Top Dermatologic Issues in Primary Care

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EXPLORE HEALTHCARE SUMMIT

OU Health | Stephenson Cancer Center

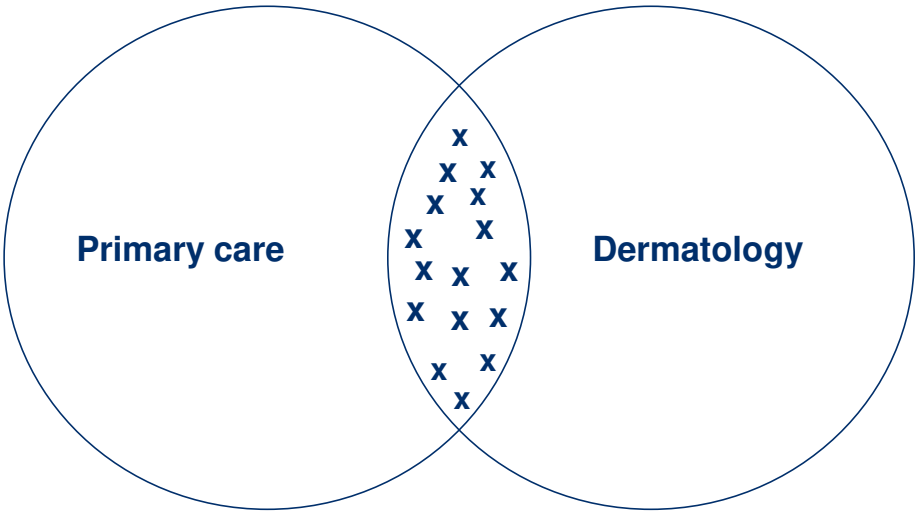
Disclosure

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

Goals of today's talk

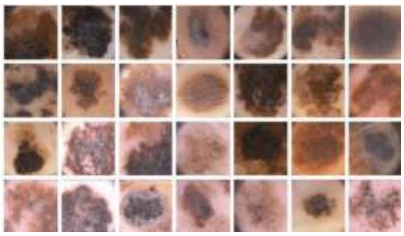
- Emphasize dermatologic diagnoses in primary care setting
- NOT to review the entirety of relevant dermatology
- Emphasize the essential role of a biopsy in making a diagnosis

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.

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

A quick tour through the world of dermatologic morphology

What are the most common derm 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.

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.

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

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

Acne vulgaris vs rosacea – diagnosis

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

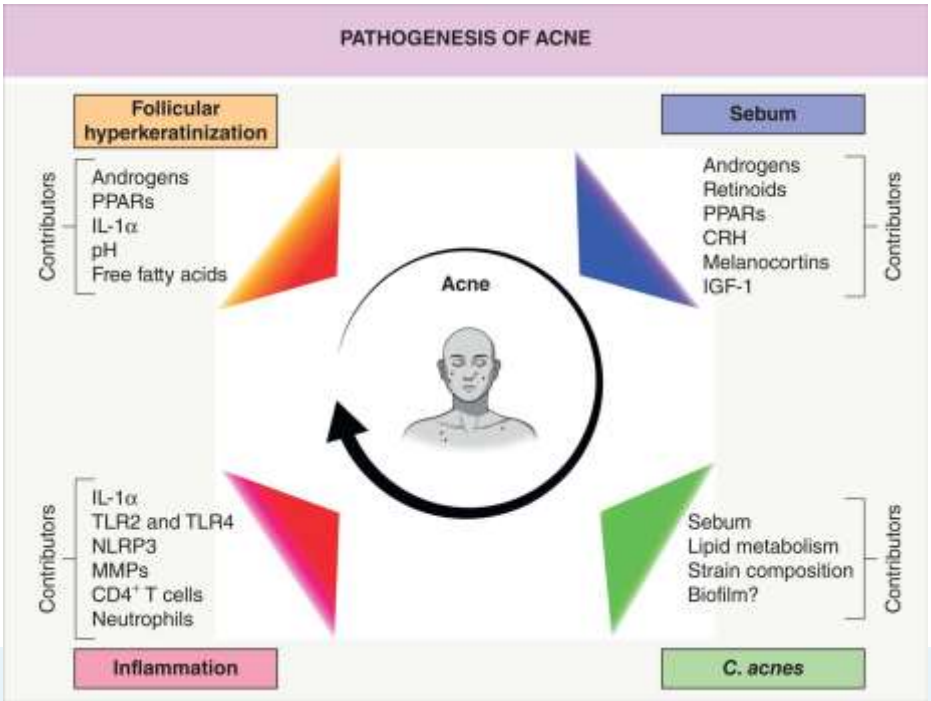
- Individuals at increased risk for the development of acne include:
  - those with an XYY karyotype
  - or endocrine disorders
    - Polycystic ovarian syndrome
    - Hyperandrogenism
    - Hypercortisolism
    - Precocious puberty

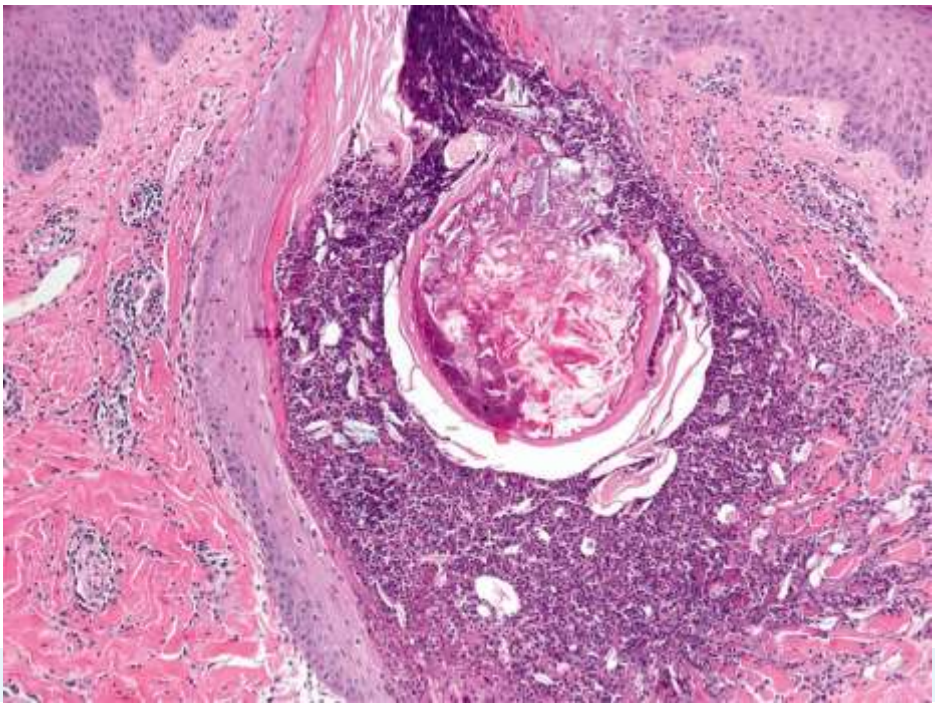
      Patients with these conditions tend to have more severe acne that is less responsive to standard therapy

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***.


PATHOGENESIS OF ACNE
Follicular hyperkeratinization
Contributors
Androgens
PPARs
IL-1α
pH
Free fatty acids
Sebum
Androgens
Retinoids
PPARs
CRH
Melanocortins
IGF-1
Contributors
Acne
Inflammation
Contributors
IL-1α
TLR2 and TLR4
NLRP3
MMPs
CD4+ T cells
Neutrophils
C. acnes
Contributors
Sebum
Lipid metabolism
Strain composition
Biofilm?



Treatment of acne

Acne activity	Treatment	
	Initial	**Follow-up**
Mild: Several papules, pustules, and/or comedones; no nodules. 	**First-line** Topical retinoid or BPO ± topical antibiotic† or Topical retinoid + BPO ± topical antibiotic† **Alternative topicals** Dapsone Clascoterone Azelaic acid*	**For additional control:** Increase strength of topical retinoid or Change from wash to leave-on BPO or Add or replace with another topical agent

Treatment of acne

Moderate: Multiple papules and pustules; few scattered nodules; variable comedones

First-line

Topical retinoid + BPO

± topical antibiotic, oral antibiotic[¶], and/or oral hormonal therapy[¶] (female patients)

Alternative topicals

Dapsone

Clascoterone

Azelaic acid*

For additional control:

See above for topical options

Add oral antibiotic[¶]

and/or hormonal therapy[¶] (female patients)

or

Change to isotretinoin

Treatment of acne

Severe: Numerous papules and pustules; multiple nodules; variable comedones 	**First-line** Topical retinoid + BPO + oral antibiotic[4] ± hormonal therapy[5] (female patients) or Isotretinoin **Consider alternative topical (see above)**	**For additional control:** Change to isotretinoin

Treatment of acne

Very severe: Numerous nodules with conglobate or hemorrhagic lesions; ± systemic symptoms (acne fulminans) 	Prednisone ± isotretinoin (low-dose initially)	Isotretinoin (slow dose escalation)

Birth control and acne

Roles of commonly used contraceptives for acne.

ROLES OF COMMONLY USED CONTRACEPTIVES FOR ACNE	
Oral contraceptive®	**Estrogen mcg/progestin mcg**
FDA-approved as treatment for acne vulgaris	
Ortho Tri-Cyclen, Tri-Estarylla, Tri-Linyah, Tri-Sprintec, Tri-Previfem, Trinessa	Ethinyl estradiol 35/norgestimate 180, 215, 250
Estrostep, Tilia, Tri-Legest	Ethinyl estradiol 20, 30, 35/norethindrone 1000
Yaz, Gianvi, Jasmiel, Loryna, Lo-Zumandimine, Nikki, Vestura, Beyaz*	Ethinyl estradiol 20/drospirenone 3000
Clinical data to support use for acne (selected products)	
Alesse, Aubra, Aviane, Lutera, Orsythia, Vienva	Ethinyl estradiol 20/levonorgestrel 100
Diane-35†	Ethinyl estradiol 35/cyproterone acetate 2000
Yasmin, Ocella, Syeda, Zarah, Zumandimine, Safyral*	Ethinyl estradiol 30/drospirenone 3000
Natazia	Estradiol valerate 1000, 2000, 3000/dienogest 2000, 3000
May worsen acne	
Combined oral contraceptives containing more androgenic progestins (e.g. norgestrel, levonorgestrel) Progestin-only depot injections, subdermal implants, and progestin-containing intrauterine devices	

*Also contains levomefolate calcium for protection against neural tube defects.

†Not available in the US.

