry sponge and dip it in water until it is soaking wet. Next, completely cover the wet sponge with lotion. Then, lay this sponge on top of your counter.



Second, take the other dry sponge and dip it in water 'til it's soaking wet. Next, completely cover this wet sponge with cream. Then, lay this second sponge on your counter next to the first.

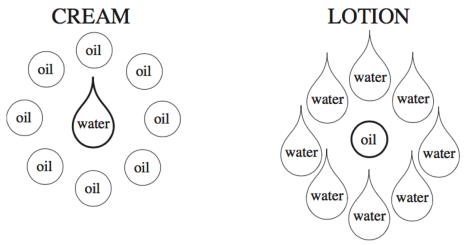
Third, turn the fan on low, wait 24 hours, and compare each sponge for moisture.

Which sponge is driest? After time in the dry air, the lotion treated sponge is drier.



Try this and find out for yourself. The lotion-covered sponge will be drier, while the cream covered sponge will be wetter inside. Why? The heavy cream seals water in to prevent water from evaporating away. The lotion allows evaporation.

A Cream Is the Best Way to Prevent Evaporation of Water



Keep this diagram in mind. Compare a cream to a lotion. The sponge stayed nice and moist with the cream outside, but dried up with the lotion outside. Why? The sponge dried out because a cream is based on a water molecule surrounded by oil molecules. The oil molecules prevent water from evaporation. On the other hand, a lotion is based on an oil molecule surrounded by water. Thus, the water can evaporate more easily. So, a heavy cream can better protect your skin moisture from evaporation. Lotion gives a brief feeling of moisture, but doesn't give the lasting cover you need to ensure effective skin barrier moisturization.

Note: In most cases, a non-comedogenic oil free lotion is the perfect daily moisturizer for a dry face, as the face comes pre-equipped with plenty of oil glands- many more oil glands than found on your body. Exception: In cases of an acute eczematous flare, when treating severe skin barrier damage, a patient may need heavy cream on the face for a season.

True vs. False Moisturization

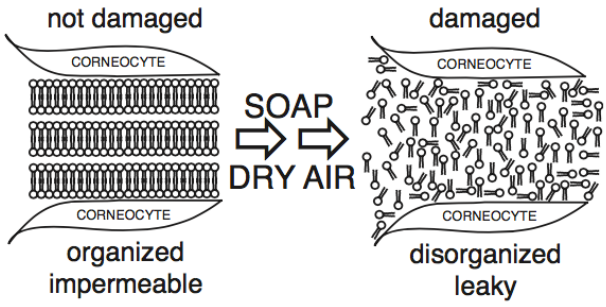
If there is true moisturization, there must also be false or inadequate moisturization. What's the difference between true and false moisturization? Go to any drug store. There are an overwhelming number of products, each claiming to moisturize the skin. It's easy to be misled about dry skin. What is the truth? As primary care resident, you should familiarize yourself with personal skin products that really work. Though a skin care company may provide advice, it may give information that's incomplete, clinically inadequate, or filled with meaningless marketing fluff. Because they are not in the same category as drugs, the FDA does not have much to say about skin moisturizers. Sadly, when it comes to dry eczematous skin, the consumer is often left alone to figure out what is true and what is false.

Medical vs. Cosmetic

As physicians, we believe that dry and sensitive skin is a medical problem and should be treated as a medical problem. However, because well-moisturized skin is very beautiful, skin moisturization is also viewed as a cosmetic issue. Thus, most of the diagnosing and care of dry and sensitive skin is in the hands of non-medical people, not physicians. As explained, even the FDA considers dry skin to be a "cosmetic" issue and classifies most dry skin moisturizers in the same category as rouge, blush, and lipstick.

We present this section on skin barrier care in hopes that you will educate your patients on skin barrier based prevention and treatment for their dry sensitive eczema prone skin.

How to Fix a Damaged Skin Barrier?

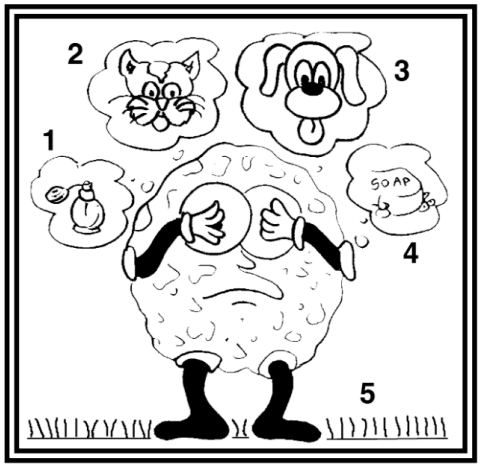


Dermatology Guidelines for the Primary Care Resident: The Essentials

In summary, with ABC skin barrier care: A- Avoid anything allergic is a concept of prevention. B-Bathe with water is a concept emphasizing restoration, and C– Cover to protect moisture is a concept of protection. Prevent, restore, and protect.

A- Avoid anything allergic.

This diagram shows you five important allergens and barrier unfriendly items to avoid.

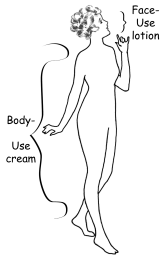


B- Bathe to restore moisture.

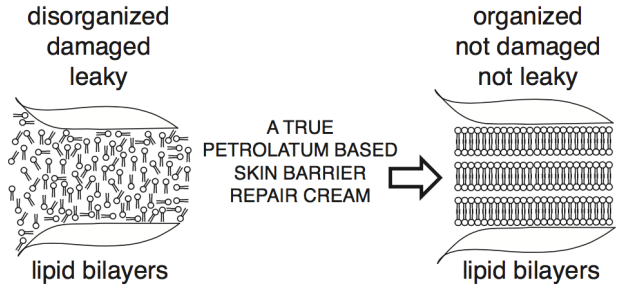
Just Add Water

- Do not use harsh soap
- Use a gentle cleanser
- Use a gentle shampoo
- Distilled water rinse
- Wring body dry
- Cover body with cream

C- Cover to protect moisture.



This is How a Damaged Skin Barrier is Fixed



Because all forms of eczema suffer with a disrupted skin barrier, skin barrier restoration and ABC care should be the foundation when treating any form of eczema in your primary care residency clinic. Yes, steroids and other medical therapies may or may not be needed, but skin barrier care should be the basis for all therapeutic interventions. With this section on skin barrier restoration and ABC care completed, we will now move on to review each of the common eczema subtypes.

Dermatology Guidelines for the Primary Care Resident: The Essentials

Suggested Personal Skin Care Products

The world of personal skin care products is often overwhelming and highly confusing. The wrong products can mean prolonged skin barrier / dry and sensitive skin frustration. Here we show photos of what we sincerely feel are safe, effective, and affordable for our dry and sensitive patients.

Cream: Lipikar Eczema

Gentle Cleanser: Toleriane

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Shampoo: DHS Clear

Face Lotion:
Toleriane Double Repair Face Moisturizer

Notes:

Toleriane Gentle Cleanser can also double as a low allergy shaving lotion. Don't use the Lipikar Cream for the face. Instead, for the face use the Double Repair Lotion. DHS Clear Shampoo is truly the lowest allergy shampoo we know of.

REMEMBER
YOUR DAILY ABC's

Dermatology Guidelines for the Primary Care Resident: The Essentials

COMMON TYPES OF ECZEMA

Contact Eczema or Dermatitis

To begin, the words “eczema” and “dermatitis” are synonymous. They have the same meeting. Years ago, doctors made a distinction between the two words, but today they are synonymous.

As its name "contact" suggests, contact eczema is the result of an allergen making contact with your skin. There are two types of contact eczema based on allergic vs non-allergic mechanisms.

First, Irritant Contact Dermatitis is a reaction that is non-allergic in mechanism. An example is contact with household bleach or battery acid. Second, Allergic Contact Dermatitis is allergic in mechanism and is a memorized Type IV delayed hypersensitivity reaction that requires previous sensitization, and is mediated by T1 cells rather than by antibodies. An example is contact with poison ivy or nickel. Systemic Contact Dermatitis is also a type 4 reaction, but occurs after eating, drinking, or breathing in a pre-sensitized allergen.

Phases of Allergic Contact Dermatitis

Sensitization phase, 5 to 21 days: An antigen-protein complex is accepted by epidermal Langerhans cells and then passes to regional lymph nodes where the antigen-protein complex is presented to naïve Th1 cells. With this, cytokines such as interleukin 2 and interferon-γ are secreted and promote clonal proliferation of the newly sensitized memory Th1 cells in nodes and are then released into systemic circulation.

Elicitation phase: When re-exposed to the same allergen, the sensitized patient will flare with allergic contact dermatitis within 12 to 48 hours

Allergic Memory

With Allergic Contact Dermatitis, interestingly, a person only rarely develops a rash after a single contact. This is the truth about allergies. The first contact almost never reacts; but, the second or the third of fourth, or ninth, etc. contact may react. Sometimes a sudden rash of Allergic Contact Dermatitis begins with the very next exposure; but sometimes it takes years to become sensitive to a certain allergen. It also works the other way around in that, what a person reacts to today, may after time, not react at all, and he or she has "lost" the memorized contact allergy.

Common Allergens

At times, the shape of the rash can give us a hint as to the allergen. For instance, a reaction to a particular dye in a shoe may affect only the sides of the feet. Or, a reaction when hiking in the forest can mark the legs with an allergic line. Common allergens that cause reactions in susceptible people, or may be the cause of a rash include:

- plants (like weeds and poison oak)
- foods (like celery and limes)
- industrial or work-related chemicals
- topical medications
- cosmetics, personal care products
- fabrics, laundry detergents, softeners
- household cleaners
- automobile cleaners

Here is a list of the most common patient allergens based on patch test results:

Balsam of Peru
Bacitracin
Cobalt
Fragrance mix
Neomycin sulfate
Quaternium 15
Formaldehyde
Gold sodium thiosulfate
Nickel
Methyldibromoglutaronitrile
Phenoxyethanol

Almost anything can cause contact dermatitis. But, a person is never allergic to something the first time they use it. It usually takes many repeated exposures before the human body can mount a true allergic reaction to an allergen. Often, a person will say, “Doctor, I can’t understand how I can be allergic to my perfume, I have been using it for 10 years.” As a primary care resident, you can explain that the person has

used the perfume for 10 years, and has finally acquired an allergy due to repeated exposures. The same holds true for contact allergic reactions to anything. A person needs to be exposed to a substance repeatedly before an allergy can manifest itself.

When faced with making a diagnosis of chronic dermatitis, a primary care resident should be able to differentiate between atopic dermatitis and allergic contact dermatitis. Atopic dermatitis is a recurrent eczematous dermatitis related to TH2 cells, asthma, and allergic rhinitis. On the other hand, allergic contact dermatitis is a TH1-cell cytokine mediated delayed type 4 hypersensitivity reaction caused by exposure to an outside allergen.

An accurate diagnosis is important from a therapeutic point of view. Children and adults with atopic dermatitis can expect chronic, recurring dermatitis. On the other hand, in persons with allergic contact dermatitis, the rash usually clears with avoidance of the inciting allergen.

Diagnosing atopic dermatitis vs allergic contact dermatitis can be a challenge in patients with symptoms of both conditions. Although there are studies to suggest that patients with atopic dermatitis are less likely to develop contact sensitization to allergens, other studies show that those with atopic dermatitis have a greater risk of developing allergic contact dermatitis.

Diagnosis & Treatment of Contact Dermatitis

The most important points to remember in the diagnosis and treatment of contact dermatitis are:

#1. Skin testing, #2. Avoidance, #3 Steroids

#1. Skin testing. By skin testing we mean either conventional patch testing or “use” testing. For your Primary Care Clinic, you may want to order a patch testing kit. With this kit, you can apply various allergen samples to the patient’s back, and test your primary care patients for allergic contact sensitivity. Another helpful tool in “use” testing. When “use” testing, you take a suspected allergen, let’s say, for example, Neosporin. You would take the Neosporin and apply it twice a day to the thin skin on the inside aspect of the elbow. After five days, if a reaction is present, then, you know that the patient is allergic to Neosporin and the patient can avoid it.

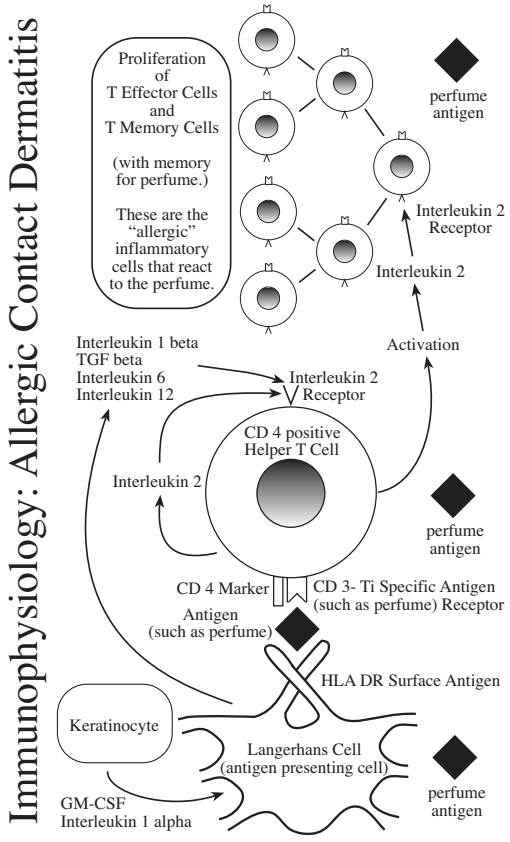
#2. Avoidance. Prevention is the best medicine. An ounce of prevention is worth a pound of cure.

#3 Steroids. Topical or systemic steroids are often needed in the treatment of allergic or irritant contact dermatitis. For more information on use of topical / systemic steroids, please read the **Steroid Therapy** section for Atopic Dermatitis.

Irritant Contact Dermatitis

As you see, irritant contact dermatitis is a first cousin to allergic contact dermatitis. The big difference is that irritant contact dermatitis is not an allergic reaction, it is a chemical reaction. Allergic contact dermatitis is an immunologic reaction. Irritant contact dermatitis is the result of local irritation rather than an acquired allergy. Examples of irritant contact dermatitis include contact reactions to battery acid and household bleach, kerosene, Dial or Zest soaps, and lye.

Immunology of Allergic Contact Dermatitis



Dermatology Guidelines for the Primary Care Resident: The Essentials

COMMON TYPES OF ECZEMA

Atopic Eczema or Dermatitis

Triad: Atopic Dermatitis, Asthma, & Hay Fever

For simplicity, think of asthma as affecting three sites: in the lungs, you get wheezing, in the sinuses, you get hay fever, and in the skin, you get atopic dermatitis. Though many skin conditions are linked to dry sensitive skin, in a child, dry sensitive skin is often associated with a triad of atopic dermatitis, hay fever, and asthma.

Findings in Atopic Dermatitis (AD)

Because itch is its first symptom, atopic dermatitis is sometimes referred to as the itch that rashes, and affects 13% of children and 2% of adults. An extra fragile, easily damaged skin barrier, high immunoglobulin E levels, and an exquisitely sensitive Th2 driven immune system result in extreme itch and a vicious itch-scratch cycle. Patients react to foods, mites, dust, staph aureus, emotional stress, and many topical allergenic items such as wool, animals, soaps, detergents, pollens, molds, and perfumes. Atopic (eczema-prone) patients are more susceptible to viral, yeast, and bacterial infections and get warts, molluscum, herpes, impetigo, and diaper rashes very easily. Nighttime itch robs these children of sleep and both the children and the parents suffer from chronic sleep deprivation.

Atopic Eczema or Dermatitis differs from allergic contact and irritant eczemas, in that atopic dermatitis is a cytokine mediated reaction that does not rely on contact. Atopic dermatitis tends to run in families and often occurs together with other types of allergic disorders such as asthma and hay fever. For example, infants, with atopic dermatitis may outgrow it by the age of 6 but later develop asthma or hay fever. Atopic dermatitis is a type of chronic dermatitis with acute flares often triggered by heat, cold, changes in temperature, sweating, emotional stress, and fatigue. One symptom that every person with atopic eczema has is a tendency towards dry sensitive skin. If you look in the Webster's Dictionary, there is no definition for the term "sensitive skin," but those who have it understand what it is. Sensitive skin is skin that dries out ever so easily. Skin moisture is not retained. Sensitive skin may ignite like a lighted match, and any little allergic thing can set it off. Sensitive skin type people are often bothered and become uncomfortable with itchy, tingly, or crawly sensitive skin.

What Areas Are Most Affected?

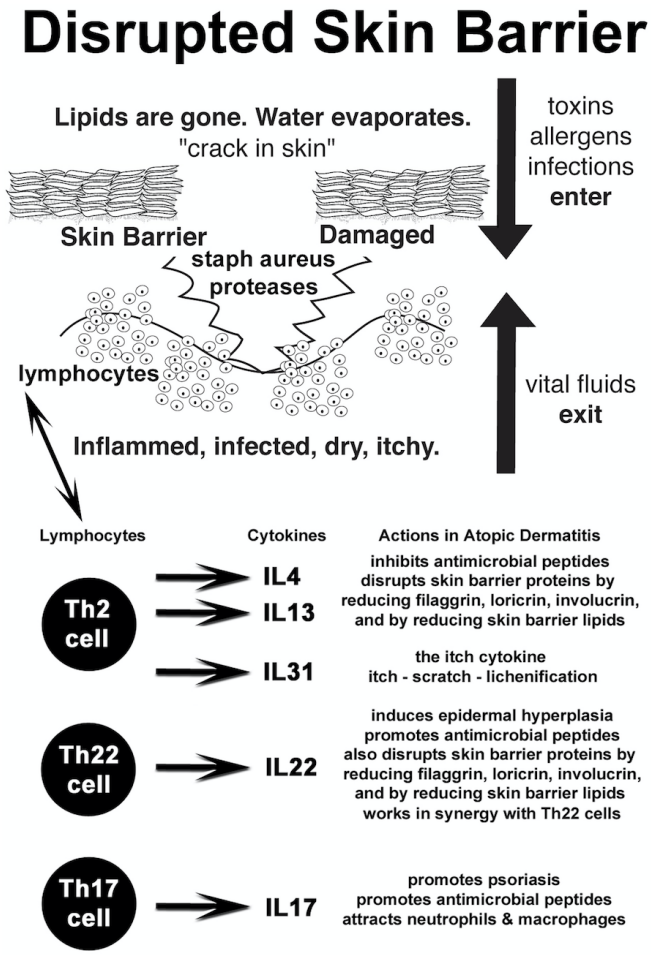
Babies from 2 months to 3 years of age develop an atopic rash that can be red and oozing especially on the scalp, face, and neck. Children ages 4 through 12 get the atopic rash on and around the scalp, neck, trunk, and genitals, and sometimes on the elbows, knees, ankles, and on the top of the hands, and feet. Older teenagers and adults get the atopic rash on the insides of the elbows, the backs of the knees, and the face. The atopic rash is mostly scaly, itchy, and dry.

Immunology of Atopic Dermatitis

A complete review of the immunopathology of atopic dermatitis is beyond the scope of the Dermatology Guidelines for the Primary Care Resident. For atopic dermatitis, the immunology flow charts are usually very involved. However, we have simplified the pathways for you and have included most of what you need to know in two helpful diagrams. Suffice it to say that atopic dermatitis is a Th2, Th22, and Th17 cell driven flare with Th2 cytokines dominating: Interleukins 4, 5, 13, and 31. Interferon gamma is decreased.

In the simplest of terms, atopic dermatitis is a chronic disease driven by both skin barrier disruption and subsequent fierce Th2 cytokine induced skin inflammation. It is thought that the initial skin barrier disruption promotes an environment in which Th2, Th22, and Th17 cells respond and release cytokines IL 4, 13, 31, 22, & 17. You also see increased IgE. The following diagram shows initial damage to the skin barrier, followed by entrance of toxins, allergens, and

infectants, the exit of water, and a resulting cascade of immunoactive cytokines and their respective interleukins and immune actions.



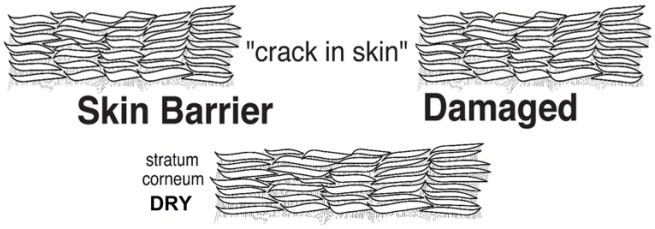
How to Manage Atopic Dermatitis

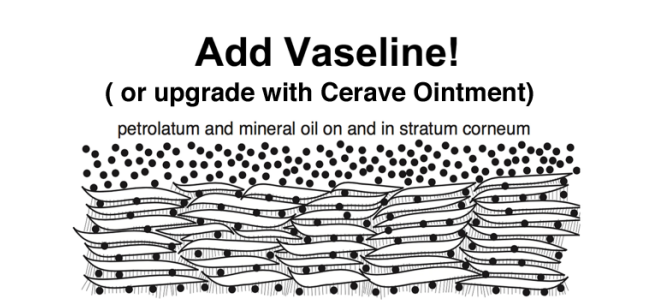
The goals of treatment in atopic dermatitis is to relieve itching, control flares, minimize risk of infection, and of course, to restore the skin barrier. Treatment methods include lifestyle changes: re the ABC's, up to date patient education, topical medications, control of staph aureus, and sometimes,s immunomodulation.

The Skin Barrier

Many dermatologists believe that it is a damaged skin barrier that allows ignition of the cytokine wildfire in AD. Thus, a healthy barrier is key in preventing AD flares. In skin of children with atopic dermatitis, there is early and potent Th cell activation. In atopic children, the non-lesional skin is hyperplastic with inflammation and activated cytokines. The filaggrin deficiency seen in lesional skin of atopic adults is not seen in children at all. In fact, atopic children do not show a filaggrin deficiency. It is interesting to note that in at-risk babies, daily emolliation starting from birth can reduce the incidence of AD by 50%.

How To Fix a Dry Damaged Skin Barrier?





Vaseline enters between your skin cells and allows your skin barrier to temporarily function again while your body heals itself.

Because atopic dermatitis is driven by a damaged skin barrier, as a primary care resident, to effectively manage your atopic dermatitis patient or any patient with an eczematous skin condition, you will need to understand and apply the basic concepts explained in the Guidelines sections covering Skin Barrier Microanatomy and Physiology and Skin Barrier Restoration. Helpful concepts of dry & sensitive care are summarized in the section under ABC Patient Education.

The purpose of ABC care is to temporarily recreate, then, restore the patient's skin barrier. Once the skin barrier is working again, the patient's atopic dermatitis will be significantly improved. At that point, careful daily skin barrier care will help to prevent future flares of atopic dermatitis. Skin barrier care, prevention, and treatment is actually straightforward and easy to understand. The difficult challenge is compliance. As you grow in the care of derm type conditions in your primary care clinic, you will find that most of the patients want to take a pill that will cure everything. No one wants the job of applying topical derm medications or skin moisturizers. They complain that topical skin care is too cumbersome, inconvenient, and messy. Thus,

compliance is always an issue. So, atopic patients get better and worse, better and worse depending on their willingness to care for their own skin barrier. Essentially, the atopic patient should be bathed daily and afterwards covered completely with heavy cream. The cream must be reapplied several times per day. The more cream, the better the patient will progress towards a functioning skin barrier. The better the barrier, the less atopic symptoms. The more closely the patient follows the ABC's, the better they will be and the less they will need chronic steroids.

Steroid Therapy

In reality, most atopic patients, for whatever reason, do not follow the ABC's; and thus, they flare. For many, it is chronic continuous topical steroids and their side effects. Truly, for atopic patients, the best care is preventative care: Keep the barrier filled and in good function. Non-compliant patients are like drivers who always run out of gas on the side of the road. They always run on empty. Why can't the driver learn that he or she always need to keep the tank somewhat filled before it runs completely empty?

When treating atopic patients with an acute flare, as a primary care resident, you can prescribe hydrocortisone 2.5 cream or ointment liberally twice a day to the entire body for 10-14 days. Regarding topical steroid medications, to prevent complications, here are a few guidelines. First, try to avoid the more potent topical steroids such as betamethasone and clobetasol. Probably the potent topical steroid most misused by non-dermatologists is betamethasone dipropionate – clotrimazole generic. The brand name for this is Lotrisone. Lotrisone is responsible for countless cases of steroid rosacea of the face, skin atrophy, and even sensory nerve atrophy. For example, topical Lotrisone is known to destroy cutaneous sensation from the genitals. So, use the lowest potency topical steroid necessary to control the symptoms. Hydrocortisone 2.5 is often a great choice. If a stronger topical steroid is needed, triamcinolone 0.1 can be used for short periods of time. Sometimes for atopic flares it is very helpful to give a three or four day burst with topical Triamcinolone 0.1 cream or ointment. After the burst, you can go back to hydrocortisone 2.5. Triamcinolone 0.1 and hydrocortisone 2.5 are both available in 80 gram tubes and one pound jars. You can dispense according to the size of each patient and the duration of treatment.

Now, there is a certain amount of corticosteroid phobia among primary care residents; and, rightfully so. But, the concerns can be minimized by sticking to triamcinolone 0.1 for short durations of time, and hydrocortisone 2.5. Remember that any fluorinated steroid such as triamcinolone can cause rosacea, so make it a point to keep topical triamcinolone meds off of the face and off of the genitalia. Also, to prevent cutaneous atrophy avoid triamcinolone on thin skin areas.

Another very helpful patient educational point: Hydrocortisone 2.5 & triamcinolone 0.1 are drugs. They are medications. So, your primary care patients should be taught: Do not use topical steroid meds as skin moisturizers. Way too many patients use hydrocortisone 2.5 cream and triamcinolone 0.1 cream as moisturizers and this potentiates their risk of side effects. Rather, your primary care patients should learn to use healthy OTC skin moisturizers for their daily care.

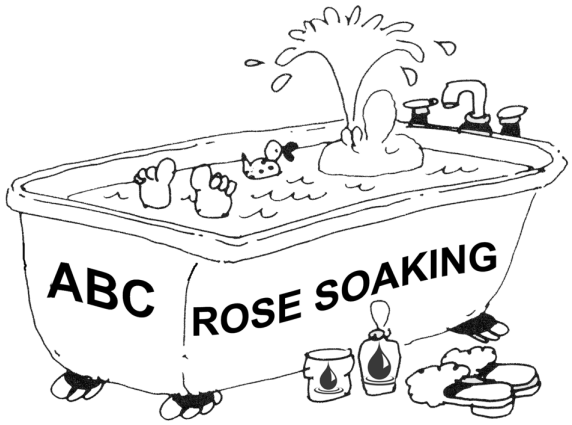
In addition to topical steroid medications, there are three other topical medications helpful in the treatment of atopic dermatitis. These have no steroid side effects, but price and coverage may be an issue. First, there is the anti-inflammatory inhibitor of phosphodiesterase 4 (PDE4) called 2% generic crisaborole ointment (brand Eucrisa). Second, is topical Elidel Cream 1% (generic pimecrolimus). Third, there is generic tacrolimus topical 0.1% Ointment (brand Protopic).

To help control itch, oral antihistamines are especially helpful; and, because they prevent the release of histamines, should be taken daily and

not just as needed. Benadryl is great for peds patients. Zyrtec is great for adult patients. Systemic steroids are often helpful for patients with a flare of atopic dermatitis. Once or twice and occasionally three times a year is not concerning for steroid use in atopic patients; however, a frequent need for systemic steroids can open a big bag of steroid problems for the patient. At this point you may need to seriously review his or her compliance with the ABC's, and / or consider a referral for possible cytokine modulation therapy.

Instead of emphasizing steroids for your primary care patients, you should teach them healthy skin barrier care. If they care for their skin barrier on a regular basis, they may not need steroids.

Bleach Bath Therapy



A better way to phrase this type of therapy is "Swimming Pool Water Therapy." Remember, staph aureus is one on the key ignitors in the cytokine wildfire of atopic dermatitis. Remove staph, and you get less atopic fire. Here is why bleach baths work: Bleach baths decrease the load of staph aureus. So, as a primary care resident, learn how to educate your patients on the benefits of bleach baths. It's easy. Before starting bleach baths, first take and send off a cutaneous culture swab to rule out any overt bacterial infections. Just add one teaspoonful of regular strength household bleach like Clorox to a gallon of water. Because many tubs hold different amounts of water, you may need to figure out just how many gallons of water it would take for your bath tub. Just remember, one teaspoonful of household bleach to a gallon of water. Now, if you want to fill up a gallon container, add a teaspoon of bleach, and pour it over the patient, that's OK. In addition to the bleach bath, you can also control staph aureus carriage, by prescribing mupirocin ointment twice a day to the inner nares, axillae, and groin. For the acute atopic flare, you may also cover your patient with anti-staph oral antibiotics.

When to Refer Your Atopic Dermatitis Patient?

When should you refer your atopic dermatitis patient to a clinical dermatologist? Firstly, how can the dermatologist help your patient? The dermatologist can provide a second opinion to confirm the diagnosis of atopic dermatitis. Sometimes the patient is given the diagnosis of atopic dermatitis, but, may actually have a diagnosis of cutaneous candidiasis, pityriasis rubra pilaris, mycosis fungoides, fungal id reaction, or some other dermatologic diagnosis that can clinically appear as atopic dermatitis. An accurate diagnosis means accurate treatment.

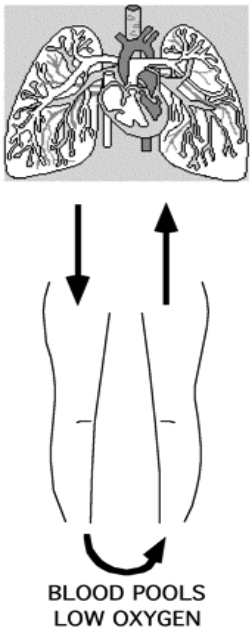
Second, if the diagnosis of atopic dermatitis is confirmed, and, if your patient is beyond topical skin barrier care, topical steroids, oral antibiotics, and bleach baths, you may want to consult dermatology. A dermatologist can help fine tune any ABC educational points your patient needs to know. A dermatologist can reinforce the plan for skin barrier restoration. A dermatologist can prescribe systemic steroids or more potent topical steroids as needed. A dermatologist can treat further with wet dressing therapy, more potent topical steroids, systemic steroids, or cytokine modulating medications. And very important, some atopic dermatitis patients have such a compromised skin barrier and severe secondary excoriations that virulent infectants can pass through the barrier to cause sepsis. In this case, the patient may need ICU admission and an in-house hospital dermatology consultation.

Dermatology Guidelines for the Primary Care Resident: The Essentials

COMMON TYPES OF ECZEMA

Stasis Dermatitis

Stasis dermatitis is a rash stemming from low skin oxygen due to edema, varicose veins, poor circulation, venous insufficiency, the use of harsh soap, and severe serious lack of adequate skin barrier moisture and function. Dry itchy stasis dermatitis usually occurs in chronically swollen legs with poor circulation. Often, your patient sits all during the day with his or her legs dangling down not elevated up and not circulating adequately.



Stasis Dermatitis: What is it?

The patient is usually older than 50 and is usually female with multiple pregnancies, a history of thrombosis, obesity, hypertension, renal disease, CHF and sedentary with a history of previous leg surgeries or injuries. Leg veins have one way valves which help to push the venous blood up the legs and back to the heart. As we age, the one-way valves begin to fail, and blood does not so easily return to the heart. Instead, the blood tends to pool in the legs. With stasis dermatitis, the patient suffers with poor circulation. The legs become sore after standing for long periods. Dry varicose veins begin to protrude or ulcerate. Swelling is seen especially around the inner aspect of the ankles. The legs then begin to swell. The skin become red and itchy especially around the ankles. The skin barrier is disrupted. Cellulitis and dermatitis may ensue. Stasis associated conditions include heart failure, venous insufficiency or any type of compromised circulation. Physical exam may show erythema, scaling, edema, and decreased peripheral pulses. Work up may include Doppler flow studies and cardiac work up for CHF.

Here is a helpful way to explain it to your primary care patients. Normally, your heart pumps fresh oxygenated blood to your legs and feet. The blood is supposed to go down, and is then supposed to return for new oxygen. Stasis dermatitis usually occurs in swollen legs with poor circulation. In stasis dermatitis, the blood is unable to quickly return to the heart for fresh oxygen. The result: Blood tends to pool at the feet, ankles, and lower legs. Because of this, there is low oxygen to the skin tissues of the legs, and thus, stasis dermatitis develops. The skin barrier is disrupted and the skin becomes dry and flaky. Thus, skin barrier care is in order.

Note: Stasis dermatitis is worse in diabetics. Also, if affected areas are injured, or if a person applies firm continuous pressure to the area, the blood is pushed out, the skin suffocates and an ulceration or "pressure sore" may develop. Pressure sores require therapy above and beyond that of regular stasis dermatitis. The patient may need turning every two hours and a wound care specialist.

How Is Stasis Dermatitis Treated?

Think about it: Anything that helps circulation will also help stasis dermatitis. Anything that prevents circulation will also make stasis dermatitis worse. Think about your legs and what you do with them. Are you helping circulation or are you hindering circulation? Leg elevation and / or walking are the two best activities you can do for your stasis dermatitis. Sitting with your legs dangling is the worst thing you can do for your stasis dermatitis.

Dermatology Guidelines for the Primary Care Resident: The Essentials

Soap Is a Big Enemy!

One very important factor that can worsen stasis dermatitis is the use of harsh soap. Harsh or excessive soap will actually make stasis dermatitis worse by stripping the skin barrier. Dial, Zest, Lever, Safeguard, Ivory, bath gels, and Irish Spring are among the worst. Soap removes your skin barrier lipids, which are cholesterol, ceramide, and free fatty acids needed to prevent loss of moisture. If your lipids are removed, your legs develop cracks, and fissures, and become dry and inflamed. Actually, plain water is often just enough to cleanse the skin of the legs.

Stasis Dermatitis Can be Treated by the Four E's

Emolliation
Elevation
Exercise
Elastic Hose

Emolliation: A different word for bathing and moisturizing the skin. “To make the skin soft.” Patients with stasis dermatitis may bathe or shower once daily: Follow the methods described in the Guidelines The “ABC’s” section.

Exercise: Keep your leg muscles always working to increase circulation. Your calf muscles are important as they help pump blood back to your heart. When you use your calf muscles, you are actually helping to bring fresh oxygen back to your leg tissues. The more oxygen the better!

Elevation: When not walking, you should keep your legs elevated. You may want to purchase a small stool to take with you. A drummer’s stool can be very helpful. They are easy to carry and have a soft padded top. Don't ever let your legs dangle as in an airplane or long car trip. Do not stand in one position for very long. Common sense will show you that circulation will improve the more you keep your legs elevated and the more you move your leg muscles. If possible, try a regular walking program.

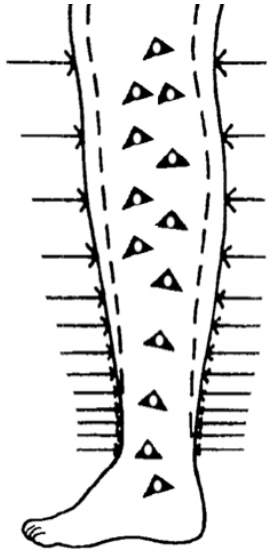
Elastic Hose: To reduce swelling and improve circulation, you can prescribe special compression elastic hose called Jobst support hose available at the pharmacy. The elastic hose prevents outward expansion and prevents blood from pooling and helps return blood to the heart for fresh oxygen. An Unna boot can also compress like the elastic hose, but is impregnated with zinc oxide and is not as friendly for skin barrier therapy.

Other treatment considerations will vary for each patient. Some patient may have excoriations or wounds that will require certain wound care such as antibiotic ointments and wound dressings. Some patients may need a culture swab to rule out infection. Some patients may require po antibiotics for treatment of cellulitis or infected wounds. Some may need treatment of varicose veins. We suggest a low threshold for sending a needy patient to vascular surgery.

Finally, patients may be concerned about the discoloration that often develops in stasis dermatitis. You can explain that the brownish reddish discoloration is due to hemosiderin (from hemoglobin breakdown) deposition from red blood cells that have leaked with increased stasis pressure into the tissues. This leakage occurs when the patient sits with dangled legs, so encourage the patients to keep legs elevated.

Encourage the Patient to Be Patient

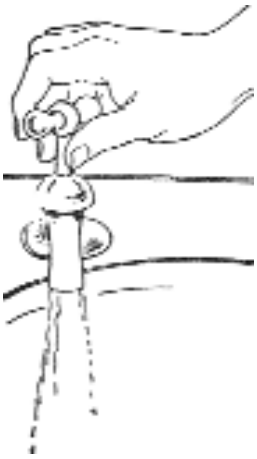
Encourage your patient to be consistent and closely follow the four E’s. Stasis dermatitis can take months to improve. If the patient does not see improvement after two months, please check for compliance with the EEEE’s and ABC’s and consider a vascular surgery evaluation.



Dermatology Guidelines for the Primary Care Resident: The Essentials

Hand Eczema

Hand eczema may occur from frequent hand washings or from use of strong solvents or soaps at home or work. The hands can become easily eczematized. And, for your patient, the most important question will always be, "What am I touching?" As you see, eczemas may occur in a variety of different body locations, and may require area specific treatments. The hands are one of the busiest parts of your body and when it comes to making contact with allergens and irritants, they can get in a lot of trouble. Hand eczema is a subset of contact dermatitis and can be either allergic or irritant. So, for hand eczema, you can refer to the Contact Dermatitis Guidelines for extra helpful information.



Question:
"Doctor, my hands are in and out of water all of the time and I have a very itchy hand rash, doctor, do you have any helpful suggestions?"

Answer: Yes, there is help. Hand dermatitis, also known as hand eczema, is a common dry and sensitive skin condition that is often worsened by frequent hand washing or exposure to water. Dry and Sensitive hand eczema sufferers experience extreme inflammation, irritation, burning, peeling, dryness, redness, cracking, itching, fissuring, and sometimes bleeding of the hands. Hand dermatitis is grouped into four basic types, and all four types may occur together or simultaneously.

First, allergic conditions may result in **allergic contact hand dermatitis**. An example is a nurse who wears latex gloves and develops an allergy to latex. **Second**, caustic or other harsh substances may cause a non-allergic hand dermatitis called **irritant hand dermatitis**. An example is a mechanic who works with gasoline and comes home every day with burning irritated hands. **Third**, emotional stress coupled with an innate predisposition may trigger a blistering itchy hand eczema called **acute and recurrent vesicular hand dermatitis**. Older names for this type of hand eczema include pompholyx and dyshidrosis. The word "pompholyx" is descriptive and derives from the Greek word for "bubble." The word dyshidrosis derives from the notion of "faulty sweating" because at one time, many dermatologists attributed this hand rash to blockage of sweat glands. We now know that this is not true. "Vesicular hand dermatitis" is a much better name for this age-old condition. Vesicular hand dermatitis has tiny pinpoint bubble-like blisters on the sides of fingers and palms. Vesicular hand dermatitis blisters tend to run in certain families. The little blisters itch intensely and tend to increase with mental stress. For example, you can often see vesicular hand dermatitis in an accountant around tax time. **Fourth**, soapy hand washing can deplete the skin barrier lipids and can cause lipid-depleted **xerotic hand dermatitis** as barrier lipids are damaged.

In addition to the four types of hand dermatitis, the hands can also suffer from drying hand conditions such as: diabetes, atopic (eczema-prone) dermatitis, yeast or fungal infections, chronic bacterial infections, psoriasis, scabies, drug induced rashes, and other dry / sensitive conditions of the hands. Because soap strips skin lipids, soap is the #1 enemy. Dry air is big enemy #2. Thus, each of these drying hand conditions will benefit from daily ABC skin barrier care.

