

Top Dermatologic Issues in Primary Care

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Disclosure

- I have served as a consultant for Castle Biosciences
- I have served as a consultant for Aegle Therapeutics

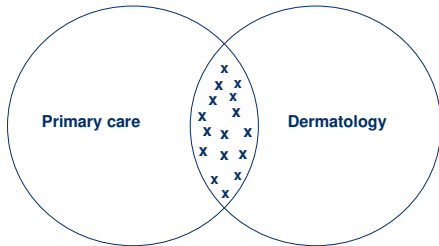


Goals of today's talk

- Emphasize dermatologic diagnoses in primary care setting
- NOT to review the entirety of relevant dermatology
- Emphasize the essential role of a biopsy in making a diagnosis

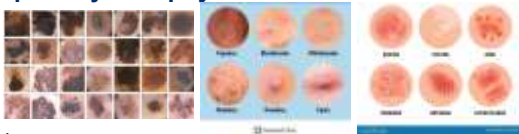


Scope of this talk



Our why:

• “Skin conditions are the most common reason for a new presentation to a primary care physician”*



*Roux E Le, Edwards PJ, Sanderson E, Barnes RK, Ridd MJ. The content and conduct of GP consultations for dermatology problems: A cross-sectional study. *Br J Gen Pract.* 2020;70(699):e723–30.

A quick tour through the world of dermatologic morphology

What are the most common dermat diagnoses in primary care?

Study in 2022: on the National Ambulatory Medical Care Survey (NAMCS) between 2007 and 2016, the most recent years available:

- The NAMCS is an ongoing survey which provides objective information about the use of ambulatory medical services in the United States.
- The survey is conducted annually by the National Center for Health Statistics (NCHS) at the Centers for Disease Control and Prevention (CDC).
- The NAMCS surveys a large, generalizable sample of physicians and non-physician providers and has achieved high response rates of up to 77%.

Ahn CS, Allen MM, Davis SA, Huang KE, Fleischer AB, Feldman SR. The National Ambulatory Medical Care Survey: A resource for understanding the outpatient dermatology treatment. *J Dermatolog Treat.* 2014;25(6):453-458.

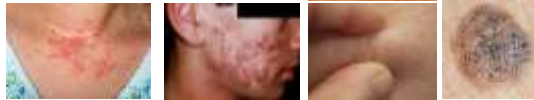
Arafa AE, Anzengruber F, Mostafa AM, Navarini AA. Perspectives of online surveys in dermatology. *J Eur Acad Dermatol Venereol.* 2019;33:511-520.

❖ Grada et al. *J Clin Aesthet Dermatol.* 2022 May; 15(5): E82-E86. 7

The most common skin diagnoses in primary care

• In the population-based, cross-sectional analysis using the National Ambulatory Medical Care Survey between 2007 and 2016:

- The five most common skin diagnoses among all medical specialties were
 - contact dermatitis
 - acne vulgaris
 - actinic keratosis
 - "benign neoplasm" of the skin
 - epidermoid cyst



❖ Grada et al. *J Clin Aesthet Dermatol.* 2022 May; 15(5): E82-E86. 8

Other "Top" Dermatologic Issues for Primary Care

- Identify a skin malignancy
- Identify eczematous, psoriasiform, lichenoid, and drug-induced conditions
- Identify potential autoimmune connective tissue diseases
- Identify autoimmune bullous dermatoses

- Barriers to sampling the skin in primary care
 - Requires proper set up, equipment for procedures, photography/triangulation of lesions, proper sample containers (ex. Michels media for direct immunofluorescence).
- Delay in referral / wait times for patients to be seen by dermatology
- Delay in diagnosis and treatment

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A bit of a deeper dive into

The most common issues

- Acne vulgaris
- Epidermoid cyst
- "Benign" neoplasms of the skin
- Actinic keratosis
- Contact dermatitis

Other top issues

- Cutaneous malignancy
 - Basal cell carcinoma
 - Squamous cell carcinoma
 - Melanoma
- Refractory inflammatory dermatoses
 - Eczematous
 - Psoriasiform
 - Lichenoid
- Autoimmune connective tissue diseases
 - Ex. cutaneous lupus
- Autoimmune bullous diseases
 - Ex. bullous pemphigoid





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Acne vulgaris vs rosacea – diagnosis





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acne





acne



Acne – severe, cystic



Acne

- Multifactorial of pilosebaceous unit
- Psychosocial impact
 - likelihood of self-consciousness, social isolation, anxiety disorders, depression, and even suicidal ideation
- Acne vulgaris affects ~40–50 million individuals each year in the US alone, leading to an estimated annual cost in the US of at least \$2.5 billion
- peak incidence during adolescence, acne affects ~85% of young people between 12 and 24 years of age



Dermatology, Bologna, 5th edition.



Risk factors for more severe acne

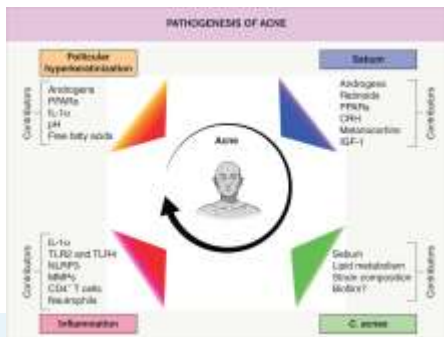
- Individuals at increased risk for the development of acne include:
 - those with an XYY karyotype
 - or endocrine disorders
 - Polycystic ovarian syndrome
 - Hyperandrogenism
 - Hypercortisolism
 - Precocious puberty
- Patients with these conditions tend to have more severe acne that is less responsive to standard therapy

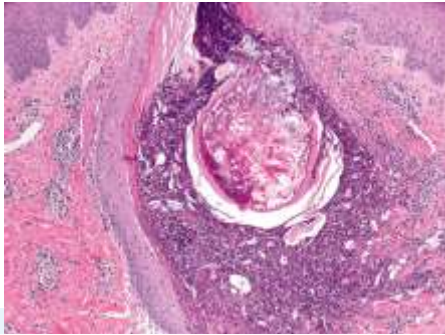


Genes found to have a possible link to acne via genome-wide association studies (GWAS) and other methods include those encoding components of the tumor transforming growth factor- β (TGF- β) pathway, other inflammatory mediators, and regulators of androgen metabolism

Dietary factors for acne?