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

OTHER FEATURES: Acne Vulgaris

- Most prevalent in adolescents and young adults
- Variable distribution on face
- Frequent shoulder, chest, and/or back involvement
- Sequelae of postinflammatory hyperpigmentation, postinflammatory erythema, and scarring
- Association with hyperandrogenic disorders (eg, polycystic ovarian syndrome)

OTHER FEATURES: Rosacea

- Most prevalent in adults >30 years old
- Centrofacial distribution (cheeks, nose, chin)
- Ocular involvement (eg, symptoms of eye irritation, eyelid erythema, conjunctival injection, crusting, recurrent hordeolum or chalazion)
- Sensitive skin
- Flushing

KEY CONCEPTS

Acne vulgaris and rosacea are common causes of inflamed papules or pustules on the face. Recognition of other characteristic features is helpful for distinguishing these conditions. Patients may exhibit some or all of the displayed features.

Distinguishing between acne vulgaris and rosacea is important because of differences in the approach to patient evaluation and treatment. For example, an assessment for signs of associated hyperandrogenism (eg, menstrual irregularity, hirsutism, virilization) is an important component of the initial evaluation of female patients with acne vulgaris, particularly in the presence of severe, sudden-onset, or recalcitrant acne. In patients with rosacea, an assessment for signs or symptoms of ocular involvement is important for identifying patients who may benefit from ophthalmologic examination.

UpToD

Acne vulgaris vs rosacea – treatment

Acne

- 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 flares
- 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 Winlevi (clascoterone) – androgen receptor inhibitor

Rosacea

- Start topical metronidazole gel
  - If fails, consider topical ivermectin (Soolantra)
- Dermatologist: can perform lasers (example PDL to target hemoglobin in telangiectasias)
- Wash with sensitive skin cleaners (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 – a2 adrenergic receptor agonist)
- Identify and reduce triggers as much as possible (alcohol, spicy foods, heat, stress, etc)
- Refer to ophthalmology if ocular involvement

Isotretinoin for severe cases

27

Epidermoid inclusion cysts - diagnosis

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

Epidermoid inclusion cysts - differential

Pilar cyst

Dermoid cyst

Lipoma

Pilomatricoma

Ganglion cyst

Cysts? Unfortunately not.

Pajaziti, L., Hapçiu, 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

Benign neoplasms of the skin (examples)

Acrochordon/skin tag

Intradermal nevus

Dermatofibroma

Neurofibroma

Seborrheic keratosis

Seborrheic keratosis

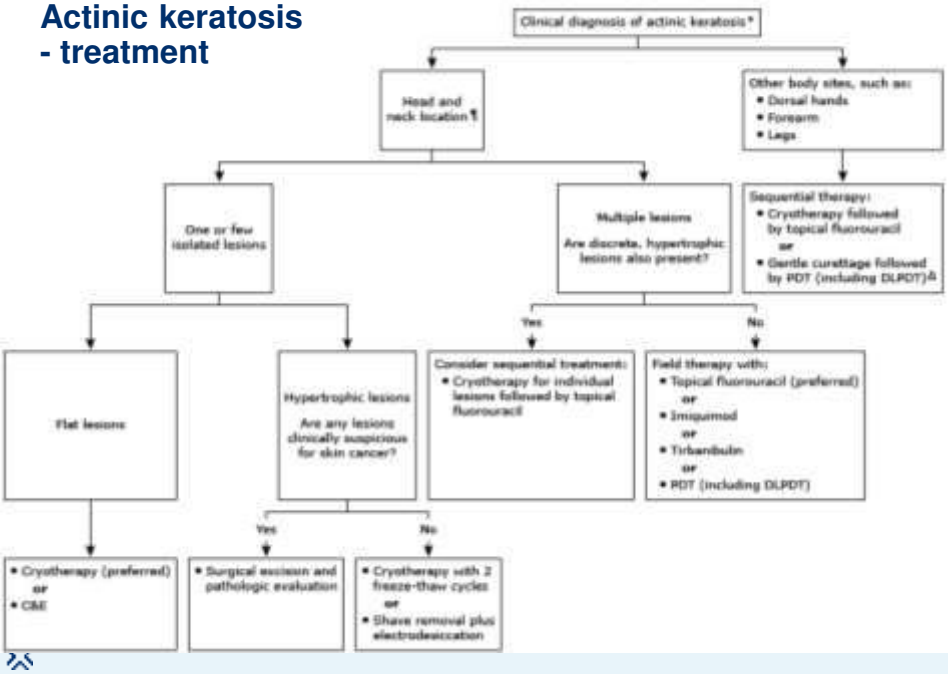
“Pyogenic granuloma” (lobular capillary hemangioma) vs other?