ABC Daily Hand Care Moisturizing Method

Explain to your patients: If you are affected, here is a helpful twice a day ABC recipe for daily care of the dry and sensitive skin of your busy hands:

A: Avoid. Stay away from hand allergens. Cleanse your hands with lukewarm water rather than hot. To wash, you may use a gentle, lipid preserving skin cleanser such as Toleriane, excellent. To avoid soap in public places, carry your gentle cleanser with you wherever you go. Another fine gentle hand cleanser is one called Guitar Hands® Cleansing Lotion. Guitar Hands® Clinical Lipid Therapy® is formulated with actual lipids and is designed for guitar players who require perfect hands for their music. But, you don't have to be a guitar player to enjoy healthy hands. Guitar Hands® Cleansing Lotion is available on line at www.guitarhands.com. Two last comments: 1. Guitar Hands® is nice to use

because it is rinse optional. In other words, with Guitar Hands® you *can* rinse your hands with water, but you're not required to rinse with water. 2. Guitar Hands® also helps replace barrier lipids.

B: Bathe. Bathe your hands. First, rinse with tap water, then, soak your hands in pure distilled water for three minutes. Let the distilled water penetrate the dry skin of your hands. After you cleanse, apply a heavy cream to seal in moisture.

C: Cover. Liberally apply a heavy fragrance-free cream like Lipikar Eczema Cream. Or, for more severe hand eczema, apply Cerave Ointment. At night, apply cream and cover hands with cotton gloves. Use cream, not lotion. Lotion evaporates too easily and does not protect as well as cream.

"How do I use my creams and medicines?"

Explain to your patients: When undergoing treatment, remember this very important truth: Every time you wash your hands, you also wash away your important skin lipids, medications, and creams. So, don't forget to reapply your creams and medications several times per day. Hands need continuous care. For severe hand rashes, your patients should reapply their topical medications after each and every hand washing.

As their rash improves, your patients can use the steroids less often. One mistake hand dermatitis patients often make, is that many use the steroid cream, forever as a moisturizer. A steroid cream is not meant for prolonged use. You should warn against this, as steroids can thin their skin with long-term use. Explain to your patients: Once your hands improve, you should maintain frequent hand moisturization with a heavy cream.

Hand Dermatitis Prevention

These guidelines on prevention and treatment work for all four types of hand dermatitis. Explain to your patients three important tips to prevent Hand Eczema: 1. Only cleanse hands with a gentle cleanser like Toleriane or Guitar Hands® Cleansing Lotion. 2. Use hand cream like Lipikar Eczema Cream, not lotion, twice a day whether or not your hands are dry. 3. Use gloves when needed. Very very important: Ruin your gloves, not your hands.

Explain to your patients: Prevention begins at home and in your personal life. If you have an automatic dishwasher, use it. Wash your dishes by machine, not by hand. Avoid direct contact with cat or dog hair, grass, weeds, latex, gasoline, diesel, turpentine, paint, paint thinner, and floor, furniture, metal, and shoe polishes. Avoid irritating solvents. When working, if allergic to latex, wear vinyl or cotton, not latex gloves. Buy gloves at the pharmacy or hardware store and always wear them for protection. Do not let your hands contact soap or shampoo in the bath, at work, or in public restrooms. Carry your gentle skin cleanser wherever you go. People with severe hands may have to wear gloves when shampooing hair or when washing their body in the shower. Note: Rings collect soap, and can worsen hands by trapping allergens. So, remove rings when doing housework and before washing hands. Also, remember to cover your hands with cotton gloves whenever possible. Take care of your hands and they will take care of you.

After hand eczema has improved, continue daily hand care. There is no fast "magic" cure. Think ABC: **A- Avoid:** Avoid anything allergic: Hands can worsen again when exposed to nickel, perfumes, dyes, hair sprays, shampoos, anti-perspirants, grasses, plants, laundry products, dog and cat hairs, chemicals, aloe vera, acrylic nails, nickel, elastic, latex, diaper wipes, and leather. **B- Bathe:** Soak hands in distilled water for three minutes and cleanse with a gentle cleanser such as Toleriane, or Guitar Hands®. **C- Cover:** Apply a heavy cream like Lipikar Eczema Cream several times a day and keep hands covered at night with cotton gloves.

One extra point for prevention and treatment of vesicular hand dermatitis: Stress control. Your patient may need either a clinical psychiatrist or psychologist depending on the severity of his or her emotional stress.

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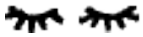
Eyelid, Nummular, & Xerotic Eczema Subtypes

Eyelid Eczema



Q: "I have dry itchy eyelids, any suggestions?"

A: Eyelid Dermatitis: Itchy eyelids are usually due to an allergic or irritating substance that touches the eye area. So, eye areas need extra avoidance of allergic items. Remember, the eyelid skin is very thin and is highly susceptible to penetration by allergens and irritants. Thus, eyelids are easily irritated. Be careful to not rub your eyelids with your hands. It's easy to forget. You may touch a dog or cat, soap, grass, or other allergen, and then, you may rub your eyes with your hands. What happens? You transfer the dog or cat dander from your hands to your eyelids. Because of your eyelid's thin skin, your eyelids become itchy. Your hands may not itch because the skin of your hands is thicker. Potential eyelid irritants include any type of eye drop, soap, lotion, or cosmetic used on face or scalp. Also, eliminate perfumed shampoos, conditioners, hairsprays, and face cleansers. Identify all these and avoid.

Treatment of eyelid eczema is strict ABC barrier care and soothing topical steroid care. In most cases hydrocortisone 1.0 is a good choice.

Nummular Eczema AKA Discoid eczema

Nummular eczema consists of dry itchy coin shaped scaly red patches mainly on the legs and back. The word "nummus" means "coin" in Latin, and is a descriptive term as the lesions are round in shape. The nummular lesions range in size from .5 to 4 inches in diameter. Lesions occur mostly on the legs but also occur on the trunk, arms, hands, and feet. They may become secondarily infected with staph. Men are more likely to have nummular eczema than women.

Nummular eczema is more common in colder climates where furnaces are turned on and the air is dry. Nummular eczema may be due to islands of severe xerosis scattered throughout a sea of non-xerotic skin. The xerotic islands have a damaged skin barrier, and thus, healthy skin barrier care is important in prevention and treatment. Contact allergens may also play a role. If a patient has a sensitivity to a certain allergen, the skin will only clear when the person avoids that allergen. Also, anything that dries the skin will make nummular eczema worse; for example, Accutane Rx.

Treatment of nummular eczema is strict ABC care and topical steroid care. If infection is suspected, then cutaneous cultures and antibiotic therapy may be indicated. Triamcinolone is a good choice in most cases. More resistant cases may require stronger topical steroids or systemic steroids.

Xerotic Eczema AKA Asteatotic Eczema

Asteatotic eczema also called xerotic eczema, or eczema craquelé, asteatosis, and the dry skin rash, occurs in skin that is chronically dry and inadequately moisturized. This rash can develop in patients who scrub their skin with too much Dial, Zest, Irish Spring, Ivory, Safeguard, Lever and bath gels. "Asteatotic" means no oil. The skin barrier is empty. Because it often occurs in winter, it is sometimes referred to as winter itch. Thus, asteatotic eczema is seasonal. For example, you would expect to see it in New York in the winter with furnaces turned on, but not in Hawaii in the summer. In eczema craquelé, the skin is so dry, that it has the appearance of a dry lake bed. You see large reticulated patches of dry skin like a big angulated dried out puzzle sitting on a table.

Treatment of asteatotic eczema is strict ABC skin barrier care and topical steroid care. Ammonium lactate 12% cream may also be of helpful use. If infection is suspected, then cutaneous cultures and antibiotic therapy may be indicated. Triamcinolone is a good choice in most cases. More resistant cases of asteatotic eczema may require stronger topical steroids and/or systemic steroids.

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Neurodermatitis AKA Lichen Simplex Chronicus

Ogden Nash once said, "Happiness is having a scratch for every itch." And, neurodermatitis AKA lichen simplex chronicus AKA LSC starts as a simple itch. The itch will begin anywhere on the body: On the ulnar forearm, anterior tibial leg, shoulder, or nape of the neck. The anal and genital areas are especially common. The itchy spot is often one single spot that stands out and yells, "Scratch me, scratch me, scratch me, please!" So, the patient begins to itch, and scratch and an intense itch-scratch cycle ensues. The itch is often associated with emotional stress. For many, the spot is itchiest when the patient is trying to relax or sleep. In fact, the itch can awaken the patient at night.

In neurodermatitis, it is rare for the patient to have more than two distinct itchy LSC spots. This point helps differentiate neurodermatitis from similar multifocal itchy conditions such as urticaria.

When nodules develop, nodular neurodermatitis is also referred to as prurigo nodularis. Chronic rubbing changes the architecture of the skin. As the itch-scratch cycle continues, the site becomes thick and leathery. This is called lichenification. Hence, the term lichen simplex chronicus. Lichen is a descriptive term from the Latin word for "tree moss." And descriptive it is, as neurodermatitis does resemble moss growing on a tree. Thus, in neurodermatitis you see thickened epidermal cell proliferation like that of psoriasis, but the cellular transit time is not as fast. You also see increased mitochondrial enzymes in epidermal skin cells and an increase in the number of melanocytes of the basal layer. It is obvious that continuous, repeated self-induced scratching plays an important role in the psoriatic like epidermal cell proliferation seen in chronic neurodermatitis. And, the successful treatment of lichen simplex chronicus is almost impossible if the repeated chronic scratching behavior is not stopped.

Treatment of Neurodermatitis

Treatment of neurodermatitis should involve skin barrier care, the ABC's, topical and/or intralesional steroids, and stress management. Antihistamines can also be tried, especially the sedating type. For primary care residents, to prevent unneeded treatments, a biopsy may be helpful to define the diagnosis. For example, we know of one resident who excised a large 5 cm section of LSC thinking it was skin cancer. A biopsy could have prevented this.

Topical steroids under occlusion can be of big help. The best is a steroid-impregnated tape, generic flurandrenolide, brand Cordran, applied once daily and left in place for up to12 hours. Cordran also prevents the lesions from being scratched. And, this in itself may help. If you can keep the patient from scratching the area for a certain period of time, in many cases, the itch-scratch cycle will subside. Intralesional kenalog can also help significantly.

Other treatment considerations of LSC include treating secondary infections, scarring, open wounds, and hair loss. These affects are reasons to intervene early with ABC care, steroids, and stress management.

Finally, let's talk about our friends, the psychiatrists. Think about the name of this chronic disorder "neurodermatitis." Even the name implies that it is a psychological-neurological condition. And thus, we would like to emphasize the importance of psychiatry in the management of neurodermatitis. Psychiatrists are boarded in both psychiatry and neurology and because emotional stress and obsessive-compulsive behavior may play a role in neurodermatitis. The psychiatrist may give the patient needed clinical psychologic therapy and may prescribe appropriate psych medications.

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Lip Licker's Eczema

Lip licker's eczema is usually seen in children who continuously lick their dry lips. The licking completely destroys their skin barrier, and eczematous inflammation ensues. Of course, the treatment is to stop the licking and apply heavy cream or ointment to restore the barrier. Hydrocortisone 2.5 cream can also help, as can zinc oxide ointment.

Suggested Personal Skin Care Products

Cream: Lipikar Eczema

Gentle Cleanser: Toleriane

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Shampoo: DHS Clear

Face Lotion:
Toleriane Double Repair Face Moisturizer

Notes:

Toleriane Gentle Cleanser can also double as a low allergy shaving lotion. Don't use the Lipikar Cream for the face. Instead, for the face use the Double Repair Lotion. DHS Clear Shampoo is truly the lowest allergy shampoo we know of.

REMEMBER
YOUR DAILY ABC's

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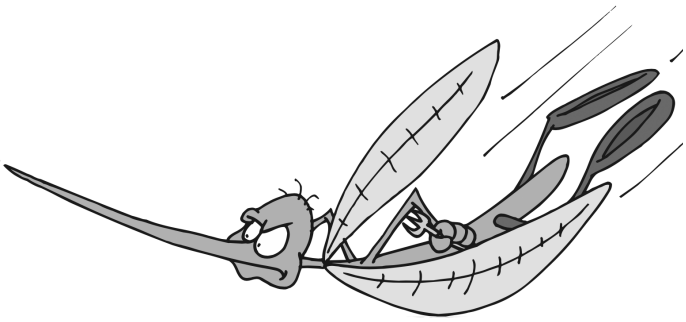
Urticaria Guidelines



What are the Symptoms?

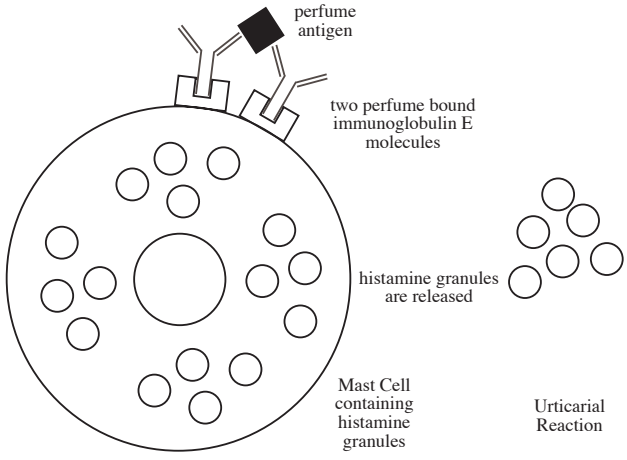
A mosquito bite causes histamine release and is a good example of acute urticarial, or hives. Urticaria is a vascular skin response showing crops of smooth raised, red blanching, haloed, itchy dermal wheals which may come and quickly go if acute, or persist for years if chronic. Crops of hives may come up in a few minutes and may last for 12+ hours. They may disappear, and then, may break out anew in another location.

The lesions of urticaria show sharp, serpent-like borders surrounded by blanching erythema. The more intense hives have a white blanched center. Typical hives range in diameter from several millimeters to over 1 inch, and individual hives usually last for no more than 8 to 12 hours. During the interval of one outbreak, hives characteristically appear and disappear in different places on the body. And, lesions that remain for more than 24 hours in a given spot may not be true hives.

Deeper hives AKA “angioedema” can affect the tongue, pharynx, larynx, and gut in addition to the face and extremities. Angioedema usually burns, and is often caused by similar mechanisms as the lesions of urticaria. However, instead of the reaction being dermal, like in urticaria, the reaction occurs in the deep dermis and subcutaneous tissue and shows mainly swelling. Angioedema and urticaria may arise together or singly, and may each herald anaphylaxis. Thus, it is important that the patient’s airways be fully examined for patency.

Acute vs Chronic Urticaria

The typical breakout of urticaria lasts for no more than several days and is known as acute urticaria. True acute urticaria lasts for no more than 6 weeks. A recurrent attack or one lasting for more than 6 weeks is considered chronic urticaria. The mast cell is the #1 histamine releasing cell in urticaria. The basophil cell is #2. In general, urticaria can occur via either immunologic or nonimmunologic mechanisms. In immunologic urticaria (see diagram below) antigens bind to IgE on the mast cell’s surface resulting in degranulation and histamine release. Once systemically released, histamine binds to H1 and H2 receptors which result in swelling and itch via dilated arterioles, constricted venules, and increased capillary permeability. Non-immunologic urticaria does not depend on IgE binding to a mast cell or basophil. For example, aspirin may induce non-immunologic urticarial histamine release via a drug related mechanism where its effect on arachidonic acid pathways results in histamine release from mast cells. In addition, physical effects such as stroking the skin to cause dermatographism may also incite histamine release via tactile mast cell degranulation.



Other types of nonimmunologic urticaria include heat or cold induced urticaria, solar urticaria, exercise induced urticaria, vibratory urticaria, aquagenic urticaria, hepatitis or EBV induced urticaria, and cryoglobulinemia induced urticaria. In addition to immunologic and nonimmunologic urticaria, there are also cases of autoimmune urticaria associated with

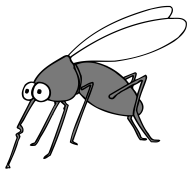
systemic lupus, dermatomyositis, polymyositis, Sjogren's syndrome, antithyroid antibodies, and Still's disease. The occurrence of chronic urticaria with H. pylori infection and celiac disease, has also been reported. Patients with internal neoplasia and malignancies, such as lymphoproliferative neoplasias, monoclonal gammopathies, Schnitzler's syndrome, other hematologic malignancies, and solid tumor malignancies can also present with urticaria.

Urticarial Vasculitis

In its most severe form, urticaria can present as urticarial vasculitis, which is urticaria plus vasculitis together. The most common causes of urticarial vasculitis include: ACE inhibitors, penicillin, sulfonamides, fluoxetine, cimetidine, diltiazem, thiazides, potassium iodide, and non-steroid inflammatory drugs. Autoimmune disease, such as SLE and Sjögren's syndrome may show urticarial vasculitis. Urticarial vasculitis has also been reported with IgA and IgM monoclonal gammopathies, mixed cryoglobulinemias, and, viral illnesses such as hepatitis B, hepatitis C, and Epstein Barr viral infections.

Acute Immunologic Urticaria

Out of the many and various different clinical presentations of urticaria, acute immunologic urticaria is the most common type urticarial reaction that occurs, and this usually resolves within six weeks. However, because hive-like reactions are not always limited to the skin, more serious urticarial symptoms can also occur. And, the same urticaria like reactions can also occur in the trachea, larynx, and bronchi. Dangerously, when histamine release affects these sensitive structures, respiratory obstruction can occur. Certain patients may be allergic to certain foods such as shrimp or lobster and can have, in addition to hives, acute anaphylaxis with difficulty breathing- a medical emergency.



Dermatographism

You can diagnose histamine filled mast cells with a simple skin test. Take a blunt object such as a wooden tongue blade. Using the wooden tongue blade, draw an "X" on your patient's bare back. Wait a minute. A red, elevated "X" on the back indicates "dermatographism," which is a sign of hives. The mast cells were physically degranulated by the tongue blade.

What Causes Hives?

When evaluating your primary care patient with urticaria, you should consider all potential causes. Although most patients do not have an identifiable cause, you should review anything the patient may touch, eat, or breathe. Because urticaria can manifest with both immunologic and non-immunologic mechanisms, hives can be a reaction to many types of possible offenders. As a primary care resident, you may want to consider questioning environmental factors such as an insect bites, certain foods, pollens or molds in the air, and other air borne agents as causes.

You may also want to review the patient's medications. ACE-inhibitors, aspirin, Motrin, Advil, opiates, and codeine have been associated with urticarial. Other inciting factors include insect stings, perfumes, cold, heat,

exertion, infections, digestive disturbances, and emotional stress. Cigarettes, cosmetics, hand lotions, contraceptives, toothpastes, gargles, mouthwashes, and scalp products are also possible causes of hives. Part of treatment should be for the patient to keep a diary of the foods they eat, the items they touch, their medications, and known exposures to anything they are allergic to. Notes should include the personal care items they use, the medicines they take, the time and occasion when they break out with hives, and the duration of their urticarial lesions.

Simple hives are commonly associated with the eating of foods such as a dish of strawberries or steamed crab. Hives will recur each time such foods are eaten again, particularly if alcoholic beverages are consumed with the meal. Other foods to be suspected include eggs, chocolate, nuts, fresh pork, soy, wheat, or milk, cheese (except cottage cheese), wine, tomatoes, navy beans, cabbage, onions, mushrooms, grapes, pineapple, fresh berries, fresh fruits, oranges, grapefruit, lemons, ginger, peppers, tea, coffee, mint, spices, & menthol. Sulfite salts, metabisulfites, and dyes can also cause hives. It is obviously impossible to eliminate all such items under ordinary circumstances. Thus, you can explain to your patients the understanding that if they can associate eating of any of these foods with the hives, that may be important diagnostic info.

Penicillin, aspirin, Motrin, opiates, and sedatives are the most common medications which cause hives. However, most other medications can also cause hives including laxatives, eye drops, and OTC vitamins.

Infections such as dental abscesses, tonsillitis, sinusitis, cystitis, cholecystitis, and ringworm can cause hives. Viral infections are second only to drugs as a cause of hives in children. The infections may be mild and overlooked, or it may be an occult infection. Just know that all viral, parasitic, fungal, or bacterial infections can cause hives.

Emotional stress and mental strain are very frequent causes of hives. In many patients, the emotional problems are well concealed or suppressed, and it is only with great difficulty that a patient can relate emotional causes to his or her itchy skin problem.

Chronic Urticaria

Patients with a personal or family history of asthma, hay fever, or migraine headaches seem to be susceptible to stubborn, chronic hives. Once the cause is identified, the best advice is to avoid subsequent exposure if possible. Please understand that the cause of chronic urticaria is not identifiable in as many as 70 to 85 percent of cases. Known inciting causes of chronic urticaria include drugs and, less frequently, foods, inhalants, pollen, and infestation with parasites. Stress and anxiety are often thought to be very important factors. Why some people react with hives to stress is not known. Despite the most diligent of efforts, the cause is not found in more than 75% of cases of chronic urticaria.

While we have simplified urticaria by classifying immunologic & nonimmunologic, in reality, it is not so simple. There are other types of hives, but for the Guidelines, we have presented a simplified view.

An interesting fact about chronic urticarial patients: When tested, about 40% of patients with chronic urticaria will patch test positive to contact allergens. And, in all of these chronic urticaria patients, allergen avoidance leads to complete remission of their chronic urticaria within 1 month.

Thus, patch testing for contact sensitization is helpful when managing chronic urticaria. If the patient can just avoid the allergen, he or she may be cured.

Hives Can Be a Symptom of Sensitive Skin

Hives may play a role in sensitive skin, as people with sensitive skin have increased immune reactivity & increased immunoglobulin E “IgE” action in their skin. Atopic patients can experience acute hives with their increased IgE levels. However, the incidence of chronic hives is not increased in atopic patients.

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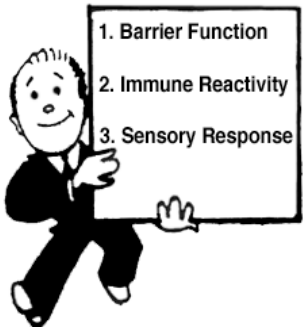


So, people with sensitive skin can experience hives but hives do not necessarily mean that the patient has atopic dermatitis. Interestingly, hives can occur separately from atopic dermatitis and separately from any other form of eczema. Though they are both symptoms of sensitive skin, you should think of hives and eczema as two separate entities as hives has a different mechanism than eczema.

Contact Urticaria Syndrome

As we review the topic of urticaria, we thought it would be helpful for the primary care residents to understand a bit about contact urticaria. From time to time, clinically, you will see patients that don't completely fit the design of true urticaria. These patients are sitting somewhere in between true contact dermatitis and true urticaria. Dr. Howard Maibach of UCSF studied this condition and coined the phrase "contact urticaria." These are patients who, when they touch a certain allergic something, instead of getting the classic poison oak vesicular eczematous eruption react quickly with brisk urticaria. Contact urticarial reactions erupt in minutes after contact exposure to the agent. The patient feels burning, tingling, swelling, and itching with the formation of a classic wheal and flare of urticaria. Interestingly, the total serum IgE level is not changed. As in typical urticaria and typical contact dermatitis, contact urticaria has both immunologic and a nonimmunologic varieties. Immunologic contact urticaria is a type 1 hypersensitivity reaction mediated by IgE antibodies specific to the antigen, and thus, requires presensitization. Also, for immunologic contact urticaria, the radioallergosorbent test for allergen-specific IgE, ie: RAST, is often positive. On the other hand, nonimmunologic contact urticaria can't be diagnosed by RAST.

Patients with immunologic contact urticaria are usually treated with antihistamines and / or an Epipen injection if needed. Dangerously, these immunologic contact urticaria patients can progress to anaphylaxis. The immunologic patients are the type of patients who can become anaphylactic simply by touching latex gloves. Nonimmunologic contact urticaria is the most common contact urticaria reaction. It does not require prior sensitization in order to erupt. The exact process of nonimmunologic contact urticaria is poorly known. The latest studies show that nonimmunologic contact urticaria may be prostaglandin mediated. The reactions of both immunologic and nonimmunologic contact urticaria may vary with the contacted site, the concentration, and the exact allergen.

How to Treat Hives

First things first: The evaluation and management of your patient for any possible signs or symptoms of anaphylaxis is of the utmost importance. Airway, breathing, circulation must be checked. If there is any compromise, of course, that takes precedence. We suggest keeping Epi-pen or similar available and 911 ready if ever needed.

Other than in cases of anaphylaxis, the most important goal in the treatment of hives is to identify the actual cause of the hives. This may be impossible. But, whenever there's an identifiable trigger in hives, that triggering agent should be eliminated. Simple avoidance of these allergic items is usually the first and best way to control hives in sensitive skin people. This is where the ABC's can play a role, at least the A-Avoid component.

So, if your patient is using any obvious histamine releasers, he or she should be made aware and d/c them. If you cannot find an obvious offender such as aspirin, ibuprofen, perfume, laundry detergent, food, contact allergen, etc., you will need to do a work-up. You may need to check a chest x-ray, sinus series, stool for O&P, C1Q inhibitor enzyme assay, ANA, hepatitis screen, and others as you see clinically relevant.

Dermatology Guidelines for the Primary Care Resident: The Essentials

In primary care, workup should be done to rule out the many possible underlying causes of your patient's urticaria.

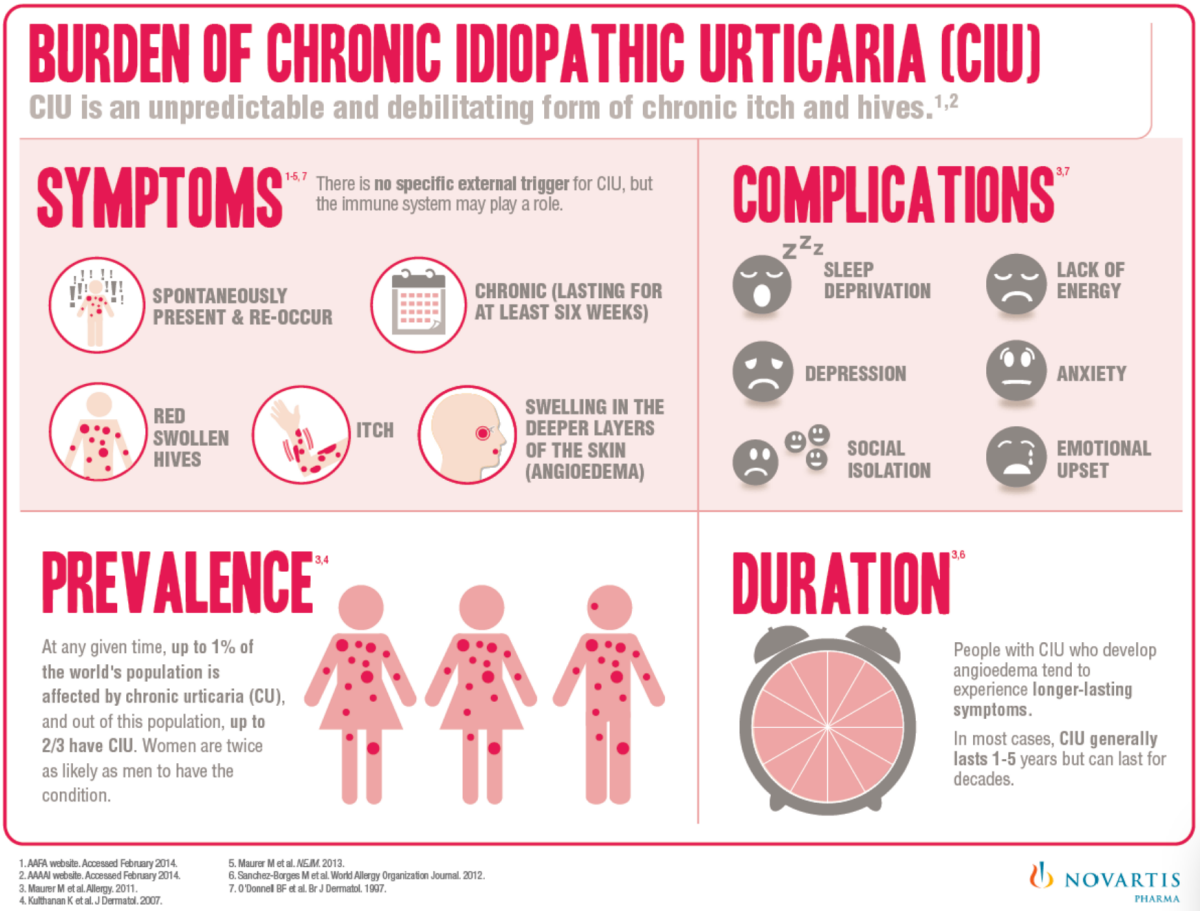
Until the offender is found and eliminated, and if work up is negative, you may prescribe H1 antihistamines to relieve symptoms. It's interesting to note that atopic dermatitis patients feel better if they take daily antihistamine therapy, and so do patients with urticaria. Because the antihistamines block histamine release at the mast cell, and not at the histamine target, patients should be taught that their antihistamine meds should be taken daily and not prn. Once the histamine is out of the mast cell, it is out and working, so explain that it is best to prevent the release of histamine by taking the med daily. Take sedating antihistamines at bedtime, and non-sedating antihistamines in the AM. You may also prescribe an H2 blocker such as Zantac or similar. For the occasional flare, a steroid injection or pack may bring needed relief if the patient has no contraindications. In addition, Singulair or similar may be tried.

When to refer?

If the patient is not better and you need additional help with management, a biologic agent such as Xolair, or with patch testing, you may refer to dermatology. One important point to note is that your treatment resistant chronic urticaria patient *could* have an underlying malignancy such as a lymphoma, monoclonal gammopathy, or solid tumor. Please rule out internal malignancy before sending to derm.

Xolair

When the cause of urticaria cannot be identified, internal malignancy has been ruled out, and all else fails, Xolair may be the answer. For Xolair, we suggest a referral to dermatology or the allergist. Xolair is an injectable biologic agent, a recombinant humanised anti-IgE monoclonal antibody. Xolair inhibits the binding of IgE to the high-affinity IgE receptor located on the surface of mast cells and basophils. Xolair is fda approved for the treatment of chronic idiopathic urticaria. Here is information from Novartis, the company who makes Xolair.



Urticaria & Angioedema Summary

Note: In general, if your patient's case of urticaria is not straightforward, a detailed history, thorough physical examination, skin biopsy, autoimmune, hematologic, and serologic blood tests, radiologic investigation of chest and sinuses, autoimmune work-up, elimination diet, and skin testing all may be required to determine a cause for urticaria. Before referring to derm, the family medicine resident may try:

History

Hives may be caused by the following:

1. **Drugs:** penicillin, opiates, ibuprofen, and aspirin most common.
2. **Foods:** nuts, fish, dairy products, tomatoes, shell fish, and berries.
3. **Infections and infestations;** occult infections of the sinuses, dental region, gall-bladder, tonsils; hepatitis, syphilis, mononucleosis, candidiasis, and parasitic infestations.
4. **Internal diseases;** lupus erythematosus, rheumatoid arthritis, carcinomas, internal malignancies of any kind, leukemia, and lymphoma.
5. **Psychogenic factors.**
6. **Genetic predisposition;** hereditary angioedema and familial urticarias.
7. **Physical factors;** local or total-body exposures, heat, ultraviolet light, vibration, pressure, and dermatographism (friction).

Diagnostic Data

❏ A sinus series may show chronic sinusitis, a CXR may show infection or tumor. Dental x-rays may show an abscess.

❏ Check CBC, Chem panel, Hepatitis Screen, C1Q inhibitor esterase assay, Total Ig's, IgE, RAST to various foods, dust, danders, trees. grasses, molds, etc. (call the lab for a list of RAST panels), stool for O and P, ANA panel, Sed Rate, cANCA, pANCA, HSV I and II titers, CMV titers, EBV titers, hepatitis panel, Rheumatoid factor, and Cryoglobulins as appropriate.

Therapy

❏ Evaluate and treat any positive historical, medical, or lab related findings.

❏ Antihistamines: Non-sedating q AM Sedating q PM.

❏ Patients should be asked to avoid any exposure that even remotely seems to worsen their urticaria. Aspirin, opiates, and Motrin are common exacerbating factors, and patients should be taught to avoid these. Patients should follow a low allergy life style. This means avoid any type of fragrance, room deodorizers, automobile fresheners, wash clothes in pure baking soda, no fabric softeners, no dryer sheets. The patient should avoid soap, especially Dial, Zest, and Irish Spring. Use Toleriane Cleanser. The patient should use fragrance free DHS Clear shampoo and conditioner, and the patient should beware of pet allergies.

If there is no relief, or the diagnosis is in question:

❏ Please refer the patient to dermatology or to the allergist. Please document all therapies you have tried. Please document any positive historical, medical, or lab related findings as listed above.

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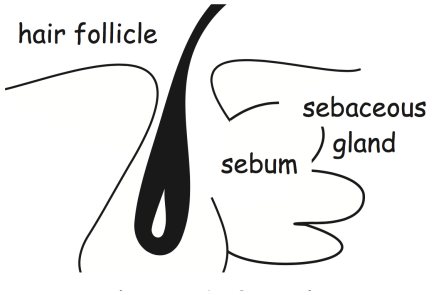
Seborrhea Guidelines



Seborrheic Dermatitis - Q & A

As a primary care resident, you will often encounter a patient with an itchy face and scalp. Most of these patients will have seborrhea. Seborrheic dermatitis AKA "seborrhea" is one of the five most common skin conditions you will see in your primary care clinic, and can affect all ages. In the Guidelines, we will try to answer some of the more common questions your patients may have about seborrhea. Bottom line: Therapy will require consistency, patience, time, and daily washing of the hair and face.

What is seborrheic dermatitis?

Seborrheic dermatitis is chronic, scaling, itching, and inflammation of the sebaceous oil glands. It is most commonly found in people with oily skin. Seborrhea is not contagious to others, but seborrheic dermatitis may be associated with emotional stress. Literally, "seborrhea" means "freely flowing oil" and refers to "sebum," the oily secretion of the sebaceous glands. The sebaceous glands make and release sebum. This fatty oil makes a thin film over the skin's surface and prevents excess water loss from the skin. An early form of seborrheic dermatitis may be nothing more than simple dandruff. As seborrhea worsens, the sebaceous glands become more and more inflamed and more sebum is produced. When dandruff becomes oily, it is referred to as seborrhea. Seborrheic dermatitis is usually itchy. Tiny white scales form on the scalp, face, and ears and fall onto shoulders. It is embarrassing for the patient to see these white scales on their dark-colored clothing. TV and magazine ads can make your primary care patients feel self-conscientious. Instead of a simple localized scalp condition, scaly crust-like patches may form around the hairline, behind the ears, in the external ear canals, around the neck, or on the chest and back. Seborrhea can happen anywhere that sebaceous glands occur. These areas become inflamed, irritated, and uncomfortable.

How does seborrheic dermatitis appear?

Seborrheic dermatitis appears like red-yellow patches of skin with a thin, greasy scale. The patches form in groups in oily skin areas. Warm and moist areas such as the creases of the neck, groin, armpits, and inframammary areas may have larger red, itchy patches.

Which body parts are most affected?

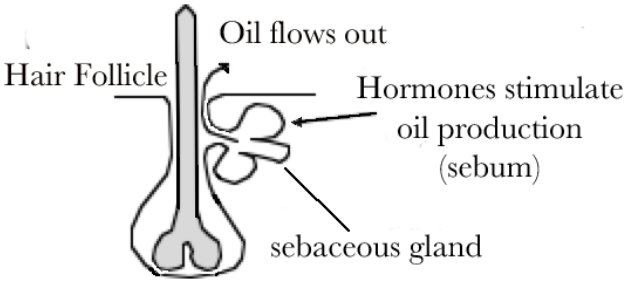
Because seborrheic dermatitis is related to sebaceous gland activity, the areas affected most are those parts of the body which have the greatest number of sebaceous glands: the scalp, face, neck, armpits, center of chest, back, umbilical region, and groin.

What are the more common age groups?

Sebaceous gland sebum production varies with age. It is high in newborns up to one year of age. Thus, infants develop "cradle cap," where the scalp is covered with greasy, yellow crusting. Cradle cap is treated much like adult seborrheic dermatitis, with topical steroids and zinc shampoo. The skin of the diaper area can also be affected by seborrheic dermatitis. However, sebaceous gland activity decreases after infancy and is inactive until puberty. Thus, during

childhood, seborrhea is rarely seen. During puberty, sebaceous glands increase in size and become active. Thus, adolescents are prone to develop seborrhea. In most patients, adult levels of sebaceous gland sebum production are reached by age 25. Though sebaceous glands remain active in men until older age, female sebaceous gland activity generally decreases after menopause.

What causes seborrheic dermatitis?



The cause of seborrheic dermatitis is unknown. Though seborrhea is not contagious, it may be related to growth of malassezia yeast often found on normal skin in minimal amounts. With the increased scaling and oil in seborrheic dermatitis, malassezia yeast can increase and promote sebaceous inflammation. In addition to yeast, there are other factors that contribute to seborrhea:

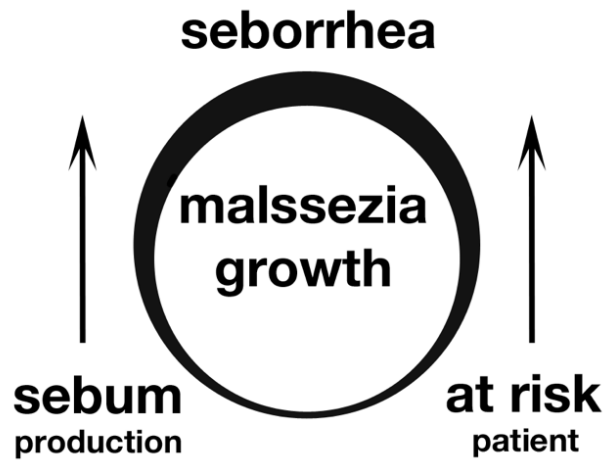
1. Heredity
a. Down syndrome
b. Family history
2. Mental stress
a. Psychiatric disorders
3. Sebaceous Gland activity
a. Birth
b. Puberty
4. CNS Disorders, Neurogenic Factors
a. Parkinsonism
b. Strokes
c. Brain injuries
5. Obesity
6. Immunosuppression
a. Immunosuppressing medications
b. HIV infections
6. Metabolic / Nutrition
a. Zinc deficiency
7. Skin Barrier Dysfunction

Is seborrhea related to other skin disorders?

The answer is, “Yes.” Patients with psoriasis may have seborrheic dermatitis. However, psoriatic patches are fine, dry, and white. Also, patients with acne often have seborrheic dermatitis, as acne, is related to sebum production. In addition, many patients with Rosacea and perioral dermatitis have seborrhea. Thus, patients with seborrhea may also need therapy for psoriasis, acne, or rosacea.

Who gets seborrhea?

Seborrheic dermatitis is a skin condition that comes and goes depending on colonization with malassezia, androgen levels, sebaceous gland activity, and sebum production. Thus, depending on the patient’s particular time of life, he or she may oy may not have a problem with seborrhea.



What is the treatment for seborrhea?

The one most important thing your patient can do to get rid of and prevent seborrhea is to wash their hair and face every day. Why is this? The washing removes old sebum and malassezia, and thus, prevents the metabolism of sebum by malassezia. You see, *malassezia* has strong lipase activity and the lipase hydrolyzes human sebum triglycerides to release unsaturated fatty acids oleic acid & arachidonic acid. These by-products cause unwanted parakeratosis, irregular lipids, a faulty corneocyte envelope, and a damaged skin barrier with resulting inflammatory cytokines IL-1α, IL-6, IL-8, and TNF-α with inflammation. Washing may be a

problem for certain patients, but daily washing removes old sebum and malassezia. You can explain to your patient the analogy of butter left on the table. Over time, it spoils and turns rancid. In the same way, sebum, if left unwashed, will turn rancid, spoil, and cause itchy seborrheic inflammation. There are some people who shampoo once per week or once per month and never get seborrhea. Unfortunately, if your patient is the one who suffers with seborrhea, he or she will need to wash their hair every day. Your patient should not expect their seborrhea to go away unless their hair is washed daily. In addition to frequent hair washing, perhaps the most annoying thing about seborrheic dermatitis is that it's a recurring condition. The bottom line: Though there is no known cure for seborrheic dermatitis at this time, a great deal can be done to control its symptoms. Most patients will need some form of long-term treatment. Generally, there are several helpful measures to find relief.