- The relationship between diet and acne remains controversial.
- Several observational studies in different ethnic groups have found that the intake of milk, especially **skim milk**, is positively associated with acne prevalence and severity.
- Exacerbation of acne with the use of **whey protein supplements for bodybuilding** has also been reported.
- **Vitamin B12 supplementation** can potentially trigger the development of acne or an acneiform eruption by altering the transcriptome of skin microbiota, leading to increased production of proinflammatory porphyrins by **Cutibacterium acnes**.





Treatment of acne

Acne severity	Treatment	Follow-up
Mild: Isolated papules, pustules and/or comedones on forehead. 	First-line Topical retinoid or BPO ± topical antibiotic* or Topical retinoid ± BPO ± topical antibiotic* Alternative options Benzoyl peroxide Clascortene Isotretinoin [†]	For additional control: Increase strength of topical retinoid or Change from retinoid to benzoyl peroxide or Add or replace with another topical agent

Treatment of acne

Moderate: Multiple papules and pustules, few isolated nodules, variable comedones. 	First-line Topical retinoid ± BPO ± topical antibiotic, oral antibiotic [†] , and/or oral hormonal therapy [†] (female patients) Alternative options Benzoyl peroxide Clascortene Isotretinoin [†]	For additional control: See above for topical options Add oral antibiotic [†] and/or hormonal therapy [†] (female patients) or Change to isotretinoin
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Treatment of acne

<p>Severe inflammatory papules and pustules, nodules, nodulocystic acneiform eruption</p> 	<p>First-line Topical retinoid + BPO + oral antibiotic* + hormonal therapy[†] (female patient) or Isotretinoin Consider alternative topical treatment</p>	<p>For additional control: Change to isotretinoin</p>
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Treatment of acne

<p>Keep severe inflammatory nodules with comedones to lowest degree possible, a cystic acneiform eruption</p> 	<p>First-line + second-line (see first table)</p>	<p>Second-line (see first table)</p>
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Birth control and acne

Index of progestin ^a and androgenicity ^b for acne	
INDEX OF ANDROGENICITY (A) AND ANDROGENICITY FOR ACNE	
First-line progestin^a	Lowest androgenicity
FDA-approved oral contraceptives for acne vulgaris	
Drife 30, Coles, Tri-Haloella, Tri-Linda, Tri-Spiron, Tri-Provera, Triamex	Minimal androgenicity (see table)
Estrostep, Miki, Tri-Lignin	Minimal androgenicity (see table)
Tri, Triam, Triamex, Triamex, Triamex, Triamex, Triamex	Minimal androgenicity (see table)
Other oral contraceptives for acne vulgaris	
Amex, Amex, Amex, Amex, Amex, Amex	Minimal androgenicity (see table)
Amex [®]	Minimal androgenicity (see table)
Amex, Amex, Amex, Amex, Amex, Amex	Minimal androgenicity (see table)
Amex	Minimal androgenicity (see table)

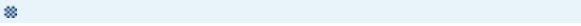
^aOral contraceptives containing norethindrone, norgestrel, or norgestimate.
^bProgestin, only depot formulations, additional progestin, androgenicity containing for medical devices.
^cAmex contains norethindrone acetate for protection against oral contraceptive.

See website for details.



Summary pearls (acne)

- Topical Rx (most common) – benzoyl peroxide, topical clindamycin, retinoid (adapalene, tretinoin, tazarotene)
- Oral Rx (most common) – Doxycycline, spironolactone or isotretinoin
- Prior to referral to dermatology
 - If isotretinoin candidate, discuss abstinence or birth control methods for people who can get pregnant
 - If suspect strong hormonal component, consider referral to endocrinology
 - Polycystic ovarian syndrome, Hyperandrogenism, Hypercortisolism, Precocious puberty

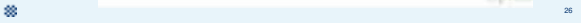


Acne vs Rosacea

ACNE (VULGARIS): Key Features	ROSCEA: Key Features
<ul style="list-style-type: none"> • Most common skin condition affecting adolescents • Prevalence 8-10% in US • Triggers include: stress, diet, lack of sleep, hormones • Acne is characterized by follicular hyperkeratinization, inflammation, and sebum production • Associated with hyperandrogenism (testosterone) and insulin resistance 	<ul style="list-style-type: none"> • Most common skin condition affecting middle-aged adults • Prevalence 10-15% in US • Triggers include: stress, diet, lack of sleep, hormones • Acne is characterized by follicular hyperkeratinization, inflammation, and sebum production • Associated with hyperandrogenism (testosterone) and insulin resistance

KEY DIFFERENCES:

- Acne is characterized by follicular hyperkeratinization, inflammation, and sebum production
- Rosacea is characterized by persistent facial redness, flushing, and papules/pustules
- Acne is associated with hyperandrogenism and insulin resistance
- Rosacea is associated with hyperandrogenism and insulin resistance



Acne vulgaris vs rosacea – treatment

- | | |
|--|--|
| <p>Acne</p> <ul style="list-style-type: none"> - Daily wash with benzoyl peroxide-containing wash (Ex. CeraVe with benzoyl peroxide) or salicylic acid wash - Topical clindamycin solution, gel, or lotion - Daily retinoid (ex. OTC adapalene gel, or tretinoin creams) – a pea-sized amount only across entire face at night - Oral medications: doxycycline 100 mg BID (or minocycline) for up to 1 month, can consider refills for flare-ups - Hormonal driven: start with spironolactone 50 mg daily, increase to 100 mg daily as tolerated (consider checking potassium; warn of side effects; not for use in woman trying to get pregnant) - Also consider topical tretinoin (tretinoin) – androgen receptor inhibitor | <p>Rosacea</p> <ul style="list-style-type: none"> - Start topical metronidazole gel <ul style="list-style-type: none"> - If fails, consider topical ivermectin (Soolantra) - Dermatologist: can perform lasers (example PDL to target hemoglobin in telangiectasias) - Wash with sensitive skin cleansers (Cetaphil, CeraVe, Vanicream, etc). - Can consider long-term, low dose doxycycline 50 mg daily, or 40 mg Oracea (slow-release) - Can consider vasoconstrictors (topical brimonidine – α_2 adrenergic receptor agonist) - Identify and reduce triggers as much as possible (alcohol, spicy foods, heat, stress, etc) - Refer to ophthalmology if ocular involvement |
|--|--|



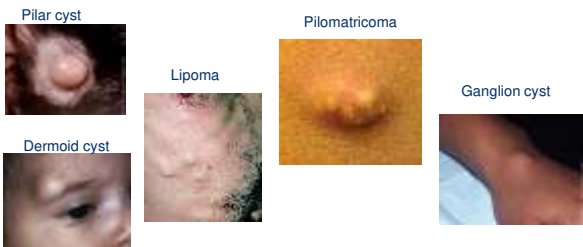
Epidermoid inclusion cysts - diagnosis



Beware of the "cyst" – if deeper with no punctum, it may not be a "cyst"

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Epidermoid inclusion cysts - differential



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Cysts? Unfortunately not.