Lobular capillary hemangioma

Spitzoid melanoma

Actinic keratoses

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

www.contactdermatitisinstitute.com

The other “Top” issues

Skin cancer – The “big 3” – diagnosis - clinical

Basal cell carcinoma

Squamous cell carcinoma

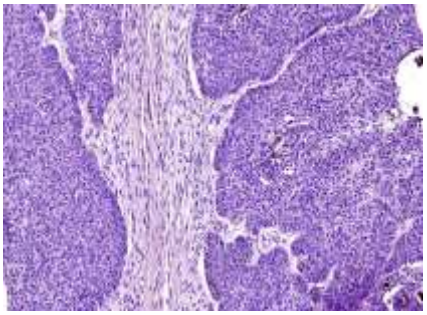
Melanoma

Skin cancer – The “big 3” – diagnosis - dermatopathology

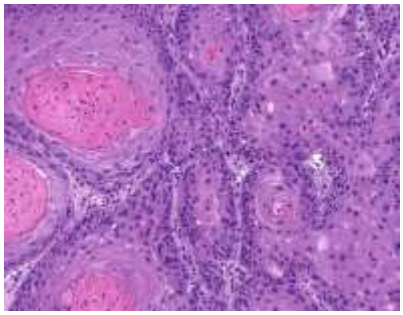
Basal cell carcinoma

Squamous cell carcinoma

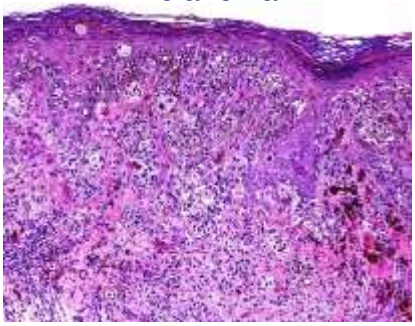
Melanoma



Ber-EP4+



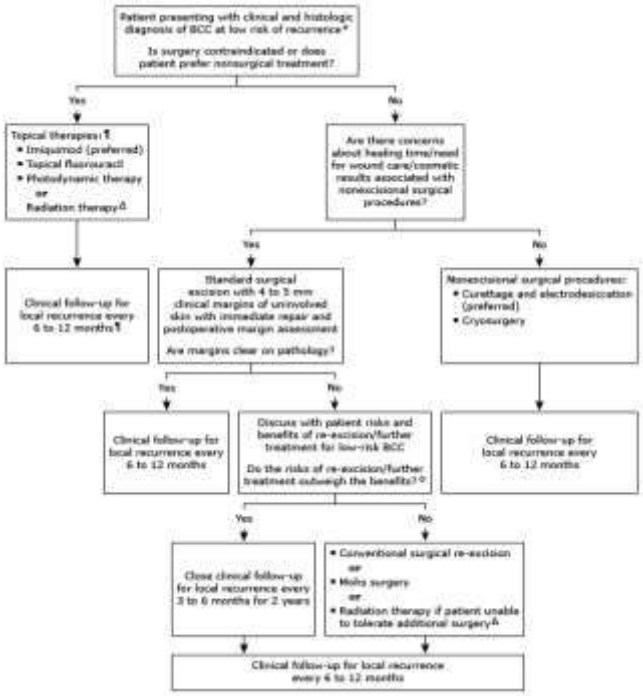
P40 +, Ber-EP4 -



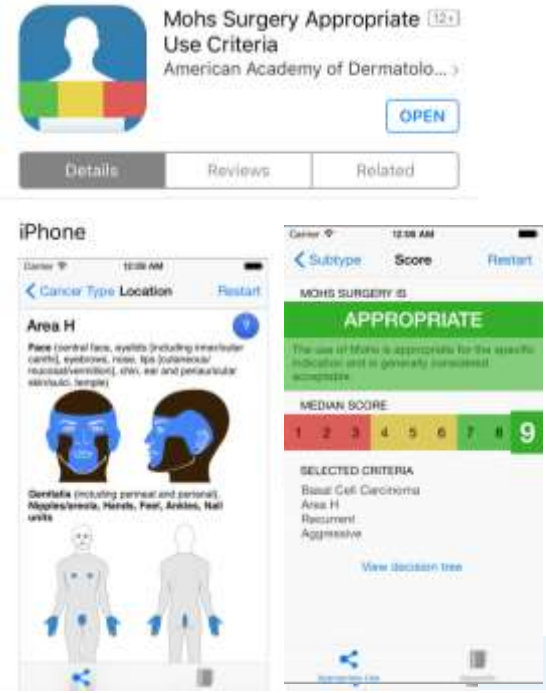
Sox-10+, PRAME+

39

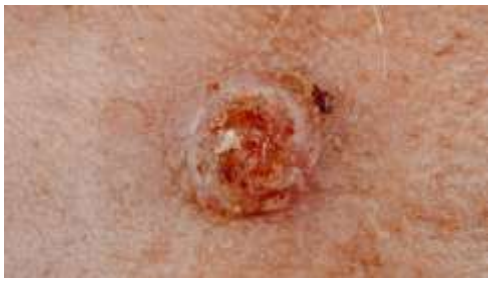
Skin cancer/BCC - treatment

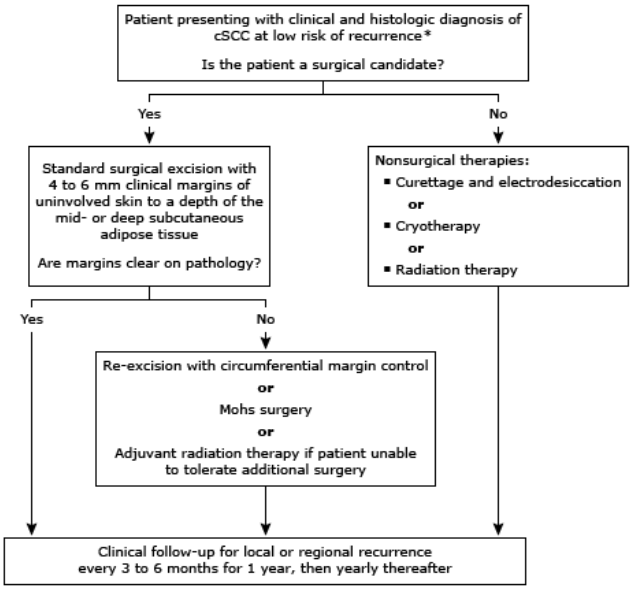


Appropriate Use Criteria for Mohs

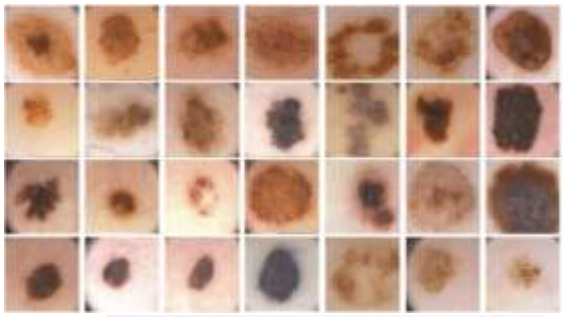


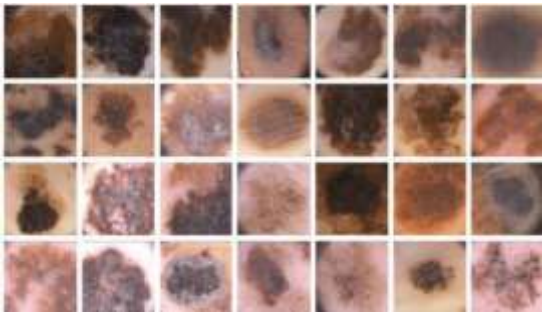
Skin cancer/SCC - treatment

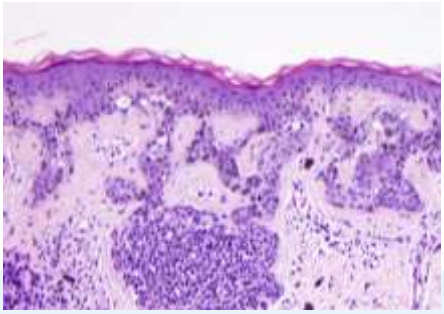


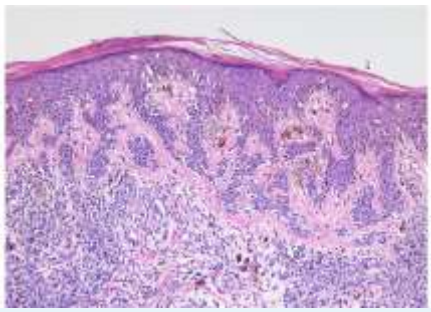


The melanocytic diagnostic dilemma









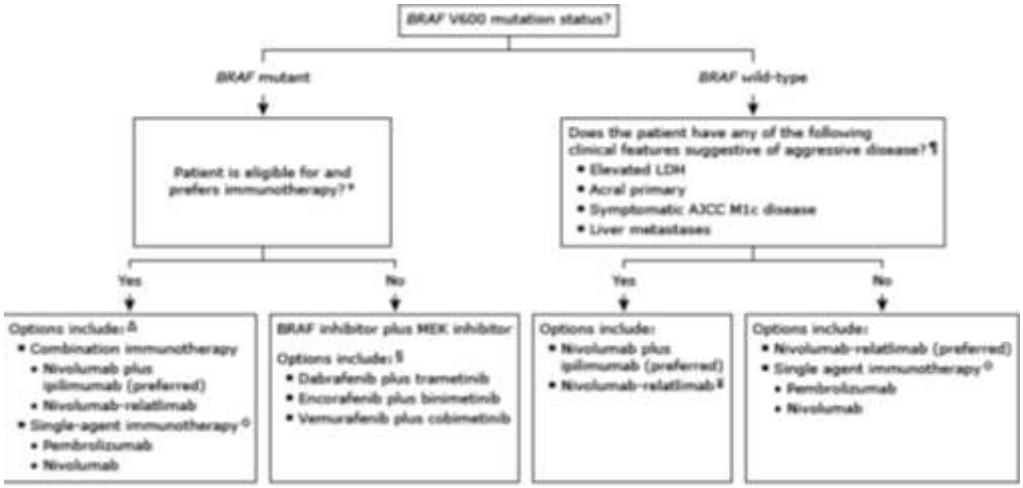
Melanoma- staging

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

T Category	Thickness	Ulceration status
Tis (melanoma *in situ*)	Not applicable	Not applicable
T1	≤1.0 mm	Unknown or unspecified
T1a	<0.8 mm	Without ulceration
T1b	<0.8 mm 0.8–1.0 mm	With ulceration With or without ulceration
T2	>1.0–2.0 mm	Unknown or unspecified
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

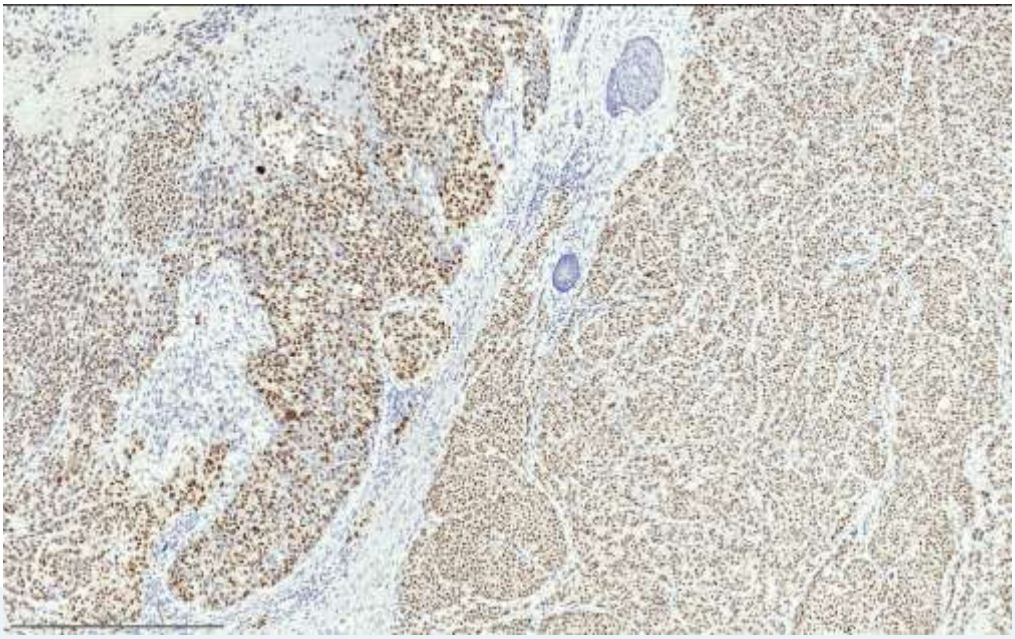
Gershenwald, Scolyar, et al. Melanoma. In Amin, M.B., Edge, S.B., Greene, F.L., et al. (Eds.) AJCC Cancer Staging Manual. 8th Ed. New York: Springer; 2017

Melanoma- treatment

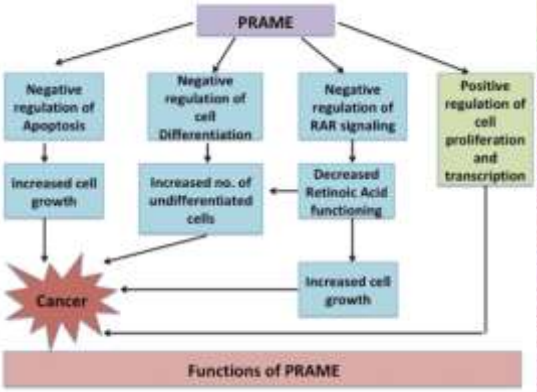


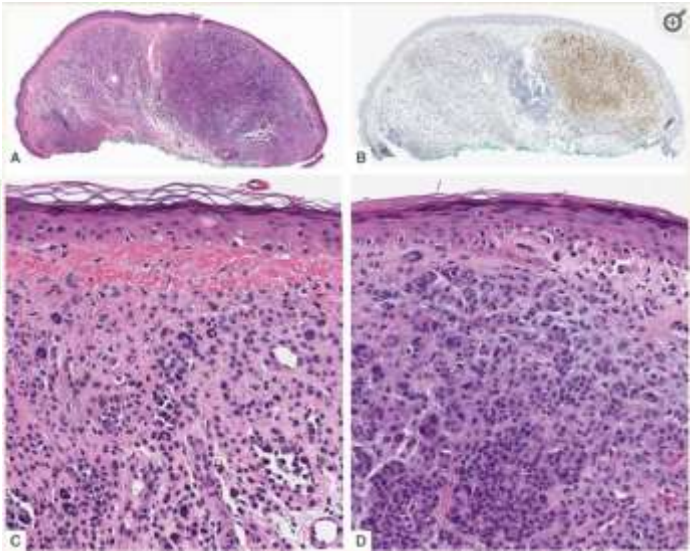
45

PRAME (PReferentially-expressed Antigen in MElanoma)



PRAME in melanoma



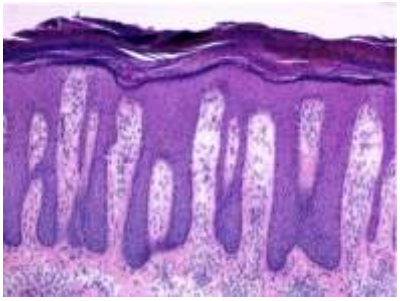
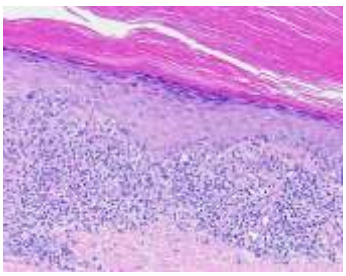


47

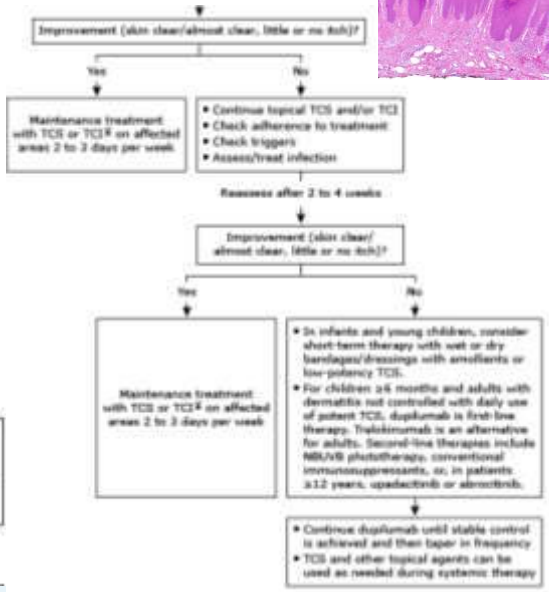
Eczematous vs psoriasiform vs lichenoid - diagnosis