Medications

Topical therapies for seborrheic dermatitis include:

Topical steroids: Hydrocortisone 2.5 solution

Topical antifungals: Ketoconazole 2% gel

Medicated shampoos: Zinc, Tar, and / or antifungal shampoos. Note: Zinc is best for lighter colored hair, tar is best for darker colored hair. Antifungal shampoos include those containing either ketoconazole or ciclopirox

Extra virgin olive oil. Apply liberally to the scalp before bedtime, shampoo in the morning. Olive oil helps to displace rancid sebum oils.

Notes

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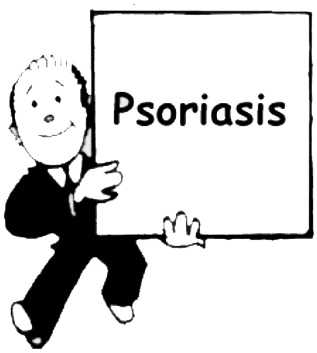


PSORIASIS Q & A

DEFINITION

Psoriasis is a common, recurring, autoimmune skin condition in which the skin develops rosy silvery plaques and scales. Psoriasis most often affects the scalp, trunk, arms and legs. Lesional pain, itching, cracking, and bleeding are common. The palms and soles may also be involved. The nails can be involved with pits and thickening, as can the joints with swollen arthritis pain. The face is usually spared. Psoriasis is a persistent skin disease that can flare and remiss with seasons. The name “psoriasis” is derived from the Greek word for "itch." The skin becomes inflamed, producing thickened areas with silver scales on the scalp, elbows, knees, and lower back. In some cases, psoriasis is so mild that people are unaware they have it. On the other hand, widespread psoriasis may cover large areas. Psoriasis cannot be passed from person to person, though it is more likely to develop in families. In the United States, upwards of 8 million people have psoriasis, and many new cases occur each year. Unfortunately, patients with psoriasis are at risk for metabolic syndrome, and thus, patients have increased incidence of hypertension, diabetes type 2, and heart attacks. Psoriasis is also associated with inflammatory bowel disease, depression, and psoriatic arthritis.

HOW DOES IT BEGIN?

Psoriasis often starts as red itchy skin, a scaly scalp, or as scaly areas of the elbows and knees. About 60% of people with psoriasis have a positive history. Psoriasis can affect anyone. One third of new patients are less than 20 at the onset. And, psoriasis occurs equally in males and females. Beneath the plaques, the skin is erythematous and easily bleeds. Hair loss is unusual. Finger and toenails have subungual yellow-brown spots and superficial pitting. There may be onycholysis. Severe psoriatic flares can present with arthritis, fever, chills, and pustules.

Metabolic syndrome is a common comorbidity in psoriasis patients. Metabolic syndrome is a risk factor based clinical complex associated with insulin resistance, obesity, and excess belly fat. The patients have coronary heart disease, hypertension, heart failure, diabetes, fatty liver, and dyslipidemia. Thus, your psoriasis patients must be evaluated re their cardiovascular status. And, it is wise to rule out diabetes in your psoriasis patients. Incidentally, there have been lawsuits against doctors who did not inform their patients about the association of metabolic syndrome with psoriasis.

WHAT CAUSES PSORIASIS?

Psoriasis is the result of an aberrant TNF driven cytokine response in which excessive and unneeded cutaneous and synovial inflammation takes place. Tumor Necrosis Factor was first discovered as an immunologically active circulating factor that caused tumors to necrose, hence the name, Tumor Necrosis Factor, AKA TNF. As the actions of TNF were further elucidated, TNF was found to be one of the first mediators of the body’s inflammatory response and it was found that TNF orchestrates local and systemic inflammatory responses by initiating cytokine cascades, especially of cytokines IL-12, 17, and 23. Where is TNF found? TNF is produced primarily by activated monocytes, macrophages, and mast cells; and also in lesser amounts by T cells, B cells, NK cells, fibroblasts, hepatocytes, splenocytes, cells of the ovary, epidermal cells, and thymic stromal cells. TNF in large amounts is also stored in mast cells granules. In case of a cancerous or infectious invasion, to protect the body, all of this TNF is ready and waiting for release. Thus, TNF mediates the body’s response to fever and

sepsis. TNF is also a cancer killer. So, TNF is a good thing. But, when it comes to TNF, if there is too much, TNF can also make everything worse. For example, too much TNF can actually promote septic shock, too much TNF can actually promote cancer, and too much TNF can promote psoriasis, ulcerative colitis, Chron's, inflammatory bowel disease, rheumatoid arthritis, ankylosing spondylitis, and hidradenitis suppurativa. Thus, TNF is a key player in fighting certain conditions, plus TNF is a key regulator in the pathogenesis of several autoimmune diseases. Of critical importance is the actual systemic level of TNF. Too much, or too little, and the body is in trouble. So, in the human body, TNF balance is critical. And so, you see, psoriasis is the result of an aberrant TNF driven cytokine response igniting excessive and unneeded inflammation.

Five Types of Psoriasis

Plaque psoriasis: Plaque psoriasis is the most common type of psoriasis comprises about 80% of patients.

Guttate psoriasis: Guttate psoriasis is common in childhood, especially after a strep infection, and shows small pink spots.

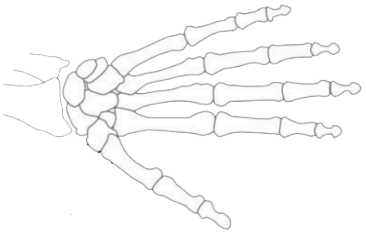
Pustular psoriasis: Pustular psoriasis is more common in adults and shows white, pus-filled blisters and large areas of inflamed skin. Pustular psoriasis typically comes with a psoriatic flare.

Inverse psoriasis: Inverse psoriasis includes patches in the axillae, inframammary areas, in the groin, and around the genitals.

Erythrodermic psoriasis: Erythrodermic type psoriasis is a severe form of psoriasis that arises with a psoriatic flare. This type can be life-threatening with high output cardiac failure, so patients are often admitted to the hospital.

PSORIATIC ARTHRITIS

Ask your patient about joint pain. About 15% of psoriasis patients suffer with psoriatic arthritis. This type of arthritis can lead to permanent joint deformity. The gold standard of treatment is biologic therapy with a TNF inhibitor such as Humira. The nice thing about Humira anti-TNF action is that Humira can actually reverse psoriatic joint deformities, radiographically.



WHAT TRIGGERS PSORIASIS?

Stress, alcohol consumption, physical injuries, medications, and infections can all trigger flares of psoriasis. Offending meds include anti-hypertensives such as beta blockers.

Is psoriasis contagious? Despite the psoriatic association with streptococcal sore throat, and chlamydia induced Reiter's syndrome, psoriasis is an inflammatory skin condition, not an infection, and cannot be transmitted from person to person. Physical trauma such as scratching, though, can cause the disease to appear in different areas of the body of the same individual. This is called "Koebnerization."

WAYS TO TREAT PSORIASIS

Today, the most efficacious treatments for psoriasis will involve biologic anti-TNF meds such as Humira (adalimumab), and interleukin cytokine modulating types of therapy such as Stelara (12/23) and Cosentyx (17). These meds require certain expertise on the part of the prescriber. The most important concerns would be exacerbation of infections. Biologic agents can reduce the ability to fight infections and thus, infections can be amplified. For example, tuberculosis can be reactivated. A simple pharyngitis can progress to a peritonsillar abscess. So, careful monitoring is needed. Very rare side effects of biologics include congestive heart failure, lupus, hepatitis, lymphoma, hematologic cytopenias, demyelination, and polyneuropathy. In patients with any of these problems, biologic use should be carefully reviewed. Despite these very rare adverse

reports, biologics meds should be viewed as a relatively safe and effective form of treatment for the vast majority of psoriasis patients for whom they are prescribed.

Methotrexate, cyclosporine, and acitretin are also possible treatment options for psoriasis, but these also have potential severe side effects.

Here is an enlightening quote by Psoriasis expert, Craig Leonardi, MD: "Methotrexate is a drug that the insurance industry says we have to flow through on our way to biologic drugs. But if complete clearance is your goal, this is an exercise in futility. These patients will never, ever get to complete clearance – or it's at least very unlikely. We shouldn't be asked to go through methotrexate on our way to anything. We shouldn't be asked to use methotrexate at all. We should be bypassing it."

Thus, with so many side effects associated with systemic treatments, for the primary care resident, the treatment of psoriasis will consist mainly of medicines applied to the skin. The goal is to reduce inflammation and to slow down rapid skin cell division. Moisturizers loosen scales and help control itching. Diet? Special diets have not been successful. Treatment is based on the severity of the psoriasis. Different types of treatments and several visits may be needed. As a primary care resident, you may prescribe topical medications applied to the skin containing cortisone, vitamin D, tar, or anthralin. These may be used in combination with natural sunlight. Sunlight exposure helps most people with psoriasis, but it must be used with caution.

PASI-- Psoriasis Area and Severity Index-- A patient's PASI score is a measure of his or her psoriasis severity and extent. The PASI is a common way used to measure efficacy in clinical psoriasis research and treatments. But, PASI does not evaluate psoriatic arthritis. Treatment results of PASI 90 indicate 90% clearance of psoriasis. Treatment results of PASI 100 indicate 100% clearance of psoriasis.

PASI evaluation consist of two clinical steps:
1) Calculate total involved body surface area.
2) Assess the severity of lesions for erythema, induration (thickness), and scaling.

Rebound Flare—Before we proceed with discussing treatment, we would like to discuss one of the most important mistakes that primary care physicians make in their treatment of severe psoriasis. A patient comes in with a raging psoriatic flare and the primary care physician loads the patient with prednisone or solumedrol or other systemic steroids. The patient cools down and the psoriasis soon clears…But then, like a tornado, the patient gets a rebound flare of psoriasis even worse than before. Just remember, steroids cause a rebound flare of psoriasis. For extreme flares, it is best to use IM methotrexate or an anti TNF biologic like Humira combined with topical care.

Types of Treatment-- Topical Steroid creams, ointments, and lotions may clear psoriasis temporarily and control it in many patients. Less potent preparations should be used on more sensitive areas of the body such as the genitals, groin, and face. More potent preparations will usually be needed to control lesions on the scalp, elbows, knees, palms and soles, and parts of the torso, and may need to be applied under occlusion. Side effects of potent topical steroids include thinning of the skin, dilated blood vessels, bruising, and skin color changes. Stopping these medications suddenly may result in a psoriatic flare. After many months of treatment, the psoriasis may become resistant to the steroid preparations. Once again, beware and do not fall into the trap of using systemic steroids in the treatment of psoriasis. Upon discontinuation of the systemic steroid, the patient will often experience a severe flare of psoriasis, sometimes requiring hospitalization.

Scalp Treatment--The treatment for psoriasis of the scalp depends on the seriousness of the disease. All scalp psoriasis patients should use some type of anti-psoriatic shampoo such as salicylic acid shampoo, tar shampoo, or zinc shampoo. Careful, tar shampoo turns white hair to yellow. **Vitamin D**-- Vitamin D, calcipotriene, is available as topical. It is useful for individuals with localized scalp psoriasis. Limited amounts should be used to avoid side effects. Ordinary Vitamin D oil, as one would buy in a drug store, is of no value in treating psoriasis. Calcipotriene is a synthetic form of vitamin D3 used for

treating mild to moderate psoriasis. Sold as Dovonex in the U.S., this prescription medication is available in a cream, an ointment and a scalp solution. It is not known for working quickly, but it is effective and safe for long-term control of psoriasis, with few side effects. The drug is not recommended for treating psoriasis on the face, and it can cause temporary skin irritation. To avoid too much medication being absorbed internally, people are advised not to use more than 100 grams of Dovonex cream or ointment or 60 ml of scalp solution per week. Another silver bullet for the treatment of scalp psoriasis is Enstilar Foam. Enstilar Foam is a combination betamethasone-calcipotriene foam that works wonders on the scalp.

Another helpful point: If vitamin D is useful in the treatment of psoriasis, it makes sense that all patients with psoriasis should keep their serum vitamin D levels within normal range.

Sunlight: Five minutes of direct sunlight each day will often help psoriasis.

Topical Vitamin A: Topical tazarotene cream can be prescribed by itself, but more and more dermatologists are prescribing it in combination with a topical steroid. The results are better, and the side effects are reduced, especially the skin irritation that tazarotene can cause. The drug may also cause the psoriatic plaque to turn red before it clears, but this is a normal reaction and it will go away. Tazarotene is a prescription topical retinoid (or vitamin A derivative) fda approved for treating mild to moderate plaque psoriasis. Sold in the U.S. under the brand names Tazorac and Fabior, this medication is available in a gel in two strengths: 0.1% and 0.05%. Tazarotene only needs to be applied once per day, and, tazarotene can be used to treat scalp psoriasis and nail psoriasis, as well.

Biologic Agents: Before the biologics, psoriasis patients suffered greatly. The disfigurement, joint pain, skin discomfort, and social isolation was more than many could endure, and psoriasis took its toll with decreased quality of life, even increased suicides in psoriasis patients. Thankfully, we now have the biologic agents. Today, essentially, no one needs to suffer with severe debilitating psoriasis.

Humira- Today, there are many different biologic agents for psoriasis, some work on TNF, others work on IL 12/23 & 17 inflammatory cytokines. Out of the many biologics available, most dermatologists consider Humira to be the gold standard for safety, efficacy, and ease of use. With 20+ years of clinical trials and use, Humira has one of the longest track records of all the biologics. Interestingly, as a reference point, and because Humira is the gold standard, most of the newer biologics that come out are usually compared to Humira. Here are a few important facts about Humira (Adalimumab):

Humira AKA adalimumab is a biologic TNFα inhibiting anti-inflammatory human monoclonal antibody that binds to tumor necrosis factor alpha (TNFα). In psoriasis, TNFα usually binds to TNFα receptors, leading to the psoriatic inflammatory pathway. By binding to TNFα, adalimumab reduces the psoriatic inflammatory response. Humira is fda approved for treating psoriasis and psoriatic arthritis. And, Humira is also fda approved for treating ankylosing spondylitis, Chron's disease, ulcerative colitis, hidradenitis suppurativa, and JRA, AKA, juvenile rheumatoid arthritis.

Humira provides first line efficacy with rapid and sustainable skin clearance. 52% of patients achieve PASI 90 scores by 16 weeks and 17% achieve PASI 100 scores by week 16. Long term patients were able to achieve PASI 90 (57%) and PASI 100 (38%) after 3 years. Humira also provides first line arthritic joint protection. 90% of Humira treated patients had no radiographic progression of psoriatic joint destruction at 48 weeks. Long term data at 144 weeks showed 79% of patients had no radiographic progression of psoriatic joint destruction, thus, demonstrating long term efficacy. One of the reasons that all of the other psoriatic biologics are compared to Humira is that more than 1 million patients have been treated with Humira. With 20+ years of clinical trial experience and 100+ global clinical trials, the 1st line safety data for Humira is the largest ever published safety database across 10 indications of any drug in the history of the fda. In fact, of the Esprit Registry data, a 10-year ongoing registry following over 6000 Humira

patients, just like those who walk into your clinic with all comorbidities, the 8-year interim analysis showed no new safety signals. Finally, Humira has first line managed care coverage, 97% of managed care insurance groups prefer Humira across the board nationally on all dermatology drug formularies.

Humira's website makes it easy for the patients.

HUMIRA® adalimumab

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Injection training the way you choose.

Watch the self-injection demonstration videos.

You can get step-by-step instructions for injecting HUMIRA now. Select which injection option you use, and then watch the demonstration video.

Injection training support is always available.

Your Nurse Ambassador* can help with any questions you may have about injecting—from setting up an in-home injection training visit to connecting you with online resources.

Don't yet have a Nurse Ambassador? **Sign Up for HUMIRA Complete**. Once enrolled, you'll be connected with your own Nurse Ambassador, who can point you to many treatment resources, including injection training. You can also get a sharps container and mail-back disposal kit for your used HUMIRA Pens or syringes—at no additional cost to you. **Watch this short video** to learn how to use your container, and what to do when it's getting full.

Your Ambassador will call you within one business day. But if you need to speak to someone sooner, call **1.800.4HUMIRA** (1.800.448.6472) to speak with a registered nurse. Nurses† are on call to provide injection help and assistance to patients taking HUMIRA.

*Nurse Ambassadors do not give medical advice and will direct you to your health care professional for any treatment-related questions, including further referrals.

†On-call nurses are available at **1.800.4HUMIRA** (1.800.448.6472) for assistance Monday through Friday, from 8 AM to 8 PM Eastern Time. At all other times, a nurse will return your call within 1 hour.

In This Section

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- Resources for You
- Save on HUMIRA
- Check Your Insurance Coverage
- Insurance Explained
- Your HUMIRA Ambassador
- How to Inject HUMIRA
- Sign Up for HUMIRA Complete

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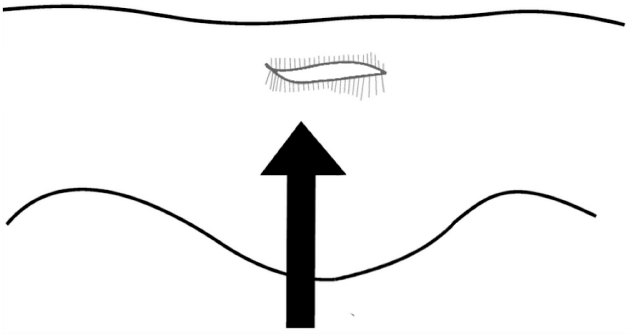
Skin Growths
Section 2

Dermatology Guidelines for the Primary Care Resident: The Essentials

Skin Growths



Benign Growths

In this section of Dermatology Guidelines for the Primary Care Resident: The Essentials, we bring to you the topic of "Skin Growths." You may wonder why we present so many different growths. Here is our thinking: One of the most common reasons a primary care patient visits his or her doctor will be to ask, "Doctor, is this growth OK?" And as a primary care physician, you will need to answer their questions. And, even though most of their skin growths will be skin tags and non-malignant nevi, you should be aware that there are many other different non-malignant and malignant possibilities, and that sometimes, a simple shave or punch skin biopsy may answer their questions.

Acrochordons

Acrochordons AKA skin tags or fibroepithelial polyps are growths of extra skin with no atypia. Acrochordons are pedunculated skin-colored or hyperpigmented papules, soft and non-tender. They occur in all humans of every race. The most frequent locations are axillae, neck, groin area, and the eyelids. Skin tags are sometimes seen in children with certain clinical syndromes or can be bothersome. Other than this, skin tags have no clinical importance and no treatment is required, other than for cosmetic considerations.

Becker's Nevus (Smooth Muscle Hamartoma)

A hamartoma is considered to be a disorganized non-functioning skin growth made of mature tissue that normally occurs within the parent organ in which it is growing. In other words, a hamartoma of the skin is composed of all the cellular elements that make up normal skin tissue. Plus, a hamartoma of the skin develops at the same growth-rate as normal skin, and thus, does not erode adjacent tissues with neoplastic pressure as do cancerous skin growths. Becker's nevi are hamartomas of smooth muscle origin, and are considered to be acquired nevi without melanocytic nevus cells. Becker's nevi most frequently appear on the shoulders, back, chest or upper limbs of young boys. True Becker's nevi appear as hairy hyperpigmented macules with hypertrichosis in the areas of hyperpigmentation. The diagnosis is made clinically, but sometimes, a biopsy is required to confirm that there is no risk of malignancy and that no treatment is required.

Dermatofibroma

The classic look of a dermatofibroma is usually enough to give a correct clinical diagnosis. A DF is a solitary round papule or nodule, usually < 1 cm in diameter, usually brown, or sometimes red with a sharply defined edge. A dermatofibroma is thought to be due to an old insect or arthropod bite, which causes a fibrous tissue reaction. The exact cause is unknown. Dermatofibromas are almost always seen in adults. A dermatofibroma is non-malignant and requires no therapy.

Eccrine Syringoma

An eccrine syringoma is a very familiar non-malignant skin growth. ES's are usually located on the lower eyelids and cheeks of adults. They are of no clinical significance, and are usually ignored in clinics. Eccrine syringomas are < 0.5 cm, flesh-colored papules that are usually multiple and bilateral. Eccrine syringomas are believed to be derived from actual eccrine sweat ducts. Inheritance may be autosomal dominant.

Ephelides & Lentigines

Ephelides, AKA freckles, are non-malignant color changes. They usually appear in childhood in fair-skinned patients and usually patients with red or blond hair color. Freckles represent an area of increased melanin deposition. On the other hand,

a lentigo (plural lentigines) represents a sun-induced proliferation of melanocytes. In a true lentigo, melanocytes and melanin replace the basal cells of the basal cell layer. No nevus cell theques are formed. A lentigo has no malignant potential. Solar lentigines usually arise in the adult population, and are also more frequent in light-skinned patients on sun-exposed areas of the body. Some lentigines blend together to form big lentigines. No treatment is required with the exception of sun avoidance and oral vitamin D.

Epidermoid Cyst

An epidermoid cyst can also be known as an epidermal inclusion cyst, an epidermal cyst, and a follicular infundibular cyst. The term “sebaceous cyst” is a misnomer, because the cyst does not originate from a sebaceous gland. A true epidermoid cyst is usually derived from the follicular infundibulum of a hair follicle. A true epidermal inclusion cyst is derived from implantation of epidermis into the dermis or subcutaneous fat, thus, distinguishing the two. An epidermoid cyst is a non-malignant true cyst with a stratified squamous lining of epithelial cells and granular cell layers. The central cavity is filled with keratin debris. An epidermoid cyst derives from a hair follicle infundibulum. Depending on the size of the cyst and state of infection, cysts sometimes need incision and drainage.

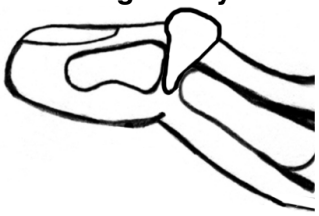
Epidermal Nevus

An epidermal nevus is a non-malignant epidermal hamartoma that most frequently occurs as a verrucous plaque, but can be widespread with related systemic findings. An epidermal nevus has the tendency to follow Blaschko’s lines of embryologic development and is associated with epidermal cell migration during embryologic development. An epidermal nevus sometimes appears in children as a single linear verrucous plaque, and is made of proliferating epidermal cells. Most epidermal nevi are a bit pigmented. Treatment is not usually required.

Fibrous Papule

A fibrous papule is an angiofibroma. Solitary fibrous papules are normal but can appear like other lesions, thus, a biopsy is sometimes done. Classic fibrous papules are usually small, dome shaped, and vascular. The usual location is the face, nose, and chin. No treatment is required.

Ganglion Cyst



A ganglion cyst is a familiar non-malignant fluid-filled cyst that occurs over a joint, and is an extension of the underlying synovium of the joint. Most ganglion cysts have no symptoms, but will sometimes cause pain if large enough. With its proximity to nerves and tendons, it may be good to have an orthopedist evaluate a ganglion cyst. Needle aspiration of joint fluid can be diagnostic.

Hidrocystoma

A hidrocystoma is a common non-malignant skin growth that is most frequently found along the margin of the eyelids. Most often, a small hidrocystoma appears as a soft, solitary, symptom free papule. It is believed that a small portion of the eccrine duct is occluded and eccrine fluid accumulates. Hidrocystomas are sometimes biopsied to rule out a basal cell carcinoma or other pearly entities.

Keloid & Hypertrophic Scar

A keloid is a non-malignant skin growth formed of post injury scar tissue. Inflammation can play a role, such as in acne. Technically, a keloid is a scar that grows beyond the borders of the original injury. Hypertrophic scars, conversely, are scars that stay within the borders of the original scar. The usual treatment is an intralesional steroid injection or silicone patch.

Lipoma

A lipoma is a solitary lump of fat that grows beneath the dermis. Lipomas are slow growing, mobile nodules and tumors, usually without symptoms, but can be tender if hit or bumped. The differential diagnosis of a lipoma includes cysts and other mobile type. Most lipomas can be treated with simple excision, but most of the time,

no treatment is required. Lipomas are usually encapsulated. A liposarcoma is usually **not** encapsulated. And rarely, a liposarcoma can mimic a lipoma. Thus, one should always be on the look-out for a non-encapsulated liposarcoma.

Melanocytic Nevi

For those just beginning their education in dermatology, the term "melanocytic nevus' is often an enigmatic phrase, i.e. a mystery. The number of melanocytic nevi in each patient is related to the whiteness of a patient's skin and the amount of childhood sun exposure. The whiter the skin, the more nevi. The more childhood sun, the more nevi. What exactly is a melanocytic nevus? In simple terms, a melanocytic nevus is a hamartoma of the skin.

By definition, a hamartoma is not a true neoplasm, but is a benign, disorganized defect of embryologic development. A true hamartoma has no function and is a tumor-like growth made of cells that normally occur in the parent organ in which it grows. In other words, a true hamartoma of the skin is composed of all the cellular elements that comprise normal skin tissue. And, a skin hamartoma develops at the same growth-rate as normal skin. Thus, a hamartoma does not erode adjacent tissues with neoplastic pressure as do cancerous skin growths. Melanocytic nevi are uncommon in infancy, then, gradually increase in childhood; and, reach peak numbers in young adulthood. Towards the end of life, melanocytic nevi gradually disappear like a sunset in the skin of senior age patients.

Junctional, Compound, & Intradermal Nevi

Melanocytic nevi are identified as either junctional, compound, or intradermal based on their location. Melanocytic nevi can be present at birth as congenital melanocytic nevi, but usually, first develop during childhood years when nevus cells proliferate at the dermoepidermal junction. Nevus cells, i.e. variant types of melanocytes without dendrites, tend to join together and proliferate to form clusters. At this early stage, the nevus is called **a junctional nevus**, as it is located within the dermoepidermal junction. Junctional activity then continues through early adulthood. With maturity, the nevus cells move to involve both the basal cell layer and the dermis. At this point, the melanocytic nevus is referred to as a **compound nevus**. After this stage, with increasing age of the patient, the junctional nevus cells cease proliferating and the nevus cell clusters move deeper into the dermis to become an **intradermal nevus**. Finally, in the end, some intradermal nevi end up as common skin tags.

Terms

Nevus Cell
Melanocytic nevi are formed of clusters and nests of nevus cells. A nevus cell is a non-dendritic melanocyte derived from the neural crest. A nevus cell is larger than the common melanocyte, and tends to form clusters or nests. Nevus cells can also move throughout the body via a non-cancerous "metastatic" transport mode by way of lymphatics and blood vessels.

Theque
A theque is a group of nevus cells, usually four or more in number. Melanocytic nevi develop when theques collect together as clusters and nests.

Melanocyte
A common melanocyte is a single neural crest derived melanocyte with dendrites. Common melanocytes produce melanin and are smaller than nevus cell melanocytes. They usually remain evenly dispersed and individual within the basal cell layer. Common melanocytes do not usually form clusters or nests, but remain in the basal cell layer present in a ratio of about one melanocyte cell to 30 basal cells.

Junctional Nevus
A JN consists of theques clustered within the dermoepidermal junction.

Compound Nevus
A CN consists of theques in the dermoepidermal junction and also in the dermis.

Intradermal Nevus
An IDN consists of theques situated in the dermis.

Congenital Melanocytic Nevus
By definition, small congenital melanocytic nevi are < 2 cm in size. Medium congenital melanocytic nevi are between 2-20 cm in size,

and giant congenital melanocytic nevi are AKA "bathing trunk" nevi, and, though rare, do have an increased risk of malignant transformation.

Blue Nevus

Blue nevi are non-malignant hamartomatous melanocytic growths with a deep dark blue color. Blue nevi, interestingly, are made of nevus cells with dendrites. Blue nevi can occur at any age, and malignant change is rare, but possible.

Nevus of Ota

A nevus of Ota is a non-malignant hamartoma of melanocytes and is most likely due to aberrant embryologic migration of melanocytes. On the face, a nevus of Ota appears as a blue macule with an indistinct border that fades into normal colored skin. Thus, a nevus of Ota is like a Mongolian spot. Biopsy is rarely required. A nevus of Ota can sometimes transform malignantly, so they should be monitored. Sometimes, a nevus of Ota can be disfiguring, so that laser therapy may be sought. Otherwise, treatment is usually not required.

Spitz Nevus

A Spitz nevus, AKA a spindle-cell nevus, occurs most frequently in children. Spitz nevi are non-malignant nevi with little, but some, malignant potential. The Spitz nevus is a melanocytic growth from spindle-shaped melanocytes. It is brown in color with regular borders, smooth, and shaped like a dome. Mostly single, they most frequently occur on the distal leg. Complete excisional removal and dermpath evaluation are recommended to rule out melanoma.

Dysplastic Nevus

A dysplastic nevus (DN), though a benign diagnosis, can be a precursor (precancer) to melanoma in situ and actual melanoma. Other names for a DN include Clark's nevus, atypical nevus (AN), atypical melanocytic nevus, and nevus with architectural disorder. The tendency to grow DN's is often inherited, such as in familial atypical multiple mole melanoma syndrome (FAMMM). These patients have a positive family history of melanoma and a significant increase in malignant melanoma.

There are those who say that melanomas arise from normal appearing skin. This is true, but, though the skin may "appear" as normal, it is obviously not normal. You see, melanoma can arise from just a few nevus cells that have gone bad, and those nevus cells are often undetectable to the human eye. Up to half of all clinical melanomas do develop from an atypical appearing precursor nevus, and we know, prevention is always the best medicine. Thus, it is important for the resident to identify and understand DN's and provide proper care.

In simple terms, a DN is in a gross and microscopic continuum with melanoma in situ (MIS), and, essentially, resembles a MIS histopathologically, but the DN's melanoma-like features have not yet attained those of a cancer. Thus, the DN is not considered to be a melanoma in situ (MIS). Dermatopathologists often refer to DN's as having mild atypia, moderate atypia, or severe atypia, and **beyond** these three grades of histopathologic atypism, the dermatopathologist will refer to the lesion as a melanoma in situ. After invasion into the dermis, the lesion will be referred to as an actual melanoma with a certain depth of invasion i.e. the Breslow's depth reported in millimeters.

So, a "precancerous" dysplastic nevus shares similar histopathologic and clinical features with melanoma in situ, but is not severe enough to be labeled an actual melanoma in situ or a true malignant melanoma.

A Continuum

To the naked eye, a DN is in continuum with MIS and has Melanoma–like features:

- "Fried egg-like" papular center surrounded by flattened macular areas of the periphery.
- Cobblestoned tactile surface appearance.
- Asymmetry: One half does not match the other half as to color, surface, and edges.

● Varying colors of pink, tan, and brown suggest a DN, while red, white, & blue colors suggest malignant melanoma.

● Irregular ill-defined border.

● Diameter > 5 mm

Dysplastic Nevus Work Up

Gross Visual Inspection

If an atypical appearing mole displays the above six atypical features, then, a dysplastic nevus is suspected. Of course, a true melanoma usually displays the classic ABCD's of asymmetry, border, color, and diameter. Regarding severity of appearance, a dysplastic nevus is usually a step or more **less** severe than is a true melanoma. But, the dysplastic nevus, on visual inspection, is in continuum with melanoma in situ. And thus, you have those who look for the dysplastic nevus that is the "Ugly Duckling" i.e. that one DN that looks "the ugliest" on visual examination. The ugly duckling is usually the mole that is biopsied.

Dermatoscopic Inspection of Atypical Nevi

Atypical Nevi: Structural Patterns include:

- Reticular Patterns
- Globular Patterns
- Homogeneous Patterns
- Homogeneous - Reticular Patterns
- Homogeneous - Globular Patterns
- Globular - Reticular Patterns

Atypical Nevi: Distributions of Pigmentation

- Uniformly Distributed
- Centrally Hypopigmented
- Eccentrically Hypopigmented
- Centrally hyperpigmented
- Eccentrically hyperpigmented
- Multifocal hypopigmented
- Multifocal hyperpigmented

Dermatopathologic Evaluation

Just as there is a continuum with DN to MIS on gross examination, there is also a continuum with dysplastic nevus to melanoma in situ on microscopic examination. A dysplastic nevus, AKA a *nevus with architectural disorder*, will show histopathologic changes consistent with varying degrees of architectural disorder. A few of the more atypical histopath changes may include:

●Melanocytic hyperplasia with elongation of rete ridges with basal layer proliferation of melanocytes and nevus cells.

●Increased nesting of the melanocytes distributed at the dermoepidermal junction.

●Bridging of melanocytic nests across adjacent rete ridges.

Atypical Nevi: Management

There are differing opinions on how to manage dysplastic nevi. Some dermatologic clinicians sequentially photograph DN's and monitor their development in this way. Others track them with a dermatoscope. Other clinicians biopsy only the "ugly duckling" which would be the "worst looking" of the bunch. Others do not biopsy unless the DN looks like an incipient melanoma.

We deploy a more aggressive approach. Yes, very very few of the DN's will ever go on to form melanoma. But, if you are one of the minority that does get a melanoma, you will wish to the stars that your doctor would have removed every single atypical mole that ever appeared on your body. Thus, our suggestion is to biopsy and remove any DN that looks grossly atypical. We remove each in its entirety with a deep shave biopsy or small excision. As for re-excisions, if the pathology margin is not clear, for mild or moderate atypia, we may or may not do a re-excision depending on if the base and periphery of the biopsy site was adequately electrodessicated at the time of the biopsy. For severe atypia, unless the lesion had already undergone a complete excision for margins, we always do a re-excision, even if the biopsy margins are clear. From time to time the pathologist reading the slide may suggest that a

rebiopsy be done for clear margins. In those cases, per the pathologist, we always rebiopsy.

Regarding sequential photos: photos do not halt the progression of an undiagnosed melanoma. Regarding dermatoscopy, in a legal court for malpractice, dermatoscopy will not provide unequivocal proof that the lesion is or is not a dysplastic nevus. Dermatoscopy can give you the idea that a lesion is a DN, but, only an actual histopathologic slide interpretation can give you the actual diagnosis, and this path diagnosis will hold up in court, while dermatoscopy will not.

Follow Up Care

Patients with dysplastic nevi should be reviewed every 3 to 6 to 12 months depending on severity and number of nevi. On exam, look for new or changing nevi. Also look for any recurrence of previously biopsied nevi, and any ugly ducklings. Patients should be educated as to the ABC's of melanoma detection and how to look for ugly ducklings. Patients with a family history of melanoma or those with the atypical nevus syndrome will need genetic counselling and more frequent follow up visits. Of course, sun safety education is of top importance for prevention.

Nevus Sebaceous

Nevus sebaceous, AKA Nevus sebaceous of Jadassohn, is a non-malignant hamartomatous growth that appears in infancy or childhood. The Nevus sebaceous of Jadassohn has a risk of malignant transformation into BCC after puberty, thus, basal cell carcinoma may develop within a Nevus sebaceous of Jadassohn. Most Nevus sebaceous of Jadassohn growths are solitary and usually located on the scalp. Nevus sebaceous is considered to be a hamartoma of the adnexal skin structures, and appears as a yellow cobble stoned like plaque with related overlying hair loss. Complete surgical excision is required.

Milia

Milia (plural) milium (singular), from the Latin root word for "a seed," are tiny, superficial epidermal inclusion cysts, white in color, that are non-malignant. Newborns have milia that resolve in time. In adults, milia most frequently occur around the eyelids. No therapy is required.

Pilar Cyst

A pilar cyst is a non-malignant growth of the scalp. Pilar cysts are often multiple, and develop from the keratinized **outer root sheath** of the hair follicle. Patients may present to their physician with an enlarging cyst of the scalp. A pilar cyst is not an epidermal inclusion cyst. Malignancy is a small possibility, as malignant pilar tumor of the scalp can also arise from the outer root sheath. Surgical removal is the treatment of choice.

Pyogenic Granuloma

A pyogenic granuloma can occur after a localized skin injury. PG's can also occur with pregnancy. A pyogenic granuloma is a vascular growth, AKA a lobular capillary hemangioma. The patient often presents with a beefy red tender bleeding nodule encircled with a collarette scale. PG's can become infected and sometimes arise in mucosal areas and periungually. Treatment is destruction.

Seborrheic Keratosis

A seborrheic keratosis is an epidermal tumor caused by growing keratinocytes, and, are the most frequently seen of all non-malignant skin growths. Seborrheic keratoses can develop in many shapes and sizes, and are mostly found in patients older than 40 years of age. Seborrheic keratoses have no malignant possibility, but can mimic other skin growths such as melanoma. Seborrheic keratoses require no treatment, unless irritated. Cryotherapy or electrocurettage are used to destroy seborrheic keratoses.

Porokeratosis

A porokeratosis presents as an annular plaque with a peripheral ridge, usually circular, in various shapes and sizes from small to large. They can be solitary or multiple, and primarily appear on sun exposed areas of skin. The solitary variety is most commonly a Porokeratosis of Mibelli. The multiple variety is known as DSAP which is an acronym for disseminated superficial actinic porokeratosis. Dermatopathology slides show the classic coronoid lamella in all lesions of porokeratosis. Treatment is sometimes difficult. Liquid nitrogen or 5 fluorouracil therapy are often helpful. Ammonium lactate cream can be helpful.

Dermatology Guidelines for the Primary Care Resident: The Essentials

Malignant Growths

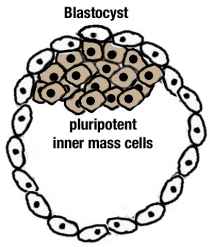
Basal Cell Carcinoma

BCC is the #1 most common malignancy in humans, and is caused by repeated ultraviolet light exposure. Ultraviolet light enters normal basal cells to damage and mutate the dna. Adenosine–Thymine dna bonds become thymine–thymine “dimers” that convert healthy basal skin cells into mutated basal skin cells.

Many clinical varieties of BCC occur, including superficial, pigmented, nodular, sclerotic, infiltrating, morpheaform, basosquamous, cystic, adenoid, keratotic, sebaceous, basosquamous, apocrine, eccrine, and fibroepithelial subtypes. The usual BCC is described as a “ppp” pink pearly papule with telangiectasias, a rolled border, and “rodent” ulceration, and are located mostly on sun-exposed areas. BCC’s can rarely metastasize. Pigmented BCC’s can mimic melanoma. At times, a BCC can form from a preexisting actinic keratosis.

What Causes BCC?

To understand basal carcinoma and its pathogenesis, you will need to understand a bit of embryogenesis, stem cells, and the hedgehog pathway. At the stage of a human blastocyst, we depart upon our developmental journey with embryonic stem cells that are undifferentiated, pluripotent, and indefinitely propagatable.



The embryonic stem cell’s devoted purpose is embryogenesis. Embryonic stem cells eventually reach their pinnacle, and we mature with a different version of stem cell, the adult stem cell, AKA the somatic stem cell. Adult stems cells are undifferentiated, self-renewable, and multipotent. Their function is to replenish expired cells and replace lost or injured tissues. Adult stem cells are able to regenerate themselves indefinitely and regenerate appropriate cells from each bodily organ in which they reside.

The Hedgehog Pathway

The hedgehog pathway was originally discovered as an important orchestrator of embryonic stem cell development. The hedgehog pathway was first found when researchers were studying body segmentation in drosophila melanogaster. When the hedgehog pathway induced gene signaling proteins was switched off, the drosophila embryo ended up developing into what looked like a hedgehog; and thus, you can understand the origin for the name “hedgehog.”