❖ Pajaziti, L., Hapciu, S.R., Dobruna, S. et al. Skin metastases from lung cancer: a case report. BMC Res Notes 8, 139 (2015). <https://doi.org/10.1186/s13104-015-1105-0> 30

Benign neoplasms of the skin (examples)



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"Pyogenic granuloma" (lobular capillary hemangioma) vs other?



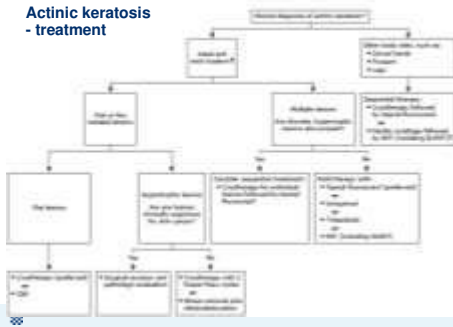
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Actinic keratoses



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Actinic keratosis - treatment



In office treatment with liquid nitrogen

freezing time 5 to 10 seconds or more, depending upon lesion size and thickness, with the "ice ball" extending at least 1 mm beyond the clinical margin of the lesion

single freeze-thaw cycle is adequate for thin lesions, while a double freeze-thaw cycle is required for thicker lesions

Contact dermatitis - diagnosis



- Common contact allergens include plant allergens, metals, fragrances, acrylates, medicaments, and preservatives.

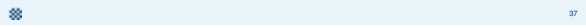
History and geometric distribution are important

Useful resource: Contact Dermatitis Institute (www.contactdermatitisinstitute.com)

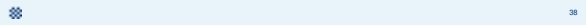
Contact dermatitis – treatment/ avoidance



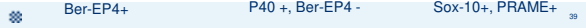
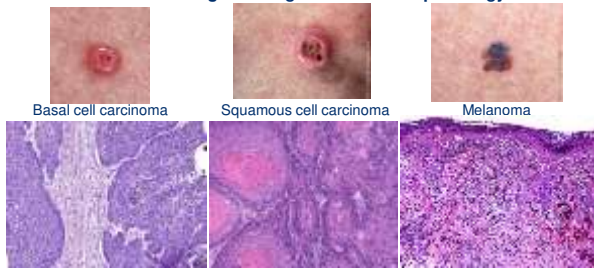
The other "Top" issues



Skin cancer – The "big 3" – diagnosis - clinical



Skin cancer – The "big 3" – diagnosis - dermatopathology



Skin cancer/BCC - treatment

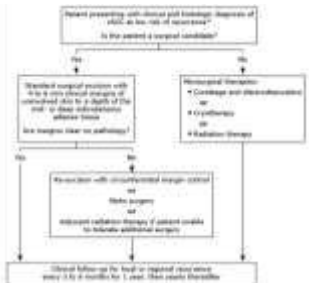


Appropriate Use Criteria for Mohs



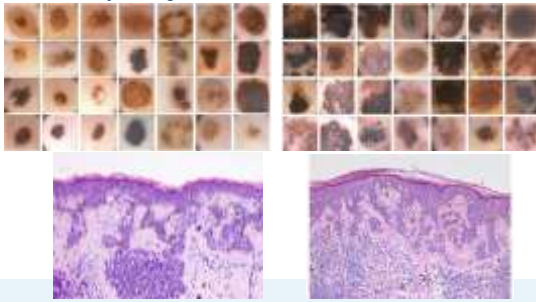
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Skin cancer/SCC - treatment



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The melanocytic diagnostic dilemma



Melanoma- staging

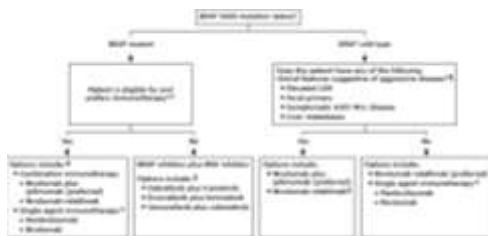
Definition of Primary Tumor (T) - AJCC 8th Edition



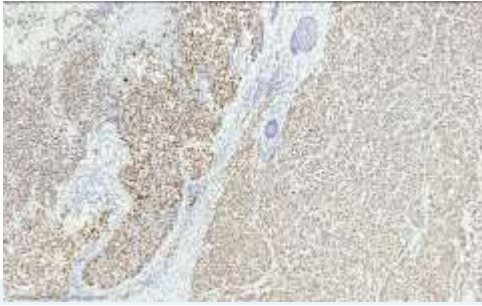
T Category	Thickness	Ulceration status
T0 (melanoma in situ)	Not applicable	Not applicable
T1	≤1.0 mm	Unknown or unspecified
T1a	≤0.8 mm	Without ulceration
T1b	≤0.8 mm	With ulceration
T2	1.0-2.0 mm	With or without ulceration
T2a	1.0-2.0 mm	Without ulceration
T2b	1.0-2.0 mm	With ulceration
T3	2.0-4.0 mm	Unknown or unspecified
T3a	2.0-4.0 mm	Without ulceration
T3b	2.0-4.0 mm	With ulceration
T4	≥4.0 mm	Unknown or unspecified
T4a	≥4.0 mm	Without ulceration
T4b	≥4.0 mm	With ulceration

From: American Joint Committee on Cancer. AJCC Cancer Staging Manual, 8th ed. New York: Springer; 2017.