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

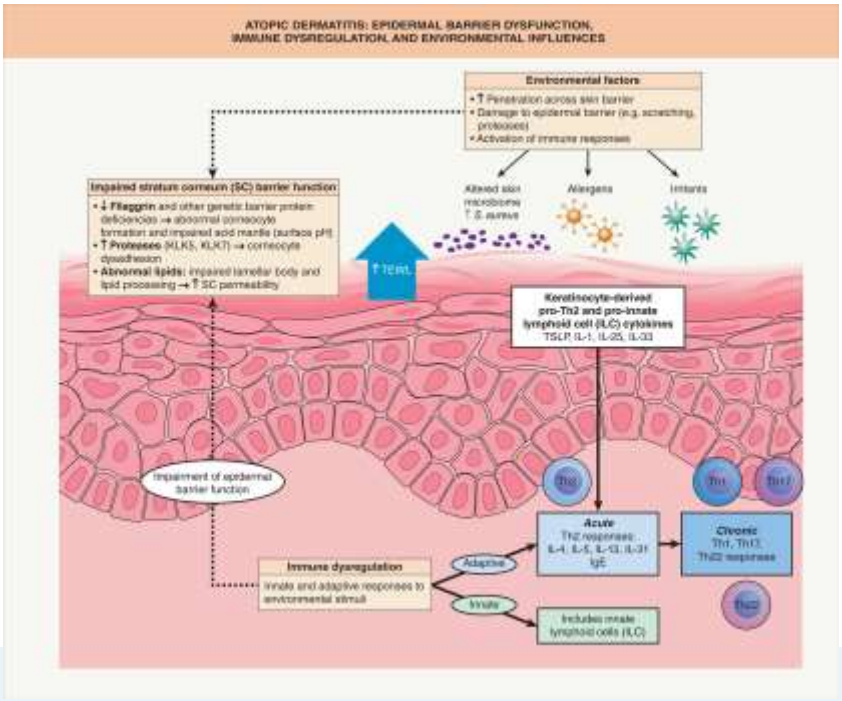
Trimodal distribution in atopic dermatitis

- 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)


ATOPIC DERMATITIS: EPIDERMAL BARRIER DYSFUNCTION, IMMUNE DYSREGULATION, AND ENVIRONMENTAL INFLUENCES
Environmental factors
• ↑ Penetration across skin barrier
• Damage to epidermal barrier (e.g. scratching, protease)
• Activation of immune responses
Impaired stratum corneum (SC) barrier function
• ↓ Filaggrin and other genetic barrier protein deficiencies → abnormal corneocyte formation and impaired acid mantle (surface pH)
• ↑ Proteases (KLK5, KLK7) → corneocyte dysadhesion
• Abnormal lipids: impaired lamellar body and lipid processing → ↑ SC permeability
Altered skin microbiome ↑ S. aureus
Allergens
Irritants
↑ TEWL
Keratinocyte-derived pro-Th2 and pro-innate lymphoid cell (ILC) cytokines
TSLP, IL-1, IL-25, IL-33
Impairment of epidermal barrier function
Th2
Th1
Th17
Acute
Th2 responses: IL-4, IL-5, IL-13, IL-31, IgE
Chronic
Th1, Th17, Th22 responses
Th22
Immune dysregulation
Innate and adaptive responses to environmental stimuli
Adaptive
Innate
Includes innate lymphoid cells (ILC)

Staph in atopic dermatitis

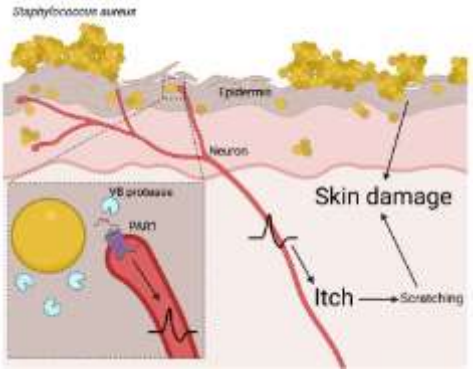
- IL-31 is a Th2 cytokine that is highly expressed in lesional skin and serum of patients with AD as well as in other pruritic skin disorders such as prurigo nodularis.
- Cutaneous exposure to staphylococcal superantigen rapidly induces IL-31 expression in atopic individuals, establishing a link between 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

S. aureus drives itch and scratch-induced skin damage through a V8 protease-PAR1 axis

Liwen Deng • Flavia Costa [10] • Kimbria J. Blake [10] • ... Rithwik Ramachandran • Alexander R. Horswill • Isaac M. Chiu [11] • Show all authors • Show footnotes

DOI: https://doi.org/10.1016/j.cell.2023.10.019 • Check for updates



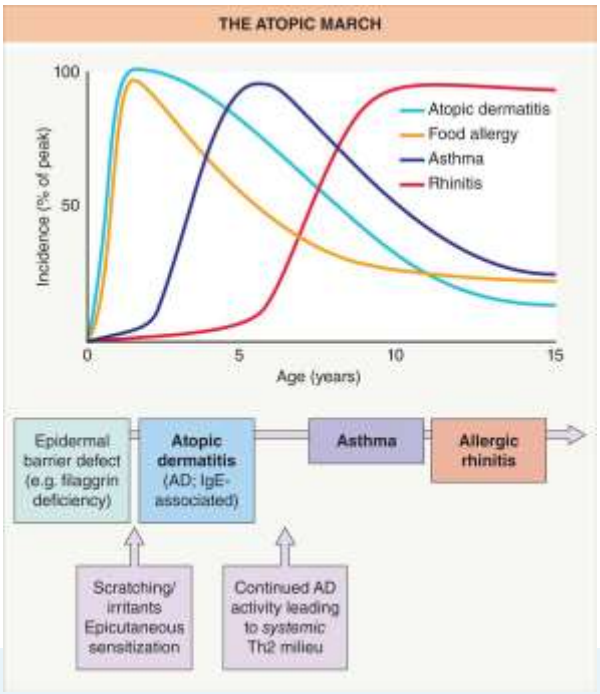
Microbiome in atopic dermatitis

- 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, filaggrin 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.


THE ATOPIC MARCH
100
50
Incidence (% of peak)
0
5
10
15
Age (years)
Atopic dermatitis
Food allergy
Asthma
Rhinitis
Epidermal barrier defect (e.g. filaggrin deficiency)
Atopic dermatitis (AD; IgE-associated)
Asthma
Allergic rhinitis
Scratching/ irritants Epicutaneous sensitization
Continued AD activity leading to systemic Th2 milieu

Genetics and AD

SELECTED CANDIDATE GENES FOR ATOPIC DERMATITIS	
Candidate gene(s)	**Defective protein(s)**
Genes encoding epidermal proteins	
FLG	Filaggrin (loss-of-function variants; see text)
FLG2	Filaggrin family member 2
SPINK5	Serine protease inhibitor LETKI
KLK5/SCTE, KLK7/SCCE	Kallikrein-related peptidases 5 &7/stratum corneum tryptic &chymotryptic enzymes
CLDN1	Claudin-1
SPRR3	Small proline-rich protein 3
TMEM79	Transmembrane protein 79 (mattrin)
KIF3A	Kinesin family member 3A
Genes encoding immunologic proteins	
FCER1A	Fc fragment of high-affinity IgE receptor I, α chain
TLR2, 4, 6, 9	Toll-like receptor-2, -4, -6, and -9
IRF2	Interferon regulatory factor 2
IL4, 5, 12B, 13, 18, 31	Interleukin-4, -5, -12B, -13, -18, and -31
IL4RA, IL5RA, IL13RA	Interleukin-4, -5, and -13 receptors, α subunits
GM-CSF	Granulocyte–macrophage colony-stimulating factor
CD14	Monocyte differentiation antigen CD14
DEFB1	β-defensin 1

Genetic associations with atopic dermatitis (continued)

GSTP1	Glutathione S-transferase P1
CMA1	Mast cell chymase
CCL5/RANTES	Chemokine (C-C motif) ligand 5/RANTES
TSLP	Thymic stromal lymphopoietin
CARD11, CARD14	Caspase recruitment domain family members 11 and 14
RETN	Resistin
MIF	Macrophage migration inhibitory factor
VDR	Vitamin D receptor
CYP27A1, CYP2R1	Cytochrome p450 family members 27A1 and 2R1

Common presentation in atopic dermatitis

Variation in clinical presentation

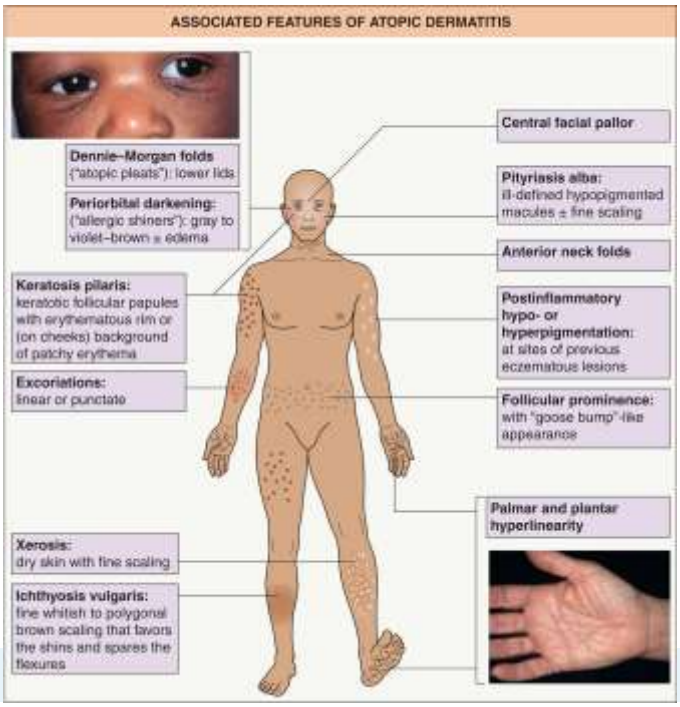
Atopic dermatitis

Chronic atopic dermatitis, lichenification

Atopic dermatitis - lichenification

Variation in atopic dermatitis

Other common sites of involvement


ASSOCIATED FEATURES OF ATOPIC DERMATITIS
Dennie–Morgan folds ("atopic pleats"): lower lids
Periorbital darkening: ("allergic shiners"): gray to violet–brown ± edema
Keratosis pilaris: keratotic follicular papules with erythematous rim or (on cheeks) background of patchy erythema
Excoriations: linear or punctate
Xerosis: dry skin with fine scaling
Ichthyosis vulgaris: fine whitish to polygonal brown scaling that favors the shins and spares the flexures
Central facial pallor
Pityriasis alba: ill-defined hypopigmented macules ± fine scaling
Anterior neck folds
Postinflammatory hypo- or hyperpigmentation: at sites of previous eczematous lesions
Follicular prominence: with "goose bump"-like appearance
Palmar and plantar hyperlinearity