Later, researchers noted varying hedgehog protein concentrations in different sections of the fly’s body segments, so that, the various sections of the developing drosophila embryo each had different hedgehog protein levels. Also, the differing hedgehog protein concentrations enabled each individual segment to, respectively, develop each into its own unique shape and size. In other words, the hedgehog protein levels were responsible for orchestrating embryogenesis in the fly to develop its unique correct form, correct size, and correct body part locations.

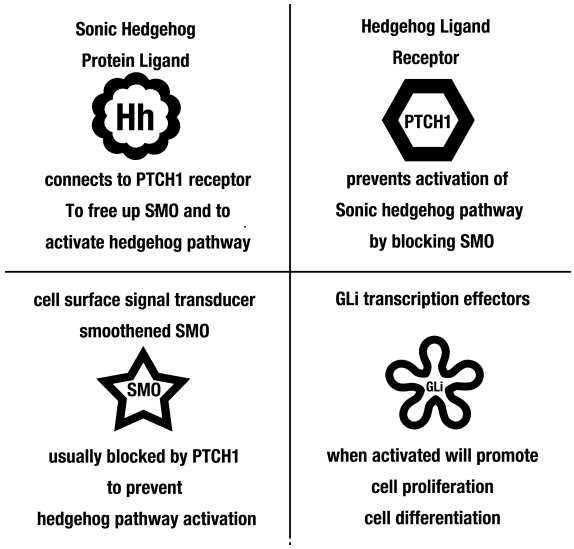
After fruit fly research, the same hedgehog pathways were also found to orchestrate embryogenesis in mammals, and then, was extrapolated to humans. For example, one study showed that hedgehog signaling enabled correct formation of the digits of the right and left upper extremities in a developing embryo.

When embryonic development had finished its course; then, the adult stem cells took center stage, and the hedgehog pathway was found to be the orchestrator of adult stem cell function, as well. The adult stem cell population was given notoriety for its functional role in the maintenance and regeneration of mature tissues.

The Hedgehog Components

In mammals, the essential components of the hedgehog pathway comprise three hedgehog ligands: sonic hedgehog, desert hedgehog, and Indian hedgehog. There is also a cell surface signal transducer called smoothened (SMO), and three GLi transcription effectors.

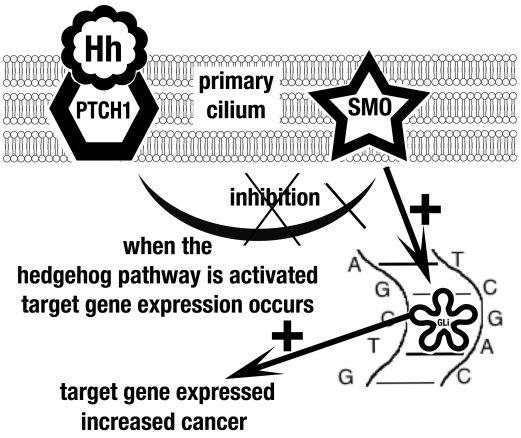
Dermatology Guidelines for the Primary Care Resident: The Essentials



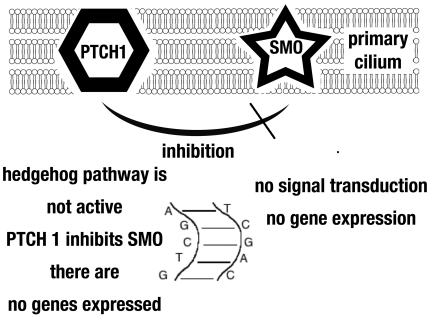
To begin, hedgehog interactions take place on a cell membrane surface called the primary cilium, a subcellular membrane that provides a specific location for the interactions to occur.

The first component of the pathway is the sonic hedgehog protein ligand. Each hedgehog gene codes for a protein ligand, and, the best understood is the “sonic” hedgehog protein ligand. Normally, the sonic hedgehog protein ligand is absent, and when absent in human tissue, the sonic hedgehog pathway is not activated. Thus, no stem cell proliferation and no cellular differentiation takes place. But, during embryogenesis and also when normal tissue regeneration is needed for whatever reason via adult stem cells, or, if there is a cancerous PTCH1 gene mutation, the sonic hedgehog protein ligand can connect with the patched 1 receptor protein. The PTCH1 gene provides codons for transcription of the patched 1 receptor protein. Receptor proteins have defined binding sites into which specific ligand proteins can fit like a key fits a lock. Sonic hedgehog is the protein ligand for the patched 1 receptor. When connected, ligands and receptors will unlock signals to control cell development and function.

Patched-1 receptor protein, connected to sonic hedgehog, as a unit, functions to orchestrate embryologic development. This unit controls cell growth, cell specialization, and organogenic shapes and patterns of the embryo via embryonic stem cells. This unit also orchestrates adult tissue renewal via adult stem cells.



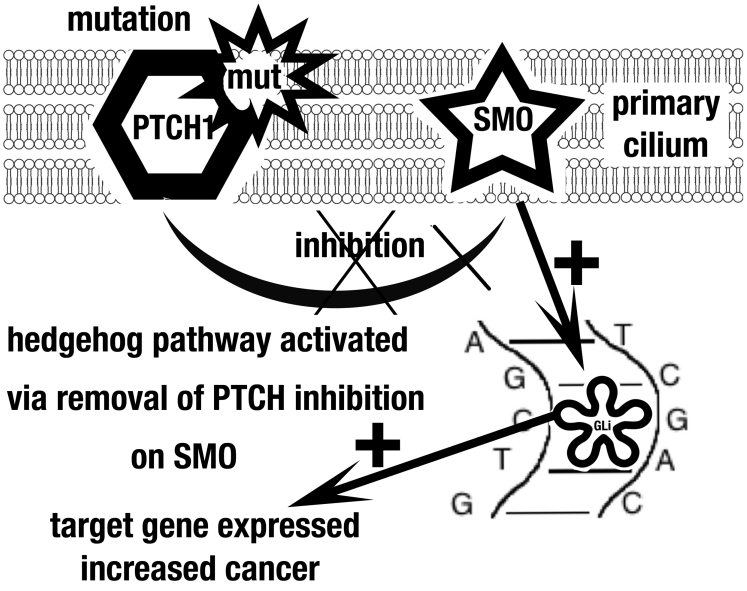
The second component is the hedgehog ligand receptor referred to as the patched 1 receptor protein and the PTCH1 gene which codes for it. Normally, without the hedgehog ligand binding, the patched 1 receptor protein spends most of its time inhibiting the smoothened (SMO) signal transducer, and in this way, the unbound patched 1 receptor protein prevents activation of the sonic hedgehog pathway. By this mechanism, a healthy patched 1 receptor works as a tumor suppressor that inhibits tumor cell progression by inhibiting SMO. In the normal resting phase, the patched 1 receptor prevents triggering of the sonic hedgehog pathway by continuously inhibiting smoothened (SMO). In the absence of the sonic hedgehog protein ligand, smoothened (SMO) is inhibited by the unbound patched 1 receptor. So, when sonic hedgehog is not present, patched 1 prevents stem cells from growing and proliferating. But, when Sonic hedgehog is connected to the patched 1 receptor protein, patched 1 stops suppressing cell proliferation via its effect on SMO. Because it prevents cells from proliferating uncontrollably, the PTCH1 gene is referred to as a tumor suppressor gene.



When the sonic hedgehog protein ligand is present, it will connect to the patched 1 receptor on the cell’s surface, and will direct the patched 1

receptor to move inside the cell, and the patched 1 receptor will dissolve inside the cell. This action frees up SMO, which is also within the cell. When patched 1 is dissolved, SMO moves from the intracellular location to the extracellular area, where it will then trigger the hedgehog signaling pathway which leads to target gene protein production via GLi transcription to promote cell proliferation, differentiation, specialization, and tissue maintenance.

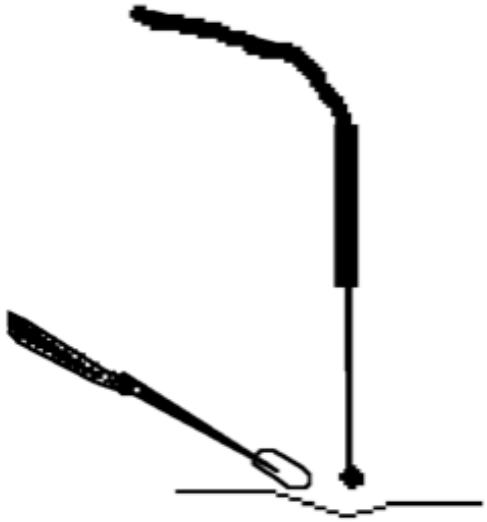
Another point, whenever the PTCH1 gene is mutated, and there is no inhibition of SMO, there will also be no tumor suppression, as the hedgehog pathway will be activated, and you see rapid tumor cell progression.



The sonic hedgehog pathway is implicated in at least 25 types of cancer. The oral medication FDA approved for the treatment of BCC is Vismodegib, a hedgehog signaling pathway inhibitor. Vismodegib AKA Erivedge works by binding to SMO, so that SMO cannot activate the hedgehog pathway.

How Is BCC Treated?

Depending on each unique case, most BCC's can be treated with curettage and electrodessication, true cautery, cryosurgery, excisional surgery, and radiation. Superficial BCC's can be treated with topical five fluorouracil or imiquimod. Mohs is an excisional technique that allows the highest cure rate with tissue sparing and smallest possible scar. In Mohs, the pathology specimens are cut like "baloney" rather than the usual "bread-loaf" cut. The "baloney" cut allows the Mohs surgeon to better visualize the entire perimeter of the specimen and better see the surgical margins. Large BCC's can be treated with oral chemotherapeutic agents, one of which is named Erivedge, another Odomzo. They focus on the hedgehog pathway. An important point for primary care residents to note: It is common for dermatologists to see BCC patients who had been inadequately electrodessicated and curretaged "EDC." These patients can have recurrence of tumor with spread to salivary glands, blood vessels, and local nerves. They can also have damaging infiltrative spread. If you do plan to treat these patients in your future clinic with EDC, to prevent possible tragic sequellae, we suggest you have thorough education and training prior to attempting EDC treatments.



Keratoacanthoma

A keratoacanthoma is rapidly growing malignant keratinocytic squamous cell tumor of the skin developed from epidermal keratinocytes. At times, a typical keratoacanthoma can auto-resolve in few weeks on its own, but often they are aggressive with invasion and metastases. Keratoacanthomas most often appear on sun-exposed areas, and are quick to enlarge and can develop into crateriform nodules with a thick keratin plug. A skin biopsy can confirm the diagnosis. Excisional removal with a clear margin is usually curative. Keratoacanthomas are known to aggressively reoccur after treatment.

Dermatology Guidelines for the Primary Care Resident: The Essentials

Squamous Cell Carcinoma

After BCC, SCC is the second most frequent skin cancer in humans. SCC can also occur in many variants, including in situ, Bowen's disease, Bowenoid papulosis, erythroplasia of Queyrat, basosquamous, and invasive SCC's of various stages of differentiation, for example, poorly differentiated, moderately differentiated, and well differentiated SCC. Clinically, SCC has the ability to metastasize, mostly to local lymph nodes. SCC occurs in chronically sun-damaged skin and can form from a preexisting actinic keratosis. SCC's can occur with mutations of the tumor suppressor geneTP53. SCC's usually appear as a patch or plaque and can show ulceration.

Definitive treatment depends on the level of SCC differentiation and the degree of tumor invasion. Therapy can include topical 5-fluorouracil chemotherapy or imiquimod for in situ lesions, surgical excision, electrodessication & curettage, Mohs micrographic surgery, radiation therapy, and chemotherapy. A very important point for primary care residents to note: It is common for dermatologists to see SCC patients who had been inadequately electrodessicated and curretaged "EDC." These patients can have recurrence and either local or distant metastatic spread. If you do plan to treat these patients in your future clinic with EDC, to prevent possible tragic sequellae, we suggest you have thorough education and training prior to attempting electrodessication and curettage treatments.

Kaposi's Sarcoma

Kaposi's sarcoma is an endothelial cell malignancy most commonly seen in older men and HIV immunocompromised patients and is associated with HHV8. Kaposi's sarcoma is a locally invasive cancer that is fatal if not treated. Kaposi's sarcoma usually appears as red to purple macules, papules, plaques, and nodules anywhere on the body. Internal organ invasion has been seen. The most common treatment is localized radiation therapy, but chemotherapy is sometimes used for widespread disease.

Paget's Disease

Paget's disease is a rare malignancy that occurs in areas of apocrine glands. Paget's can appear as mammary or extramammary**.** Paget's disease is an intraepidermal adenocarcinoma, that can also herald other underlying malignancies such as underlying breast cancer. Paget's disease is slow-growing and usually presents as an itchy, burning, shiny red-pink patch. Biopsy is required. Treatment of Paget's disease depends on the stage of the tumor.

Merkel Cell Carcinoma

Merkel cell carcinoma is an aggressive neuroendocrine malignancy. Merkel cell carcinoma develops from Merkel cell mechanoreceptor nerve endings within the skin. The lesions usually occur on the sun exposed head and neck, and sun exposure is a causative factor. Merkel cell carcinoma appears as red to purple papules or plaques that rapidly grow in size and often ulcerate. The prognosis is poor due to high post op recurrence and rapid metastasis. If you see a patient with an itchy areolar rash, don't assume that it's eczema. Treatment is excisional removal with wide margins, sometimes followed by radiation and chemotherapy.

Sebaceous Carcinoma

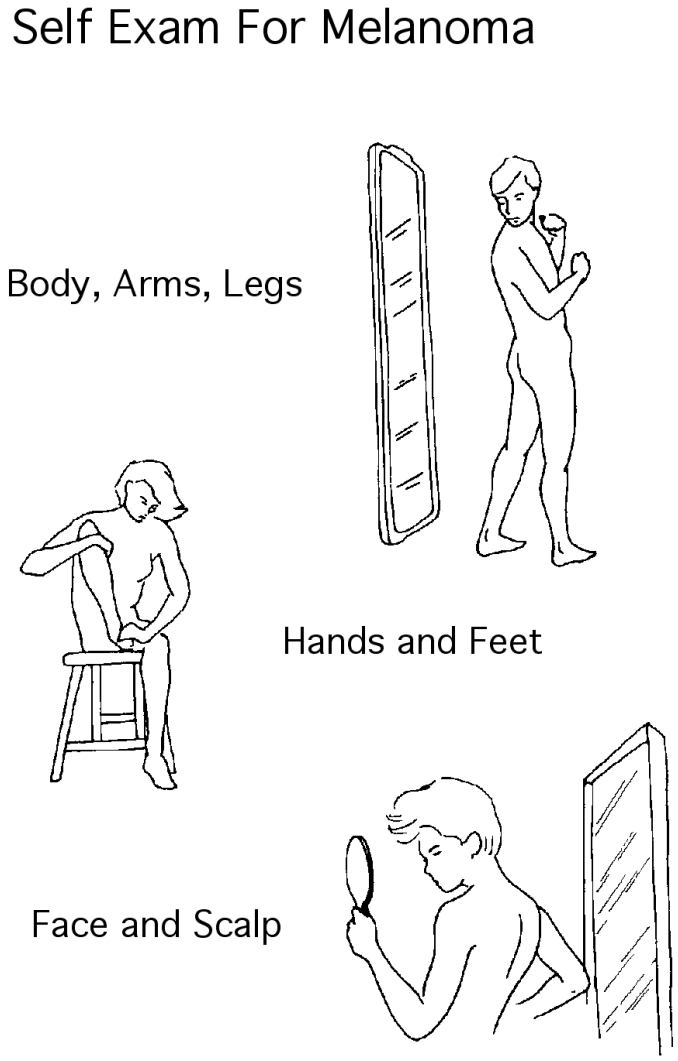
Sebaceous carcinoma is a highly malignant, aggressive sebaceous gland cancer. Sebaceous carcinoma is most frequently seen around the eyelids in older females. Sebaceous carcinoma often mimics BCC around the eyes, and can be seen in Muir-Torre Syndrome. Histopathology sebaceous carcinoma shows a high degree of infiltrative growth, with deep invasion into subcutaneous tissues. Treatment is surgical with sometimes adjuvant radiation and chemotherapy.

Melanoma

Melanoma is a fatal skin cancer of ultraviolet light exposed melanocytes, and often metastasizes via lymphatics and blood. Melanoma in situ is a lesion that has not yet crossed the basement membrane zone. The classic patient is a red or blond hair, blue eyed, freckled person who had tanned or burned in the sun or tanning parlor. Malignant melanoma is rare in children and teens. The leg is the most frequent location in women and the back is the most frequent location

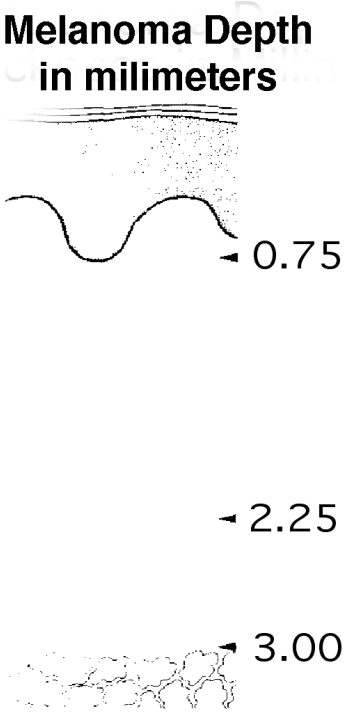
in men. Most malignant melanomas develop in previously normal skin. About 30% of mm's develop in a dysplastic melanocytic nevus. A small percentage develop in congenital nevi.

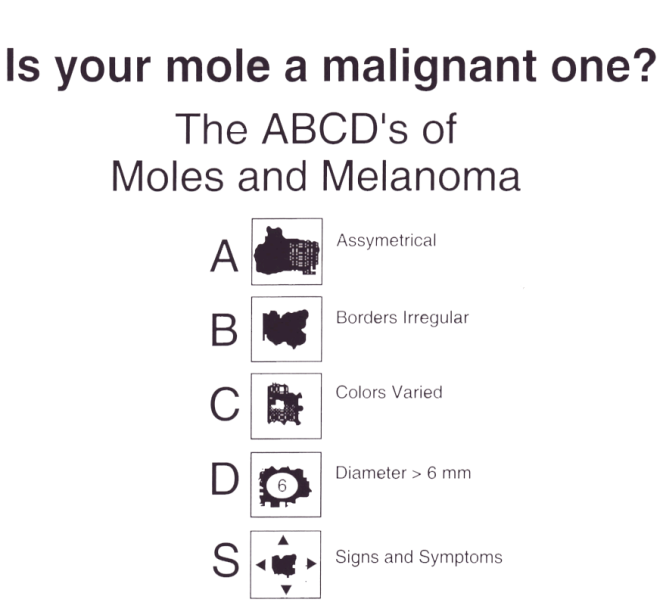
It is important to teach your patient how to examine their own body for atypical nevi. Please read our Guidelines section on atypical nevi.



The treatment and prognosis of melanoma depends mostly on the Breslow's thickness and the presence or absence of ulceration. Thus, as a primary care physician, it is important to note ulceration on the chart, and, when biopsying to rule out melanoma, it is important to obtain a deep biopsy specimen down to the fat.

Melanoma treatment is beyond the scope of the Derm Guidelines. Primary care physicians, dermatologists, ENT's, general surgeons, radiation oncologists, and medical oncologists usually work together as a team for the patient in the treatment of melanoma.





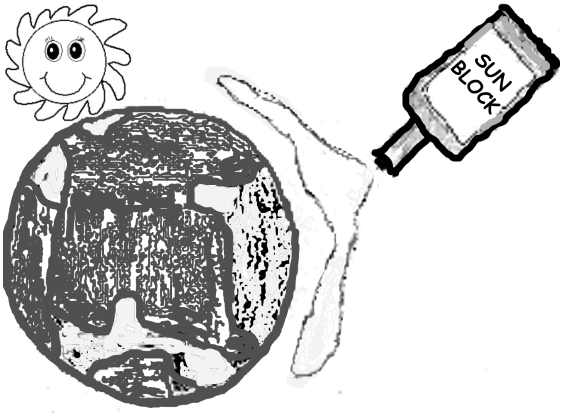
Sarcomas

Just for your awareness, there are sarcomatous skin cancers, for example, malignant fibrous histiocytoma, dermatofibrosarcoma protuberans, pleomorphic sarcoma, atypical fibroxanthoma, and leiomyosarcoma.

Our list of skin cancers is not 100% complete, as a comprehensive skin cancer review is beyond the scope of this Guidelines book. We mention these rare skin cancers to make you aware that there are, in fact, many skin cancers other than the usual basal cells, squamous cells, and melanomas.

Notes

Dermatology Guidelines for the Primary Care Resident: The Essentials



PROTECTION FROM THE DAMAGING SUN

Too much sunlight can be dangerous. Being outside on a warm, sunny day is one of life's great pleasures, but getting too much sun can be dangerous. Excessive sun exposure can result in painful sunburn, but can also lead to other serious health problems, including melanoma, a life-threatening form of skin cancer. Melanoma is one of the fastest-growing forms of cancer in the U.S. New melanoma cases in the U.S. have more than doubled over the past two decades. In addition to melanoma, excessive UV exposure can lead to premature aging of the skin, cataracts, non-melanoma skin cancers, and immune system suppression. Special concerns for children: Because many of the sun's worst effects do not appear until later in life, hindsight show us that it is very important to protect children and teenagers from overexposure to UV radiation. The majority of most people's sun exposure occurs before age 20, and studies increasingly suggest a link between early exposure and skin cancer as an adult. The following precautions can help ensure that adults and children avoid UV-related health problems, both now and later in life. Started early, each of these steps will become an easy habit.

❶. **Avoid the Midday Sun.** The sun's UV rays are strongest between 10 a.m. and 4 p.m. Avoid these hours. Plan outdoor activities for early morning or late afternoon.

❷. **Wear Long Sleeves and a Brimmed Hat.** A hat with a wide brim offers good sun protection to your eyes, ears, face, and the back of the neck. Wear long-sleeved shirts and long pants when out in the sun. Choose tightly-woven materials for greater protection from the sun's rays.

❸. **Apply a Zinc Oxide Based Sunscreen** before every exposure to the sun, and reapply frequently and liberally, at least every two hours, as long as you stay in the sun. **Use a sunscreen** during high altitude activities such as mountain climbing and skiing. At high altitudes, where there is less atmosphere to absorb the sun's rays. **Don't forget to use sunscreen** on overcast days. The sun's rays can be as damaging to your skin on cloudy days as they are on sunny days. **Individuals at high risk for skin cancer** such as outdoor workers, and fair-skinned people should apply sunscreen daily.

❹. **Wear Sunglasses** that Block UV Radiation. Sunglasses that block UVB will greatly reduce sun exposure that can lead to cataracts and eye damage.

❺. **Avoid Tanning Salons.** UV light emitted by tanning booths causes sunburn and premature aging, and increases your risk of developing skin cancer.

❻. **If you develop and allergic reaction** to your sunscreen, change sunscreens. Try zinc based.

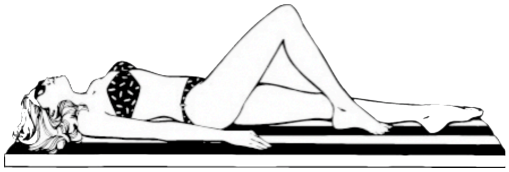
❼. **Beware of Reflective Surfaces!** Sand, snow, concrete, and water can reflect more than half the sun's rays.

❽. **Photosensitivity** increased sensitivity to sun exposure is a possible side effect of certain medications, drugs, cosmetics, and birth control pills. You may need to take extra precautions.

❾. **Protect Children! Keep wee ones out of the sun.** Begin using sunscreens on children at six months of age, and then allow sun exposure only with caution. **Teach your children sun protection early.** Sun damage occurs with each sunburn and accumulates over the course of a lifetime. Your baby will one day be an adult.

❿. **Watch for the UV Index.** The UV Index, by the National Weather Service provides a forecast of the expected risk of sun exposure.

Dermatology Guidelines for the Primary Care Resident: The Essentials



Actinic Keratoses (Precancers)

The risk of actinic keratoses progressing to squamous cell carcinoma or basal cell carcinoma has been well documented. If neglected, precancerous spots have about a 20% or greater chance of changing into skin cancer. Actinic keratoses are precancerous, and to prevent skin cancer from developing, we believe they should be treated. There are many different ways to treat actinic keratoses. Cryo spray is the most common way, however, there are other effective methods for the treatment of actinic keratoses. Therapeutic modalities include electrodessication, true cautery, trichloroacetic acid (TCA), blue light, CO2 laser, Solaraze, Picato, and 5-FU.

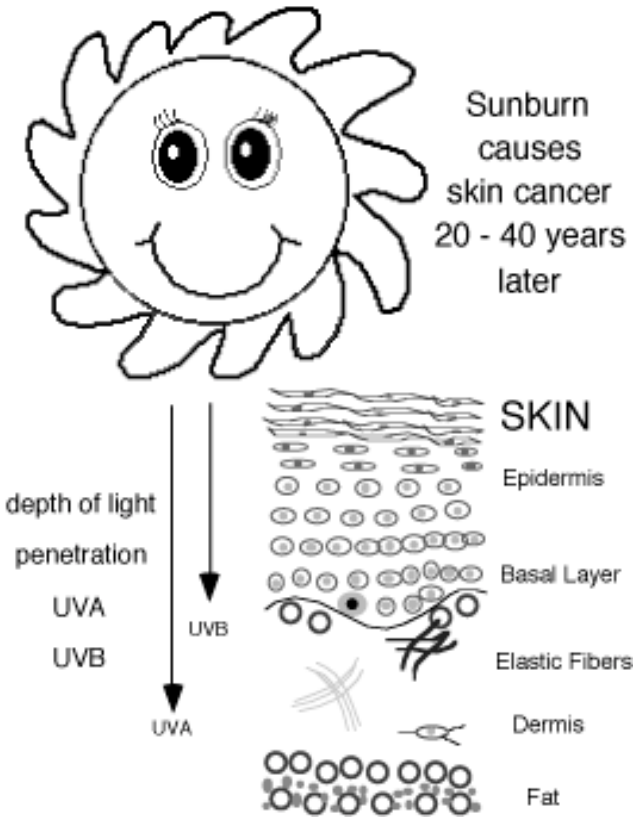
New primary care residents often ask, "How do dermatologists treat actinic keratosis?" We will explain. First, we examine the patient to make sure the lesions are true AK's and nothing else; then, dermatologists treat each patient according to each's unique individual findings.

Because many patients have innumerable lesions, most dermatologists prefer to treat actinic keratoses patients little by little. With baby steps, the patient is not so heavily engulfed with pain and discomfort. Certainly, each patient will tolerate a different level of pain, but, if we try to treat all AK's at once, the patient usually becomes frustrated and overwhelmed. Thus, our AK treatment philosophy is "By the yard, it's hard, but, inch by inch it's a cinch." Remember, actinic keratoses are like weeds. They come and go and come and go and will always grow even after you treat them. Thus, in treatment, we focus on keeping AK's under control little by little. In other words, it is impractical to think that one can get rid of all actinic keratoses with one treatment all at once during one office visit. Because actinic keratoses can cover a great many square inches of a sun exposed body, actinic keratosis therapy is best undertaken in an ongoing periodic basis, regularly, and little by little. And, you can certainly use more than one modality. Each patient is different as to what they want, what they need, and what they can tolerate. You may see the need for liquid nitrogen every three months, but the patient may refuse. Thus, you can offer one of the other modalities. Certain hypertrophic AK's may require a biopsy and electrodessication. Other AK's may be isolated to specific areas such as the nose and ears, and you may want to try Picato. Other AK's may be diffusely spread on the forearms and you may see the need for 5 FU or Solaraze. The patient may have a bald head loaded with AK's and you might use TCA. It is good to learn how to use the different modalities. With regular AK follow up visits you can detect and treat developing lesions as they grow close to cancer. We find that regular periodic visits and the little by little method is a great way to prevent the skin cancers from growing out of control.

For the primary care resident just starting out and treating AK's in his or her clinic, we would like to suggest Picato and 5FU. Excluding liquid nitrogen, they are really the easiest modalities. For one thing, practically speaking, liquid nitrogen is difficult to keep. If you don't use a lot of it, it can be expensive and can easily go to waste. For the primary care resident, true cautery pens can be an option to liquid nitrogen.

First, let's review Picato. Picato is a plant derived medication FDA approved for the treatment of AK's. Many patients prefer plant based meds. Picato is topical ingenol mebutate gel for the treatment of actinic keratoses. Ingenol mebutate → ingenol-3-angelate, a diterpene ester extracted and purified from the milk weed plant *Euphorbia peplum,* which produces a milky sap. Picato causes death of precancerous cells, but, the exact mechanism of Picato is not known. Picato comes as 0.015% liquid and is applied once daily for 3 consecutive days for AK's of the face and scalp. Picato also comes as 0.05% liquid and is applied once daily for 2 consecutive days for AK's of the body, arms, and legs. Picato is easy for your patient to

use. It only requires 2 or 3 days, and patients like that. Picato is also highly effective and does the job. Compared to 5FU, Picato is the easier choice for patients and the doctor. A few points to understand about Picato. First, about 15% of patients cannot tolerate a third application to the face. That's OK. Patients should be fine with just two applications. Second, explain to your patient to avoid areas near the eyes. Also, wash hands after applying Picato, and do not touch eyes and do not apply Picato near the eyes. Third, Picato is designed for the treatment of small areas only. Explain to your patient that Picato is a little by little way of treating their AKs. One single treatment with Picato is meant to only be for a 2 inch by 2inch- area of skin. Thus, Picato therapy is slow, but tolerable and highly effective.



Now, let's talk about 5-fluorouracil. Generic 5-FU does an incredible job, and is well tolerated if the patient uses 5-FU by the little by little method. In other words, just do small areas at a time. Every two weeks, do a small area of 3 x 3 square inches, and then, every ten days, change to another new 3 x 3 square inches small area of treatment. Now, regarding 5FU, you may have heard people say, "I'll never use that stuff again!" But, that is mostly because the person did their whole face all at once. That is too much!!! It's like drinking a whole bottle of hot sauce in one gulp. NO! Note: We suggest only small drops of hot sauce to flavor your food, and similarly, only 3" x 3" small amounts and small areas of 5-FU to treat precancers.

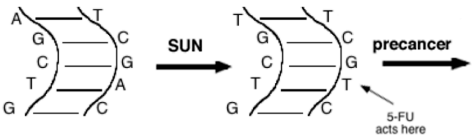
Common skin precancers include:
1. Actinic (solar) keratoses
2. Actinic Cheilitis.

Actinic keratoses: (AK's) Actinic keratoses are rough, slightly raised scaly growths which may become as large as one inch or more in diameter on sun exposed areas of skin. Actinic keratoses range in color from white to red to brown and are most frequently found in older people. Actinic keratoses may appear like tiny red patches in their early stages, and later become scaly. Actinic keratoses may bleed as they evolve into skin cancers. Instead of viewing actinic keratoses as benign precancers, certain dermatopathologists consider actinic keratoses to be embryonic skin cancers, or skin cancers in their earliest beginnings.

Actinic Cheilitis: Actinic Cheilitis is a chronic precancerous condition of the lips and is most frequently found on the lower lip due to greater exposure to the sun. The lesion of actinic cheilitis is essentially an actinic keratosis of the lower lip. Actinic cheilitis typically presents as a localized dry cracked area, scaly and pale pink or white in color. Actinic cheilitis is often ignored. The patient usually looks at the lip and can't believe that their dry lip condition is precancerous. "It's been this way for years," they say. This neglect may lead to squamous cell carcinoma of the lower lip, which has a very high chance of becoming metastatic and spreading to the lymph nodes. For this reason, you should be aggressive in treating actinic cheilitis. When it comes to skin cancer, It is better to be safe than to be sorry.

What Causes Precancer? Repeated prolonged sun exposure leads to epidermal and dermal sun damage of skin. Fair skin persons will be at greatest risk. Occupations requiring prolonged exposure to the sun, individuals spending extensive leisure time in the sun, and anyone with a history of sunburn are at risk. Just like x-rays can penetrate to your bones, invisible UVA & UVB rays of the sun can penetrate under the

surface of your skin. When a person is exposed to the sun, ultraviolet rays cause damage to the DNA of skin cells. The damaged DNA forms thymine dimers. The abnormal DNA thymine dimers remain in the skin and may cause skin cancer to arise ten to forty years after a bad sunburn. Thus, precancer and skin cancer is a time-delayed reaction. Explain to patients: Skin cancer may not show up until years later.



What is 5-FU? 5-FU is not an acid and does not work by "burning out" the bad skin. 5-FU is a pharmacologically active systemic and topical chemotherapeutic agent, which seeks out and selectively destroys abnormal skin cells containing thymine dimers, and leaves the good skin cells alone. To be specific, 5-FU specifically locates thymine dimers and selectively destroys skin cells that contain thymine dimers in their DNA. How was 5-FU discovered? 5-FU is a chemotherapeutic agent originally and still used to treat solid tumors of the kidney and liver. Many years ago, 5-FU was given intravenously to cancer patients. Interestingly, when given to cancer patients with fair skin and a history of sun exposure, doctors noted that the patient's precancers turned red and disappeared. With this observation, 5-FU was then made into a topical cream, and was found to destroy precancers when applied to the skin.

5-FU destroys precancers that are visible on the surface of the skin. 5-FU also destroys newly forming precancers that are deep down and not seen on the skin's surface. As 5-FU destroys precancerous cells, there is usually considerable irritation, often within 3-4 days. Explain to your patient: Do not be alarmed about this. This is what we expect. After the irritation disappears (2-4 weeks), good skin will appear. You will notice a new smoothness to your skin. Note: Many people will refuse to use 5-FU because they do not want to appear unsightly. Many of these people have baked in the sun, and some have not, but all have severely sun-damaged skin. This sun damage took years and years to develop. Educate them: Think about it: Even if it takes a month to treat, considering the years it took to develop, isn't it worth this short amount of time to prevent skin cancer? A few weeks of unsightliness is a small price to pay to reverse many years of sun damage. If your patient is one who refuses 5-FU for simple reasons of appearance, please encourage he or she to give serious thought to skin cancer and the benefits of skin cancer prevention. Surgery in later years can be far more disfiguring than 5-FU today.

General suggestions for use of 5-FU…

Twice A Day Method: Educate your patient on how to do the Twice A Day Method. With this method, explain to your patient: You have been given a prescription for two medications. The first medication used is either 5-FU cream or 5-FU solution. The second medication used is a topical steroid cream or ointment. Please understand that 5-fluorouracil and the topical steroid are two different medications. 5-FU is used first, and will cause a "red reaction." After you have developed an adequate red reaction, you will use the topical steroid cream or ointment to cool the red reaction to bring your skin back to normal. Some refer to the topical steroid cream or ointment as the "neutralizing cream" and to 5-FU as the "burning cream." Others use the terms, “bad cream and good cream.” Educate: Please do not use 5-FU and topical steroid cream or ointment at the same time simultaneously, as they will cancel each other out. First, use the (5-FU cream) "burning cream" alone for one or more weeks to develop a red reaction, then completely stop the 5-FU. Second, use the "neutralizing cream" topical steroid cream or ointment for two weeks to bring soothing relief. It is the cool down cream.

Twice A Day Fine Points: Educate your patient: Your goal will be to use 5-FU to develop a bright red skin color at selected precancerous sites. This color change is called the "red reaction." To develop the red reaction, most patients with precancers of the face will need to apply 5-FU twice a day for 2 to 3 weeks. You should indicate the exact site and duration on the prescription you write. Lip areas may not tolerate more than 1 or 2 weeks of 5-FU. The scalp or arms may tolerate 4 or more weeks of therapy. The 5-FU should be rubbed in the

entire involved area (for example, on the face, or back of the hands, or lips, or arms, or scalp, or as instructed) twice a day. This can be done with the fingers, which should be washed off with mild soap after completing the application. If you are treating difficult lesions, or lesions of the scalp, trunk, or arms, you may need to apply 5-FU twice a day for up to 4 weeks before an adequate red reaction develops. If you are treating the lips, you may only need to apply 5-FU twice a day for one week before an adequate reaction develops. Remember the 3 x 3 rule. If your patient wants to treat a larger area than 3 x 3, it is ok with the understanding that he or she should advance the size of the treated area slowly little by little as to not create an overwhelming 5-FU area of irritation. What should the red reaction look like? The rule is: The thicker the skin, the longer it takes to develop a red reaction. Everyone asks, "How red should I get before stopping the 5-FU?" The answer is: Use 5-FU twice a day until all the precancers turn a bright pinkish red. The treated skin may look like wind burned or chapped lips. Some areas of treated skin may look like they have been sunburned. There may be crusting with more severe lesions. Explain, you may experience mild to moderate pain and discomfort. This redness should remain for three days. You may use Tylenol for pain. When the pain and discomfort is so great that you can't continue with the 5-FU, then, you've probably had enough. At that point, you may stop the 5-FU and begin to use the topical steroid cream or ointment. Regarding scales: It is OK to remove scales from the skin spots before you apply the medicine. You may rub the scales off with a cotton tip applicator.

5% 5 Fluorouracil Cream
small areas only
2 x per day x 2 weeks
then D/C and cool with
TAC .1 or HC 2.5 cream

Where to treat: Educate your patient: Try to keep the 5-FU away from the eyes, inside of mouth, and skin folds where your nose joins your cheeks. Explain: You may need to treat your lower lip if you have actinic cheilitis. When treating the lower lip, remember that the skin of the lip is thin, and there is less time needed to attain a red reaction. Sun Exposure: Be careful to avoid sun exposure while on 5-FU. Educate your patient that precancers are due to sunshine that they have accumulated over their lifetime. Sunshine may irritate the treated skin during therapy. A large hat is better protection than sunscreen. Explain, if you don't have a hat and must go out in the sun, please use sunscreen with your 5-FU. Shelf Life: 5-FU loses potency, but may still work for years after the expiration date. Small Areas Only: If your patient has any fears, unanswered questions, or apprehensions about 5-FU, we suggest for the patient to try 5FU on a small area of 1 x 1 square inch or less for a couple of weeks to get an idea of what to expect. Note: Most patients should do only small areas of 3 x 3 at a time, anyway, and change and do a new area every two weeks. This is the “baby steps” method, and it is much more tolerable than doing large areas. Remember, by the yard, it’s hard, but, inch by inch it’s a cinch.

Please note that 5-FU will not harm normal skin (skin that has not been sun damaged) Normal skin should not turn red with 5-FU. Make-Up: Many women ask if they can cover the irritation with make-up. Make-up is fine, but must be applied on top of the 5-FU. Cooling: Note, explain: Do not use the topical steroid cream or ointment until an adequate red reaction occurs. In other words, when the red reaction occurs and has destroyed the actinic keratoses, then, the patient may stop the 5-FU, and cool the reaction with the topical steroid cream or ointment. How to get quick relief for the red reaction: Apply the topical steroid neutralizing cream as thick and as often as possible. Explain: It is OK to use the topical steroid cream or ointment as thick and as often as desired for two weeks as you cool the red reaction. Important: Do not use the topical steroid cream or ointment for longer than two weeks on the face, as topical steroid cream or ointment may cause rosacea (adult acne) with long term use on the face. Scarring: As a rule, 5-FU does not leave permanent scars or discoloration on the skin, but the face may have a mottled appearance for several months following the treatment. Explain to the patient

that this discoloration represents the battlefield where you destroyed the precancerous lesions. Discoloration can be prevented by careful sunscreen use during the months following 5-FU therapy. As the prescribing physician, you may also prescribe a bleaching cream such as hydroquinone. Resistant Areas: The hands, arms, and scalp respond more slowly than the face. Your patient can expect the development of mild burns, tenderness, pain, scaling, and sometimes oozing in the involved areas. These areas respond more slowly than other areas. The red reaction is the goal of treatment but is not essential for therapy. Some patients do not develop a red reaction with 5-FU. Repeat: In severe sun-damaged cases, the 5-FU course may be repeated every four months or more often for skin cancer prevention. Note: In most cases, when the patient uses 5-FU for 4 weeks and no red reaction occurs, the patient is clear.