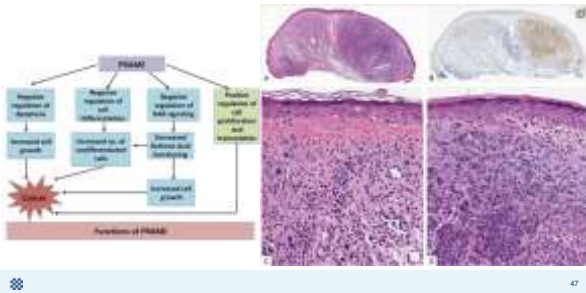
Melanoma- treatment



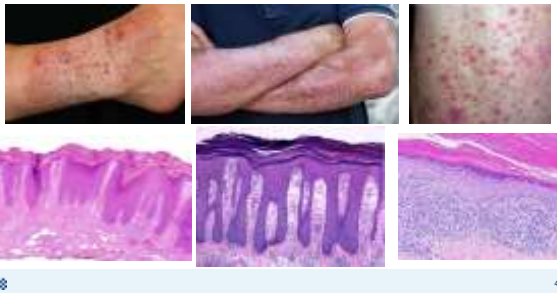
PRAME (PReferentially-expressed Antigen in MElanoma)



PRAME in melanoma



Eczematous vs psoriasiform vs lichenoid - diagnosis



Eczema / atopic dermatitis - treatment

Top pediatric dermatology issues

Chronic or Severe Eczema (Atopic Dermatitis)

Chronic or Difficult-to-Treat Acne

Psoriasis

Chronic Urticaria

Vascular Birthmarks and Hemangiomas

Pigmented Lesions and Nevi

Suspected Skin Infections

Rare or Unusual Skin Conditions:
- Uncommon genetic or autoimmune skin disorders (e.g., epidermolysis bullosa, ichthyosis, lupus).

Alopecia (Hair Loss):

Hyperpigmentation or Hypopigmentation Disorders:

Genodermatoses

Atopic dermatitis

Trimodal distribution in atopic dermatitis

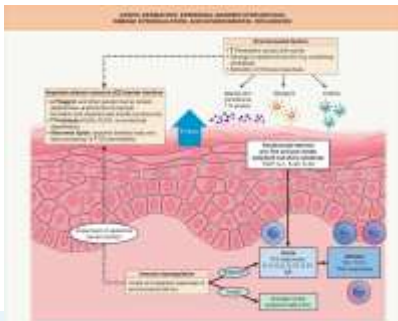
- early-onset AD:
 - defined as AD beginning in the first 2 years of life
 - most common type of AD - first 6 months of life in 45% of affected individuals, during the first year of life in 60%, and before 5 years of age in 85%
 - Approximately half of children with disease onset during the first 2 years of life develop allergen-specific IgE antibodies by 2 years of age
 - About 60% of infants and young children with AD go into remission by 12 years of age, including a group with resolution by 4-6 years of age
- late-onset AD: starts after puberty
 - Approximately 30% of AD patients overall are in the non-IgE-associated category
- AD in the elderly: a subset of AD that begins after 60 years of age



Atopic dermatitis (“eczema”)

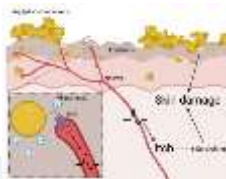
- Atopic dermatitis (AD) is the most common chronic inflammatory skin disease, and its increasing prevalence presents a major public health problem worldwide
- Characteristic features of AD include pruritus and a chronic or chronically relapsing course, usually beginning during infancy (early onset) but occasionally first developing in adulthood (late onset)





Staph in atopic dermatitis

- IL-31 is a Th2 cytokine that is highly expressed in lesional skin and serum of patients with AD as well as in other pruritic skin disorders such as prurigo nodularis.
- Cutaneous exposure to staphylococcal superantigen rapidly induces IL-31 expression in atopic individuals, establishing a link between staphylococcal colonization of the skin and pruritus.
- The heterodimeric receptor for IL-31 is expressed by keratinocytes, eosinophils, activated macrophages, cutaneous C nerve fibers, and dorsal root ganglia
- *Staphylococcus aureus* *colonization* of the skin affects lipid composition and contributes to epidermal barrier impairment
- The *S. aureus* extracellular V8 protease, which has a sequence similar to those of *S. aureus* exfoliative toxins, is also thought to degrade Dsg1



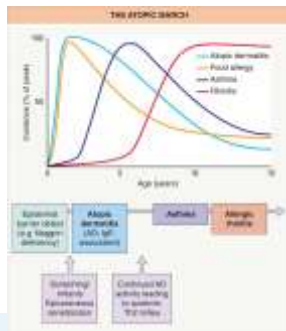
Microbiome in atopic dermatitis

- More than 90% of patients with AD have skin colonized with *S. aureus*, compared to about 5% of unaffected individuals, presumably reflecting the disrupted acid mantle, decreased antimicrobial peptides (e.g. cathelicidins, defensins), and altered cytokine milieu of AD skin.
- During AD flares, bacterial diversity decreases and the proportion of the microbiome accounted for by *Staphylococcus* spp. increases from ~35% to ~90%. Conversely, normalization of the microbial population correlates with clinical improvement in AD.
- Superantigens can promote the development of a Th2 immune response, and exotoxins with superantigenic properties are produced by up to 65% of the *S. aureus* strains that colonize AD patients.
- Compared to unaffected controls, an IgE response to the *S. aureus* superantigens enterotoxin A and enterotoxin B occurs more frequently in patients with AD. The *S. aureus* δ -toxin also stimulates mast cell degranulation and Th2 inflammation.



Microbiome in atopic dermatitis

- In addition, flaggrin deficiency increases the susceptibility of keratinocytes to *S. aureus* α -toxin-induced cytotoxicity. Lastly, *Malassezia* spp. may also contribute to inflammation in AD, and adults with severe head and neck disease often display IgE reactivity to *Malassezia* antigens.
- Alterations in the skin microbiome of AD patients related to the use of cleansers and topical immunomodulatory or antimicrobial agents may have potential effects on cutaneous inflammation and barrier function.
- Topical administration of coagulase-negative *Staphylococcus* strains with antimicrobial activity or the Gram-negative commensal *Roseomonas mucosa* has been shown to markedly reduce *S. aureus* colonization in AD patients.
- R. mucosa* application was also associated with decreased AD severity and topical corticosteroid requirement, providing the basis for bacteriotherapy as a potential AD treatment.
- In addition, treatment with UVB has been shown to reduce *S. aureus* colonization of the skin in AD patients.