Triggers/exposures in atopic dermatitis

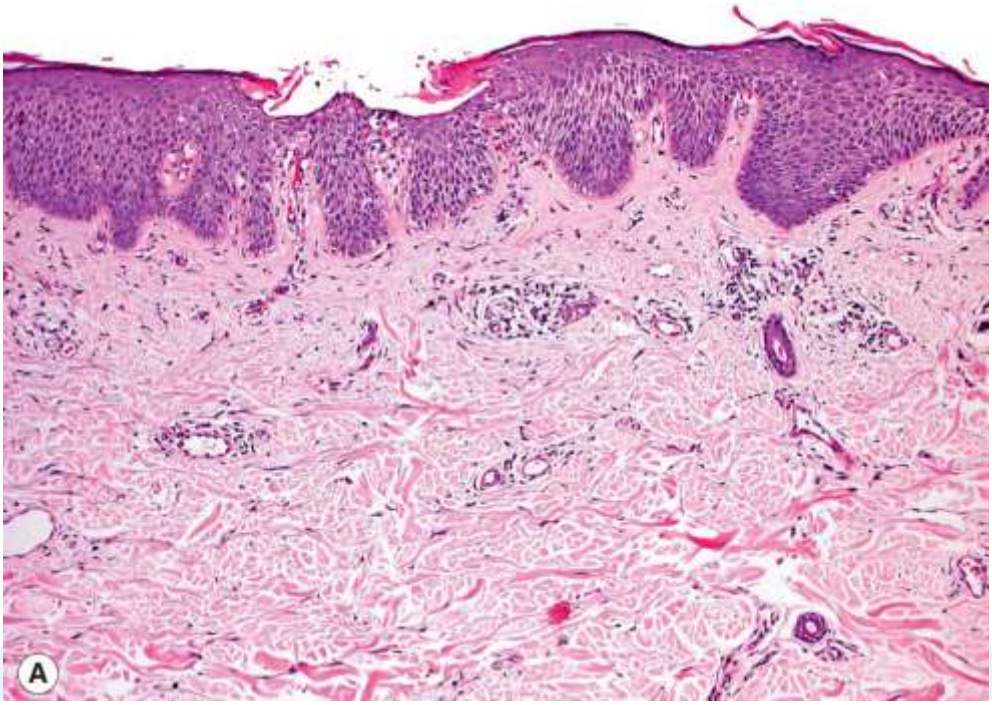
Triggers
• *Climate:* extremes of temperature (winter or summer), low humidity
• *Irritants:* wool/rough fabrics, perspiration, detergents, solvents
• *Infections:* cutaneous (e.g. *Staphylococcus aureus*, molluscum contagiosum, herpes simplex) or systemic (e.g. URI)
• *Environmental allergies:* e.g. to dust mites, pollen, contact allergens
• *Food allergies:*
• Trigger in small minority of AD patients, e.g. 10%–30% of those with moderate to severe, refractory AD
• Common allergens: egg > milk, peanuts/tree nuts, (shell)fish, soy, wheat
• Detection of allergen-specific IgE (via 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

Eczema herpeticum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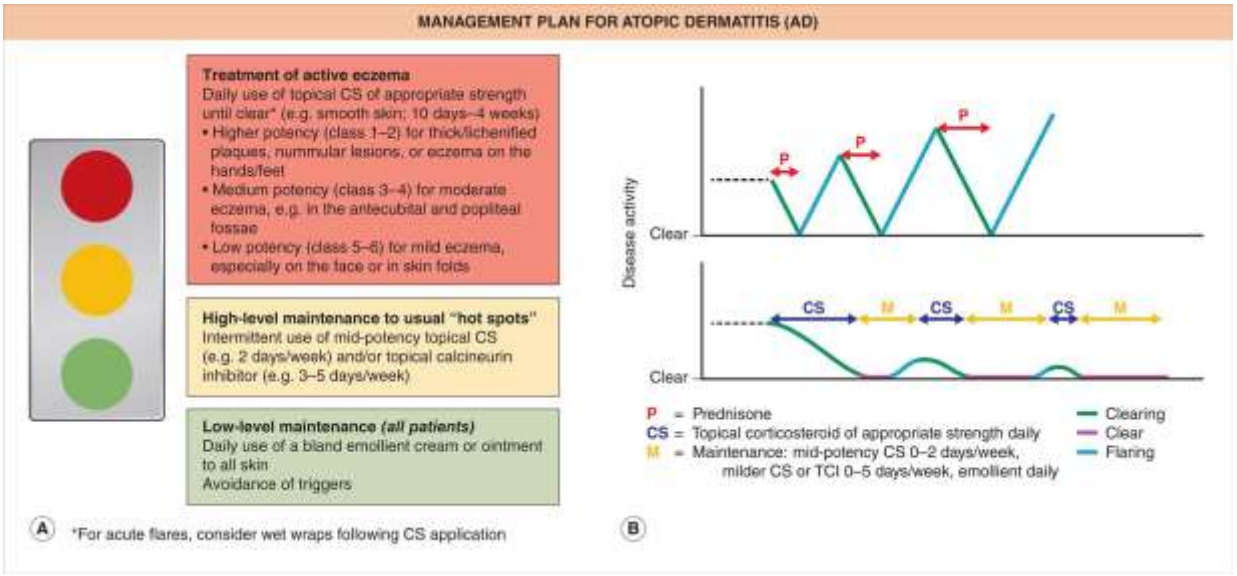

MANAGEMENT PLAN FOR ATOPIC DERMATITIS (AD)
Treatment of active eczema
Daily use of topical CS of appropriate strength until clear* (e.g. smooth skin; 10 days–4 weeks)
• Higher potency (class 1–2) for thick/lichenified plaques, nummular lesions, or eczema on the hands/feet
• Medium potency (class 3–4) for moderate eczema, e.g. in the antecubital and popliteal fossae
• Low potency (class 5–6) for mild eczema, especially on the face or in skin folds
High-level maintenance to usual "hot spots"
Intermittent use of mid-potency topical CS (e.g. 2 days/week) and/or topical calcineurin inhibitor (e.g. 3–5 days/week)
Low-level maintenance (all patients)
Daily use of a bland emollient cream or ointment to all skin
Avoidance of triggers
A
*For acute flares, consider wet wraps following CS application
Disease activity
Clear
P
P
P
Clear
CS
M
CS
M
CS
M
P = Prednisone
CS = Topical corticosteroid of appropriate strength daily
M = Maintenance: mid-potency CS 0–2 days/week, milder CS or TCI 0–5 days/week, emollient daily
Clearing
Clear
Flaring
B

THERAPEUTIC LADDER FOR ATOPIC DERMATITIS (AD)	
Topical therapies	Evidence
Moisturizers	1
Corticosteroids	1
Calcineurin inhibitors	1
Crisaborole	1
JAK inhibitors e.g. ruxolitinib (FDA-approved), delgocitinib (approved in Japan)	1
Phototherapy	
Narrowband UVB, UVA–UVB, UVA1	1
Systemic therapies	Evidence
Dupilumab	1
IL-13 inhibitors, e.g. tralokinumab, lebrikizumab	1
JAK inhibitors, e.g. upadacitinib, abrocitinib, baricitinib	1
Cyclosporine (short-/intermediate-term)	1
Azathioprine	1
Mycophenolate mofetil/enteric-coated mycophenolate sodium	1/2
Methotrexate	1/2

Systemic corticosteroids (short-term for severe acute flares; "rebound" exacerbations often occur upon discontinuation)	2
Omalizumab	2*
Nemolizumab† (anti-IL-31 receptor A; not currently FDA-approved)	1‡
Rituximab	2
Interferon-γ	‖
IVIg	2*
Adjunctive therapies	
Wet wraps, open wet dressings, or soaks combined with topical corticosteroids for acute flares	
Dilute sodium hypochlorite (bleach) baths	
Treatment of associated bacterial, viral, or fungal infections	
Oral antihistamines for associated conditions (e.g. dermographism, rhinoconjunctivitis) and sedative effects	
Leukotriene antagonists¶	
Sodium cromoglycate (topical or oral)¶	

Key to evidence-based support: (1) prospective controlled trial; (2) retrospective trial or large case series; (3) small series or individual case reports.

*No significant benefit was found in a small controlled trial.

†Benefit for pruritus in patients with moderate to severe AD.

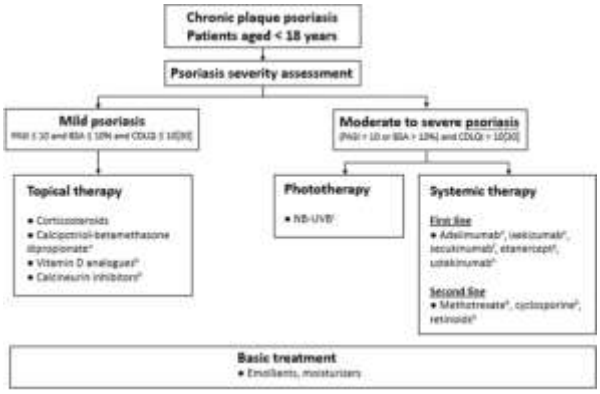
‡Although found to be effective in one randomized controlled trial, results of other studies have been inconsistent (see **Ch. 128**).

¶Inconsistent demonstration of efficacy in controlled trials.

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

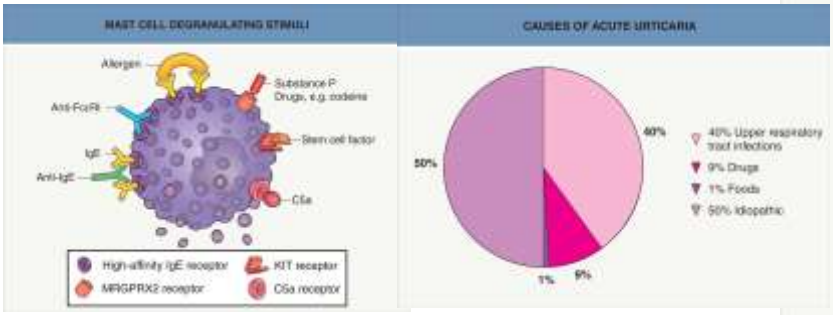


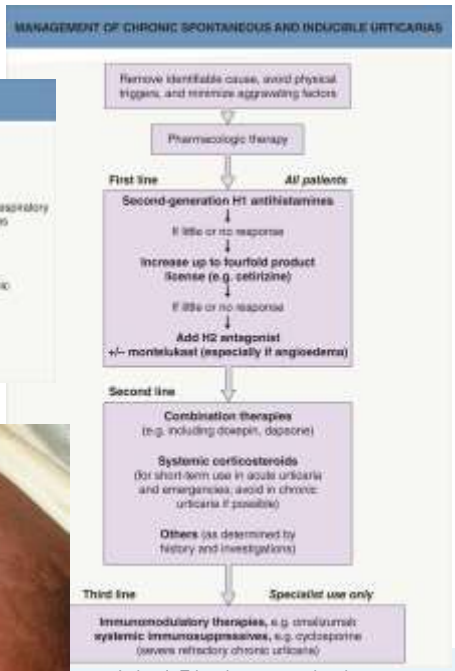
Labs: CBC, CMP, Hep panel, Quant TB

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 Arcutis' ZORYVE® (roflumilast) Cream 0.3% for Treatment of Psoriasis in Children Ages 6 and older