Quotations on 5-fluorouracil therapy

"5-FU has the distinct advantage of unmasking clinically hidden microscopic lesions." Detlef K. Goette, M.D.

“There just isn't any modality as effective for sun damage as 5-FU. When lesions are numerous, 10 or more, there is no physical technique, including freezing as practical for treating so many lesions. There just isn't any modality as effective for sun damage as 5-FU. In well over 3,000 patients treated with 5-FU, I have never had any significant residual scarring, even with ulcerations. With other available modalities, there is a greater likelihood of residual atrophic scars that can't be covered by make-up, and often require further treatment. Occasionally, there may be a bit of hyperpigmentation following 5-FU treatment. But that is virtually eliminated if the patient uses a sunscreen. The bottom line is that the more severe the skin damage, the more severe will be the patient's reaction, and the more benefit the patient will derive. Quite a bit has been made about contact allergy to 5-FU. Allergy is a real problem, but its incidence is very small. I recently tabulated that in 3,000 treated patients I had only 3 bonafide allergic reactions.” Wayne Simmonds, MD, PhD.

"I think 5-FU is a grossly under-used drug with fantastic effects." Albert Kligman, M.D.

Picato Information

Even though most dermatologists use 5-FU for their treatment of AK’s, for the primary care physician treating actinic keratosis, Picato may be the better choice. Why is this? The answer is simplicity. Picato is so much easier for the patient to use. 5-FU has a much more difficult learning curve. Picato is quicker, and there is not as much “hand holding” with Picato as there is with 5-FU. Picato comes in small tubes.

For patients interested in “natural” medications, Picato is simply the extract of milk weed sap.

Here is a bit of company literature on Picato.

Is your mole a malignant one?

The ABCD's of Moles and Melanoma

A  Assymetrical

B  Borders Irregular

C  Colors Varied

D 

Diameter > 6 mm

S  Signs and Symptoms

Signs and Symptoms

❶ Positive family history of melanoma
❷ Family members with many moles
❸ History of a sunburned mole
❹ A mole that is:
- ✳ Growing
- ✳ Changing in color
- ✳ Bleeding
- ✳ Itching

Two ABCD's positive out of five may be a sign that your mole needs a biopsy.

How to Biopsy a "Rule-out Dysplastic Nevus."

Before you biopsy, ask if the patient is on blood thinners or if the patient has any artificial body parts that would require appropriate pre-biopsy planning.

Important Points

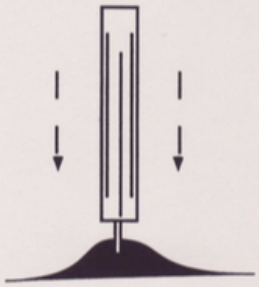
When you biopsy a pigmented lesion, or any skin lesion, please remember two important points about a punch biopsy or a deep shave biopsy:

1. **Depth:** The biopsy sample must go down as deep as the fat. This will allow the pathologist to determine a Breslow's thickness if the lesion is a melanoma. Please note that a small punch in the center of a lesion **will not** cause a skin cancer to metastasize.

2. **Width:** The biopsy sample must include **the most** suspicious part of the lesion. It is best to remove the entire lesion, but, to prevent an unneeded scar, it is often reasonable to take a small punch biopsy specimen of the lesion. If you are sampling a small portion of an atypical mole, please be sure to sample *the most* atypical part of the mole. For larger lesions, you may need to take two or more punch or deep shave biopsy samples from different areas for complete histopathology.

Method

A. Prep with alcohol and inject one cc of xylocaine with a #27 needle. Inject below and around the skin lesion. Wait a moment for adequate anesthesia.



B. Gently enter the lesion with a 2 or 3 mm punch or deep shave with a #15 blade. Use forceps to remove a small sample. Put the sample in a formalin bottle from the lab.

C. With a cotton tip, apply a drop of Monsel's Solution or Drysol (order from pharmacy) to stop any bleeding. Some bleeding may need hot cautery. Deeper biopsies may need a small stitch.

D. Patient Education: The wound may be cared for with hydrogen peroxide, Polysporin, and a BandAid bid. Explain that direct, firm pressure is first aid for a bleeding wound.

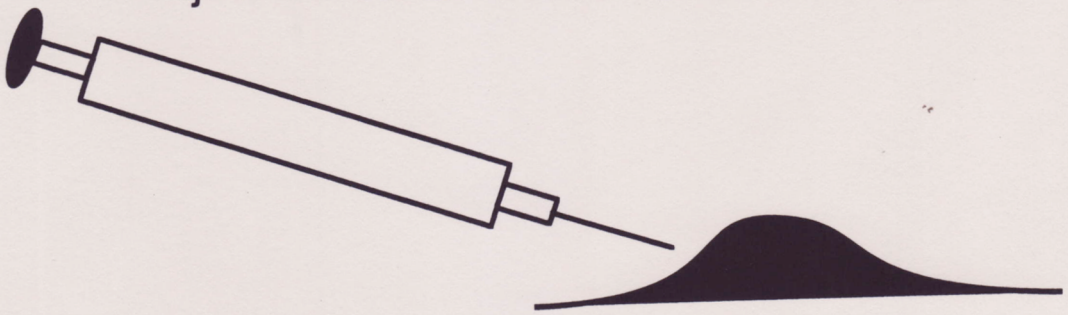
Dermatology Guidelines for the Primary Care Resident: The Essentials

Shave Biopsy Technique

You may not know the exact diagnosis, but you can do a simple biopsy to find out.

Before you biopsy, ask if the patient is on blood thinners or if the patient has any artificial body parts that would require appropriate pre-biopsy planning.

A. Prep with alcohol and inject one cc of xylocaine with a #27 needle. Inject below and around the skin lesion.



B. Gently make a **horizontal** (2 mm or more deep) shave with a #15 blade. Use forceps to remove a small sample. Put the sample in a formalin bottle for the lab. Ask the lab to supply the formalin bottles.

C. With a cotton tip, apply a drop of Monsel's Solution or Drysol (order from pharmacy) to stop any bleeding.

D. The wound may be cared for with hydrogen peroxide, Polysporin, and a BandAid bid.

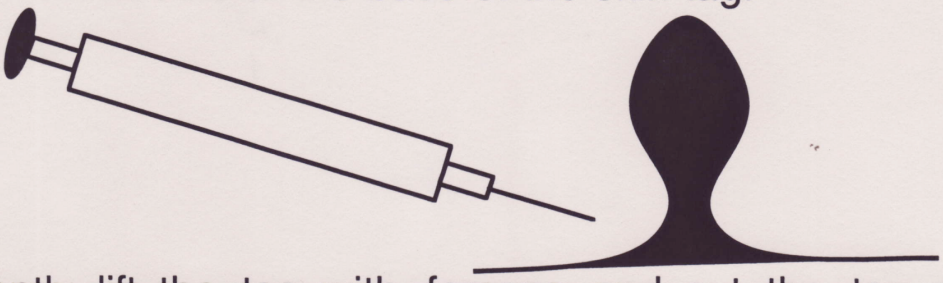
Please Note: If Monsel's or Drysol or pressure do not give adequate hemostasis, you may need to use true cautery.

Skin Tag Removal

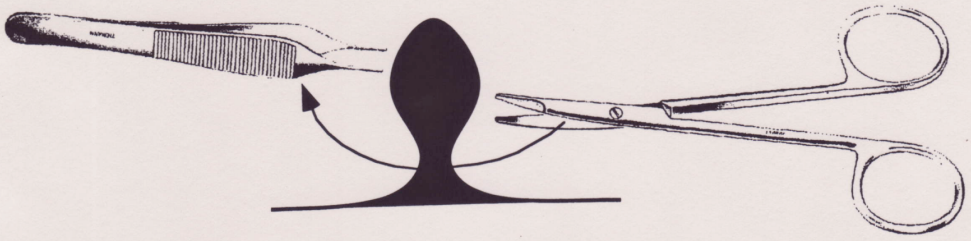
Before tag removal, ask if the patient is on blood thinners or if the patient has any artificial body parts that would require appropriate planning.

Skin tags can be removed with scissors, or a 15 blade. First, sterilize the area with alcohol or Hibiclens and anesthetize the base of the tag with a small amount of xylocaine. Second, lift the tag with forceps and cut the tag at the bottom of the stalk. Third, apply a drop of Monsel's Solution or Drysol (order from pharmacy) to stop any bleeding.
ed sheet.

A. Prep with alcohol and inject a small amount of xylocaine with a #27 needle at the base of the skin tag.



B. Gently lift the tag with forceps and cut the tag at the bottom of the stalk. Use forceps to remove the tag.



C. With a cotton tip, apply a drop of Monsel's Solution or Drysol (order from pharmacy) to stop any bleeding.

D. The wound may be cared for with hydrogen peroxide, Polysporin, and a BandAid bid.

Please Note: If Monsel's or Drysol or pressure do not give adequate hemostasis, you may need to use true cautery.

Derm Medical Supplies for the Primary Care Clinic

What supplies should you stock for your primary care derm clinic? Most primary care offices have basic instruments for suturing. Most have xylocaine and syringes. Most have Bandaids, Neosporin, and gauze. Most do not have liquid nitrogen. Here is a list of supplies you will need to stock for your primary care derm patients. You can buy these from Moore Medical Supply 1-800-234-1464. This company sells good quality yet inexpensive supplies for use with your primary care derm patients. Also, there are three items on this list that you will need to get from the clinical lab (Labcorp, Quest, etc.)

2 mm Disposable Skin punch Moore Medical item # 52437
(Use to biopsy anything on the skin from nevi to rashes.)

Disposable Curette Moore Medical item # 60134
(Use to curette skin cancers, warts, and molluscum.)

High Temp Cordless Surgical Cautery Moore Medical item # 41112
(Use to burn warts, molluscum, and to stop bleeding.)

Drysol Solution Moore Medical item # 77458
(Use to control bleeding.)

Wart Stick Moore Medical item # 73360
(Use to treat warts. You can sell this to the patients.)

LMX 5% Cream Moore Medical item # 77432
(Use for topical anesthesia. You can sell this to the patients.)

#27 gauge needle Moore Medical item # 53814
(Use inject xylocaine.)

#30 gauge needle Moore Medical item # 11157
(Use inject xylocaine.)

Verruca-Freeze Moore Medical item # 80792
(Use to treat warts, molluscum, and certain AK's)

1% Xylocaine Moore Medical item # 69162
(Use to inject local anesthesia.)

Adson Forceps Moore Medical item # 78027
(Use to pick up skin culture specimens, skin biopsy specimens or skin tags.)

Iris Scissors Moore Medical item # 37839
(Use to cut any skin lesion or to obtain a nail fungal culture.)

Throat Culture Tubes: order from the Clinical Lab
(Use to obtain a skin bacterial culture to send to the lab.)

Sterile Urine Jars: order from the Clinical Lab
(Use to obtain a skin fungal culture to send to the lab.)

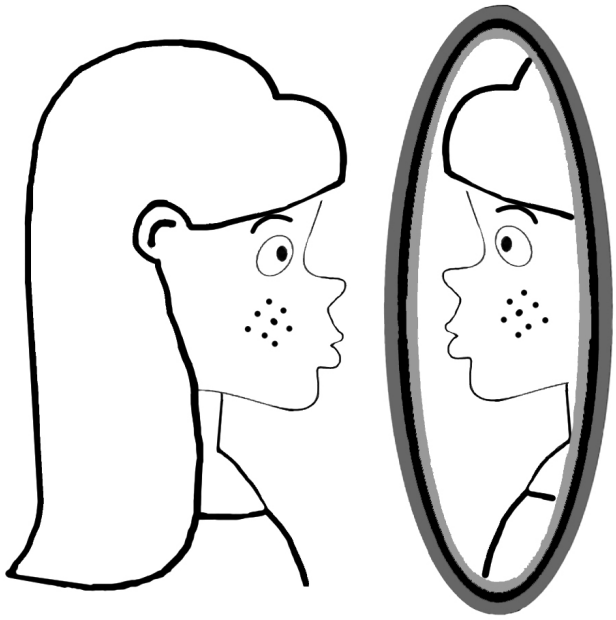
Formalin Bottles: order from the Clinical Lab
(Use for skin biopsy samples that are sent to the lab.)

Sebaceous Gland Conditions
Section 3

Dermatology Guidelines for the Primary Care Resident: The Essentials

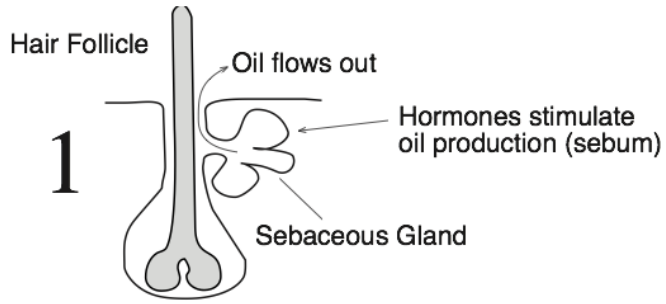
Acne Care Guidelines

Oh no, zits! 85% of teens get acne, and for a teenager, basic acne is one of the most common reasons for visiting a doctor. A majority of teens will seek help from their primary care doctor first. So, as a primary care resident, you need to be ready. High school students were interviewed and asked what was most important to their appearance. A thin body, muscles, beautiful hair, etc., or clear skin. Clear skin was voted most important. A recent TV documentary on the biology of human sexuality indicated that clear skin is one of the first features of human beauty and sexual attraction. Thus, clear skin is important to self-image, social bonding, and self-worth. With this in mind, our goal is to help you to show your patients how to have the clearest skin possible.



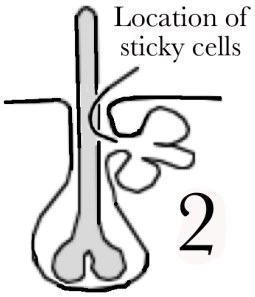
What is acne? We know common acne as blackheads, pimples, and zits. Acne is medically defined as an inflammatory disease of the hair follicle and its sebaceous glands. The word "acne" is derived from the Greek word "acme," meaning, "point." And acne appears as many "points," on the skin ranging from comedones ("blackheads" or "whiteheads") to solid bumps, pustules, pimples ("zits"), and even large cysts. Acne mainly affects the face, but may also appear on the shoulders, back, and chest. As you know, acne is a common skin disease of adolescence, when hormones change and fluctuate. Boys are slightly more prone to develop acne than girls and often suffer more severe acne. Those whose parents have had bad acne may also have a tendency to experience severe out-breaks. The presence of acne is almost universal in teenagers of all races. Other than teenage acne, other forms of acne also exist, including adult female acne, comedonal acne, and adult acne: Rosacea.

What causes acne? Acne is commonly seen in teens and young adults, and sometimes in infants. During teen years, the same hormones that trigger puberty also trigger changes in the skin's oil glands. The hormones are male type hormones, androgens, and are responsible for production of an oily substance, sebum, which normally softens and lubricates the skin. Please see diagram 1.

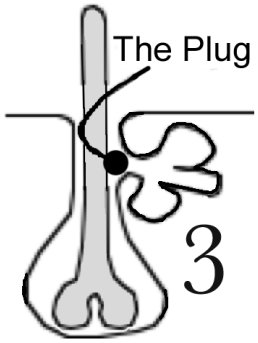


Acne starts within the sebaceous glands, oil glands that connect to hair follicles. The sebaceous glands are usually dormant from birth until puberty. Then, under the influence of hormones, they burst into activity, pouring sebum up through the follicles onto the skin's surface, causing the face and nose to be shiny with oil. This process is more or less normal in adolescence. Most teenagers have oily facial skin. As long as the oil can freely flow out onto the surface of the skin, the teen will have no major problem with acne. So then, what causes acne? Acne lesions develop when oils become hardened and blocked and cannot flow out freely. The hard oils form a plug. Unfortunately, acne prone people produce a sticky glue-like substance that is deposited between skin cells and tends to block the openings of oil glands.

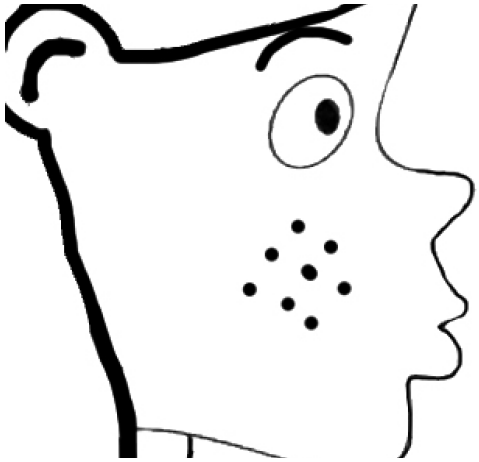
This substance forms the “sticky plug.” See diagram 2.



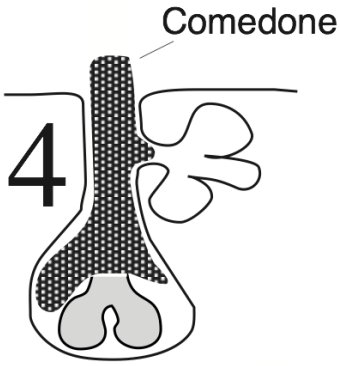
In people with acne, the cells are "sticky" and are not easily shed. The pores become plugged with a sticky plug of sebum, oils, and dead skin cells. These "sticky" cells tend to run in families with acne prone skin. As oily sebum travels out the hair follicle towards the surface of the skin, the sticky plug becomes larger. This sticky plug results in an open comedo, also known as a blackhead. See diagram 3.



A blackhead is formed when sebum, oil, and dead cells are exposed to surface air. Blackheads look like tiny black dots on the surface of the skin. It is very important to realize that the black color of the blackhead is not dirt.

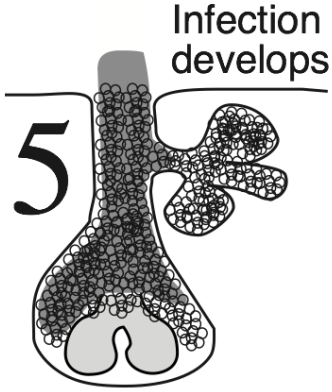


Small white dots on the surface of the skin are called whiteheads. A whitehead contains a sticky plug, oils, and dead skin cells, but has not been exposed to the air, and remains white instead of black in color. A whitehead is closed, and is called a closed comedo, comedone, or milium. See diagram 4.



Blackheads and whiteheads can be extracted.

What happens then? The plug may reach the opening of the outlet and appear as a black dot on the skin. This is an "open comedo" or "blackhead." The dark color is not dirt, as once commonly believed. It is thought to result from chemical changes due to air exposure. If the new comedo does not push itself to the surface, but remains just below the skin, it appears as a tiny, rounded, white elevation. This is a "closed comedo" or "whitehead." See Diagram 5.

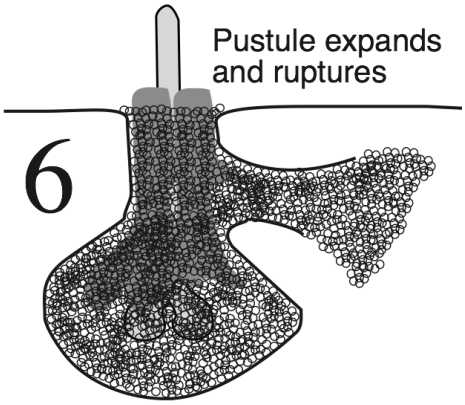


Both "blackheads" and large "whiteheads" may remain in the skin for a long time. In mild cases comedones may be the only manifestation of acne. The most important point to understand with this discussion is that acne is caused by blockage of pores. Thus, anything external that blocks pores will also cause acne. For example, mineral oil and petrolatum blocks pores by

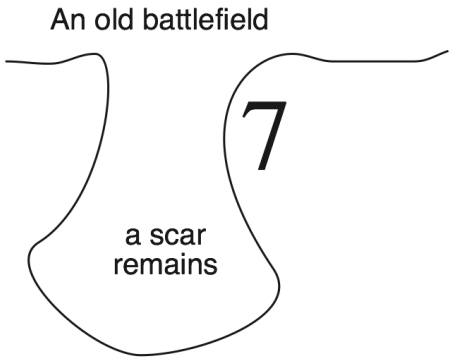
enhancing sticky plugs, thus blocking free flow of oil out of the pores. Acne = Androgens + Blocked pores + Infection.

As the follicles remain plugged, bacteria called propionibacterium acnes infect the follicles. The bacteria digest the oils and release free fatty acids. Free fatty acids cause irritation, inflammation, and pustules. In certain cases, large plugged follicles may burst and oil may leak out into surrounding skin, causing scars.

A large cyst can become like an over inflated balloon, which eventually explodes. See diagram 6.



This bursting effect can cause permanent scars and may leave skin depressions, ice pick like scars, and holes in facial skin. Our first goal is to prevent scarring as we treat acne. Lifelong scar prevention is our most important goal. Hormones will eventually subside and so will acne, but scars will remain if they are not prevented during adolescence. See diagram 7.



Will the acne go away?

Yes, acne will usually go away by itself. In girls, acne is usually at its worst from the ages of 14 to 17. In boys, acne reaches its peak in the late teens. Thereafter, its severity decreases and acne usually subsides by the early twenties. However, in some cases, the deeper lesions may leave a small, depressed scar or a lumpy scar. While the condition is active, acne may alternately erupt and then improve. When to refer to dermatology? When you see scaring or potential for scarring, it is a good idea to refer to a good complexion specialist dermatologist. A complexion specialist dermatologist is one who has extra expertise in treating of acne.

Think of active acne as a battlefield on your patients face. The patient is warring against bacteria and inflammation as they try to inflame and infect their teenage face. After the battle is over, and if the battle has been long and drawn out, the battlefield may show many areas of destruction. There may be holes and craters. These represent battle scars. As a primary care resident, to prevent permanent scars, your goal is to help your patient win and end the battle as quickly as possible. Acne will go away in time but scars will remain for life!

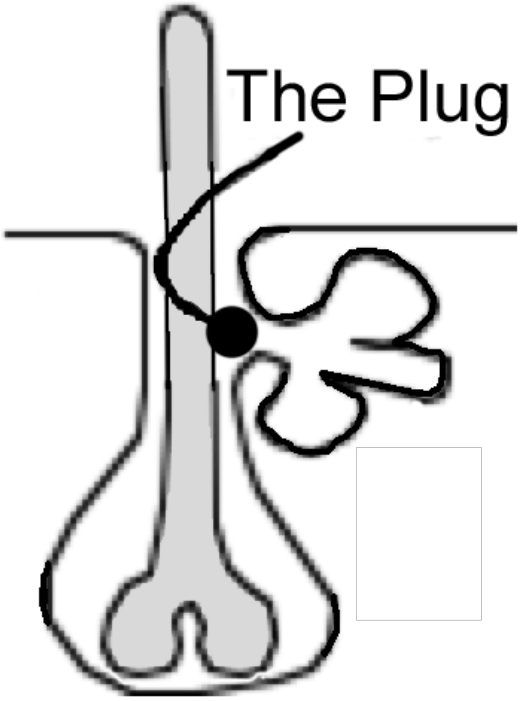
Thick Oil vs Thin Oil

Everybody has oil glands in their face, and for practical purposes, you can think of acne prone people as those who have a tendency to produce “thick oil” in their skin.; and, those people with less acne are those who produce “thin oil.” Thick oil leads to formation of the “sticky plug” shown in diagram # 3. In actuality, it’s much more complicated than this, because it’s really more than just oil. There are other factors as well, such as “sticky” epidermal cells, but for the sake of learning, you can think in terms of thick vs thin oil. The people with thick oil usually end up with plugged hair follicles that become infected, and thus get acne. So, if your patient has acne, then, as a primary care, you want to do everything possible to keep your patient’s pores clear and oil “thin.” One of the best ways to “thin” the oil is to use Retin-A or Epiduo. Retin-A is generic tretinoin and Epiduo is brand adapalene-benzoyl peroxide.

What things make acne worse?

Stress: Several factors may aggravate acne. First, are psychological factors & emotional

stress. Just as emotions can aggravate other skin conditions, such as eczema and psoriasis, emotions can also aggravate acne. Moreover, a teenager under emotional pressure may unknowingly pick at his or her pimples or repeatedly rub his or her face. Severe or prolonged emotional tension may aggravate acne. That's why acne may flare up before a math examination, or under stress of high school or college. Stress can increase acne hormones in your body (androgens or cortisol). Occasionally, a boy or girl may become upset over their perceived unsightly appearance. This psychological stress may in turn aggravate the acne further. It is advisable to take daily measures to reduce stress. Here are a few suggestions for your teenage patients: When things bother you, talk with your friends and relatives. Eat right. Enjoy a hobby. Exercise regularly.



Hormonal Factors: In many young men, a long list of muscle building androgenic steroids may aggravate acne. In girls, because of the influence of hormonal factors, acne may also worsen shortly before their regular monthly menstruation. Also, androgens are not the only hormones that promote thick yellow sebum production and influence the development of common acne vulgaris. Progesterone, a common female hormone produced by the ovary after the egg is released, can increase acne immediately before menstruation. Oral contraceptives, which contain various amounts of progestin (synthetic progesterone), can also cause pimples or make an existing acne condition worse. Under stress, depression, or anxiety, cortisol hormones may increase acne. Certain cortisol hormones can play a role in adult females with acne. Finally, certain women may have small ovarian cysts that produce excess androgens and acne. This condition is called Poly Cystic Ovary. If there is any question, you can order blood tests to rule out hormonal imbalances.

Food Factors: Are chocolate and fatty foods bad for acne? Not necessarily. These once forbidden foods seem to have little effect on the course of acne; so, daily diet is not usually restricted. Rarely, however, there are a few exceptions. Explain to your patients: If you find that certain foods, such as chocolate, fatty foods, excessive amounts of milk, sweets, or other foods seem to aggravate your acne, it is best to avoid them. In general, the best diet for those with acne is a well-balanced one. **Other acne aggravators include:** Travel Factors: Traveling to a different atmospheric climate may cause acne to flare. Drugs: Acne like skin conditions may result from the use of certain drugs, primarily bromides, iodides, fluorides, corticosteroids, androgens, and drugs used to treat epilepsy. Industrial chemical factors: Industrial substances, including coal tar, cutting oils, cooking oils, and petroleum oil, may block pores and cause severe acne. Local Factors can aggravate acne. Pressure on the skin from clothing, backpacks, shoulder pads, headbands, sport masks, and helmets can aggravate acne by friction. Similarly, resting the chin or cheek on a hand while doing homework or talking on the phone can cause a breakout. Finally, excessive sweating can worsen acne.

Mythical factors: While all the above factors have been proven to promote acne, or worsen an existing acne condition, certain other factors are widely believed to cause acne, but have no solid scientific evidence behind them. Eating chocolate, nuts, buttered popcorn, or greasy foods will not, in all people, increase acne. But, the oil from these, if touched onto the face, will block pores and cause comedonal acne.

Because of oil on the face, working over a deep-fat fryer in a fast-food restaurant may also

worsen acne. Rubbing popcorn butter over your face will also aggravate acne. Routine sexual activity does not prevent or clear acne, and masturbation does not cause it. Although, in men and in women under constant sexual stimulation, acnegenic androgen hormone levels increase in large amounts. Thus, some men and women may see more acne with sex.

How about cosmetics?

Cosmetic factors are the most obvious. Cosmetics with oils or wax are more prone to cause acne or aggravate a pre-existing acne condition. Any type of cream, cleanser, moisturizer, or makeup foundation that is not specifically labeled "oil-free" should not be used by at risk acne prone teenagers. Products labeled as “oil free” should be scrutinized before using. If the word "oil" appears in the ingredient list, the product shouldn’t be used. Of course, water based makeup is usually safe. Certain cosmetic products may be categorized as being comedogenic or non-comedogenic. Comedogenic products have a tendency to block pores and can promote acne. Mineral oil is the most common pore-blocking ingredient. Petrolatum, cocoa butter, and lanolins are also common pore blockers. Acne patients should not use mineral oil or petrolatum containing products on their face. Rather, they should use products that are listed as non-comedogenic. On a more positive note, water or gel based sunscreens that are primarily alcohol are usually a better choice than tanning oils, creams, or lotions. Mascara, lipsticks, and lip-glosses may have oil or wax. Many cosmetics, especially certain cleansing creams and moisturizers have greasy bases that can aggravate acne. Even certain oily "brilliantines" used on the hair may drip onto the forehead and help cause blackheads. It is important to avoid oily substances on your face by using water-based make-up instead of oil-based make-up, and by avoiding contact of skin with oily hair.

The cosmetics don't, as many people think, actually plug the pore or follicular opening. What cosmetics do appear to do is to alter the cells of the follicle, making them more likely to stick together and form plugs or comedones. This enhances the sticky cells described above. It is probably best to use as few cosmetics as possible for the time being. (One exception might be the cosmetics used to cover acne blemishes. They are usually made of a non-oily liquid base with powder, and seldom cause any harm.) If you must use cosmetics, use those labeled as water based "non-comedogenic." When moisturizing, always your patient should use a non-comedogenic oil free moisturizer.

Will vigorous washing help my acne?

You can explain to your patient: If you feel your skin is especially greasy, wash your face gently two or three times a day with a mild non-irritating gentle cleanser such as Aquanil Cleanser. But, do not rub and scrub. Forceful rubbing may only make matters worse. A mature comedo may be a few millimeters deep. No amount of rough scrubbing will dislodge it. The damage you cause to the follicle may in fact cause it to rupture, producing scar forming inflammatory lesions. For male patients, a clean razor is better than an electric shaver for acne. Other sources of friction, such as leaning on or rubbing an area of the skin affected with acne, or the pressure from helmets or tight collars, belts, or backpacks may also have similar effects. Although good hygiene is suggested, surface dirt on the skin does not cause acne. Remember that oil, not dirt, causes acne.

Should the pimples be squeezed?

Definitely not by the patient or by a non-professional. Most pimples should be left alone. In some cases, you, as a physician, may drain them if you learn the acne surgery techniques to open pimples and remove comedones. This is always a minor surgical procedure which needs experience, special instruments, and a special technique. Although many teenagers (and older people) think that picking or squeezing pimples will help eradicate acne, picking may actually make the condition worse and may lead to severe skin infections, pitting, and scarring. It is much more prudent to leave the pimples alone to dry, no matter how tempting it may be to squeeze them, and let

them heal by themselves. Digging and squeezing with fingernails results in ugly scarring. In fact, many times, more damage can be done to the skin by picking and squeezing than by the acne process itself.

Is there anything that is good for acne?

You can explain to your patient: For the most part, sunshine in small amounts is good for acne. Sunlight seems to help fully developed lesions disappear and can decrease the emergence of new ones. Be careful not to burn. Sunlight, together with the beneficial effects of outdoor exercise and holiday relaxation are probably the reasons why acne seems to improve during summer vacation. Of course, too much sun can make acne worse and can lead to wrinkling and skin cancer. Thus, even sunlight is not all good. As a rule, a tan is a response from the skin saying, "I've been damaged by the sunshine." Fair-skinned blondes and redheads often have the worst damage. In addition, prolonged heat and humidity can have ill effects in fair or dark individuals especially those unaccustomed to such a climate. In a humid climate, these people may experience explosive outbreaks of severe cystic acne, called "tropical acne." Thus, Is anything good for acne? Yes, a little bit of sunshine, lots of relaxation, and plenty of water.

Antibiotics? Is Acne an infection?

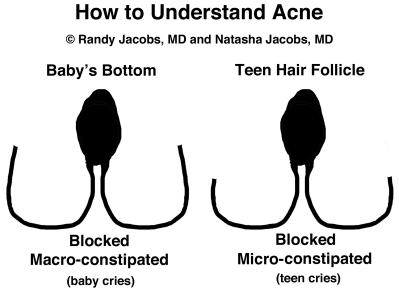
Yes, in certain ways acne is an infection. However, the usual microbes that develop on dirty cuts or scratches do not cause it. The causative organisms are tiny, often harmless microbes that are normally present in the hair follicle, especially one bacterial group called Propionibacterium acnes. Propionibacterium acnes can break down the fatty parts of sebum into fatty acid substances that leak into the surrounding skin and cause inflammation leading to pustules. Thus, antibiotic therapy is helpful in acne. As a primary care resident, you may prescribe an antibiotic that is taken internally as well as one that is applied on the skin. Antibiotics suppress Propionibacterium acnes, thus, there are fewer fatty acids produced and fewer scar forming inflammatory acne lesions. Because the skin is a difficult organ for drugs to penetrate, antibiotic therapy may be needed for one to three months, or more, depending on the severity of acne. You can remind your patient to inform you if any reactions develop or if they are pregnant, nursing, or trying to get pregnant. If given an oral antibiotic for acne, the patient should ask the pharmacist for a written handout on its proper use, benefits, and side effects.

Acne is Micro-constipation.

Strange but true, acne is actually a form of micro-constipation. “What?” The teen asks, “I have a constipated face?” Yes, you do! The concept of facial constipation may be a bit gross, a bit uncomfortable for some teens to think about; but, acne is truly a form of micro-constipation. And, if the patient can understand the concept of micro-constipation; certainly, he or she will better understand how to care for his or her acne problem.

First, let’s consider macro-constipation in a little baby. Every parent has dealt with this. The baby is irritable and crying and will not stop. The baby has not poohed (defecated) in three days, and is miserable. The parent calls the doctor and the doctor suggests a suppository. That doesn’t work so; the doctor prescribes a baby enema. Voila! Like magic! A muddy river flows and the baby is happy, smiling, laughing, playing, and now, everyone can sleep at night. Macro-constipation resolved.

Now, fast forward this baby 13 years; now the baby is a teenager with acne. And, now you see micro-constipation with plugged “constipated” blocked hair follicles filled with sebum. Acne develops. The baby cries and is miserable: Inconsolable! Nobody sleeps! Now, the doctor prescribes Retin-A, or Differin, or Fabior topical retinoid meds; and, voila! Like magic! The sticky acne plug goes away. Micro-constipation is resolved. The baby is better, happy again, and now everyone can sleep at night. Here is a diagram showing macro vs micro constipation.



Dermatology Guidelines for the Primary Care Resident: The Essentials

How is Acne Treated?

Although acne usually says goodbye after adolescence, a variety of treatments are available to prevent the development and spread of acne pimples. Prevention of scars and early treatment of acne tends to be much more effective than trying to rid the skin of pits and scars later in life. For mild acne, all that may be needed is frequent, gentle cleansing with an antibiotic based facial cleanser. Washing once or twice a day can help remove excess sebum and surface oils. Explain to your patients: Do not scrub your skin too vigorously, since friction can damage the delicate hair follicles and the openings through which the sebum must flow. There are many over-the-counter topical type preparations available to fight acne. Ingredients such as benzoyl peroxide, salicylic acid, sulfur, and resorcinol induce shedding of dead skin cells and surface debris, control bacteria, open the pores, and allow the sebum to escape to the surface. Benzoyl peroxide is probably the most effective of these agents. It is available in pads, creams, gels, and lotions, and in a variety of strengths. Benzoyl peroxide not only induces peeling, it also removes comedones, increases blood flow to the area, and suppresses bacterial growth. Explain to your patients: Careful, if used inappropriately, benzoyl peroxide can be severely irritating to the skin. Stronger is not necessarily better when treating acne. Do not use a higher strength benzoyl peroxide than you need to effectively control your pimples.

If over-the-counter preparations do not help, you may prescribe Tretinoin (Retin-A), a derivative of vitamin A, which is a topical retinoid prescription drug that is very effective in treating blackheads and whiteheads by removing comedones and interfering with the formation of new ones. Retin A somehow makes the sticky plug fluids less sticky. Thus, the sticky plugs are not able to form. Tretinoin should be used only after your patient has read and understood the proper use of Retin-A. Retin-A was the original topical retinoid developed years ago by Albert Kligman of the University of Pennsylvania. Differin and Tazorac are newer topical retinoid medications, and are in the same family as Retin A. Differin and Tazorac are used like Retin A. With Retin A, Differin, and Tazorac it may take up to four months before your patient sees results. Explain: Do not be discouraged. Topical retinoid medications are preventative, not curative. Once the old acne cells have been replaced by Retin A, Differin, or Tazorac primed new cells, your patient will have less acne as long as they continue to use their medication. Explain: If you stop the medication, you will see the acne return in three or four months. So, do not stop Retin-A until you are completely out of puberty.

Topical antibiotics, such as erythromycin, tetracycline, doxycycline, or clindamycin (cleocin), are particularly effective against bacteria living within the hair follicles, and can reduce the amount of irritating free fatty acids in the hair follicle. However, topical antibiotic therapy may not be enough to eradicate severe, cystic inflammatory acne. Rather, since an oral antibiotic can better penetrate the underlying skin tissue, oral antibiotic therapy is sometimes necessary to clear tough infected areas. It may take several weeks of treatment before the antibiotic's therapeutic effect is seen.

Females who do not respond to antibiotic therapy may benefit from estrogens contained in oral birth control pills, which decrease the size of sebaceous glands and reduce sebaceous oil production. Yaz is an oral contraceptive that is also FDA approved for the treatment of acne.

Isotretinoin (Accutane), like tretinoin, is also derived from vitamin A, but is taken orally. Accutane strongly inhibits sebum production and the growth of bacteria within the sebaceous glands. Although very effective, Isotretinoin must be used under strict medical supervision because it can cause severe birth defects. Isotretinoin must not be used by anyone who is pregnant. And, for women who may become pregnant (i.e. all women of childbearing potential), strict birth control must be practiced during Accutane therapy and for two months after discontinuing Accutane.

A few other acne treatments are available for you to learn and use. These include acne extractions, in which large acne lesions are drained and whiteheads and blackheads are

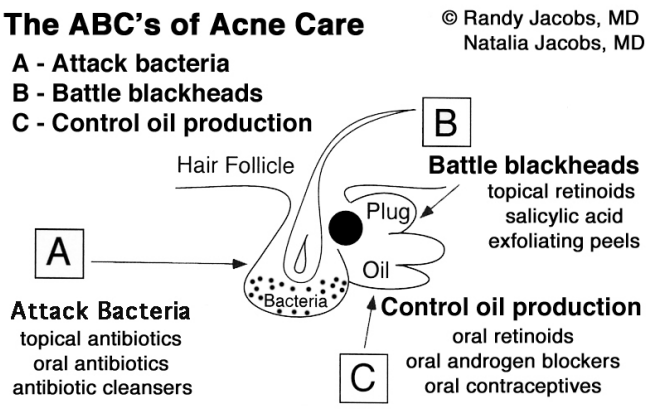
removed using special instruments. Light chemical peels, which peel the skin to exfoliate blackheads. Finally, hydrocortisone injections into enlarged acne cysts can result in a flatter pimple. Caution, cortisone injections can cause loss of subcutaneous fat, and depressed skin areas around the injection site. Thus, it is best to inject nothing stronger than hydrocortisone.

The ABC's of Acne Therapy

In summary, let's review acne pathogenesis & put it all together in a treatment focused way.

#1. A-Attack Bacteria- Control Infection.