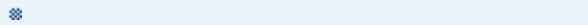
Genetics and AD

SELECTED CANDIDATE GENES FOR ATOPIC DERMATITIS	
Candidate gene(s)	Effect(s) on pathogenesis
Genes encoding epidermal proteins	
<i>FLG</i>	Flaggrin (loss-of-function variants, see 100)
<i>FLJ2</i>	Flaggrin family member 2
<i>ITPA</i>	Human protein inhibitor of LTAI
<i>KL1</i> / <i>KL2</i> / <i>KL3</i> / <i>KL4</i> / <i>KL5</i> / <i>KL6</i>	Keratins (select keratins 5, 6, 16/18/20/24/25/29) (see 2)
<i>CHST2</i>	Chondrin 2
<i>SCN7A</i>	Small proline-rich protein 2
<i>TNFRSF10</i>	Tumor necrosis factor receptor 10
<i>KLHL3</i>	Keratin family member 31
Genes encoding immunologic proteins	
<i>FCER1A</i>	Fc fragment of high-affinity IgE receptor 1 α -chain
<i>CCR1</i> , <i>CCR2</i> , <i>CCR3</i>	Chemokine receptor 1, 2, 3 (see 5)
<i>IRF1</i>	Interferon regulatory factor 1
<i>IL4</i> , <i>IL13</i> , <i>IL4</i> , <i>IL13</i>	Interleukin 4, 5, 13 (see 13, 18, 200-2)
<i>IL18R1</i> , <i>IL18R2</i> , <i>IL18R3</i>	Interleukin 18, 18, 18 receptor 1, 2, 3
<i>LRN</i> / <i>CDP</i>	Cleavage of pro-matrilin during vitronectin binding
<i>CD4</i>	Receptor for Mycobacterium tuberculosis CD4
<i>IL27RA</i>	IL-27 receptor α



Genetic associations with atopic dermatitis (continued)

FCER2	High-affinity Fcε receptor 2
CMA4	Blau and atopy gene
CYA11/RANTES	Chemokine (C-C motif) ligand 11/RANTES
TSLP	Thymic stromal lymphopoietin
CARD34, CARD14	Caspase recruitment domain family member 11 and 14
IL27	Interleukin 27
ITP1	Intercellular adhesion molecule-1 inhibitor 1
VDR	Vitamin D receptor
CYP24A, CYP24B	Cytochrome P450 family members 24A and 24B



Common presentation in atopic dermatitis



Variation in clinical presentation



Atopic dermatitis



Chronic atopic dermatitis, lichenification



Atopic dermatitis - lichenification

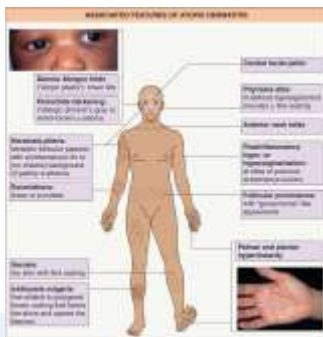


Variation in atopic dermatitis



Other common sites of involvement





Triggers/exposures in atopic dermatitis

Triggers

- Climate: extremes of temperature (drier or warmer), low humidity
- Diet: acids, acid/rough fibers, perspiration, detergents, soaps
- Fungal/yeast infections (e.g. *Staphylococcus aureus*, *Molluscum contagiosum*, *Tinea ringworm*) or scabies (e.g. *SAR*)
- Environmental allergens: e.g. to dust mites, pollen, contact allergens
- Food allergies
- Trigger in small minority of AD patients, e.g. 20%–30% of those with moderate to severe, refractory AD
- Common allergens: egg, milk, peanuts/tree nuts, fish/shellfish, soy, wheat
- Detection of allergen-specific IgE (in blood and skin prick tests) does not necessarily mean that allergy is triggering the patient's AD



Keratosis pilaris co-existence with atopic dermatitis



Pityriasis alba



Superinfection

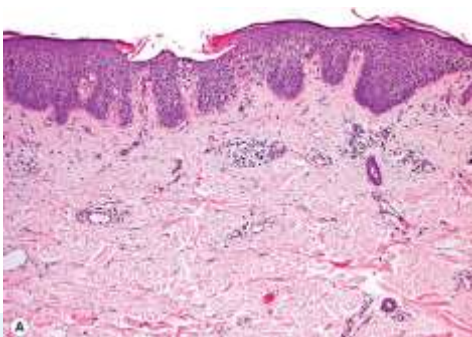


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Eczema herpeticum



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MANAGEMENT PLAN FOR ATOPIC DERMATITIS (AD)

Treatment of active eczema
Daily use of topical CS of appropriate strength and frequency should begin -> Avoid a period higher potency (class 1-3) for flared period of time, symptoms return, or recovery at the flare/flare

Highest maintenance to avoid "hot spots"
Increased use of mid-potency topical CS (e.g. 2 treatments daily) until control is achieved (e.g. 5.0-10.0%)

Lowest maintenance and patient safety
Daily use of low-potency topical or systemic CS if still flared

For acute flares consider wet wraps following CS application

F = Flare
CR = Topical corticosteroid of appropriate strength/daily
+ = Maintenance - not topical CS (e.g. dupilumab, oral CS or T1/2-0-Approved, emollient wrap)
 — Flare
 — Clear
 — Flare



THERAPEUTIC LADDER FOR ATOPIC DERMATITIS (AD)	
Topical therapy	1st line
Moisturizers	A
Corticosteroids	B
Calcineurin inhibitors	C
Crabapple	D
JAK inhibitors e.g. ruxofinib (FDA-approved), abrociclib (pending approval)	A
Biologics	
Interleukin 17A (IL-17A) (TYK2, TYK2, TYK2)	B
Interleukin 4/13 (IL-4/13) (Dupilumab)	1st line
Small molecules	
Hydroxyethylamine (HEH)	A
IL-2 inhibitors, e.g. tralokinor, bimekizumab	B
JAK inhibitors, e.g. upadacitinib, filgotinib, tofacitinib	C
Cyclosporin (short-term use only)	A
Acupuncture	D
Mycophenolate mofetil/everolimus/rapamycin/everolimus	D
Moisturizers	D



Systemic corticosteroids (short term for severe acute flares, "rescue" treatment when flare severe again (avoid chronic))	A
Dupilumab	1st line
Interleukin 13 (and IL-4) receptor A, not currently FDA approved	2
Hydroxyethylamine	B
Interleukin 17A	C
TYK2	1st line
Biologics	
Wet wraps, open wet dressings, or occlusive treatment with topical corticosteroids for acute flares	
Other occlusive dressings (band-aids) help	
Treatment of associated bacterial, viral, or fungal infections	
Oral antihistamines for associated conditions (e.g. sleep disruption, (hives/angioedema)) and sedative effects	
Lactation management?	
Breastfeeding options beyond or not?	