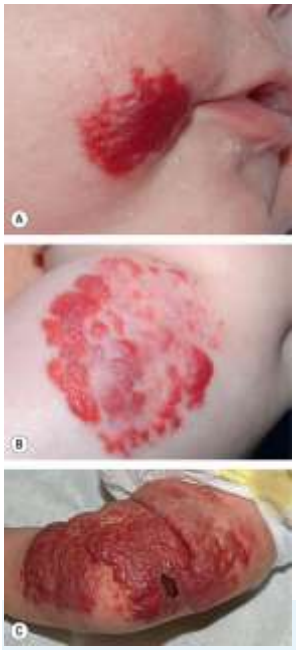
Chronic Urticaria



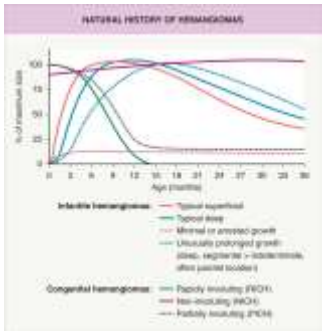


Labs: IgE levels; tryptase levels

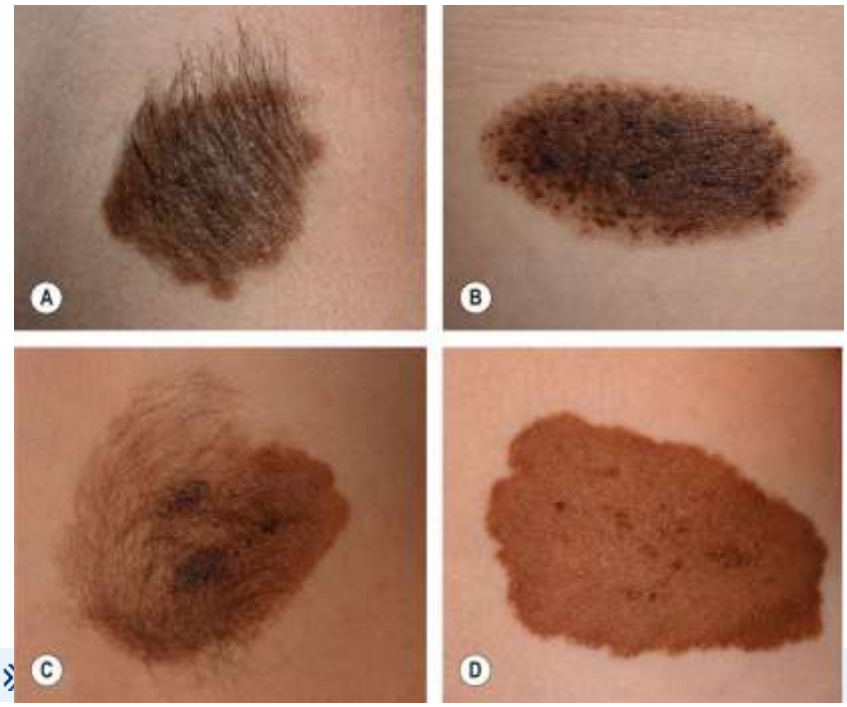
Vascular Birthmarks and Hemangiomas



	Infantile hemangiomas	Vascular malformations
Clinical	• Usually absent at birth or subtle prominent lesion present; only occasionally well formed • Rapid proliferation in first 1–2 months of life • Spontaneous involution over years	• Usually evident at birth • Slower expansion, with increase in area proportionate to the child's growth • Persists into adulthood
Epidemiology	More common in: • Girls (3:1) • Premature and/or low birth weight infants • Infants whose mothers were older, underwent chorionic villus sampling, or had preeclampsia	No sex or gestation predilection
Pathology	**Proliferating**: endothelial cell hyperplasia, lobule formation, mast cells, prominent basement membrane **Involuting**: fibrofatty tissue replacement, decreased mast cells when fully involuted	Dependent upon type; often irregular vascular channels
Immunohistochemistry	Positive for GLUT1, Lewis Y antigen, merosin, and Fcγ RII, Wilms tumor protein 1 (WT1)	Negative for GLUT1, Lewis Y antigen, merosin, and Fcγ RII; NTI



Pigmented Lesions and Nevi



Small: Less than 1.5 cm
Medium: 1.5–19.9 cm
Large or giant: 20 cm or more

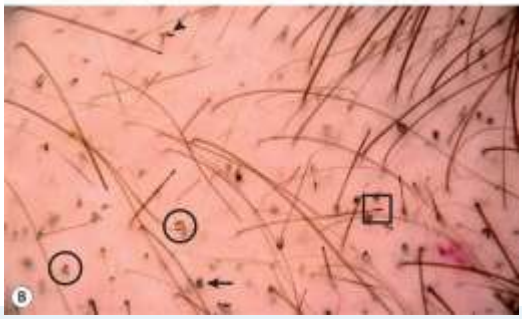
Risk 6-20 % - MM

Alopecia (Hair Loss):

Alopecia areata

JAK inhibitors are making waves.
Ritlecitinib (Litfulo) has been drastic in improving in our patient population

*Beware of syphilis



trichotillomania

Pigmentation disorders

- 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

Nevus depigmentosus

Psoriasis

Seb derm

Pityriasis lichenoides chronica

Atopic dermatitis

Neonatal lupus

Discoid lupus

Cutaneous infections

Paronychia (staph)

abscess

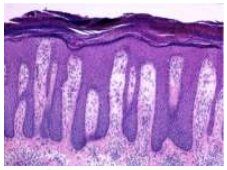
Peri-anal strep

Necrotizing fasciitis
(Grp A b-hem step)

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



Biologic	Other Compatible Conditions	Contraindications	Dosing	Approx. Cost (First Year)	Common Adverse Reactions (>10%)	Efficacy - Primary Outcome and Long term Outcome[a]
Adalimumab (Humira) TNFi	Pregnancy Breastfeeding PsA Crohn's/UC[b,c,e]	Active TB or other severe infections Malignancies Hepatitis B Demyelinating disease Heart Failure[d,e]	Every 2 wks (SC)[a]	$21,359[f]	Injection site rxn Headache Skin rash Antibody Development URTI/Other infections[e]	PASI 75 @ Week 16: 71-79% Loss of adequate response[a] @ Week 52: 35%[a]
Certolizumab pegol (Cimzia) TNFi	Crohn's Disease PsA Pregnancy Breastfeeding[a]	Active TB or other severe infections Heart failure[d,e]	Every 2 wks (SC)[a,g]	$19,271[a]	Headache Nausea Antibody development URTI/Other infections[e]	PASI 75 @ Week 16: 73-88% % of PASI 75 responders maintained until Week 48: 80-86%[a]
Etanercept (Enbrel) TNFi	PsA Pregnancy Breastfeeding[b,c,e,f]	Hypersensitivity to etanercept Patients at risk of sepsis syndrome[a]	Twice weekly for 3 mos, then once weekly (SC)[a]	$23,963[a]	Injection site rxn Headache Skin rash URTI/Other infections[e]	PASI 75 @ Week 12: 47-48% PASI 75 @ Week 96: 53%[a]
Infliximab (Remicade) TNFi	Crohn's/UC PsA Pregnancy Breastfeeding[b,c,e,f]	Severe infections[e] Heart failure[d,e]	IV Infusion at 0, 2, and 6 wks, then every 8 wks after[a,g]	$30,960[a]	Infusion rxn Headache Antibody development Gastrointestinal symptoms URTI/Other infections[e]	PASI 75 @ Week 10: 75-80% PASI 75 @ Week 50: 59-61%[a]
Ixekizumab (Taltz) TNFi	Crohn's/UC PsA/AS/RA[a]	Severe infections Heart failure Pregnancy Breastfeeding[a]	IV Infusion at 0, 2, and 6 wks, then every 8 wks after[a,g]	$21,900[a]	Infusion rxn Headache Antibody development Gastrointestinal symptoms URTI/Other infections[e]	Not reported (refer to infliximab)[a]

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

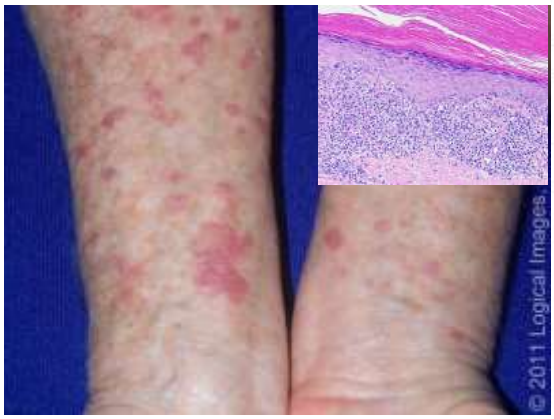
Psoriasis – treatment - biologics

Biologic	Other Compatible Conditions	Contraindications	Dosing	Approx. Cost (First Year)	Common Adverse Reactions (>10%)	Efficacy – Primary Outcome and Long term Outcome*
Brodalumab (Siliq) IL-17i	PsA Hepatitis B/C Pregnancy Breastfeeding*	Crohn Disease Hypersensitivity to brodalumab*	Weekly for 3 wks, then every 2 wks (SC)*	$16,000*	URTI/Other infections*	sPGA 0/1 at Week 12: 70-80% % of sPGA responders maintained until Week 52: 79-83%*
Ixekizumab (Taltz) IL-17i	PsA Hepatitis B/C*	Hypersensitivity to ixekizumab Pregnancy*	Every two wks until week 12, then every 4 wks (SC)*	$25,825*	URTI Injection site rxn*	sPGA 0/1 at Week 12: 71-83% % of sPGA responders maintained until Week 60: 73%*
Secukinumab (Cosentyx) IL-17i	PsA Hepatitis B/C Pregnancy Breastfeeding*	Hypersensitivity to secukinumab IBD TB Chronic Infection*	Loading dose weekly for 4 wks, then every 4 wks after (SC)*	$26,520*	URTI/Other infections*	PASI 75 at Week 12: 75-87% % of PASI 75 responders maintained until Week 52: 81-84%*
Guselkumab (Tremfya) IL-23i	PsA (phase II RCT) Breastfeeding*	Hypersensitivity to guselkumab Active infection Untreated hepatitis B Hx of lymphoproliferative malignancy HIV Pregnancy*	Once at wks 0 and 4, then every 8 wks after (SC)*	$21,610*	URTI/Other infections*	PASI 90 at Week 16: 70-75% % of PASI 90 responders maintained until Week 48: 69%*
Ustekinumab (Stelara) IL-12/23i	PsA Crohn Disease Pregnancy Breastfeeding*	Active infection Untreated hep B Hx of lymphoproliferative malignancy Hypersensitivity HIV*	Once at 0 and 4 wks, then every 12 wks after (SC/IV)*	$22,960*	Antibody development URTI/Other infections*	PASI 75 at Week 12: 67% % of PASI 75 responders maintained until Week 52: 69%*
Risankizumab (Skyrizi) IL-23i	Crohn Disease (Phase II RCT)*	Hypersensitivity Pregnancy*	Once at wks 0 and 4, then every 12 wks after (SC)*	$24,675*	Antibody development URTI/Other infections*	sPGA 0/1 at Week 16: 84-88% sPGA 0/1 at Week 52: 87%**