Antibiotics: To attack the bacteria, you can prescribe oral or topical antibiotics. Topical antibiotics include benzoyl peroxide and sulfa meds available as either topical gels, suspensions, or cleansers in various strengths. To prevent irritation, use the lowest potency meds to do the job. Topical erythromycin & topical 1% clindamycin come as lotions, gels, and solutions. Topical antibiotics are applied once or twice daily to the surface of the skin to help fight the bacterial component of acne. Oral antibiotics are indicated for more severe inflamed, pustular or papular types of acne. Your patient may need them for one to three months or more depending on the severity of the acne. You may need to prescribe a few different antibiotics before you find the one that works for your patient. Remember, acne is an infection caused by the bacterium P. Acnes and potentiated by pore blockage. Certain strains of P. Acnes may be resistant to even the most expensive antibiotics. Finding the best antibiotic for your patient's acne is like finding the right key to fit a lock. Your patient should ask the pharmacist for an information sheet that explains any antibiotic side effects. Most important with minocycline, tetracycline, and doxycycline: The cycline antibiotics can accelerate tanning and can cause severe sunburn. When taking these, the patient should avoid sun and supplement with oral vitamin D.



#2. B-Battle Blackheads- Remove Plugs.

Topical retinoid medications Retin A, Differin, and Fabior have a unique way of decreasing the stickiness of blocked pore cells, and thereby promote the removal of sticky plugs. You will also notice that topical retinoids cause scaling. This scaling also helps to remove the plugs. Think about it... If you remove the plug, you will not get a buildup of sebum. Your sebum will flow out normally, and acne will not develop. What a concept! This is like removing the blockage from a plugged kitchen sink... Everything will flow better. Topical retinoids all act in a similar way. They are drugs that prevent acne from developing. Explain to your patients: Do not expect immediate results from topical retinoids as these drugs are only preventative. In reality, it takes at least 16 weeks to see results from Retin A, Differin, or Fabior. Sometimes topical retinoids may actually make acne worse before your frustrated acne patient sees improvement. They may bring all the acne to the surface before they see positive results.

#3. C- Control Oil Production.

If prescribed topical and oral antibiotics, plus topical retinoids do not bring enough relief, and especially if scarring is of concern, after careful consideration, the patient may need control of oil production. For females, this is where oral contraceptive and androgen blocking type meds can help. For both males and females this is where isotretinoin can help.

Oral Contraceptives for Female Acne

In females, both the adrenals and the ovaries can produce increased amounts of androgenic hormones to cause acne. The androgens stimulate oil production, and thus, oral contraceptives can be used to decrease oil production. Yaz birth control pill is a combination (contain both an estrogen plus a progestin) oral contraceptive pill that is FDA approved to prevent pregnancy. Yaz is also FDA approved in the treatment of acne in females 14 years or older who have attained

menarche. Yaz contains a low dose of ethinyl estradiol plus drosperinone, an antiandorgenic progestin. Yaz does not contain actual spironolactone. The progestin, drosperinone is a spironolactone analogue similar to spironolactone in its ability to block androgens.

Estrogens are known to block androgens by promoting increased sex hormone binding globulin, which binds androgens and decreases the serum levels of free testosterone and dihydroepiandrosterone sulfate (DHEAS). Estrogens also inhibit 5 alpha reductase and thus, prevents conversion of milder testosterone to potent dihydrotestosterone. Estrogens also inhibit both ovarian and adrenal androgen synthesis by way of decreased LH. The FDA has approved three brand name combination oral contraceptives for the treatment of acne: Ortho Tri Cyclen, Estrostep, and Yaz. Of these, Yaz contains the most potent antiandrogenic progestin hormone, and theoretically should show the very most antiandrogenic effects. However, the studies show that all three of these oral contraceptives are about equal.

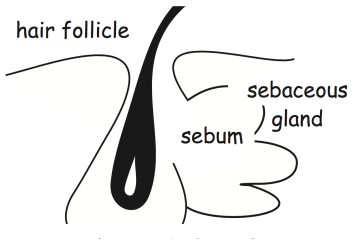
Spironolactone is actually not FDA approved for acne, and has a black box warning. Many dermatologists use it in their management of acne, but because of its black box warning for tumorigenesis, and can cause feminization of a male fetus exposed during the late first trimester, we will leave spironolactone out of the Dermatology Guidelines for the Family Medicine Resident. If spironolactone is needed for your acne patient we suggest that you refer the patient to dermatology.

So, for female patients with acne who are candidates, as a primary care resident, you can prescribe one of the three oral contraceptive FDA approved for acne. And, incidentally, pelvic exams and pap smears are no longer required when prescribing oral contraceptives. Side effects of all oral contraceptives include weight gain due to fluid retention, breast tenderness, decreased libido, mood changes, unscheduled bleeding, and melasma; not to mention the side effects of DVT in patients >35 years of age who smoke. In a large 2016 study, Yaz was shown to be of no greater risk for DVT than the other acne approved oral contraceptives. The risk of breast cancer is controversial, while the risks of ovarian & endometrial cancers are decreased.

Isotretinoin for Acne

Another way to block oil production is by way of isotretinoin. Isotretinoin, AKA Accutane, is associated with teratogenicity. It is also associated with premature closure of the epiphyses and has a black box warning by the FDA. Accutane management requires a special certification through a government program agency called ipledge. Accutane is actually FDA approved for acne, but has a black box warning. Of course, many dermatologists use it in their management of acne, but because of its black box warning for teratogenicity, also, a listing for suicide, and the possibility of premature closure of the epiphyses, we will leave isotretinoin out of the Dermatology Guidelines for the Family Medicine Resident. If isotretinoin is needed for your patient's acne, we suggest that you refer the patient to dermatology.

Most acne medications, for example Retin-A, Differin, and Fabior, are very irritating to the skin barrier and can cause drying and scaling. Thus, teenagers may become frustrated and may want to discontinue the medications. Explain to them: Don't do this! Just use the right moisturizer, and/or decrease the amount of medication applied, and the irritating dryness should improve. For example, when using topical retinoids, we suggest that the patient begin with once or twice a week applications before bedtime, and then, slowly increase to every night only as tolerated over the course of a month. Also, it is important to moisturize using the correct facial lotion that does not block pores. Many face lotions contain paraffin, oils, and pore blockers that can make acne worse.



Dermatology Guidelines for the Primary Care Resident: The Essentials

Rosacea Guidelines

ROSACEA: Q & A

For adults, getting acne can be a very difficult problem. Often, this happens to patients who never had acne during their teen years. The problem can be an embarrassment and a personal handicap, especially in social or business situations. There is help for patients.

What is rosacea?

“Acne Rosacea”, is a dermatologic condition that causes redness, pustules, papules, and inflammation of the facial skin. Rosacea is commonly, but mistakenly, attributed to alcohol consumption or may be referred to as "adult acne” as there is sebaceous inflammation.

How do I recognize rosacea?

Rosacea usually begins as a persistent sunburn like facial redness and flushing. As rosacea progresses, pimples appear on the face in the form of small, solid red bumps (papules) and pus-filled bumps (pustules). Pustules and papules may be accompanied by a condition called telangiectasia- thin, red lines caused by dilated blood vessels on the surface of the skin. Unlike acne, there are no blackheads or whiteheads, and this disease affects primarily the central portion of the face, particularly the center of the forehead, the chin and the lower half of the nose. In more advanced cases of rosacea, and more often in men, a condition called rhinophyma develops. A bulbous, enlarged red nose and puffy cheeks characterize rhinophyma. Comedian W. C. Fields had a good example of this condition, if you recall his nose. In addition to skin involvement, rosacea may also affect the mucous membranes of the eyes. Patients may complain of eye soreness or grittiness, a sign of ocular inflammation, or conjunctivitis. Untreated ocular rosacea may lead to a more serious complication called "rosacea keratitis," which may ruin vision. If symptoms are not relieved, refer your patient to an ophthalmologist.

Who is affected?

Fair skinned women ages 30 to 50 are most susceptible to rosacea. Men are affected almost as frequently. Most patients tend to be of northwest European descent, especially Celtic. Other risk factors include a family history of rosacea and "sensitive skin." Teenagers who are unusually sensitive to basic drug store cosmetic products and acne medications are potential candidates for rosacea in later years. One should keep in mind that rosacea may develop in the absence of any known predisposing factors. Rosacea may also be aggravated by long-term use of potent topical steroids or by using fluoride toothpaste.

What are the aggravating factors?

The exact cause of rosacea is still unknown, although studies have shown that many factors can irritate the condition. As a general rule, anything that increases facial redness will tend to worsen rosacea. Alcohol consumption of any type, spicy foods, hot drinks (including tea and coffee), and smoking will all complicate the problem by causing blood to rush to the affected areas, and aggravate flushing. It's important to note that, although alcohol may worsen a case of rosacea, symptoms may be just as severe in someone who does not drink at all. Certain drugs like nitroglycerin, theophylline, and niacin may dilate cutaneous blood vessels, cause flushing, and may aggravate rosacea.

How is rosacea treated?

Helpful hint: In some cases, before treating, you may want to check a dsDNA to rule out lupus.

Rosacea can be treated, however, there is no cure. Thus, preventative factors play a role. Stress, alcohol, fatty foods, cheeses, nuts, hot temperature foods, and hot, spicy foods, may flare rosacea. Since the majority of those affected by rosacea are unfamiliar with it, identifying the disease is the first step in treating it. When left untreated, rosacea may worsen and may become difficult to control. Self-diagnosis and treatment are not recommended as some over-the-counter skin applications may magnify the problem. Total therapy usually combines a sensible diet, avoidance of known aggravating factors, and appropriate medications. As a primary care resident, you may initially prescribe topical and/or oral medications. You can recommend that your patients cleanse their facial skin with Toleriane gentle cleanser and cool water. Explain: Do not use hot water. Rosacea therapy requires daily care. If your patient is diligent and cares for their rosacea on a day-by-day basis, your prescribed treatment programs can alter the progress of rosacea and can prevent late stage complications such as rhinophyma and scarring.

What are the medications?

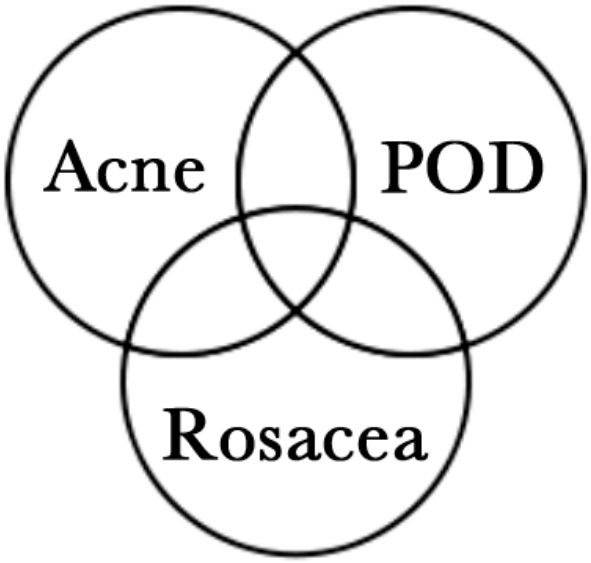
Topical antibacterial agents may be prescribed including topical metronidazole, tetracycline, clindamycin, erythromycin, and sulfa based cleansers alone or in combination with a mild topical steroid to decrease redness (hydrocortisone 2.5 lotion). Oral antibiotics may be useful in the treatment of rosacea. Tetracycline derivatives, minocycline, and doxycycline are most commonly used. Erythromycin can also be used. Avoid tanning and excess sun while using these. Topical Soolantra (ivermectin) is beneficial as it treats the demodex component of rosacea. Topical azelaic acid (Finacea) is also very helpful.

What about advanced therapy?

Surgical treatment of rhinophyma may be accomplished by dermabrasion, scalpel surgery, electrosurgery, or laser surgery. Telangiectasias (small dilated blood vessels) may be treated with a small electric needle, laser, or surgical ligation. You can refer resistant cases to dermatology.

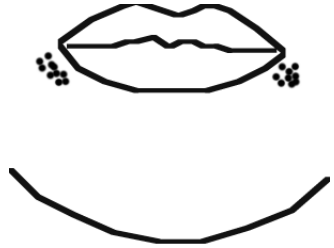
Notes

Acne, peri-oral dermatitis, and rosacea can blend together.



Dermatology Guidelines for the Primary Care Resident: The Essentials

POD: PERIORAL DERMATITIS



Definition

Perioral dermatitis, POD, has been defined as a persistent erythematous eruption of the skin composed of multiple tiny papules and little papulopustules, distributed primarily around the mouth. Perioral dermatitis is also a fairly recent dermatologic diagnosis. POD was not widely recognized until the mid 1950's. POD was given its present name in 1964, and the name describes the distribution of the rash: Perioral.

What Causes POD?

The exact cause of perioral dermatitis is unknown. Attempts to blame certain microbial agents have been unsuccessful. Some have claimed that sensitivity to the sun is the main cause, but the sun's role is now doubted with lack of substantiation in many cases.

Hormonal etiology has been suspected, as POD usually affects young women of child-bearing age, and has been associated with the use of oral contraceptive pills. Thus, hormonal etiologic evidence is there, however, hormones do not fully explain POD because many patients who have never used contraceptive pills get it, and others did not improve after discontinuing them.

Other perpetuating postulations for POD include: Fluorinated toothpastes, cosmetic ingredients, emotional stress, and "picking" at the perioral region. For certain, most observers are convinced that POD results from prolonged therapy with fluorinated topical corticosteroids applied for any reason to the perioral region.

Interestingly, you can also see the same POD like eruption on the scrotum after prolonged use of fluorinated topical steroids to the groin.

What Does POD Look Like?

Perioral dermatitis is predominantly a disease of young women. The condition consists of a circumoral eruption of many minute papules, pustules, and various tiny papulopustules on an erythematous and sometimes scaly base. The lesions spread irregularly in clusters, or as plaques of erythema, edema, and micropustules until they completely surround the mouth. The extent of involvement varies from a few inconspicuous lesions to a disfiguring eruption involving most of the area bordered by the nasolabial fold and sides of the chin. In most cases, the perioral area alone is involved. It is characteristic that there is a narrow zone of normal skin sparing the area around the vermillion border of the lips. At times, perioral dermatitis may involve the forehead, eye areas, the entire face, and even the groin. Around the eyes, it is referred to as periocular dermatitis.

How Does POD Progress?

The course of perioral dermatitis is one of fluctuations with recurrent flares of intense erythema and crops of papules. The primary lesion is a small pin-head sized papule or papulopustule that is either red or flesh-colored. These will dry up leaving a varying degree of scaling of the area. Mild to moderate itching and burning may be present. The affected area is often intolerant of sunlight and almost always to wind, heat, chlorinated pool water, and even hot water. Soaps and cosmetics may cause irritation, and even simple topical applications are badly tolerated. The total duration of the dermatitis can vary from months to years.

How Is POD Diagnosed?

The distinctive appearance of well-established perioral dermatitis usually allows for no confusion with any other facial dermatosis. Differential diagnosis of perioral dermatitis includes: Seborrheic dermatitis, rosacea, contact dermatitis, acne vulgaris, and adult female acne.

Dermatology Guidelines for the Primary Care Resident: The Essentials

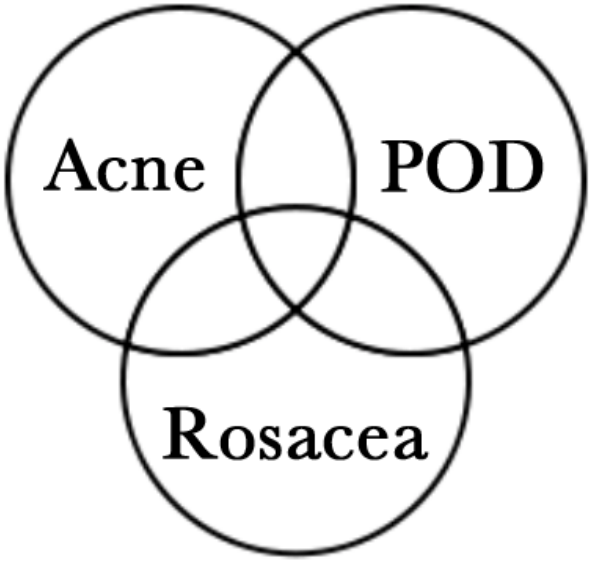
How Is POD Treated?

Perioral dermatitis has proved to be one a most difficult complexion skin condition to treat. Perioral dermatitis is usually not responsive to topical medical treatments. The essential first step is: discontinuation of all strong topical steroid medications used on the face. Oral tetracycline 250 mg 2 to 4 times daily for **2 to 3 months** has been effective in several studies. Doxycycline, is also effective. Topical antibiotics may be of benefit when combined with oral antibiotics. Mild topical steroids may actually help.

Often, POD comes when someone uses their hands to apply potent topical steroids to another part of their body, and then touches their face with their hands. Remember, with POD, don't ever touch your face with your hands. Always use a tissue to touch your face. Once controlled, POD is usually maintained in nearly all patients. Some patients may need additional work. Patience is important if POD is to be controlled.

Notes

Acne, peri-oral dermatitis, and rosacea can blend together.



Skin Microbiology
Section 4

Dermatology Guidelines for the Primary Care Resident: The Essentials

Skin Infections & Infestations

HERPES SIMPLEX: Q & A

Introduction

Infection with HSV is one of the most widespread infections in the world. Every day, if you notice, you might see people with a cold sore above their lip. HSV-1 infection is the number one cause of oro-labial herpes “cold sores”, while HSV-2 is the #1 cause of most cases of genital herpes. Seroprevalence of HSV-2 is lower and mostly appears after the age of sexual activity. Herpes virus has the ability to produce latent, but lifelong infections by entering immunologically protected nerve tissues. Intermittently, there are episodes of reactivation that allow viral transmission from person to person. HSV infections are classified as either “first episode” or “recurrent." The vast of majority of infected people remain asymptomatic, otherwise, infection presents as grouped vesicles-blisters on erythematous base. The lesions are usually painful, and eventually form yellow crusts. The crusts gradually fall off and leave slowly fading areas of red. The onset of herpes is often accompanied by itching, burning, stinging, or deep pain, fever, malaise, and local lymphadenopathy. The whole process takes about 10 to 14 days. The pain disappears several days before the ulcers heal, which usually occurs by 1 week on the average. Scars can form, but usually do not. There are variable degrees of illness. The very first infection with type 1 herpes virus usually happens in childhood. Herpes virus can also propagate in broken skin on any part of the body, and is a common infection of wrestlers, hence the term herpes gladiatorum. Children with eczema are also prone to herpes infection. Herpes virus can easily gain entrance through damaged skin and establish an infection in all involved areas, resulting in serious illness. Girls are prone to herpes infections of the vagina and labia. The blisters and ulcers of herpes simplex are filled with viral particles and are contagious until they heal. Reactivation of infection usually appears under following conditions: During menses, overexposure to sunlight, colds, and other upper respiratory infections, fever from any source, physical or emotional stress, reactions to drugs of foods, and injuries.

Caution

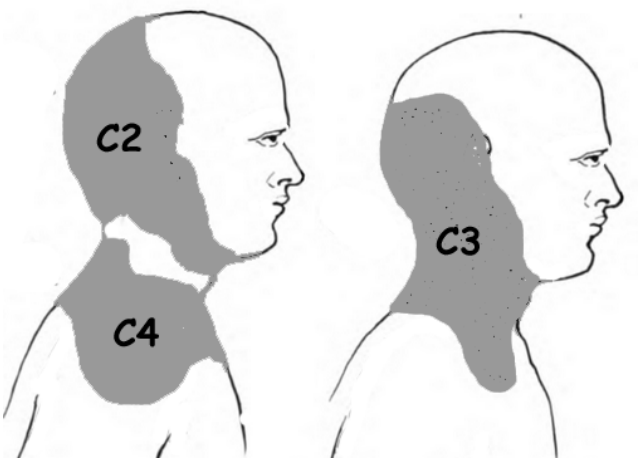
Explain to your patients that, if they have an active cold sore, please do not touch a baby or immunocompromised individual. There are cases where a grandmother or grandfather or a mother or father kiss their baby grand child or child. Afterwards the baby gets herpetic meningitis and dies. Please explain these facts to your patients.

How are herpes infections treated?

Medications for the treatment of herpes include Valtrex, Zovirax, and Famvir. The medications work best when started early. To date, no vaccine has been developed to prevent herpes infections from occurring, and no cure has been developed. Recurrent attacks of facial herpes simplex may be decreased by avoiding excessive sun exposure and by using a good sunscreen.

Notes

Dermatology Guidelines for the Primary Care Resident: The Essentials



Herpes Zoster

Zoster is caused by reactivation of varicella zoster virus. After primary infection or vaccination, the virus remains latent in the sensory dorsal root ganglia. The most common inciting factors of reactivation are immunosuppression and age-related deficiency of cell-mediated immunity. Thus, incidence increases with age. Classically, Herpes Zoster occurs unilaterally within distribution of the dermatome of a cranial or spinal sensory nerve. Most commonly affected dermatomes include thoracic (55%), cranial (20%), lumbar (15%), and sacral (5%).

Cutaneous lesions are preceded by several days of pain in the affected area. Rarely, but occasionally, an eruption may be painless. Skin symptoms initially present as papules and plaques of erythema, then within hours, plaques develop blisters that may become hemorrhagic, necrotic, or bullous. In some cases, patients may have pain, but no skin lesions. Interestingly, there is a correlation with the severity of pain and the extent of skin lesions, and elderly patients tend to have more severe symptoms of pain and may be more refractory to treatment.

Duration of the disease depends on three factors:

1. The patient's age.
2. The severity of eruption.
3. The presence of immunosuppression.

Sometimes lesions may develop on mucous membranes within the mouth of a patient with zoster of the mandibular maxillary division of the trigeminal nerve, or in the vagina in zoster of the S2/S3 dermatome. Also, zoster may appear on recent surgical scars. Post-herpetic neuralgia is the most common post zoster complication and can last up to several months, or for years. Among other adverse sequellae, you may see motor nerve neuropathy in about 3% of patients, and Ramsay Hunt syndrome: Auditory symptoms and facial paralysis from facial and auditory nerve involvement.

Treatment

Antiviral therapy is the cornerstone of management of herpes zoster, the main benefit of which is reduction of duration of disease, and decrease in zoster-associated pain. Valtrex or famciclovir are usually prescribed. In severe cases, intravenous acyclovir may be indicated. Pain management is important and depends on the severity of pain. Topical analgesics, NSAIDs, opioids, epidural or sympathetic blocks, transcutaneous electrical nerve stimulation, anti-seizure medications, neuroleptics, amitriptyline, and local application of heat may also be recommended.

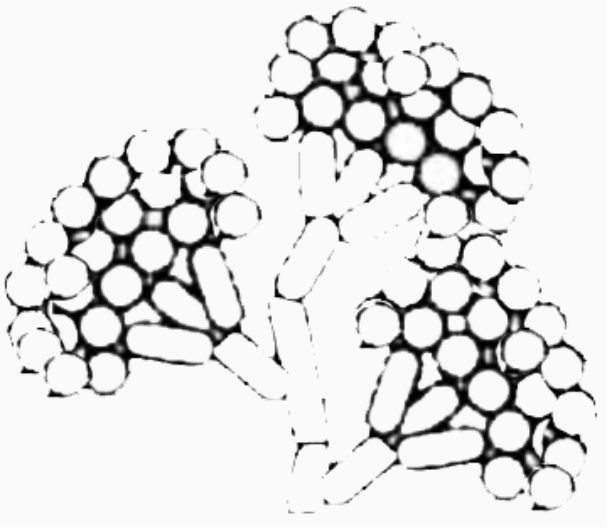
Prevention

A vaccine using the same attenuated virus as in varicella vaccine, but in much higher titers, Zostavax, has been approved for prevention of herpes zoster. Zostavax is recommended in all patients aged 60 years of age older, including immunosuppressed individuals. Zostavax vaccination reduces the incidence of zoster by 50%, and significantly lowers the pain and shortens the duration of post-herpetic neuralgia and other complications of zoster.

Notes

Dermatology Guidelines for the Primary Care Resident: The Essentials

Tinea Versicolor



Introduction

Tinea Versicolor (TV) is a cutaneous yeast infection caused by Malassezia furfur, which is the mycelial phase of Pityrosporum orbiculare, a yeast which is a normal skin flora. TV is slightly contagious, but the disease is usually self-generated or autogenous. TV is common in the 20's, uncommon in kids and rare in seniors. It affects both sexes the same. Steroid therapy promotes TV, as does Cushing's syndrome. TV is more noticeable in the summer when the rest of the sun exposed skin is tanned but the skin affected by TV does not tan.

What does TV look like?

TV is usually noticed as slowly enlarging scaly, whitish spots scattered over the arms, chest, and back. On light skin, the spots may go unnoticed. Fungal cells growing in large numbers on the skin prevent the skin from tanning normally; thus, as the individual's skin tans, the spots become more noticeable. The white patches are due to a bleaching chemical produced by the yeast. When they occur on the exposed areas, such as the face and neck, their appearance may be very alarming to the patient.

What are the symptoms of TV?

TV produces few symptoms. Occasionally, a patient will experience slight itching. The scaling is minimal and develops very fine scales when the patient rubs or scratches. Itching may be intense when the patient is hot.

Who gets infected?

A majority of people who get TV are teenagers or young adults. TV is very rare in the elderly and is uncommon in children, except in tropical climates where it is seen in almost all ages. People of both dark and light skin seem to be equally susceptible. Patients with oily skin may be more susceptible than those with dry skin.

Why do some have TV and others do not?

The reasons why some have TV and others do not are not clear. Since the fungus is normally present in small numbers on the skin, everyone could develop TV. Malassezia usually grows slowly in the skin and normal hygiene removes the excess number of fungal cells along with the dead skin. During summer months when the heat and humidity are high, yeast cells increase in number and the removal of scales is reduced; therefore, malassezia grows and forms small colonies on the surface of the skin. These colonies affect pigmentation, resulting in light or dark colored spots. In some tropical countries where the heat and humidity are continuously high, persons have these spots all year around. In temperate climates, TV usually disappears in the cooler and drier months of the year.

How is this infection diagnosed in the office?
TV is easily recognized. In most cases, the signs and symptoms are enough to make a diagnosis. Years ago, a simple direct examination of fine scales scraped from the lesions would confirm the presence of the fungus. Now, a CLIA certificate is required to do this test.

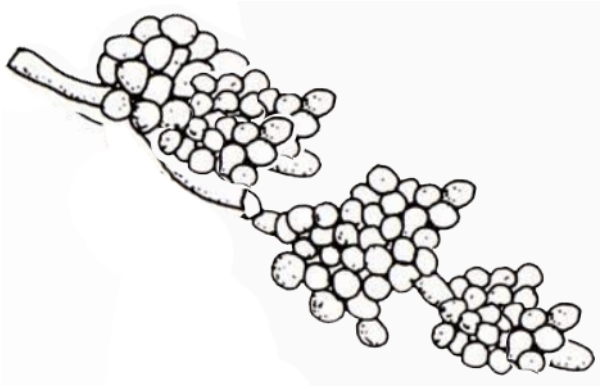
How is this fungal infection treated?

TV is easily treated with either topical or oral anti-yeast meds; but, uneven pigmentation of the skin can remain several months after the fungus leaves. To treat TV topically, ask your patient to apply 2.5% Selenium Sulfide (Selsun or Exsel) Suspension to the affected areas. Allow the 2.5% Selenium Sulfide (Selsun or Exsel) Suspension to dry for 10 minutes, and then, wash off. Repeat this process every day for ten consecutive days. **Because the depigmentation is caused by a bleaching chemical, it is important to remember that the fungus is easy to kill, but it takes many weeks to repigment the skin.** It is

possible for TV malassezia yeast to be gone, yet the bleaching agent remain for a while. It is also possible for TV to recur.

"Worst yeast infection I've ever seen!"

Notes



Candidiasis

Candidiasis, is a yeast infection due to Candida albicans, a common yeast inhabitant of the human GI tract, urinary tract, and skin. Under warm moist conditions, Candida albicans becomes a real pathogen, causing respective lesions of the skin, nails, mucous membranes, and sometimes systemic infection as well. The intertriginous areas are frequently affected. An environment with warmth, moisture, and skin maceration allows Candida albicans to thrive. The areas of skin most often involved are the bilateral perianal and inguinal folds, abdominal creases, inframammary creases, the interdigital areas, nail folds, and bilateral axillae.

Candida albicans is primarily an opportunistic yeast, acting as a pathogen when there is an impaired immune system, or where local environmental conditions encourage growth. Decreases in competing flora during certain antibiotic therapies can also favor candidal growth. Diapers, underwear liners, and occlusive body wear raise skin pH and may predispose patients to severe Candida albicans infection.

In healthy infants, mucous membranes of the mouth may be infected with thrush. In newborns, the infection may be acquired from contact with the vaginal area of the mother. In older children and adults, thrush is commonly due to antibiotic therapy, or even immunosuppression. Gray-white patches are found on the surfaces of the mucous membranes, whereas the base of the lesions is red, erythematous and macerated. At times, Candida albicans can spread to the angles of the mouth, where it causes perleche, and other intertriginous areas where it promotes intertrigo. Candida albicans is most often treated with oral nystatin suspension in babies, or a single oral

dose of the antifungal, fluconazole in adults. In immunocompromised patients, higher doses of anti-yeast medication are often needed.

Perleche, AKA angular cheilitis, shows chronic maceration and tender fissuring of the oral commissures. Perleche mostly occurs with night time drooling or poor fitting dentures. The lesions are gray-white thickened areas with erythema at the base. Anti-yeast creams are effective, but response is better if combined with a topical corticosteroid and a zinc oxide paste.

Candidal vaginitis is the most common candidal infection in women. Candidal vaginitis may develop during pregnancy, with diabetes, or secondary to antibiotic therapy. The vaginal discharge is usually thick, white, and cottage cheese like, associated with discharge, severe pruritus, burning, erythema, swelling, and maceration of external genitalia. Treatment is usually a single oral dose of fluconazole, and probiotics may also help.

Candidal intertrigo is a pruritic eruption that develops between moist skin folds, such as the groin, axillae, buttocks, infra-mammary areas, and under obese abdominal folds. The area is red and erythematous with moist patches and maceration, sometimes with pinpoint white pustules on the surface. At times, candidal infections can resemble a fungal infection, but it is less scaly and has a greater tendency towards fissuring. Topical anti-yeast medications are usually effective, but recurrence is common. Measures to promote dryness of the area are always recommended. This is one example of the age old derm saying, “If it is wet, dry it, and if it is dry, wet it.”

In babies, diaper candidiasis may be suspected by finding involvement of the body folds and the breakout of many small erythematous red desquamating “satellite” lesions scattered along the edges of larger patches. See thrush in the mouth and red satellites in the diaper area, think candida. Topical anti-yeast meds are effective, sometimes compounded with zinc oxide paste to protect against urine maceration and irritation. You may prescribe Nystatin Oral Suspension. The dose of Nystatin for children and adults is 4-6 mL four times daily- one-half of dose in each side of mouth. Infant dose is 2 mL four times daily. In infants and young children, use dropper to place one-half of dose in each side of mouth, and avoid feeding for 5 to 10 minutes. For information on babies, please see the Dermatology Guidelines Pediatric section on diaper dermatitis.

Candidal paronychia is a chronic swollen, tender inflamed infection of the nail fold that produces redness, erythema, gradual thickening along the sides of the nail. Patients can have an atopic background or diabetes. Fingers are infected more often than toes. A culture helps to confirm the diagnosis of candida yeast and differentiates candida paronychia from bacterial paronychia. Treatment of candida paronychia is topical anti-yeast creams, and oral fluconazole. Diabetic control is important. It is also essential for the patient to avoid exposure to moisture and irritants. This means latex or nitrile gloves with white cotton gloves underneath.

Systemic candidiasis may develop when a patient’s immunological defenses or skin barrier function is compromised. Those at risk include patients with various hematologic malignancies, especially leukemia’s and lymphomas, AIDS, transplant recipients, patients with indwelling intravenous catheters, patients undergoing therapy with systemic steroids, and debilitated or malnourished patients. Diagnosis is confirmed by culturing candidal organisms from body fluids and tissues ordinarily sterile for Candida. The gold standard of treatment is Amphotericin B.

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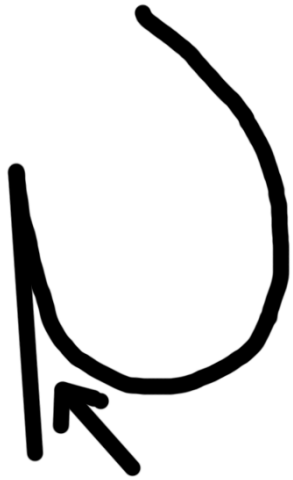
Dermatology Guidelines for the Primary Care Resident: The Essentials

Intertrigo



Intertrigo is a chronic superficial inflammatory dermatosis occurring where two skin surfaces are contacting each other in opposition. There is chronic wetness and maceration. The skin gets no air and thus it becomes like a tropical jungle that grows everything bad: Yeast and bacteria. Excessive sweating adds to the problem. Intertrigo is really a physically induced disorder, in that, if the skin folds were not touching each other, your patient would not have intertrigo. As result of friction, heat, and moisture, the affected folds become erythematous, macerated, and secondarily infected. There may be erosions, fissures, and exudation, with symptoms of burning and itching. Intertrigo is most frequently seen in hot and humid weather, and primarily in overweight or diabetic people usually located in the inner thighs, armpits, and undersides of the breasts. Tight fitting clothing makes the condition worse. The patient may find discomfort in walking because the moist areas can be painful when mechanically rubbed.

As a result of maceration, a secondary combined infection with bacteria and yeast is promoted. The bacterial component of infection may be due to Streptococci, Staphylococci, Corynebacterium, anaerobes, Pseudomonas, and other gram negatives. The yeast is most commonly Candida Albicans. Treatment is directed toward drying out the maceration. Appropriate antibiotic agents to treat the above organisms are needed and are taken orally and applied topically. Botulinum toxin type A has been used to dry out areas of recurrent macerated disease.

Management and Treatment

For the treatment of intertrigo, first, take bacterial and fungal cultures. Then, the affected body surfaces should be thoroughly cleansed, dried, and sprinkled with talcum powder or pastes containing zinc oxide. Zeabsorb is a helpful powder available over the counter at your pharmacy. Do not use corn starch as the bacteria and yeast can thrive on corn products. Continuous use of gauze strips or terry cloth towels between the affected areas will also help keep the areas dry and exposed to air. Some people find great relief by keeping a fan blowing on the area or by keeping a blow dryer (set on no heat) blowing on the area. The fan keeps the area dry and free of maceration. For the combined yeast / bacterial infection you may prescribe anti-bacterial and anti-yeast medications in addition to zinc oxide paste. The prevention of intertrigo and other fungal or yeast infections is a highly important topic for patient education.

People should make sure to wash and dry very well especially between the toes, armpits, groin, and undersides of the breasts. Showers in homes, hotels, and locker rooms should also be kept clean. In addition, the following measures can be helpful for some people with intertrigo: Cotton underwear and loose-fitting clothing are best. Explain to your female patients that they should avoid pantyhose and tight fitting under wear and clothing. Sterilize panties by washing with very hot water and detergent.

Explain to females that they need to wipe from front backwards, and wash with water each time after going to the toilet. Keep Hemoglobin A1c in check. Eat acidophilus yogurt or take probiotics. In most cases, the patient can continue with their regular life if the patient follows through with the proper medications and keeps the areas dry. In severe cases, the patient may have to rest until the infection has cleared.

Helpful Hints

Two helpful hints to share with your intertrigo patients. #1 Your patient might find quicker relief if he or she sprays antiperspirant deodorant liberally to the macerated areas twice a day. #2 Superabsorbent baby diapers are great for

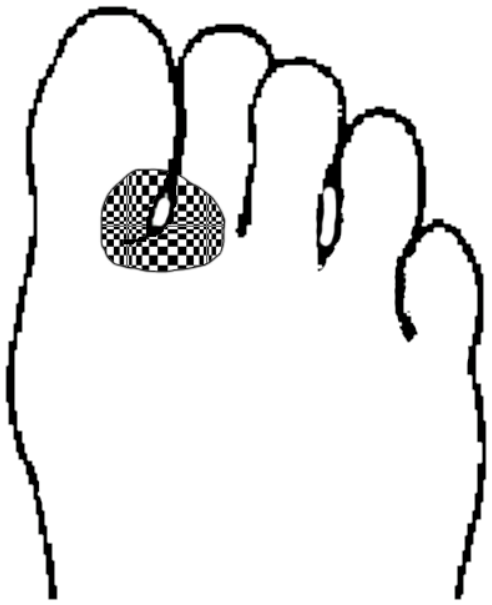
wicking up extra moisture, to keep the macerated areas dry. Use Superabsorbent baby diapers and change them often.

What to Expect

In most cases, intertrigo will clear in two to four weeks with the above program. After intertrigo clears, it is most important to keep the areas dry to prevent recurrences. Some patients are not compliant or are helpless in caring for themselves. Each patient's case will need to be evaluated individually.

Notes

Tinea Infection



Introduction

Tinea is a commonly diagnosed superficial dermatophyte fungal infection of the skin in adults and children that may affect the scalp as tinea capitis, the body as tinea corporis, the groin as tinea cruris, the feet as tinea pedis, the hands as tinea manus, or the nails as tinea unguium. Tinea corporis is diagnosed by one or more circular, sharply circumscribed, erythematous, dry, scaly patches with central clearing like rings, that give them their "ringworm" appearance. The diagnosis is easily made by finding fungus under the microscope in skin scrapings or can be cultured on Saboraud's agar. Mild tinea corporis is usually treated with topical antifungals. More severe infections require oral medication, usually griseofulvin. Please note: Griseofulvin cross reacts with penicillin, and thus, some penicillin allergic patients will need oral terbinafine or fluconazole therapy.

Tinea capitis

Tinea capitis is distinguished by dry thick scaling and well circumscribed puffy areas of alopecia and broken hairs, with prominent posterior cervical lymphadenopathy.

Wood's lamp can reveal fungal fluorescence and is useful as a diagnostic tool. Tinea capitis is also treated with oral griseofulvin or terbinafine and requires four months of oral treatment.

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Tinea Corporis & Tinea Cruris

Fungal Infections: Body & Groin: Tinea corporis and tinea cruris are fungal infections of the body & groin. When the infection involves the upper thighs or groin, it is called tinea cruris, also known as "jock itch."

Tinea corporis can occur in all ages. Sometimes, people acquire the infection from pets like dogs or cats. Sometimes patients can acquire tinea from human contact, or rarely, the soil. The infection usually occurs on the trunk, limbs, or face, tinea facei, and the arms or neck.

Tinea corporis and tinea cruris can occur anywhere on the body. Tinea cruris can be aggravated by tight clothing, obesity, and warm weather. Humidity tends to grow dermatophyte fungal organisms.

Tinea Manus & Onychomycosis

Fungal Infection: Hands & Nails: Tinea Manus is a chronic fungal infection of the hands, usually Trichophyton rubrum. Tinea Manus is almost always connected to a pre-existing Tinea Pedis, or fungal infection of the feet. When one sided with tinea pedis, the condition is called Two Foot One Hand Disease. One of the most difficult to treat forms of infection, onychomycosis, destroys the nail plate.

Majocchi's Granuloma

Most tineae infections are superficial, but Majocchi's granuloma is a tinea infection that goes deeper than usual. Majocchi's granuloma appears as deeply set papules associated with hair follicles.

"Id Reaction"

"Autosensitization dermatitis," also known as "autoeczematization," also known as the "Id Reaction" is the vesicular rash sometimes seen in patients with acute fungal infections of the feet. The vesicular eruption involves the sides of the fingers but can involve the entire body. Some feel that the "Id Reaction" is a systemic allergic reaction to the fungi or some antigen formed during the inflammatory process.

How are tinea infections treated?

Treatment of Tinea corporis can consist of topical antifungal creams or lotions such as ketoconazole and clotrimazole, or oral medications. Oral meds include griseofulvin for non-penicillin allergic patients, terbinafine, and fluconazole.

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PARONYCHIA

What Is paronychia?