Do not use topical corticosteroids if a patient is breastfeeding, but dupilumab is safe because of oral and topical use.

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My checklist in atopic dermatitis treatment

- 1. Skin cleanser
- 2. Detergent type
- 3. Anti-staph/bacterial strategy; "microbiome" strategy
- 4. Anti-inflammatory strategy
- 5. Emollient strategy
- 6. Anti-itch/pruritus strategy



Psoriasis



Lab: CBC, CMP, Hep panel, Quant TB

Biologics

- Etanercept (anti-TNF): Approved for people 4 years and older
- Ustekinumab (anti-IL-12/23): Approved for people 12 years and older
- Secukinumab (anti-IL-17): Approved for people 6 years and older
- Ixekizumab (anti-IL-17): Approved for children 6 years and older
- FDA Approves Acute: ZORYVE® (roflumilast) Cream 0.3% for Treatment of Psoriasis in Children Ages 6 and older

Chronic Urticaria

Lab: IgE levels, tryptase levels

Vascular Birthmarks and Hemangiomas

Pigmented Lesions and Nevi

Alopecia (Hair Loss):



Alopecia areata

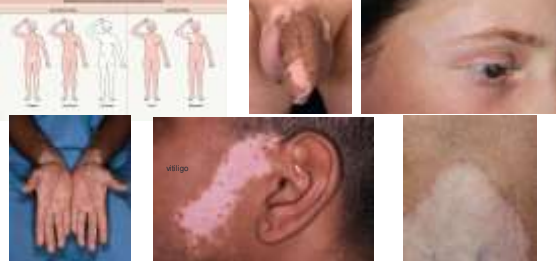
JAK inhibitors are making waves. Ritlecitinib (Liftuvo) has been drastic in improving in our patient population. *Beware of syphilis



trichotillomania

Discoid Lupus

Pigmentation disorders



vitiligo

ruxolitinib cream or by its brand name Opzelura, is the first and only FDA-approved topical JAK inhibitor for treating vitiligo in people ages 12 and older.



Linear nevoid hypopigmentation

Nevoid depigmentous



Psoriasis

Atopic dermatitis

Seb derm

Neonatal lupus

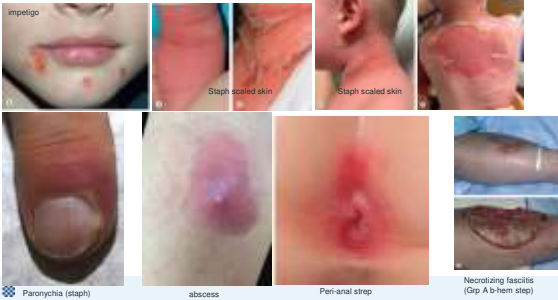
Lichen striatus

Discoid lupus

Pityriasis lichenoides chronica



Cutaneous infections



impetigo

Staph scaled skin

Staph scaled skin

Paronychia (staph)

abscess

Peri-anal strep

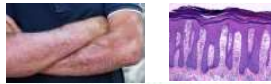
Necrotizing fasciitis (Grp A b-hem strep)

Overall pearls prior to referral

- History in referral (duration, associated symptoms, previous treatments, family history)
- Photography in referral (far away and close up); measurement of sizes of lesion serves as comparison and can help determine growth
- Treatments: including OTC or at-home remedies, along with Rx treatments
- Consider hormonal imbalance as contributing factors (endocrinology)
- Birth control discussion prior to isotretinoin referral vs abstinence



Psoriasis – treatment - biologics



Agent	Drug Classification	Indications	Usage	Major Side Effects	Common Laboratory Tests	Warnings - Precautions and Long-term Risks
Adalimumab	Tumor Necrosis Factor (TNF) Inhibitor	Plaque psoriasis, Psoriatic arthritis, Ankylosing spondylitis, Axial spondyloarthritis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
Etanercept	Tumor Necrosis Factor (TNF) Inhibitor	Plaque psoriasis, Psoriatic arthritis, Ankylosing spondylitis, Axial spondyloarthritis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
Infliximab	Tumor Necrosis Factor (TNF) Inhibitor	Plaque psoriasis, Psoriatic arthritis, Ankylosing spondylitis, Axial spondyloarthritis	Injection (IV)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
Secukinumab	Interleukin-17 (IL-17) Inhibitor	Plaque psoriasis, Psoriatic arthritis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
IgA1	Interleukin-23 (IL-23) Inhibitor	Plaque psoriasis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
Guselkumab	Interleukin-23 (IL-23) Inhibitor	Plaque psoriasis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
Risankizumab	Interleukin-23 (IL-23) Inhibitor	Plaque psoriasis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding
Tremfya	Interleukin-23 (IL-23) Inhibitor	Plaque psoriasis	Injection (IM)	Headache, Rash, Infection, Tuberculosis, Heart failure, Liver enzyme abnormalities	None	Contraindications: Active tuberculosis, Hepatitis B, HIV, Active malignancy, Pregnancy, Breastfeeding



<https://www.skintherapyletter.com/psoriasis/education-tool-biologics/>

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Psoriasis – treatment - biologics

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<https://www.skintherapyletter.com/psoriasis/education-tool-biologics/>

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Drug-induced lichenoid dermatitis – treatment