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

Drug-induced lichenoid dermatitis – treatment

1. Eliminate potential drug causes



2. Topical steroids
3. Wide range of immunosuppresives

Group of drug	
Antimicrobial substances	Aminosalicylate sodium, ethambutol, griseofulvin, ketoconazole, streptomycin, tetracycline, trovafloxacin, isoniazid
Antihistamines (H_2-blocker)	Ranitidine*, roxatidine
Antihypertensives/antiarrhythmics	**ACE inhibitors** (captopril, enalapril), doxazosin, **beta blockers** (propranolol, labetalol, sotalol), **methyldopa**, prazosin, nifedipine, **quinidine**
Antimalarial drugs	**Chloroquine**, **hydroxychloroquine**, quinine
Antidepressives/antianxiety drugs/antipsychotics/antiseizure medications	Amitriptyline, carbamazepine, chlorpromazine, levomepromazine, methopromazine, imipramine, lorazepam, phenytoin
Diuretics	**Thiazide diuretics** (chlorothiazide and hydrochlorothiazide), furosemide, spironolactone
Antidiabetics	Sulfonylureas (chlorpropamide, glimepiride, tolazamide, tolbutamide, glyburide)
Metals	**Gold salts**, arsenic, bismuth, mercury, palladium, lithium
Nonsteroidal anti-inflammatory drugs	Acetylsalicylic acid, benoxaprofen, diflunisal, fenclofenac, flurbiprofen, ibuprofen, indomethacin, naproxen, sulindac
Proton pump inhibitors	Omeprazole, lansoprazole, pantoprazole
Lipid lowering drugs	Pravastatin, simvastatin, gemfibrozil
Tumor necrosis factor-alpha antagonists	Infliximab, adalimumab, etanercept, lenercept
Checkpoint inhibitors	Nivolumab, pembrolizumab, atezolizumab, ipilimumab
Miscellanea	Allopurinol, bleomycin, cinnarizine, cyanamide, dapsone, hydroxyurea, hepatitis B vaccine, imatinib, immunoglobulins, interferon alfa, l-thyroxin, levamisole, mesalamine, methycran, **penicillamine**, procainamide, pyrimethamine, pyritinoxine, **quinacrine**, sildenafil, sulfasalazine, terbinafine, trihexyphenidyl, ursodeoxycholic acid

The **bolded** drugs are the ones most frequently implicated.

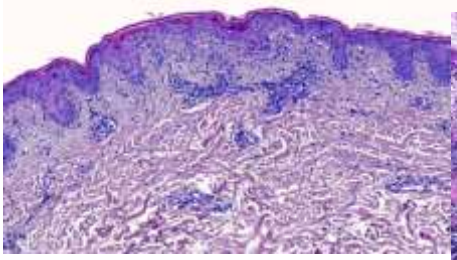
Autoimmune connective tissue disease - diagnosis

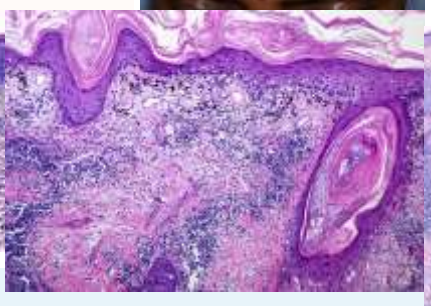
Acute cutaneous lupus

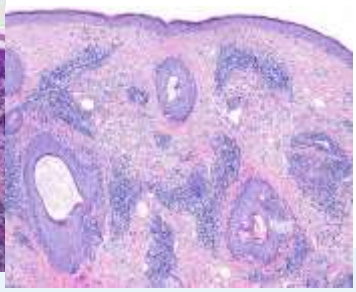
Subacute cutaneous lupus

Discoid lupus

Tumid lupus

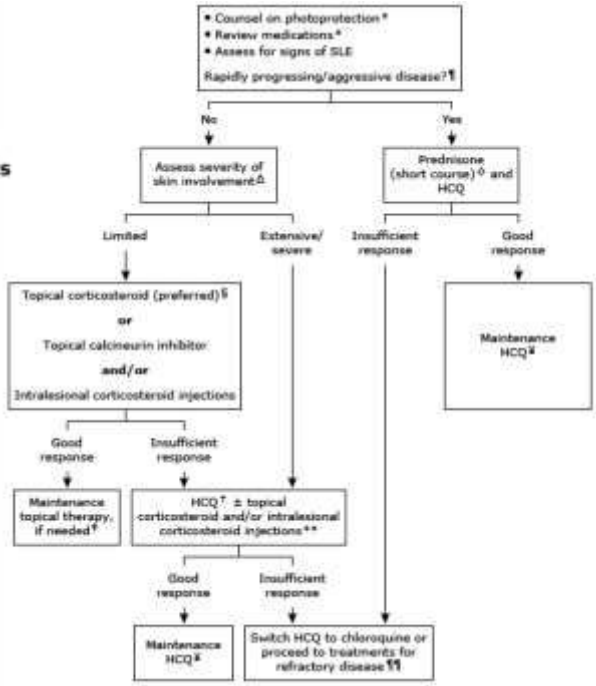






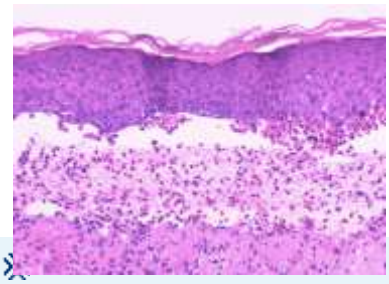
Autoimmune connective tissue disease - treatment

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

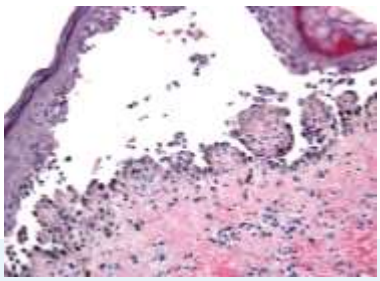


Autoimmune bullous dermatoses, examples - diagnosis

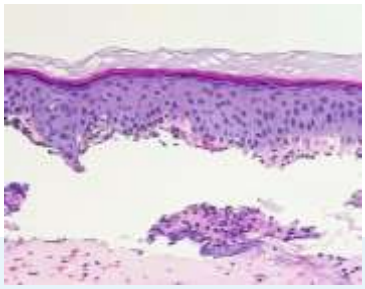
Bullous pemphigoid



Pemphigus vulgaris

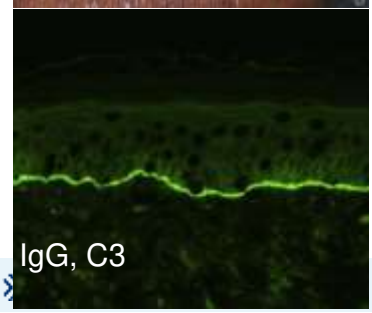


Bullous lupus

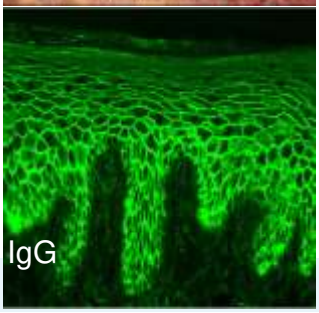


Autoimmune bullous dermatoses, examples - diagnosis

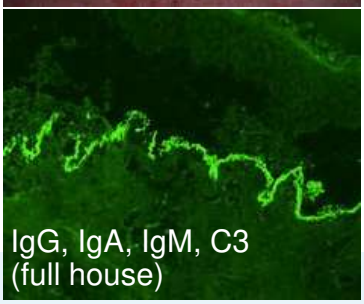
Bullous pemphigoid



Pemphigus vulgaris



Bullous lupus

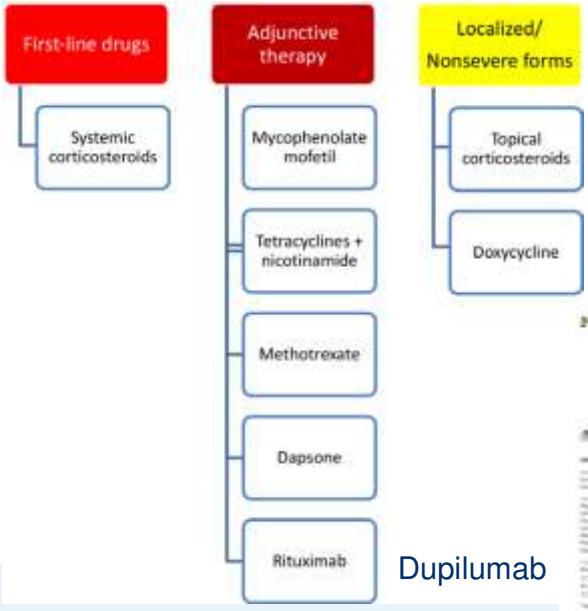


98

Medications implicated in drug-associated bullous pemphigoid

Likely association[*]	Probable association[¶]	Uncertain association[Δ]
• Alogliptin • Anagliptin • Aspirin • Biotin • D-penicillamine • Enalapril • Erlotinib • Etanercept • Everolimus • Furosemide • Ibuprofen • Levofloxacin • Linagliptin • Nivolumab • Pembrolizumab • Phenacetin • Psoralens with ultraviolet A • Rifampicin • Serratiopeptidase • Sirolimus • Sitagliptin • Teneligliptin • Tetanus toxoid • Tiobutarit • Vildagliptin	• Actinomycin D • Adalimumab • Amoxicillin • Ampicillin • Arsenic • Atezolizumab • Bumetanide • Cefazolin • Cephalexin • Chloroquine • Ciprofloxacin • Diclofenac • Dacarbazine • Durvalumab • Efalizumab • Fluoxetine • Gabapentin • Griseofulvin • Hepatitis B vaccine • Hexavalent combined vaccine • Hydrochlorothiazide • Infliximab • Ipilimumab • Lisinopril • Losartan • Mefenamic acid • Metamizole • Metronidazole • Penicillin • Rosuvastatin • Spironolactone • Sulfasalazine • Terbinafine • Ustekinumab	• Aldesleukin (IL-2) • Amantadine • Amlodipine • Anthralin (dithranol) • Aripiprazole • Captopril • Coal tar • Complementary medicines • COVID-19 mRNA vaccines • Dabrafenib • Doxepin • Enoxaparin • Escitalopram • Fluorouracil • Flupenthixol • Galantamine hydrobromide • Herpes zoster vaccine • Influenza vaccine • Iodide • Levetiracetam • Mesalazine • Nadolol • Nifedipine • Novocaine (benzyl benzoate) • Omeprazole • Placental extracts • Photodynamic therapy • Risperidone • Rotavirus vaccine • Sulfonamide • Swine flu vaccine • Timolol • Valsartan