Paronychia is an inflammatory reaction involving the folds of skin surrounding the fingernail. Paronychia may be acute or chronic, purulent, tender, and painful with swelling of tissues around the nail. Usually, Paronychia is a result of trauma from moisture-induced maceration of nail folds resulting from frequent water exposure. Paronychia starts because the moist nail grooves become secondarily invaded by bacteria or yeast.

In paronychia, the most commonly found bacteria are staphylococci, pseudomonas aeruginosa, & gram-positive streptococcus. Candida albicans is the most common yeast organism found. *Candida is suspected if the proximal nail fold is involved, while Staph is suspected if the lateral areas are involved.*

Acute paronychia

Acute paronychia develops over a few hours when a nail fold becomes painful, red and swollen. Sometimes yellow pus appears under the cuticle. In some cases, fever and painful glands under the arms accompany acute paronychia. It is usually due to Staphylococcus aureus, a bacterial infection treated with oral antibiotics. Sometimes an abscess forms and must be lanced. Acute paronychia usually clears completely in a few days, and rarely recurs.

Chronic paronychia

Chronic paronychia is a gradual process and much more difficult to get rid of. It may start in one nail fold but often spreads to several others. Each affected nail fold is swollen and lifted off the nail plate. It may be red and tender from time to time, and sometimes a little pus (white, yellow, or green) can be expressed from under the cuticle. The nail plate becomes distorted and ridged as it grows. It may become yellow or green and brittle. After recovery, it takes up to a year for the nails to grow back to normal. Chronic paronychia is due to several different micro-organisms. Often a mixture of yeasts and bacteria are present, particularly candida species and Gram-negative bacilli. The inflammation results in debris, which builds up, encouraging more infection. It mainly occurs in people who have constantly wet hands, such as dairy farmers, fishermen, bar tenders and housewives. It is more likely to occur, and more difficult to clear up, in those with poor circulation, especially during the winter months.

How Is paronychia treated and prevented?

Preventive measures are important for anyone who uses their hands in water. Preventive measures consist of cotton lined rubber gloves to keep the hands dry and warm. Explain to your patient: Avoid wet work, or use totally waterproof gloves. Keep your hands clean. Wash thoroughly after dirty work with Toleriane and water, and rinse off. Dry carefully. Don't let the skin dry out and crack. Prevent drying by applying a moisturizing hand cream frequently. Apply antimicrobial lotion regularly twice daily to the nail fold. A suitable preparation is: Polysporin lotion or spray. Topical gentamicin solution may be prescribed. You may prescribe a course of an oral anti-yeast agent or antibacterial antibiotic. Also, in severe cases, you may order an x-ray to rule out osteomyelitis. It often takes months to clear paronychia, and paronychia can recur in predisposed individuals if not prevented.

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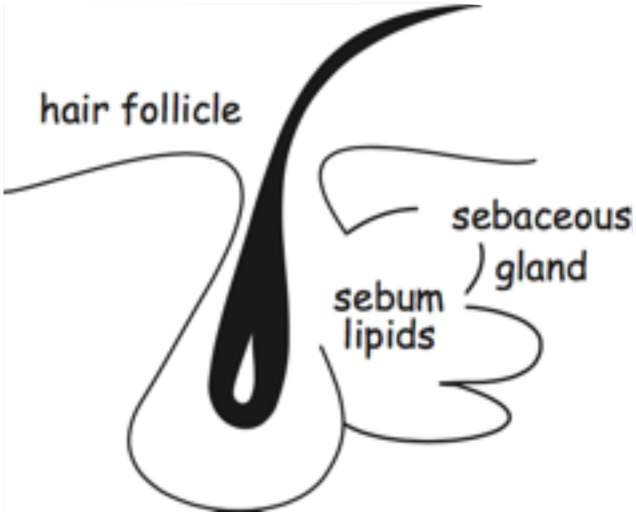
Infected Hair Follicles

Folliculitis



An infection of hair follicles, folliculitis is distinguished by follicular pustules, most times with surrounding erythema. Etiology can be bacterial, but physical and chemical factors can also come into play. You can culture folliculitis, as there is pus.

Susceptibility factors are shaving, occlusive dressings, use of oil containing products, staph colonization, and friction. The under 30's and adolescents are mostly affected, but folliculitis can infect anyone any age. Most common sites include the face, scalp, beard, underarms, butt, and lower extremities.

Two very important predisposing factors include: #1. Hot tubs. Pseudomonas folliculitis is very commonly spread at hot tub gatherings. #2. Pickers. Fingernails are some of the most contaminated parts of the human body, and picking or scratching can inoculate organisms and cause folliculitis.

Furuncles, Carbuncles, & Abscesses

Folliculitis, furuncles, and carbuncles represent a continuous thread of infection of hair follicles. You can culture them, as there is pus. Furuncles AKA "boils" are much like folliculitis, but, the infected suppuration travels beyond the follicle deep into the dermis and subcutaneous fat. Furuncles are fluctuant plus tender but usually do not cause fever, chills, and systemic signs. The furuncles will later burst and produce pus. The most common areas of involvement include those close to friction, such as the breasts, axillae, butt, and thighs. A carbuncle is a spread-out collection of furuncles with a variety of sinus tracts.

AN easy way to remember the difference between furuncle and carbuncle is to remember that a furuncle is sitting in one place, while a "car" buncle gets in a car and drives all over.

An abscess is a localized mass, a tender collection of pus and may have developed from a furuncle or carbuncle or infected cyst. The nape of the neck is the usual site. Sometimes these more complicated lesions are associated with systemic signs and symptoms of fever, tiredness, chills, malaise, and also lymphadenopathy. MRSA staphylococcal aureus carriage, diabetes types 1 and 2, and morbid obesity are susceptibility factors. Very much like furuncles and carbuncles, abscesses are exquisitely tender, fluctuant and nodular with a rim or erythematous color. However, unlike a furuncle or carbuncle, an abscess is not associated with a hair follicle.

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Superficial Bacterial Infections

Cellulitis

Cellulitis is a painful infection of the deeper layers of the skin, usually on the foot or leg in adults. You cannot culture it, as there is no pus. The infected area is red and swollen. There may be fever. Cellulitis is a nonfollicular bacterial infection but does not have the draining pus of an abscess. Tender to touch erythema, edema, and warm to touch distinguishes cellulitis. Tumor, dolor, calor, rubor. Cellulitis involves the deeper layers of the dermis and subcutaneous fat and is often manifested with systemic symptoms such as fever, chills, and overall malaise. The area of involvement depends on the age of the patient. In children, head and neck cellulitis is the most common. In adults, lower extremity cellulitis is most common. The break in skin that serves as the entry for infection, can be clinically subtle. It can be as simple as a damaged skin barrier, dry skin, an excoriation, a slight scrape or small puncture. An underlying dermatitis with a scratch, a moist fungal infection, trauma, a surgery, a foreign body, or a simple cut after trimming toenails: Each of these can allow entry of bacteria. Chronic leg swelling, venous insufficiency, diabetes, alcohol addiction, cancer, drug abuse, and immunosuppression are all susceptibility factors.

Erysipelas

Erysipelas is a more superficial type of cellulitis, less deep into the skin. Erysipelas is distinguished by redness, and by being warm to touch, tender, and extending out towards the periphery. You cannot culture it, as there is no pus. Cellulitis has an indistinct border, but with erysipelas, there is a sharply demarcated, raised border, with vesicles, tiny pustules, and sharply defined necrosis of the skin. Local lymphadenopathy, inflamed lymphatic vessels, and fever, chills, malaise are common. The face and legs are the areas most commonly infected.

Notes

Infestations

Scabies

Introduction

Scabies also known, as "Sarcoptes scabiei" is an intensely itchy rash caused by a tiny mite that burrows into the skin. It is the Sarcoptes scabiei, fertilized female mite which burrows into the stratum corneum of epidermis and deposits her eggs. The scabies mite is only 1/60 inch long, and is almost impossible to see. The scabies rash usually involves the hands, wrists, breasts, genital area, umbilicus, and waistline. Scabies is distinguished by pruritic papules, excoriations, and burrows.

This tiny mite has infested humans for thousands of years. The mite is often hard to diagnose and causes a fierce, itchy skin infestation. Scabies can strike anyone of any race or age, regardless of personal hygiene. Face and scalp are usually spared, except in infants. The burrows appear as slightly elevated, grayish, tortuous lines on the skin. Vesicles or pustules may be noted at the end of the burrow.

The eruption varies depending on the length of infestation, previous sensitization, climate, and patient's immunologic status. Lichenification, impetigo and furunculosis may be present. Scabies is acquired by close personal contact or through clothing, bedding, and furniture.

The diagnosis can be confirmed by scraping out the mite after mineral oil is applied to a burrow. Keep in mind that in the average scabies patient, there are only 11 mites involved. Thus, the chance of actually finding a mite is low. Plus, this

procedure is very time consuming in a busy clinic and is very low yield, and thus, is not routinely done. Sometimes a biopsy is done.

What causes scabies?

Humans can be infested with a number of different mite types, including bird mites, chicken mites, water mites, and mites from dogs and cats. In scabies, the mite in question is a human mite. This microscopic mite is a tiny, eight-legged creature with a round body. The scabies mite burrows deep within the skin, causing an allergic reaction similar to a small mosquito bite. The result is severe itching, intense enough to keep your patient awake all night long.

Animals can also get scabies, but, in people, scabies is almost always caught from another person who has come into close contact. The contact may be a child, a friend, or other family member. Scabies is not necessarily a condition of low-income families, neglected children, or poor hygiene. No one is immune from posh Beverly Hills to the slums. Attracted to body warmth, the female scabies mite is drawn to a human host, creating her burrows, laying her eggs and producing secretions that cause allergic skin reactions. Scabies larvae hatch from the eggs and then travel to the surface of the skin, laying in shallow burrows where they develop into adult scabies mites.

How to treat?

It may take a month before a newly infested person will notice the bothersome itching. In children, especially babies, scabies can occur anywhere on the body, extremities, or face. Infestation may involve the entire body, including the palms, soles, and scalp.

Explain to your patient that all members of the family should be treated at the same time. Use the scabies medicine. Take the sheets off the bed. Use a fresh towel in the bathroom. All items of bedding and clothing should be washed in hot water or kept in a plastic bag for a month. Spray the mattress with Rid Spray OTC.

Here are helpful tips. Explain to your patients:

Apply Elimite or permethrin 5% cream to dry skin from the neck down on adults. On children apply all over. Leave on for 8 hours on adults, and 6 hours on children or infants, then, wash off. Repeat the same in 1 week.

Ivermectin (Stromectol) oral medication has shown very good efficacy in scabies treatment and is commonly prescribed with permethrin to improve treatment outcome.

6 or 8% Precipitated Sulfur may be safely used on patients younger than two years of age and on pregnant women. Apply daily for three consecutive days.

Wash clothing, bed linens, and towels in hot water. Machine dry after treatment.

Vacuum the entire house and discard the vacuum cleaner bag.

Fumigate your house.

Flea dip your dogs and cats.

What to avoid: Don't attempt to treat scabies by scrubbing with laundry detergent, hard soaps, or by applying kerosene, alcohol, gas, or diesel fuel.

Insecticides like Black Flag or Raid will kill scabies, but can be toxic or poisonous to you.

Don't use steroids or any other creams until after Elimite is tried.

In pregnancy use precipitated sulfur 6% in petrolatum. You may also ask a pharmacist to compound this medicine for resistant scabies.

Apply to the entire body from the neck, down, nightly for three nights, and wash off after 24 hours. If symptoms have not cleared, a second application may be applied one week after the first. Dispense qty one pound.

Finally, scabies can be fatal. In April, 2018, a 93-year-old female nursing home patient in Georgia died of severe untreated scabies. The death certificate read: Cause of death- sepsis secondary to scabies.

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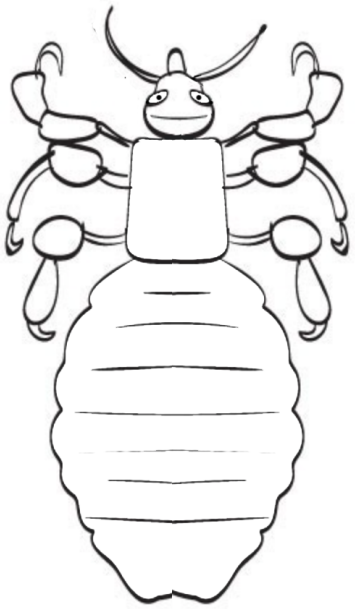
Lice



Lice are wingless nonflying insects and have been infesting humans for thousands of years. They live on human blood, exclusively, and spread by direct contact. Three variants of lice exist: The head louse, the body louse, and the pubic louse. When they eat, lice bite. Their biting causes a localized skin eruption on the host. Lice can transmit other diseases such as typhus, relapsing fever, and trench fever. These infections are rare in the USA.

Body lice are mostly seen in homeless patients and people who live in filth.

Pubic lice occur as a STD mainly seen in young sexually active patients.

Head lice live in the scalp between hairs. They are transferred by person to person contact, and from fomites, such as brushes, combs, pillows, and head rests on commercial airplanes. Lice can also be transmitted by contaminated fingernails. Patients experience severe itch of the scalp and neck.

A single louse has a life-cycle of three stages. The adult female louse lays eggs AKA “nits.” The nits hatch after eight days. The baby louse “nymph” grows to adulthood within twelve days. The adult louse lives for one month and feeds exclusively on human blood. Adult lice are about three millimeters in length and grey. The nits are yellow in color and are one millimeter in size. Without human blood, lice die in two days. Bacterial infections may occur from scratching.

Treatment: We suggest the Nix Complete Lice Treatment Kit available OTC. The Kit contains permethrin medication and educational information to help your derm patients better understand how to treat lice.

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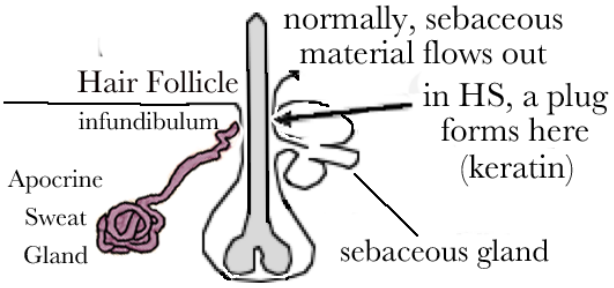
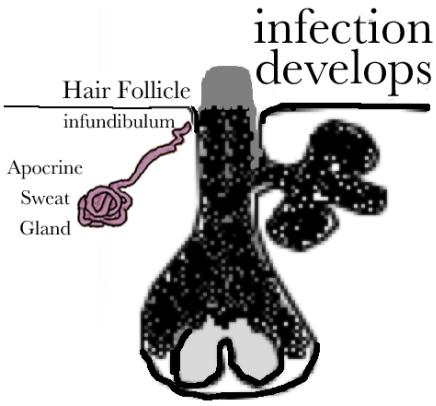
Hidradenitis Suppurativa

Hidradenitis Suppurativa (HS) is a suppurative bacterial infection that originates in the apocrine sweat glands. An apocrine sweat gland is a holocrine gland that empties out by decapitation secretion into the infundibulum of a hair follicle.



Hidradenitis Suppurativa becomes a chronic and smoldering infection with tender boils and lumps. HS extends to the subcutaneous tissue with, painful induration and scarring.



Essentially, as the infection of HS extends, skin appendages are destroyed and tunnels, sinus tracts, and canals develop in the subcutis. The canals and tracts are connected to the skin's surface via fistulae which form in the process. Painful lesions develop wherever apocrine glands are located- especially the axillae, groin, buttocks, anogenital, and inframammary areas.

Hidradenitis Suppurativa (HS) typically begins during or after puberty in fully formed apocrine glands. HS can persist until age 70 or 80 depending on the severity. The incidence of HS is 1 in 100. Women are affected more often than men in a ratio of 3:1, though women do find some relief during pregnancy. Androgen hormones are often high in women with HS, but can also be normal. Overweight patients are more likely to have HS. Also, HS is more commonly associated with metabolic syndrome and hyperglycemia.

The mechanism of HS is thought to be due to hair follicular occlusion with thick keratin. HS is part of the follicular occlusion triad- hidradenitis suppurativa, acne conglobata, and dissecting cellulitis of the scalp. If a pilonidal sinus is present, the complex is referred to as a follicular occlusion tetrad.

The Hurley system is a clinical way to document the severity of HS, and is mostly used by researchers. Hurley system describes three stages:

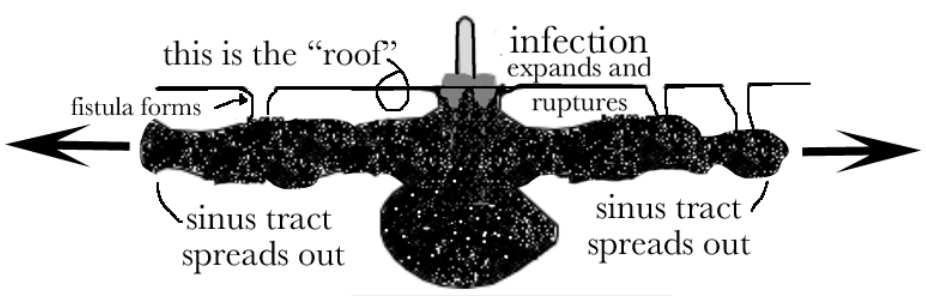
Stage 1: Stage 1 HS is the most common. Patients show single or multiple abscesses without sinus tracts or canals, and without scarring.

Stage 2: Stage 2 HS is characterized by localized abscesses, sinus tracts, canals, and scarring.

Stage 3: Stage 3 HS shows widespread involvement with extensive sinus tracts, canals, and widespread scarring.

Tumor Necrosis Factor

Yes, patients with HS do have a higher level of tumor necrosis factor, with often higher TNF levels than those of psoriasis patients. And yes, anti-TNF therapy works wonders in HS patients. Humira is now FDA approved for treatment of HS.



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Treatment of Hidradenitis

Initially, HS should be treated as a bacterial infection. Staphylococcus lugdunensis is the most common microbial pathogen. Staph aureus, corynebacteria, enterobacteriaceae, and Propionibacterium are also common colonizers. Thus, prior to antibiotics, bacterial cultures should be taken. Then, appropriate antibiotics can be prescribed, directed to the above organisms or those discovered by culture sensitivities.

Overweight patients should be encouraged to lose weight as body folds can rub together and can aggravate HS.

Short courses of oral or IM injectable steroids are sometimes added to the oral antibiotic treatment. Intralesional steroid injections are often helpful.

Topical antibiotics or antibacterial preparations of various kinds can help to prevent flares. These can include Sulfa Cleanse, Hibiclens, topical clindamycin solution, topical erythromicin solution, and benzoyl peroxide preparations.

Accutane and acitretin oral retinoids have been known to help hidradenitis suppurativa.

Surgical procedures consist of simple incision and drainage, to deroofing of sinus tracts, to small or complete surgical excision of the areas.

When to Refer?

Firstly, HS patients should be referred to a dermatologist when the diagnosis is not clear. For example, inflammatory bowel disease, Paget's disease, and shingles can sometimes be mistaken for hidradenitis suppurativa.

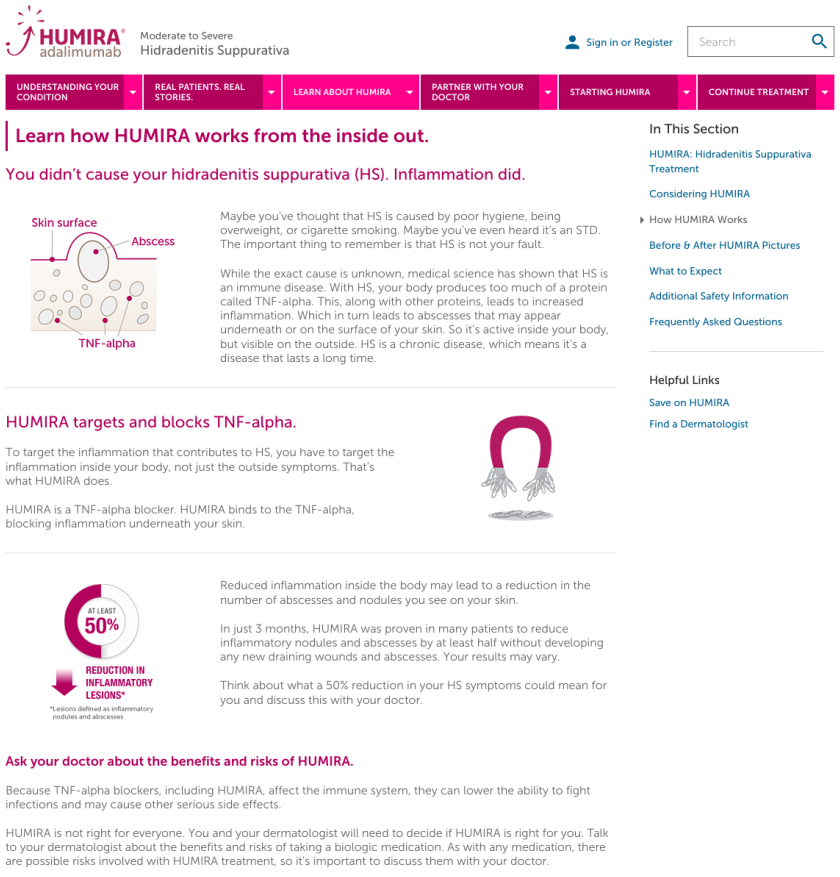
HS patients should be referred to a dermatologist when malignancy is suspected. HS patients are at increased risk of squamous cell carcinomas in the hidradenitis suppurativa involved areas.

HS patients should be referred to a dermatologist for any treatment beyond oral antibiotics and simple I & D. The dermatologist may treat with minor surgical methods or may decide to refer to general surgery for more extensive excisions.

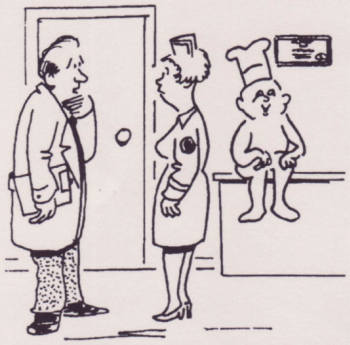
Lesions of HS are known to express five times the amount of TNF alpha compared to normal skin. Thus, Humira is a great choice for the treatment of resistant cases of hidradenitis suppurativa.

Humira is nothing less than a miracle for the treatment of severe hidradenitis suppurativa. HS is the kind of disease in which the patient usually goes from doctor to doctor looking for help. And, Humira is the answer for so many HS patients who have suffered for years in silence. But certainly, a dermatology referral is needed if anti-TNF therapy is indicated: refer for Humira.

HUMIRA adalimumab



Skin Culture



Worst fungus I've ever seen!

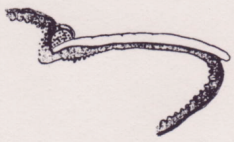
Don't Forget a Skin Culture

More and more, patients are asking about skin infections. Infomercials and magazine ads for antibiotics encourage patients to ask their doctors about fungal infections, yeast infections, herpes, and impetigo. This page is a brief review of only the more common skin infections.

How to accurately diagnose and treat? ---- Take a skin culture.

In our busy world of blood cultures, sputum cultures, throat cultures, urine cultures, stool cultures, and CSF cultures, we often forget about a simple skin culture.

How is it done?



Bacterial Culture: Use a throat swab. Rub the infected area with the swab. Send it to the lab. Try this on pustules, leg ulcers, wounds, abscesses, and impetigo.

Fungal Culture: Scrape small flakes of skin, or cut pieces of toenails. Put pieces in a sterile urine container. Send the container to the lab requesting a fungal culture. Try this on fungal infections of the feet, hands, nails, scalp, arms, legs, face, and trunk.

Yeast Culture: Same as for fungal, but, add a small amount of water to keep moist. Try this on satellite pustules near body folds, the groin, under breasts, and corners of the mouth.

Viral Culture: Requires special media available from the lab. An easier way to check for viral infection is to ask for Viral Ig G and Ig M titers. Elevated Ig M indicates a recent infection. For example, you can request Varicella, HSV, or Coxsakie titers.

AFB Culture: Can be requested with your routine bacterial culture.

Fungal Infections: Treatment Summary

Physical Exam

❏ Classic tinea corporis usually shows a scaled annular plaque. Classic athlete's feet shows interdigital scaling and maceration.

Work-up

❏ Send a skin scraping, hair root sample, or nail clipping to the lab for fungal culture. These may take four to six weeks to grow. Please review the section on how to take a fungal culture.

Topical and Oral Therapy

❏ Topical antifungals can be tried for simple tinea corporis or tinea pedis. Ketoconazole 2% comes in cream form for smooth skin areas and gel form for hairy areas. Use topicals liberally for two or more months.

Note: Simple fungal infections may take 2 to 6 months to improve. If not relieved, or if hair loss, nail loss, or severe disease occurs:

❏ If oral antifungal therapy is needed, order a CBC and Chem 14 to pre-evaluate for oral antifungal therapy. Fungal cultures must be attempted for Lamisil. Document any history of liver disease.

❏ Tinea capitis, unguim, and diffuse tinea usually require oral meds. Tinea capitis usually requires 4- 6 months of oral medication.

❏ Severe tinea of the nails usually requires long term oral antifungal therapy. You can discuss risks and benefits of long term oral antifungal therapy with the patient. Nails usually require 8 months or longer duration of therapy.

❏ You may be familiar with oral antifungal therapy with meds such as griseofulvin or Lamisil. Children require 20 mg per kg per day of Grifulvin therapy. Most adults do well with Grispeg Ultra 250 mg po QD or Lamisil 250 mg po QD. Check labs q month. The most common side effect is mild elevation of liver enzymes.

❏ If you refer to derm, please document all therapies you have tried, lab results, and culture results.

How to Use Salicylic-Acid Wart Medicine

Explain to your patients, or photocopy this sheet for them.

CHEMICAL REMOVAL OF WARTS

Mediplast or Other Sal Acid Wart Medicine and Pumice Stone Method

Here are the steps:

1. At bedtime, put the wart-destroying medicine on the warts.

2. After applying the medicine, cover your warts with adhesive tape. Use the old-fashioned fabric type of adhesive tape. The tape keeps your skin moist. The moisture softens the surface of the warts so the medicine will penetrate.

3. After a few days the outside of the warts will start to turn gray. This means the chemical has begun to destroy them. Scrape this gray wart tissue off with a pumice stone every second or third day. Do the scraping after a bath has softened the wart's surface.

4. Be sure to remove every bit of dead wart tissue; otherwise it will keep the wart-destroying medicine from reaching the living tissue underneath. Sometimes small curved scissors help in removing the dead wart tissue. Whatever you use for scraping your warts should not be used for anything else, because warts are contagious.

5. If the warts become too sore, stop the treatment for a few days.

6. If your plantar warts hurt when you stand or walk, wear a pad cut out of Dr. Scholl's Adhesive Foam (available without prescription). Cut a hole (or holes) corresponding to where the warts are. This will take pressure off.

7. Continue the treatment until you believe the warts are gone. If you can see the lines of your skin crossing the treated area, the warts are probably gone. If it turns out that after you stop treatment the warts are still there, start treating them again until you feel more certain that the warts have gone away.

8. If necessary, continue the treatment for two to four months. If the warts haven't been significantly destroyed after four months of treatment, return for different therapy.

9. In case the warts become excessively painful or infected, return at once to your doctor.

Pediatric Dermatology
Section 5

Dermatology Guidelines for the Primary Care Resident: The Essentials

Dermatology Guidelines for the Primary Care Resident: The Essentials

Pediatric Dermatoses: The Basics

CHILDHOOD EXANTHEMS

Exanthems are a common cause of common generalized rashes in children. Because of a variety of clinical presentations, exanthems pose a diagnostic challenge to even the most experienced primary care physicians and specialist physicians, alike. The morphology, distribution, and associated signs and symptoms are sometimes specific enough for a definitive diagnosis, but, nonspecific clinical findings often make the diagnosis impossible. Advances in laboratory techniques, new antiviral drugs, new vaccines, epidemic outbreaks, and new clinical syndromes have stimulated renewed interest in exanthems. With modern vaccinations, childhood exanthems are now less common; but, if any physician sees a childhood exanthem, it most likely will be a primary care physician. And, parents will want to ask their primary care physician many questions about their child's immunizations. Thus, for primary care residents, we've included this info on childhood exanthems.

Historically, exanthems were numbered in the order in which they were first differentially diagnosed from other exanthems. Thus, "first" disease was measles (rubeola), "second" disease was scarlet fever, and "third" disease was rubella (German measles). The specific disease described as "fourth" disease, the so-called Pilatov-Dukes disease, is no longer viewed as a distinct clinical entity, with some clinicians believing that it represented staphylococcal scalded skin syndrome, and others believing that it was a combined infection with both scarlet fever and rubella. Fifth disease is erythema infectiosum, and sixth disease is roseola infantum. Now, with the identification of parvovirus B19 as the cause of erythema infectiosum and human herpesvirus 6 as the cause of roseola infantum, we have identified the agents of these exanthems.

Measles AKA Rubeola AKA First Disease

Because of recent outbreaks, we have decided to include measles in the Guidelines. Measles is caused by a paramyxovirus RNA virus. The spherical virus particles are approximately 100-200 nm in diameter. Measles virus is highly labile which results in a very short survival time when outside of a host. Measles usually occurs in the winter and spring, although cases can occur all year around. Clinical symptoms are sufficient so that a specific clinical diagnosis can be made in most cases. The incubation period of measles is approximately 1 week. Three forms of measles occur clinically, typical, modified, and atypical.

Symptoms

Measles usually begin like a common cold after a seven to fourteen-day incubation period. You see sinus congestion, a runny nose, a cough, and red, irritated eyes. Two days later, although often unnoticed, Koplik spots (small red spots with bluish-white specks in the center) form inside the mouth opposite the molars. Four days later, a classic rash appears first on the face and neck, then on the trunk, arms and legs. Patients may have some sensitivity to light. After two to four days of general ill feeling, the rash, cough, stuffiness, and red eyes (conjunctivitis) abruptly improve. Measles usually run its course by the tenth day. Measles patients can have lowered resistance to infections such as bronchitis, ear infections, or other bacterial infections. Possible direct complications may include pneumonia and inner ear infections such as otitis media and mastoiditis, which can lead to loss of hearing. Encephalitis, which occurs in one out of 1,000

cases, can result in cognitive retardation. In some cases, corneal ulceration may also occur. Measles virus can be associated with Subacute Sclerosing Panencephalitis (SSPE), a slow virus infection. Slow viruses may remain dormant in humans for extended periods of time, then, may become reactivated. SSPE is a chronic brain disease of children and adolescents that can occur months to years after an attack of measles. SSPE can cause mental deterioration, convulsive seizures, coma and motor abnormalities. Three forms of measles are recognized clinically: typical measles, modified measles, & atypical measles.

Typical measles is the most common form, occurring in patients without immunization. A characteristic prodrome of 2 to 4 days of high fever, coryza, cough, and conjunctivitis virtually always precedes the onset of the exanthem. Koplik spots, the enanthem of measles appear during the prodrome and fade within 2 to 3 days after the onset of rash. These spots, are tiny, white or blue-gray specks superimposed on an erythematous base, located on the buccal mucosa, mostly adjacent to the molars.

The rash of measles begins behind the ears and at the scalp margin, rapidly spreading downward to involve the entire body. Lesions begin as discrete erythematous papules that gradually become confluent. The rash is usually not pruritic. It lasts 4 to 7 days, often with desquamation.

Fever usually begins to decline on the second or third day of the rash unless complications of infection occur. Pneumonia, diarrhea, and otitis media are the most common complications. Other complications include laryngo-tracheo-bronchiolitis, myocarditis, and encephalitis.

Modified measles occurs in partially immune patients, either infants with partial protection through maternal antibody or immunized individuals with partial vaccine failure. In these cases, the prodrome may be shorter and the rash less severe. Koplik spots help greatly in diagnosis, if present, but without them, the rash may be difficult to differentiate from other viral exanthems.

Atypical measles mostly occurred in individuals who became infected after receiving the killed measles virus vaccine which was given until 1967, but a few less severe cases have been reported in children receiving live attenuated vaccine. The abrupt onset of high fever, myalgias, and cough is followed 2 to 5 days later by a rash beginning on the extremities, which gradually spreads centrally. Atypical measles is usually papular or papulovesicular, and lesions are often hemorrhagic. Koplik spots are usually absent. A lobular or segmental pneumonia is usually present, with pleural effusions. Other findings include hepatosplenomegaly and weakness.

The exanthem of atypical measles differs from that of typical measles. The exanthem usually begins as erythematous macules and papules on the distal extremities around the palms, wrists, soles, and ankles. This centripetal pattern also occurs in Rocky Mountain spotted fever and meningococcal sepsis. It then spreads to involve the trunk and face. The lesions may continue in this pattern or progress to vesicles or petechial lesions with purpura. The vesicles appear singly or in crops with erythematous bases, mainly over the trunk, and resemble the rash of varicella.

LABORATORY FINDINGS

Diagnosis of infection is best performed by blood testing. Antibodies first appear 1 to 2 days after the rash, and peak titters are reached 2 to 4 weeks later. Uncomplicated measles infection is usually associated with a leukopenia (low white blood cell count). The leukopenia persists until recovery, after which a mild to moderate leukopenia is observed.

Cause: Measles is caused by a paramyxovirus. The virus infiltrates the nose and mouth (nasopharynx), and measles is a very contagious.

Affected Population: Measles affects males and females equally, and can occur worldwide. Supposedly on the verge of extinction in the United States, from 2001 to 2016 there were 2082 reported cases of measles in the USA.

Measles virus initially gains access to the respiratory tract. By the onset of the prodrome of measles, the virus is widely spread throughout the body. During this time, multinucleated giant cells can be seen in sputum, nasal secretions, and also lymphatic tissues.

Dermatology Guidelines for the Primary Care Resident: The Essentials

Similar Disorders

Disorders similar to measles include: Rubella, or three-day measles. Rubella is characterized by mild symptoms that may result in abortion, stillbirth, or congenital defects in infants born to mothers infected during the early months of pregnancy. Other symptoms may include a two to three-week incubation period with no recognizable symptoms, mild course of short duration, low fever, rash (less extensive than other types of measles), a reddish flush simulating that of scarlet fever which may be noticed on the face, enlargement of lymph nodes, and a normal blood count. Symptoms are usually mild in children with Rubella infection. Adults experience worse fever, discomfort, headache, weakness or exhaustion, stiff joints, and mild rhinitis. Encephalitis is a rare complication that can occur. Testicular pain is also a frequent complaint in adult males. Scarlet Fever is an infection caused by bacteria that usually affect the mouth and throat area (pharynx), but may also affect the skin or birth canal. Patients may experience headache, abdominal pain, nausea, and a skin rash. Rarely, complications are lymphocytic meningitis and hepatitis. A reddish flush may be apparent on the face, chest and extremities, with tiny red spots in some cases.

Roseola Infantum (Exanthem Subitum) is an acute disease of infants or very young children characterized by high fever, absence of localizing symptoms or signs, and appearance of red spots simultaneously with, or following, lowering of the defervescence. The mode of transmission is not known, but Roseola Infantum is caused by a neurodermotropic virus. It occurs most often in the spring and fall. Minor local epidemics have been reported. Atypical Measles Syndrome (AMS) is most common in adolescents and young adults and usually associated with prior immunization using the original killed measles vaccines, which are no longer in use.

THERAPY, COURSE, & PROGNOSIS

Treatment of measles is symptomatic. Antibiotic antimicrobial therapy is recommended when bacterial superinfection has been documented. Complete resolution of the illness usually occurs within 14 days. The prognosis for uncomplicated measles is excellent. Serious illness and death can result from secondary bronchopneumonia and encephalitis. In general, once a person is infected, there is little to do other than let measles run its course, and make the patient as comfortable as possible. The use of aspirin to treat viral diseases in children and young adults should be avoided because of the risk of Reye's Syndrome, a rare but life-threatening condition. Bed rest and a light diet seem to be of benefit. Vaccination for measles is the most effective method found to prevent outbreaks of measles.

PREVENTION

Because measles can be contracted from someone whose symptoms have not yet appeared, it is often difficult to avoid exposure. Measles ceases to be contagious four days after appearance of the rash. Vaccination is the best way to prevent measles.

Scarlet Fever AKA Second Disease

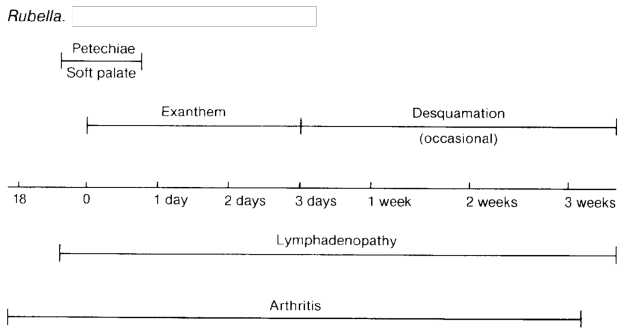
About 10% of children presenting with acute symptoms of streptococcal pharyngitis will go on to develop scarlet fever. Scarlet fever is caused by group A beta-hemolytic streptococci that exude streptococcal erythrogenic exotoxin. scarlet fever patients develop the rash 24-48 hours after the onset of pharyngeal symptoms. The cutaneous scarlet fever eruption begins on the neck, then spreads to the trunk, and finally to the extremities. The rash is characterized by fine, confluent, erythematous, blanching, macules, and rough sandpaper-like papules. There is accentuation of the rash over skin folds; and, an associated lineal petechial eruption "Pastia lines," is often present over the antecubital and axillary folds. There is facial flushing and circumoral pallor. Petechiae on the palate may occur, as well as erythematous swollen tongue papillae with a white coating on the tongue AKA white strawberry tongue. Red strawberry tongue occurs after the white coating. Desquamation begins as the scarlet fever eruption fades, especially on the face, in skin-folds, and on the palms and soles. The scarlet fever rash can last up to six weeks.

Rapid antigen tests are usually used to determine the scarlet fever and streptococcal involvement, and throat cultures may be ordered when there is

suspicion for group A streptococcal infection, and if the rapid antigen test result is negative. Either penicillin, dicloxacillin or erythromycin treatment is curative, and the prognosis is excellent.

Rubella AKA Third Disease

Rubella is also known as German Measles or the Three-Day Measles. Rubella is a contagious viral disease characterized by swelling of lymph nodes and a rash. A pregnant woman infected with rubella during the early months of pregnancy may progress to have an abortion, stillbirth or congenital defects in the infant.



Symptoms

Rubella has a 14- to 21-day incubation period and a 1- to 5-day preliminary phase in children. The preliminary phase may be minimal or absent in adolescents and adults. Tender swelling of the glands in the back of the head, the neck and behind the ears is characteristic. The typical rash appears days after onset of these symptoms. The rubella rash is similar to that of measles, but it is usually less extensive and disappears more quickly. It begins on the face and neck and quickly spreads to the trunk and the extremities. At the onset of the eruption, a flush similar to that of scarlet fever may appear, particularly on the face. The rash usually lasts about three days. It may disappear before this time, and rarely there is no rash at all. A slight fever usually occurs with the rash. Other symptoms such as headache, loss of appetite, sore throat and general malaise, are more common in adults and teenagers than in children. After-effects of rubella are rare among children, although there have been cases of joint pain, sleeping sickness and thrombotic blood clotting problems. Adult women who contract rubella are often left with chronic joint pains. Encephalitis is a rare complication that has occurred during extensive outbreaks of rubella among young adults serving in the armed services. Transient pain in the testes is also a frequent complaint in adult males with rubella.

Cause

Rubella is caused by an RNA virus (probably a toga-virus), and is spread by airborne droplet clusters or by close contact with an infected person. A patient can transmit the disease from 1 week before onset of the rash until 1 week after it fades. Congenitally infected infants are potentially infectious for a few months after birth.