1. Eliminate potential drug causes

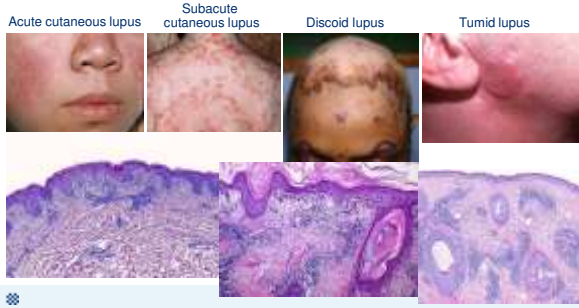


2. Topical steroids

3. Wide range of immunosuppressives

Group of drugs	Associated drug-induced lichenoid dermatitis
Antibiotics (e.g. beta-lactams)	Amoxicillin, dicloxacillin, cephalexin, erythromycin, penicillin V, tetracycline, vancomycin
Antidepressants (e.g. SSRIs)	Escitalopram, fluoxetine, paroxetine, sertraline
Antiepileptics	Carbamazepine, phenytoin, sodium valproate, topiramate, lamotrigine, levetiracetam
Diuretics	Hydrochlorothiazide, furosemide, bumetanide
Anticancer drugs	Cyclophosphamide, docetaxel, paclitaxel, vinorelbine, irinotecan, gemtuzumab, gemtuzumab, gemtuzumab, gemtuzumab
Anticoagulants	Warfarin, dabigatran, apixiban, rivaroxaban, edoxaban
Cardiovascular drugs	Losartan, lisinopril, valsartan, amlodipine, nifedipine, diltiazem, verapamil, digoxin, digoxin
Antipsychotics	Risperidone, haloperidol, amisulpride, levamisole, levamisole, levamisole, levamisole
Antifungals	Fluconazole, voriconazole, isavuconazole
Antibiotics	Amoxicillin, dicloxacillin, cephalexin, erythromycin, penicillin V, tetracycline, vancomycin
Antidepressants	Escitalopram, fluoxetine, paroxetine, sertraline
Antiepileptics	Carbamazepine, phenytoin, sodium valproate, topiramate, lamotrigine, levetiracetam
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Antipsychotics	Risperidone, haloperidol, amisulpride, levamisole, levamisole, levamisole, levamisole
Antifungals	Fluconazole, voriconazole, isavuconazole

Autoimmune connective tissue disease - diagnosis

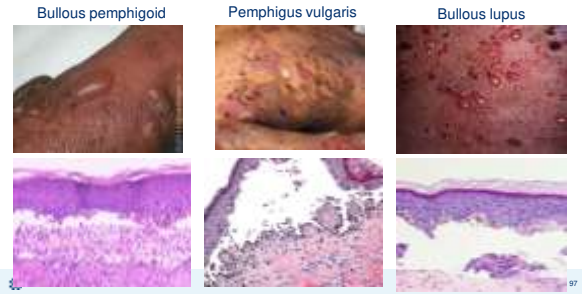


Autoimmune connective tissue disease - treatment

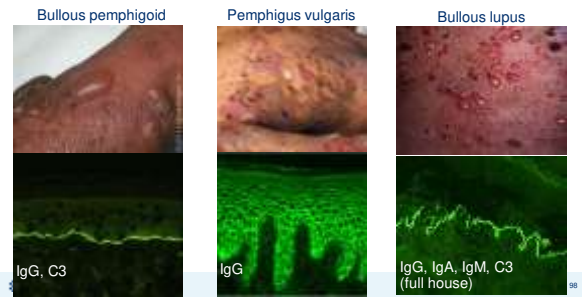
Management of discoid lupus erythematosus and subacute cutaneous lupus erythematosus in adults



Autoimmune bullous dermatoses, examples - diagnosis



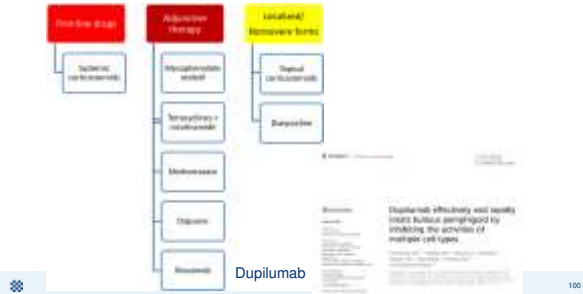
Autoimmune bullous dermatoses, examples - diagnosis



Mediators implicated in drug-associated bullous pemphigoid

Autoantigen	Antibody associated	Cellular mechanism
BP180	Anti-BP180	Direct cytotoxicity
BP230	Anti-BP230	Direct cytotoxicity
BP230-1	Anti-BP230-1	Direct cytotoxicity
BP230-2	Anti-BP230-2	Direct cytotoxicity
BP230-3	Anti-BP230-3	Direct cytotoxicity
BP230-4	Anti-BP230-4	Direct cytotoxicity
BP230-5	Anti-BP230-5	Direct cytotoxicity
BP230-6	Anti-BP230-6	Direct cytotoxicity
BP230-7	Anti-BP230-7	Direct cytotoxicity
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BP230-45	Anti-BP230-45	Direct cytotoxicity
BP230-46	Anti-BP230-46	Direct cytotoxicity
BP230-47	Anti-BP230-47	Direct cytotoxicity
BP230-48	Anti-BP230-48	Direct cytotoxicity
BP230-49	Anti-BP230-49	Direct cytotoxicity
BP230-50	Anti-BP230-50	Direct cytotoxicity

Treatment – autoimmune bullous disease – BP as an example



Basic dermatologic procedures

Shave biopsy

Punch biopsy



Types of Biopsies and Indications

<ul style="list-style-type: none"> - Pedunculated lesions (skin tags) - Dome-shaped nevi - NMSC (BCC/SCC) - Pigmented lesions (ruling out melanoma) 	<ul style="list-style-type: none"> - Connective tissue diseases (Lupus/ Dermatomyositis) - Papulosquamous disorders (psoriasis) - Blistering disorders (pemphigus) - Granulomatous diseases (sarcoid) - Vasculitis (HSP) - NMSC (infiltrating tumors) 	<ul style="list-style-type: none"> - Subcutaneous or deep dermal tumors (can do a "punch-within-a-punch") - Panniculitis (also "punch-within-a-punch") - Melanoma - Atypical pigmented lesions



Biopsy Site Selection

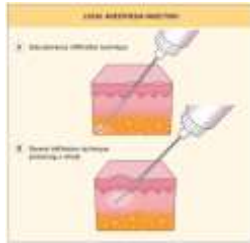
BIOPSY SITE SELECTION	
Lesion/Condition	Appropriate site
Tumor	Thickest portion; avoid necrotic tissue
Bleeder	Edge of lesion, including perilesional skin (see Fig. 0.11)
Ulcerated/necrotic lesion	Edge of ulcer or necrosis plus adjacent skin
Generalized polymorphous eruption	Characteristic lesion of recent onset (i.e. more developed lesion)
Small vessel vasculitis	Characteristic lesion of recent onset