Rubella is apparently less contagious than measles, and many persons are not infected during childhood. As a result, 10% to 15% of young adult women are susceptible if they have not been vaccinated against the disorder. Many cases are misdiagnosed or go unnoticed. Before rubella vaccine was developed, epidemics occurred at regular intervals during the spring. Epidemics occur at about 6 to 9 year intervals. Once infected by rubella, immunity is lifelong.

Erythema Infectiosum AKA Fifth Disease

What causes it? Erythema infectiosum is caused by human parvovirus B19 infection. This virus is also recognized as a cause of transient aplastic crisis in patients with hemolytic anemias, and hemoglobinopathies plus chronic anemia in immunodeficient patients, and an arthritis similar to rheumatoid arthritis, as well as **intrauterine infection and fetal death in infected pregnant women.** The virus can affect all age groups, but the classic exanthem, erythema infectiosum, is most common in school-aged children ages 5 to 15. The incubation period is usually between 4 and 14 days but may last as long as 20 days. The disease is transmitted by respiratory secretions.

How does it begin? First phase: The incubation period of erythema infectiosum is typically between 4 and 14 days, after which 50% of patients have a mild prodromal illness. 50% have mild fever less than 37.8 degrees C, headache, pruritus, malaise, and chills 8 to 10 days after inoculation. This lasts 1 to 4 days. The second phase of illness, shows with a rash and low-grade fever, and begins about 1 week later.

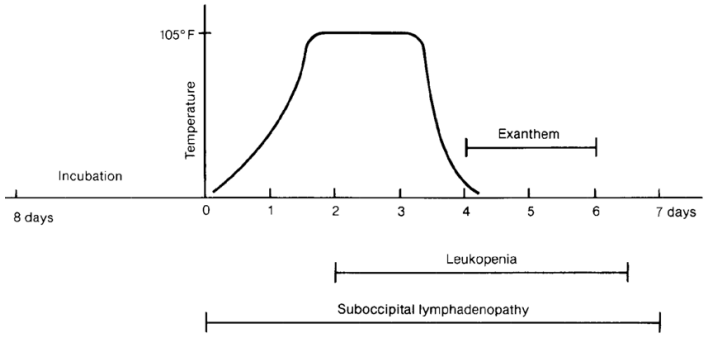
How does it progress? The characteristic exanthem of erythema infectiosum occurs in three overlapping phases. The initial phase is that of an erythematous, raised, and warm rash, prominent over the eminences of the face and often associated with a circumoral pallor. It is referred to as the "slapped cheek" rash and is rarely present in the infected adult. This eruption generally fades in 3 to 5 days. The second phase begins concurrently with the first or up to 5 days later. This symmetric rash becomes most prominent on the extensor surfaces of the extremities and on the buttocks. It may involve the palms and soles. It is characteristically a mildly pruritic, macular, and papular eruption, which clears centrally as it progresses, forming a reticular or lacy pattern. During the third and final stage, the rash may wax and wane. Recrudescence is often associated with sunlight, bathing, exercise, and traumas. Resolution of the rash is typically complete in 1 to 2 weeks. Post inflammatory hyper pigmentation may occur. These dark spots will fade in time.

How is it diagnosed? Fifth disease can be verified by identification of viral particles or by a blood test. Viral particles can be detected by counter-immunoelectrophoresis (CIE) or by the more sensitive DNA hybridization technique in serum, urine, and respiratory secretions during the prodrome. IgG and IgM parvovirus antibodies can be measured by radioimmunoassays or enzyme-linked immunoabsorbent assays. The presence of IgM antibody, or a fourfold increase of IgG antibody indicate a current infection with parvovirus. IgM remains in the serum for approximately 2 months after primary infection whereas IgG persists for years. Furthermore, the finding of IgG is evidence of immunity infection.

How is it treated? In the otherwise healthy person, no treatment is necessary for self-limited erythema infectiosum. Aplastic crisis resulting from parvovirus infection typically lasts 10 to 12 days and may require transfusions. The arthritis, common in infected adults and less common in children, responds to anti-inflammatory therapy. At present, there is no vaccine to prevent infection with parvovirus. Patients with fifth disease are contagious before the rash develops therefore, for susceptible people such as pregnant women and persons with chronic hemolytic anemias, avoidance of persons with viremia is difficult. If an outbreak of erythema infectiosum occurs in a school, it is advisable that all pregnant personnel remain at home for 2 to 3 weeks after the last case has been diagnosed.

Roseola Infantum AKA Exanthem Subitum AKA Sixth Disease

Human herpesvirus 6 causes roseola. Little infants and children less than three years of age are most commonly affected. Roseola appears with sudden onset of high fever for one to five days. During the febrile period, the children usually appear to be in no acute distress with no viral type symptoms and no rash, except for occasional cough, runny nose, and diarrhea. Once the fever breaks, you see the brisk appearance of an erythematous maculopapular rash starting on the trunk and spreading peripherally. The rash of roseola is like that of rubeola AKA measles. To characterize rubeola measles from roseola, the rash of roseola starts on the body while the rash of measles rubeola starts on the face, usually behind the ears, or mouth with Koplik spots, and moves down the body. Pediatric patients with the rash of roseola usually appear well, but those with measles appear rather ill. Roseola is a self-limited viral infection requiring no treatment other than symptomatic supportive care.



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Symptoms

The incubation period before the onset of symptoms of roseola infantum is approximately 5 to 15 days. A high fever begins abruptly and usually lasts for 3 or 4 days without an obvious identifiable cause. Convulsions are common during this period. Low levels of white blood cells (leukopenia) may occur by the 3rd day. The spleen may be slightly enlarged. The fever usually breaks on the 4th day, and a characteristic rash of red spots or elevated (macular or maculopapular) spots appears. This rash may cover the chest and abdomen, although it often appears in a mild form on the face and extremities. Temperature returns to normal at this stage, and the child usually feels and acts well. In some cases, the rash may be go unnoticed.

Factors & Therapy

Roseola tends to occur most often in the spring and fall seasons. Minor epidemics in certain geographic areas have been reported. Once a person is infected with roseola infantum, there is little to do other than let the disorder run its course, and make the patient as comfortable as possible. Medication to bring down the fever may be helpful in serious cases. However, the use of aspirin to treat viral diseases in children and young adults should be avoided because of the risk of Reye Syndrome, a life-threatening condition.

Pityriasis Rosea

About 80% of patients with pityriasis rosea start off with a solitary oval-shaped, salmon rosy colored patch, most commonly starting on the trunk. This is called the “herald patch,” and is usually 2 - 10 cm in diameter and is bordered with peripheral scaling. The herald patch is often confused for tinea corporis. This lesion may persist for several weeks before the rapid spreading of similar smaller lesions usually on the trunk, sparing sun-exposed surfaces. The rash of pityriasis rosea is usually bilateral and symmetrical, and distributed parallel to the lines of Langer in a Christmas tree-like pattern.

Pityriasis Rosea is sometimes misdiagnosed as tinea corporis because of the annular lesions with raised borders, scaling, and central clearing. Tinea corporis is usually a single plaque without eruptions of smaller lesions typical of pityriasis rosea. Pediatric patients may have a history of mild symptoms of upper respiratory infection associated with rash, and about half may complain of itching. Oral lesions are uncommon.

The rash associated with pityriasis rosea may be present for up to 12 weeks, and disappears spontaneously. Treatment is usually symptomatic and supportive. Although the cause is not completely clear, pityriasis rosea is thought to be a viral exanthem, possibly due to human herpesvirus 6 and 7, echo, coxsakievirus, and mumps virus. The differential diagnosis includes of pityriasis rosea includes secondary syphilis.

Impetigo

Impetigo is a gram positive bacterial infection of the epidermis. Primary staph or strep infections occur when the pathogen enters breaks through a damaged disrupted skin barrier, whereas secondary infections develop at the sites of earlier skin conditions which may have been scratched with infectious nails. You can see both bullous forms and nonbullous forms of impetigo. The bullous type is more often seen in newborns and renal patients and is severely contagious. Conversely, the nonbullous type is more common in children ages 2 to elementary years. At one time, *Streptococcus pyogenes* was a common cause of nonbullous impetigo, however, now, *Staphylococcus aureus* has become the more prevalent. And, *Staph pyogenes* may still be the most common cause in warmer moist climates.

Bullous impetigo is most commonly caused by *Staph aureus.* Vesicles, pustules, and thick, yellow crust is classic in the early stages of infection. The lesions are quickly spread by excoriated autoinoculation. Impetigo most frequently occurs on exposed areas of the body such as face and hands. Appropriate antimicrobials are used to cure and prevent.

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Tinea Infection

Tinea is a commonly diagnosed superficial dermatophyte fungal infection of the skin in adults and children that may affect the scalp as tinea capitis, the body as tinea corporis, the groin as tinea cruris, the feet as tinea pedis, the hands as tinea manus, or the nails as tinea unguium. Tinea corporis is diagnosed by one or more circular, sharply circumscribed, erythematous, dry, scaly patches with central clearing like rings, that give them their “ringworm” appearance. The diagnosis is easily made by finding fungus under the microscope in skin scrapings or can be cultured on Saboraud's agar. Mild tinea corporis is usually treated with topical antifungals. More severe infections require oral medication, usually griseofulvin. Please note: Griseofulvin cross reacts with penicillin, and thus, some penicillin allergic patients will need oral terbinafine or fluconazole therapy.

Tinea capitis

Tinea capitis is characterized by dry thick scaling and well circumscribed puffy areas of alopecia and broken hairs, with prominent posterior cervical lymphadenopathy.

Wood’s lamp can reveal fungal fluorescence and is useful as a diagnostic tool. Tinea capitis is also treated with oral griseofulvin or terbinafine and requires a minimum of four months of oral treatment to successfully remove the infection.

Tinea Corporis & Tinea Cruris

Fungal Infections: Body & Groin: Tinea corporis and tinea cruris are fungal infections of the body & groin. When the infection involves the upper thighs or groin, it is called tinea cruris, also known as "jock itch". Tinea corporis can occur in all ages. Sometimes, people acquire the infection from pets like dogs or cats. Sometimes patients can acquire tinea from human contact, or rarely, the soil. The infection usually occurs on the trunk, limbs, or face, tinea face, and the arms or neck. Tinea corporis and tinea cruris can occur anywhere on the body. Tinea cruris can be aggravated by tight clothing, obesity, and warm weather. Humidity tends to promote the growth of dermatophyte fungal organisms.

Tinea Manus & Onychomycosis

Fungal Infection: Hands & Nails: Tinea Manus is a chronic fungal infection of the hands, usually Trichophyton rubrum. Tinea Manus is almost always connected to a pre-existing Tinea Pedis, or fungal infection of the feet. When one sided with tinea pedis, the condition is called Two Foot One Hand Disease. One of the most difficult to treat forms of infection, onychomycosis, destroys the nail plate.

Majocchi's Granuloma

Most tineae infections are superficial, but Majocchi's granuloma is a tinea infection that goes deeper than usual. It is a follicular dermatophyte infection of the deep dermis. Majocchi's granuloma appears as deeply set papules associated with hair follicles. This tinea infection looks like a granuloma.

"Id Reaction"

"Autosensitization dermatitis," also known as "autoeczematization," also known as the "Id Reaction" is a vesicular rash sometimes seen in patients with acute fungal infections of the feet. The vesicular eruption involves the sides of the fingers but can involve the entire body. Some feel that the "Id Reaction" is a systemic allergic reaction to the fungi or some antigen formed during the inflammatory process.

How are tinea infections treated?

Treatment of Tinea corporis can consist of topical antifungal creams or topical lotions such as ketoconazole and clotrimazole, or oral medications. Oral meds include griseofulvin for non-penicillin allergic patients and terbinafine.

CHICKENPOX (Varicella)

Chickenpox is an often mild, but, highly contagious disease. It is caused by Herpes Varicella-Zoster virus (VZV). Currently, thanks to vaccination programs, exposure to someone with active shingles is the most common way for an unvaccinated child to acquire chickenpox. There is no specific antiviral medication, so therapy must be directed towards preventing possible sequellae, rather than curing the disease itself. At first, for 1 or 2 days, there will be fever, abdominal pain, and malaise. Then, skin eruptions will

appear almost anywhere on the body, including mucous membranes. Within 24 hours, the vesicles collapse and are crusted. New crops erupt over a 3 to 4-day period spreading to face and scalp and tending to spare the arms and legs. The mouth, throat, and conjunctivae are sometimes affected with varicella.

Parents need to understand that at any given time their child is likely to have old crusted spots and fresh new red ones, as well as some which are still in the blister stage. The lesions may last for 1 or 2 weeks before detaching and leaving fine scars that may remain for several months. In black children the scars are light and more noticeable because of the contrasting darkness of the surrounding normal skin. These whitish spots may persist for several years. There are variable degrees of severity in chickenpox. At one extreme, the child may have only one or two lesions, detectable only after a deliberate search. On the other hand, there are those children who may be literally covered with the pox, with little uninvolved skin remaining. Most cases of varicella lie somewhere in between.

Our opinion is that a sibling exposed to a family member with chickenpox will be prone to develop a more severe case of varicella, presumably because of a more extended prolonged exposure to the initial case, and a heavier dose of virus. However, this is not always true and seems to depend upon individual characteristics of each child's own response to infection.

Complications and sequellae if there are any, usually develop during the 2 weeks of the actual illness. Scarring is the only permanent effect that may possibly result from uncomplicated chickenpox. The scarring may occur at the site of a particularly large pox lesion or one that becomes secondarily infected with bacteria. The infectious period begins 1 or 2 days before the skin eruptions appear and continues until new eruptions stop appearing. Symptoms of infection in others appear approximately 2 weeks after exposure to the infected person.

Newborns are protected for several months from chickenpox, assuming that they received protective antibodies from a mother who had chickenpox or a vaccination before pregnancy. These antibodies in the newborn diminish and are practically gone in 10 to 12 months. By birthday number one, almost all children are susceptible to chickenpox. One of the most common sources of chicken pox infection in a baby can be contact from a family member with shingles, perhaps a visiting grandparent or parent or cousin, etc.

Chicken Pox: Vaccination

There are two doses of chickenpox vaccine for children, adolescents, and adults. Children should receive two doses of the vaccine—the first dose at 12 to 15 months of age, and a second dose at 4 to 6 years of age. Once infection has occurred, the child develops antibodies, giving almost 100 percent immunity to reinfection by the virus. After chickenpox runs its course, the virus itself goes into the latent phase, remaining for life. Reactivation of the virus later in life is responsible for outbreaks of shingles in dermatomal array.

Chicken Pox: Valtrex

What does Valtrex do? 1. Valtrex decreases the number of chicken pox lesions. The actual number of lesions decrease a little, but all lesions are not eliminated. 2. Valtrex decreases the length of time new lesions remain. 3. Valtrex decreases the number of days children have a fever. What does Valtrex not do? 1. Valtrex does not decrease the sequellae of chicken pox such as secondarily infected skin lesions. 2. As the most infectious period of chicken pox occurs before the rash appears, Valtrex does not make the patient less infectious to others. 3. Valtrex does not lower the patient’s immunity to chicken pox after the infection.

Warts

Warts are common growths on the skin caused by human papilloma virus HPV. An infection with HPV may be clinical, subclinical, or latent. Clinical lesions are those grossly visible on exam. Warts may grow on any part of the skin, where ever a contact occurs: Plantar, usually from a contaminated shower or gym floor, genital, from sexual contact, hands, usually from a contaminated handshake, and more. The most common wart location is on the hands, especially on palms and fingers. And, it makes sense:

Those are areas most often contacted from a HPV donor to an HPV receiver.

Wart lesions range in size from pinpoint to more than 1 cm, and usually present as an elevated, rounded papule with a rough surface showing tiny black dots. A wart takes one to six months to develop after exposure to the HPV virus. HPV is double-stranded, slow growing, naked DNA virus, that contains no envelope. So, it is resistant to drying, freezing or solvents, it is very contagious and is spread by direct person-to-person contact. Autoinoculation is common. When considering treatment, about 65% of warts disappear within two years without any treatment at all. The problem is contagion. If warts are untreated, they will certainly spread to others.

Common questions asked by parents

What Causes Warts?

Warts occur as a result of infection with **h**uman **p**apilloma**v**irus. Unique HPV subtypes cause warts on specific body areas.

Are there different kinds of warts?

Yes. Common warts are thick and rounded with a rough, grayish surface. They are usually found on the fingers, hands, elbows, chest, back, and knees. Plantar warts appear on the soles of the feet and, unlike other warts, grow inward. Flat warts are thin and smooth and are seen most often on the back of the hands and on the face. Genital warts affect the genitalia and rectal area of both men and women. Genital warts on a child may be an appropriate reason to notify a Child Protective Services agent.

Human Papilloma Virus (HPV) has over 75 different subtypes that are associated with various clinical presentations, recognized as: cutaneous warts, which are plantar warts serotype 1, and common warts serotypes 2 and 4. Plantar warts, serotype 1 are found on the soles of feet and tend to be located deeper in the skin and are usually painful. Common warts, serotypes 2 and 4 are found on the hands and fingers. Anogenital warts AKA condylomata acuminata, are recognized as: genital warts serotypes 6 and 11 (benign), and preneoplastic (cervical intraepithelial neoplasia, CIN) which are caused by serotypes 16, 18, 31 and 35. Anogenital warts also cause laryngeal papillomas in sexually active adults and infants delivered vaginally. Over 90% of genital warts are serotypes 6 and 11 (benign). 95% of cervical intraepithelial neoplasia cases contain HPV DNA.

Are warts contagious?
Can their spread be prevented?

Warts are contagious and are usually spread by skin-to-skin contact. Condoms may provide protection against the transmission of genital warts, but only if the condom shields the actual wart from contacting the sexual partner. Children acquire warts by touching other people with warts, usually a playmate. Plantar warts are usually contacted at the gym or from a contaminated shoe, shower, or bathroom floor.

Should warts be treated?

Yes, warts require treatment. Common and flat warts should be removed to prevent multiplication or their spread to other areas. Plantar warts can cause pain and discomfort as they grow and may make walking difficult. Finger warts can destroy fingernails and can spread. Finger warts can also develop into either squamous cell carcinoma or verrucous carcinoma.

How are warts removed?

There are a number of different therapies. Certain therapies may not be FDA recognized. First line of treatment: The wart may be coated with a topical agent, such as salicylic acid. The more potent the sal-acid, the more effective. Electrical destruction by electrodessication is widely used. Warts can also be frozen with liquid nitrogen, surgically scraped, or thermally cauterized by true cautery. Aldara is FDA approved for genital warts. Aldara is not FDA approved for common warts, but many physicians prescribe Aldara for their wart patients, especially for difficult warts of the fingers. Aldara works by stimulating natural interferons. Explain to the patient that Aldara can cause severe irritation, but the irritation will help

to destroy the warts. Also, with Aldara, some patients experience systemic flu like symptoms. If this is too bothersome for your patient, you may need to prescribe something else. For facial warts, tretinoin .025 is not FDA approved, but many physicians do use it, as the irritation tends to be helpful in removing warts from the face, especially flat warts.

Is it possible for warts to return?

Wart recurrence rates may be as high as 45%; As, treatments destroy visible warts, but may not eradicate the entire viral load, which can persist in a latent state. Also, at some point, the virus may begin replicating, causing warts to recur. Until there is a perfect drug to kill HPV, all we can do is treat visible warts and depend on the body's immune system to keep the HPV virus in check.

Prevention

Polyvalent vaccine against HPV 6,11,16,18 is highly effective and is now approved for the immunization of prepubertal girls to reduce the rate of cervical intraepithelial dysplasia in women.

Molluscum Contagiosum

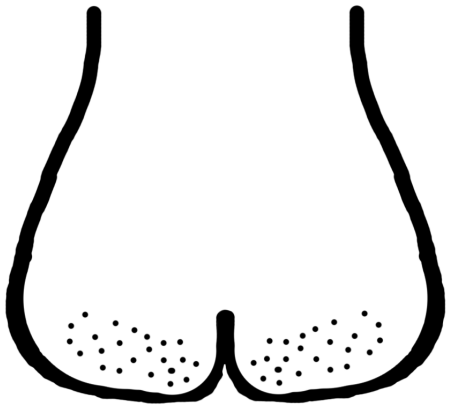
Molluscum contagiosum is a poxvirus skin infection. In adults, Molluscum infection is often a sexually transmitted disease. But can also be due to immunosuppression. Young children are most commonly affected, and most of these are not due to sexual contact. Molluscum may appear solitary or in crops, with a 2 to 7-week incubation period. The classic lesion is a dome shaped pearly papule with central umbilication. Molluscum can last for a few weeks to several months, and occasionally for up to four years. Oral lesions are rare, but molluscum may appear on the genitals and conjunctiva. Resolution is often spontaneous, but sometimes the lesions persist. Molluscum lesions spread by scratching and autoinoculation. New molluscum lesions continue to appear in various places of the body and take quite a long time to resolve. Many pediatricians do not treat them; but, some do. Unfortunately, an infected child can spread the molluscum to many other children. Molluscum is most easily transmitted by direct skin-to-skin contact, especially if skin is wet. So, swimming pools have been associated with molluscum. Because they are highly contagious, most dermatologists do treat molluscum. Please note: There are no FDA approved medications for the treatment of molluscum. Treatment can involve antiviral medications, cryotherapy, imiquimod, cantharidin, Picato, tea tree oil, and topical retinoids. Simple curettage or forceps removal are often the easiest ways to remove molluscum. Secondary infections can occur. Widespread molluscum can also occur in atopic dermatitis.

Diaper Dermatitis

When the wee one is miserable, diaper rash may be the cause. Diaper dermatitis is a lot like intertrigo in its pathogenesis and treatment, because you have a chronically macerated area of skin that develops secondary infection. The differential diagnosis of baby's diaper dermatitis includes the following diagnoses: Langerhans cell histiocytosis, acrodermatitis enteropathica, HIV, and Granuloma Gluteale Infantum. If you are sure that the patient has none of these, then, just like in the intertrigo section, you can proceed to suggest appropriate drying and anti-infective care.

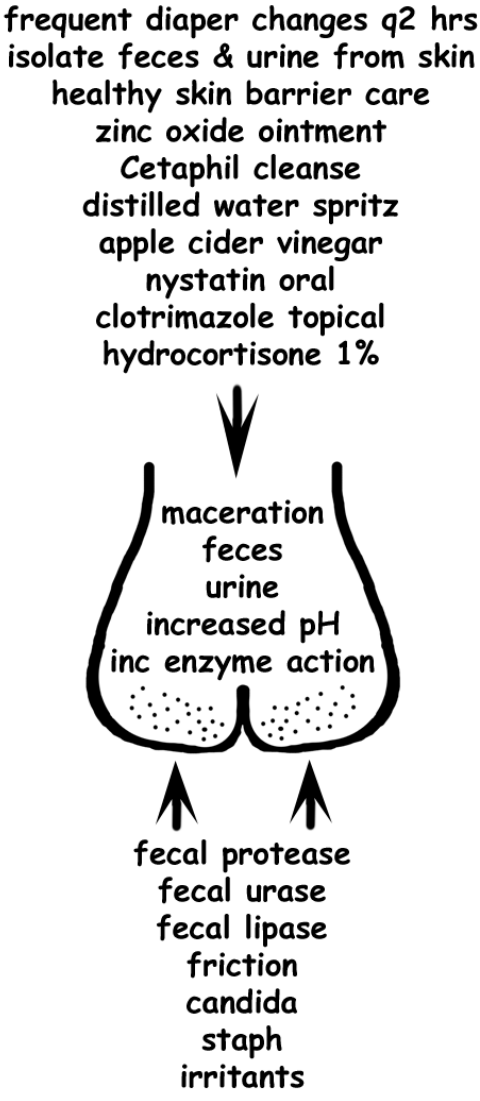
Diaper dermatitis differs from intertrigo in that in addition to an infection, diaper dermatitis also has a significant component of irritant dermatitis. And thus, your goals of treatment should be, both:

1. To decrease baby's exposure to irritants.
2. To strengthen the skin barrier against irritants.



Of note, the main irritants in diaper dermatitis are fecal proteases and fecal lipases. These enzymes irritate the skin barrier so that the barrier loses function and allows the entrance of toxins,

infectants, and allergens into the skin. They also allow the loss of vital filaggrin and natural moisturizing factor. In addition, excess urine greatly elevates the pH, causing even more lipase and protease activity. Remember, an acidic skin surface pH 4.5 – 5 is essential for the maintenance of a normal perineal microflora. To preserve the natural bacterial balance and to promote innate antimicrobial protection against invasion by pathogenic bacteria and yeasts, an acidic pH must be maintained. If there are many unchanged diapers loaded with urine, perhaps a dilute vinegar rinse would help. We suggest one teaspoon apple cider vinegar in a cup of water.



Another point in pH balance: The normal skin pH is between 4.5 – 5.5. Fecal urease breaks-down urine to form ammonia. Prolonged urine wetness leads to maceration of the stratum corneum, and further disruption of intercellular skin barrier lipids. So, regarding diaper dermatitis, what does all this point to? It basically points to the same goals we present for intertrigo. For the diaper area: #1. Prevent and treat maceration. #2. Prevent and treat secondary infections. So, the old derm saying also applies here: "If it is wet, dry it, and if it is dry, wet it."

Just like we approach intertrigo: Let's begin by taking bacterial and fungal cultures of the diaper area. This will help us to identify any specific organisms. Please note: Candida is not normal flora of the baby's perineum; it is fecal in origin. Now, if the diaper area looks candidal with red satellites, and you see a bit of thrush in the baby's mouth, you can prescribe nystatin orally to decrease the candida from the mouth and the entire gastrointestinal tract. Don't worry. Nystatin is not systemically absorbed. For Nystatin Oral Suspension, the dose of Nystatin for infants is 2 mL four times daily. In infants, use a dropper to place one-half of the dose in each side of the mouth, and avoid feeding for 5 to 10 minutes.

Offending bacteria are next on our list. It takes up to seven days to get the culture results back, so, in the meantime, what can you prescribe to start fighting bacteria? In a diaper rash with bacterial overgrowth, staph and strep will be your most common invaders. The FDA warns that antibiotic ointments containing neomycin, polymyxin, or bacitracin should not be used for diaper rash. Mupirocin is not FDA approved for babies less that 3 months of age. Clindamycin lotion can make diaper rash worse. So, we cannot suggest any of these.

Staph aureus, strep, enterobacter, and anaerobes are the most common bacterial infections seen in diaper dermatitis. When it comes to fighting possible staph and strep in a diaper rash in an infant, until a positive bacterial culture report gives you a culture and sensitivity, you can safely suggest only mild non-harmful treatments. For example, amoxiciilin has been shown to increase candida in the perineum, thus, you don't want to prescribe oral antibiotics unless indicated. It is true that zinc and copper ions have antimicrobial activity against Staphylococcus aureus *and* Candida albicans, thus, zinc oxide paste is a great way to begin diaper rash treatment. Also, decreasing the pH can inhibit bacterial growth, so you can suggest one teaspoon of apple cider vinegar in a cup of water. The parent can use this dilute apple cider vinegar to lavage the baby's bottom.

Frequent cleansing of the baby's bottom is fine, and helpful as long as you cleanse the ABC way. Cleansing away fecal proteases and fecal lipases is essential in diaper dermatitis care. A-Avoid

anything allergic. This means avoid diaper wipes. They are loaded with allergens and harsh surfactants. Instead, encourage parents to use Toleriane gentle cleanser to cleanse the baby's bottom. Use nothing scented. B- Bathe to restore moisture. Use Toleriane and rinse with water, then, spritz with distilled water. C- Cover to protect moisture. Apply liberal zinc oxide ointment.

As in any case of irritant dermatitis, skin barrier protection and restoration is essential. So, follow the ABC's of skin barrier care. We suggest that parents protect the baby's skin barrier with zinc oxide ointment applied liberally and frequently.

Keeping the diaper area dry: The question of baby powder. When treating diaper rash in an infant, the concept of baby powder sounds reasonable. Parents have been using baby powder for decades. But, is it safe? The American Academy of Pediatrics educates that **baby powder** can cause lung damage in **babies** if they inhale the particles. This is especially true of talcum based **powders**, with small particles. Talcum powder has also been associated with ovarian cancer. And other facts, cornstarch can actually grow candida. Talcum and cornstarch can both cause aspiration pneumonitis. Thus, to keep lungs healthy, we do not recommend that parents use powders in the treatment of baby's diaper rash.

Rather, to keep the baby's diaper area dry, we suggest more frequent diaper changes. Bottom line, explain to the parents to never let the baby sit around and wait with a wet diaper. With more frequent diaper changes the parent can limit the amount of time the baby's skin is exposed to urine and feces. Parents should change baby's diaper, as often as every 2 hours or sooner if the diaper is used. Exposing baby's bottom to fresh air as much as possible during the day will certainly help. Superabsorbent disposable diapers are best. Tight-fitting diapers should be avoided. Your wee one will thank you!

Finally, you can explain to the parents that they can decrease cutaneous inflammation by using a low potency topical steroid on the affected area. Prescribe hydrocortisone 1.0 ointment to baby's bottom 2 times per day up to 4 times per day if needed. Avoid potent topical steroids such as Mycolog II or Lotrisone. These potent topical steroids will result in atrophy and bothersome symptoms of steroid rosacea. Please take caution with potent topical medications.

Haemophilus influenzae

Haemophilus influenzae usually causes ear, eye, lung, and sinus infections in babies, and can also cause serious soft tissue and skin infections. Haemophilus can lead to sepsis and meningitis or pneumonia. Be on the lookout for erythematous or violaceous swellings of the orbits or cheeks of a baby or young child. Treat aggressively for possible H. influenzae type b.

H. influenzae type b infections have been largely eradicated with the help of immunization programs. However, with worldwide travel, and the increase in unvaccinated immigrants, plus possible vaccine failures, we suggest that you always consider H. influenzae type b infection, as H. influenzae type b can lead to meningitis.

Hair Loss Disorders
Section 6

Dermatology Guidelines for the Primary Care Resident: The Essentials

Dermatology Guidelines for the Primary Care Resident: The Essentials

Hair Loss Basics

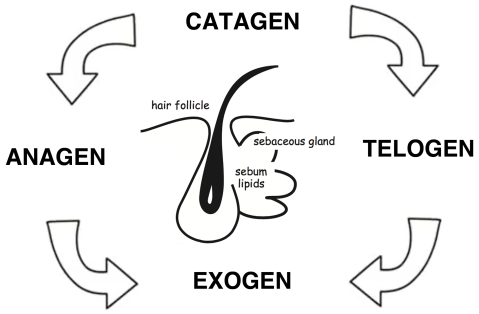
Alopecia is the dermatologic term for hair loss. Human hair grows in a cycle, and, it is normal to lose hair and grow it back during this cycle. You lose, you grow, you lose, you grow... The average non-alopecic human **scalp** has about 100,000 to 150,000 total **hairs**. Normally 80–90% are in growing phase. Unfortunately, hair loss becomes evident when you lose more hair than you grow.

Hair Growth Cycle

First, is anagen, the growing phase. In anagen, hairs grow and grow and grow. Then, in catagen, hairs rest. In telogen, hairs rest in a detached state, then, in exogen, hairs shed, and afterwards return to anagen. Out of 100,000 to 150,000 scalp hairs, it is normal to lose 50 - 100 hairs per day. So, some hair loss is just part of the natural cycle of hair growth. We normally lose an average of 50 to 100 hairs per day, but new hairs grow back to replace the lost hairs.

All hair loss is not the same, and there are various different types of hair loss. The reasons for hair loss can vary from genetics and aging to stress and disease states. Hair loss naturally happens with aging, and is slightly more common in men than women. In addition to this, hair loss can also happen as a result of a major surgery, an acute bout of emotional stress, or childbirth. Hair loss can also be caused by autoimmune conditions, in born genetic tendencies, hormonal conditions, and side effects of medications.

Hair Growth Cycle:



Anagen: Growth phase. Lasts 2-7 yrs. Determines hair length.
Catagen: Transitional phase. Lasts 1-4 months. Papilla detaches.
Telogen: Resting Phase. Lasts 1-4 months. Hair rests detached.
Exogen: Shedding phase. Hair leaves the body. Cycle continues.

Common Types of Hair Loss

Alopecia areata: Alopecia areata is a common autoimmune disease that can begin in childhood or adulthood and can affect people of all ages. In alopecia areata you see yellow dots and exclamation point hairs on the scalp.

Alopecia totalis is a process in which all scalp hairs are lost, resulting in complete balding. Alopecia universalis is a process in which all body hair is lost. In alopecia areata, the affected hair follicles are mistakenly attacked by a person's own immune system, resulting in hair loss. Because the autoimmune process in alopecia areata involves the upper aspects of hair follicles, and not the roots, in alopecia areata the hairs can regrow. In fact, most cases of alopecia areata resolve spontaneously within one year. In contrast, in scarring alopecia, the destructive process is focused around the roots of hair follicles. Thus, blood supply is destroyed and hair loss is permanent with no chance of regrowth.

Trichotillomania: Trichotillomania refers to mechanical loss of hair due to chronic compulsive twisting and twirling of hair, resulting in hair breakage. To prevent permanent hair loss, trichotillomania requires psychological therapy.

Scarring Alopecia: Scarring alopecia is AKA cicatricial alopecia. In scarring alopecia, the autoimmune process occurs around the hair follicle roots. Thus, follicles are destroyed and

hair loss is permanent with no hair regrowth. Two common diagnoses with scarring include frontal fibrosing scarring alopecia and central centrifugal scarring alopecia. Discoid lupus and lichen planopilaris can also cause scarring alopecia. Here are distinguishing features of the scarring:

- Permanent areas of alopecia with loss of visible follicular openings.
- Destruction of viable hair follicles and replacement by fibrous tissue strands.

Scarring alopecia involves potentially permanent and irreversible destruction of hair follicles, and their replacement with scar tissue. To best define the diagnosis, a 4-mm punch biopsy is indicated.

Telogen effluvium: In telogen, it is normal to lose 50 to 200 hairs per day. Telogen effluvium involves a disturbance of the hair growth cycle in which the patient loses > 200 hairs per day. Normally, about 80 to 90% of hair follicles are in anagen AKA the growing phase. In telogen effluvium, certain stressors elicit large numbers of hair follicles to switch from anagen to telogen, and you see diffuse hair shedding. The stressors may include poor diet, vitamin deficiency especially vitamin D, iron deficiency, systemic illness, drugs, childbirth, fever, weight loss, emotional stress, general anesthesia, shock, hemorrhage, and any other major stressor. If the stressor is removed or deficiency corrected, the hair usually grows back within six months.

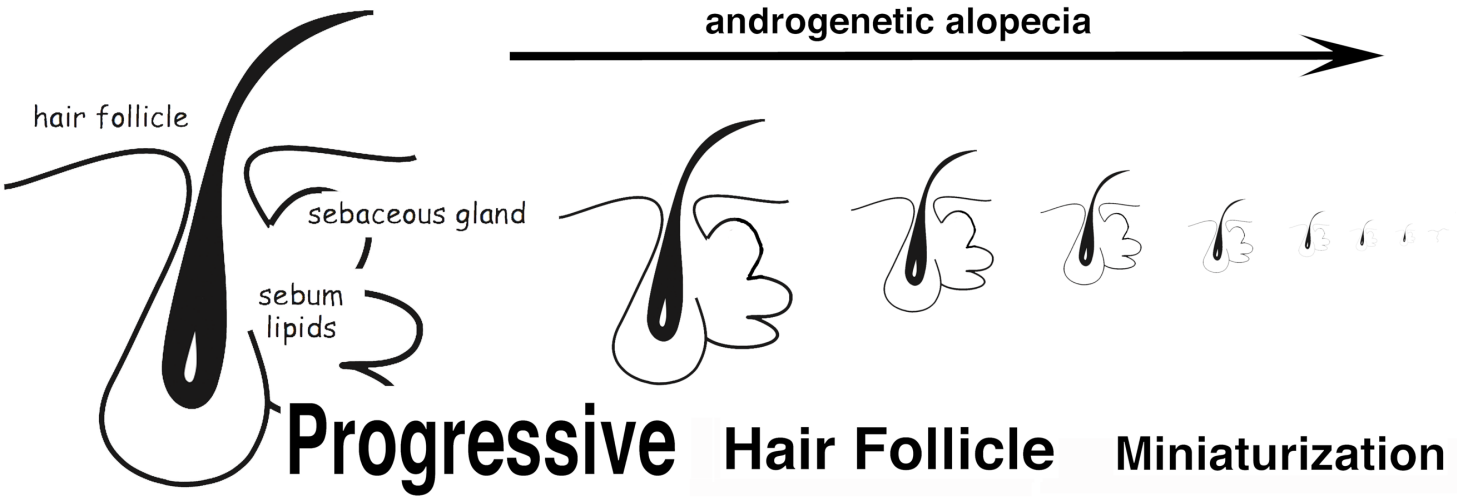
Endocrine Alopecia: Diffuse endocrine alopecia is commonly seen in hypo or hyperthyroid states, pituitary gland deficiencies, hypoparathyroidism, pregnancy, diabetes mellitus, poly cystic ovarian syndrome, and with oral contraceptive cessation.

Androgenetic Alopecia

Male pattern baldness: Male pattern baldness, also known as androgenetic alopecia, affects about 80 per cent of men by age 70. Androgenetic alopecia is due to a slow process known as progressive hair follicle miniaturization. Please see the diagram below. In both male and female androgenetic alopecia, you see variability in the diameters of individual hair strands.

Male pattern balding is better prevented than treated. Once the hair follicles are dead, they are dead and gone. In many cases, it is possible to prevent this hair follicle death. And, prevention is especially important for boys who have a strong genetic link to balding. (Mother's father is bald). If started before hair loss, topical minoxidil can prevent progressive hair follicle miniaturization.

Female pattern baldness: Female pattern balding is also known as androgenetic alopecia in women. Just as males lose hair, so can females. About 40 per cent of women show signs of androgenetic hair loss by age 50. Genetics are involved, as are hormones in patients with poly cystic ovarian syndrome and patients with lower post-menopausal estrogen levels. Interestingly, many women with androgenetic hair loss have normal androgen levels. Minoxidil OTC can help, but takes nine-months and continued use for life. Some patients complain and say, "I will not use minoxidil because once you start it, you have to use it every day the rest of your life." We remind our patients that as humans, we have many "daily duties" such as brushing teeth, using deodorant, daily bathing, eating food, putting on clothes, etc. With androgenetic alopecia, prevention is the best Rx. To better visualize, here is a diagram:



Hair Loss Guidelines

Note: See the hair growth cycle diagram

Please Document the Patient's History

❏ **Genetic:** Any history of female or male pattern balding in the family. Any type of familial hair loss is important.

❏ **Stress:** Any history of significant life stressors in the last two years, such as: Deaths in the family, divorce, auto accident, major surgeries, weight loss, crash diets, emotional trauma, inadequate nutrition.

❏ **Medical:** Any history of trichotillomania, tight braids, syphilis, iron deficiency, lupus, AIDS, syphilis, scalp conditions, anemia, acne, irregular menses, seborrhea, psoriasis, nervousness, diabetes, depression, hormonal problems, menopause, unwanted hair growth in women, alopecia areata, thyroid disorders, autoimmune conditions.

Lab work up for women

❏ Order: CBC, Chem panel, thyroid panel, hemoglobin a1c, ANA, RPR, DHEAS, estrogen, SHBG, free & total testosterone, LH/FSH ratio, 17-Hydroxyprogesterone, as appropriate.

Therapy

❏ Evaluate and treat any positive historical, medical, or lab related findings as listed above.

❏ Try over the counter Rogaine 5% foam for uncomplicated cases, and especially for androgenetic alopecia and cases of life stressors.

❏ Scarring alopecias are best referred. If you refer to derm, please document all therapies you have tried. Please document all pertinent labs, genetic, stress related, and medical history.