Patient Preparation

- Determine the type of biopsy
- Informed consent: bleeding, discomfort, infection, and scarring
- Site preparation:
 - Identification and marking
 - Time Out
 - Photograph
 - Close up for lesional details
 - Distant for identification of landmarks

Anesthesia Techniques

- Lidocaine 1% with or with out epinephrine
- Small lesions: direct infiltration of anesthetic into lesion
- Larger lesions: a field block by placing a ring of anesthesia around surgical site
- Bevel up
- Use small gauge needle (30), insert quickly at a 45° angle
- Slow injection to create an intradermal wheal, then may proceed to subcutaneous injection depending on shave vs. punch
- Additional sticks should be done through areas that are already numb
- Use smaller syringes – require lower pressure for injection
- Warm anesthetic to body temperature
- Slow injection
- Verbal and tactile distraction



❖ Bolognia et al. Dermatology

Patient Preparation Continued

- Prep
 - ETOH swab
 - Iodine
 - Chlorhexidine
- Anesthesia
 - Plane of injection
- Procedure
 - Hemostasis: Aluminum chloride, hemostatic sponge, compression, cautery, suture, ferric subsulfate
 - Label specimen bottle with formalin



❖

Punch Biopsy



❖

Instrument tie

- Needle holder is held parallel to the wound incision
- Needle end of suture is looped twice around the holder before grasping the free end of suture
- The free and needle end of the suture exchange sides across the wound
- Additional throws are done in a similar manner, except with one loop





Biopsy for direct immunofluorescence



Bullous disorder

lesional



perilesional



Vasculitis

lesional



Elston DM, Stratman EJ, Miller SJ. Skin biopsy: Biopsy issues in specific diseases.

J Am Acad Dermatol. 2016 Jan;74(1):1-16; quiz 17-8. doi: 10.1016/j.jaad.2015.06.033.





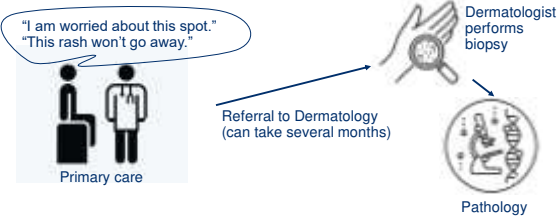
Billing/coding

Code	Description
11102	Tangential biopsy of skin (e.g., shave, scrape, saucerize, curette) single lesion
+11103	each separate/additional lesion (List separately in addition to code for primary procedure)
11104	Pluck biopsy of skin (including simple closure, when performed) single lesion
+11105	each separate/additional lesion (List separately in addition to code for primary procedure)
11106	Incisional biopsy of skin (e.g., wedge) (including simple closure, when performed) single lesion
+11107	each separate/additional lesion (List separately in addition to code for primary procedure)

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Dermatology in the Primary Care Setting

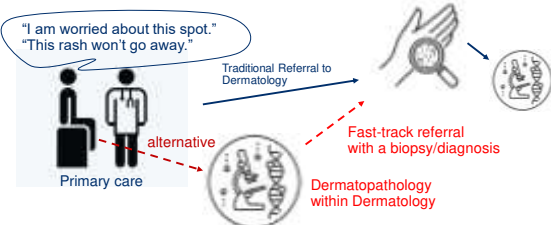
Primary care providers are in a prime position to take care of dermatologic issues.



Jeffrey D. McBride, OU Dermatology, OU Dermatopathology 113

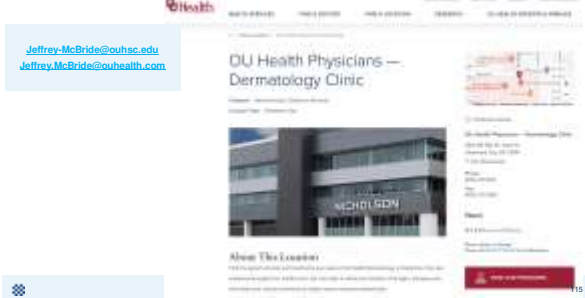
Dermatology in the Primary Care Setting

Primary care providers are in a prime position to take care of dermatologic issues.



Jeffrey D. McBride, OU Dermatology, OU Dermatopathology 114

Thank you for your attention.



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Selected references

Ahn CS, Allen MM, Davis SA, Huang KE, Fleischer AB, Feldman SR. The National Ambulatory Medical Care Survey: A resource for understanding the outpatient dermatology treatment. *J Dermatol Treat.* 2014;25(6):453-458.

Arafat AE, Anzengruber F, Mostafa AM, Navarini AA. Perspectives of online surveys in dermatology. *J Eur Acad Dermatol Venereol.* 2019;33:511-520.

Grada et al. *J Clin Aesthet Dermatol.* 2022 May; 15(5): E82-E86.

Roux E Le, Edwards PJ, Sanderson E, Barnes RK, Ridd MJ. The content and conduct of GP consultations for dermatology problems: A cross-sectional study. *Br J Gen Pract.* 2020;70(699):e723-30.

Deng L, Costa F, Blake KJ, Choi S, Chandrabalan A, Yousuf MS, Shiers S, Dubreuil D, Vega-Mendoza D, Rolland C, Deratson C, Voinin T, Baggood MD, Wesemann L, Frey AM, Palumbo JS, Wanger BJ, Galo RL, Leyva-Castillo JM, Verginolle N, Price TJ, Ramachandran R, Horvath AR, Choi IM. S. aureus drives itch and scratch-induced skin damage through a Vβ protease-PAR1 axis. *Cell.* 2023 Nov 22;186(24):5375-5393.e25. doi: 10.1016/j.cell.2023.10.019.

Elston DM, Stratman EJ, Miller SJ. Skin biopsy: Biopsy issues in specific diseases. *J Am Acad Dermatol.* 2016 Jan;74(1):1-16; quiz 17-8. doi: 10.1016/j.jaad.2015.06.033.

Dermatology. Bologna, 5th edition

UpToDate (treatment algorithms) accessed 2023-2024.

Contact Dermatitis Institute (www.contactdermatitisinstitute.com)