Treatment – autoimmune bullous disease – BP as an example



Dupilumab


Dupilumab effectively and rapidly treats bullous pemphigoid by inhibiting the activities of multiple cell types

Basic dermatologic procedures

Shave biopsy

Punch biopsy

Types of Biopsies and Indications

-Pedunculated lesions (skin tags)

-Dome-shaped nevi

-NMSC (BCC/SCC)

- Pigmented lesions (ruling out melanoma)

- Connective tissue diseases (Lupus/ Dermatomyositis)

- Papulosquamous disorders (psoriasis)

-Blistering disorders (pemphigus)

-Granulomatous diseases (sarcoid)

-Vasculitis (HSP)

-NMSC (infiltrating tumors)

-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

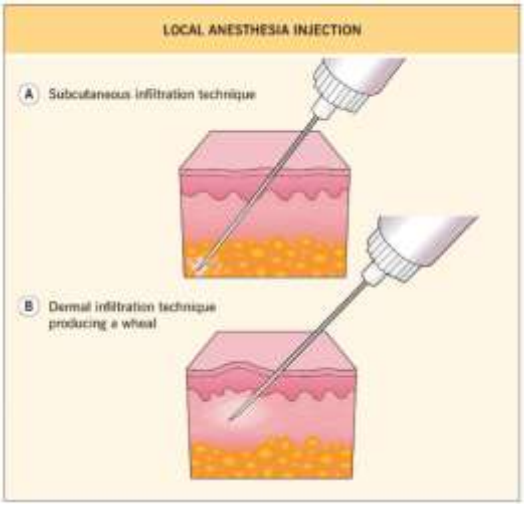
BIOPSY SITE SELECTION	
Lesion/disorder	**Appropriate site**
Tumor	Thickest portion; avoid necrotic tissue
Blister	Edge of lesion, including perilesional skin (see Fig. 0.11)
Ulcerated/necrotic lesion	Edge of ulcer or necrosis plus adjacent skin
Generalized polymorphous eruption	Characteristic lesion of recent onset (± 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



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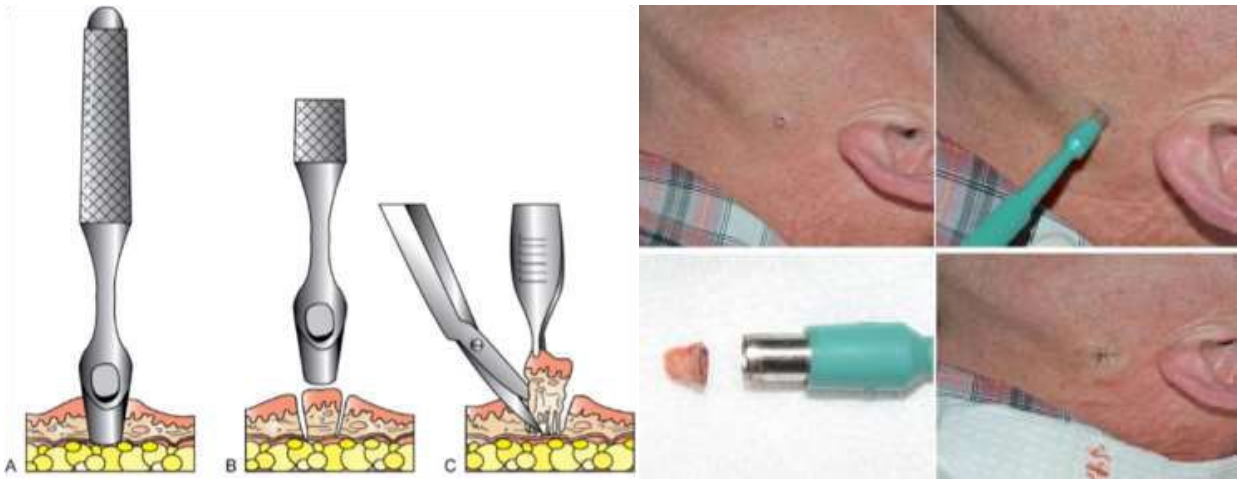
Patient Preparation Continued

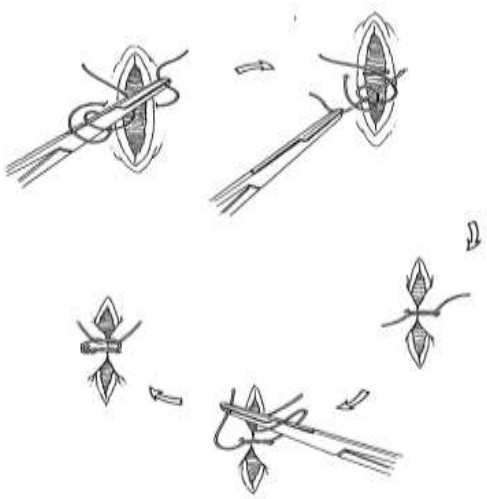
- Prep
  - ETOH swab
  - Iodine
  - Chlorhexidine
- Anesthesia
  - Plane of injection
- Procedure
  - Hemostasis: Aluminum chloride, hemostatic sponge, compression, cautery, suture, ferric subsulfate
  - Label specimen bottle with formalin

Punch Biopsy

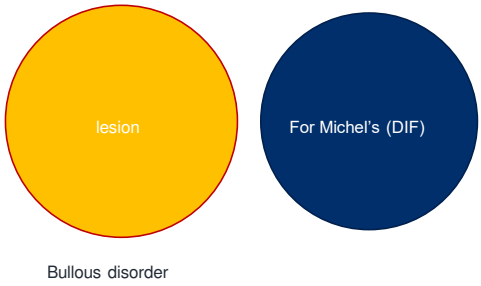


Instrument tie

- 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

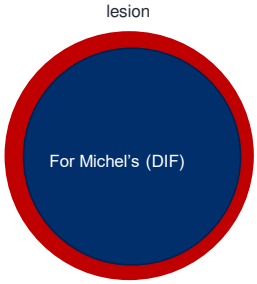


Biopsy for direct immunofluorescence



Bullous disorder

lesional perilesional



Vasculitis

lesional

CONTINUING MEDICAL EDUCATION

Skin biopsy

Biopsy issues in specific diseases

Dirk M. Elston, MD,[a] Eric J. Stratman, MD,[b] and Stanley J. Miller, MD[c]
Charleston, South Carolina; Marshfield, Wisconsin; and Baltimore, Maryland

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.

Table I. Suspected disease entities with recommended biopsy type, site, and requested laboratory tests

Disease	Recommended biopsy technique	Comments
Autoimmune bullous diseases	H&E—Sectioned removal of intact bulla if possible, or broad sectionation of periphery of bulla DIF—Perilesional skin ≤1 cm from bulla	Avoid lower extremity when possible because of delayed healing and greater risk of false-negative results
Epidermolysis bullosa	Sectioned removal of intact bulla if possible, or broad sectionation of periphery of bulla	Blisters >72 hrs old should be avoided; a fresh blister can be induced in clinically uninvolved skin, near a site where the patient usually blisters. Topical anesthetics should be avoided because they may induce artificial blistering
Vasculitis	H&E—Punch or deep shave of well-established purpuric lesion (>72 hrs old) DIF—Punch or deep shave of acute lesion (<24 hrs old)	IgA vasculitis is more likely to retain positive DIF findings in established lesions
Panniculitis	Deep incisional biopsy	Punch biopsy specimens tend to fracture, leaving inflamed or necrotic fat behind. An electric kidney power punch can overcome this limitation. A 5-mm punch is the smallest size that should be divided for culture and H&E. The edge of a necrotic focus provides a high yield for culture and special stains. The skin surface should be prepped with alcohol and allowed to evaporate. Deliver the culture specimen to the desk that handles fungal and AFB specimens
Lupus and dermatomyositis	H&E—Punch biopsy of an established lesion (>6 months old) that is still active DIF—Punch biopsy of lesional skin; choose an established lesion (>6 months old) that is still active	
SJS/TEN vs SSSS	Shave or punch biopsy including the full thickness of the epidermis	Desquamating sheets of skin may constitute an adequate specimen
Scarring alopecia	H&E—≥4-mm punch biopsy of an established lesion (>6 months old) that is still active DIF—≥4-mm punch biopsy of lesional skin; choose an established lesion (>6 months old) that is still active	For all forms of alopecia, avoid the active advancing border. Established lesions are preferred. One specimen can be bisected transversely 1 mm above the dermal/SQ junction, or it can be submitted intact for the laboratory to section transversely or with the Horizontal or Tyler techniques. One specimen can be bisected vertically—half submitted in Michel medium for DIF and half added to the formalin bottle containing the transversely bisected or intact specimen
Nonscarring alopecia	For pattern alopecia or telogen effluvium—3-4-mm punch biopsy of an established area of alopecia For alopecia areata or syphilis—≥4-mm punch biopsy of an active lesion of recent onset is preferred.	If pattern alopecia or telogen effluvium is suspected, the specimen can be bisected transversely 1 mm above the dermal/SQ junction, or it can be submitted intact for the laboratory to section transversely or with the Horizontal or Tyler techniques. For other forms of nonscarring alopecia, the specimen should be submitted intact
BCC/SCC	Shave or punch biopsy of adequate depth to show the invasive pattern and detect perineural invasion if present	In convex sites or thin facial skin, more superficial shave biopsy specimens may be appropriate. The skin should be pulled taught to provide greater control over depth. Avoid creating contour defects in sebaceous skin
Suspected melanoma	Complete excisional removal whenever possible	This may take the form of a sectionable
DFSP	Deep incisional biopsy	

Billing/coding

Code	Description
11102	Tangential biopsy of skin (e.g., shave, scoop, saucerize, curette) single lesion
+11103	each separate/additional lesion (List separately in addition to code for primary procedure)
11104	Punch 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)

Dermatology in the Primary Care Setting

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

Dermatology in the Primary Care Setting

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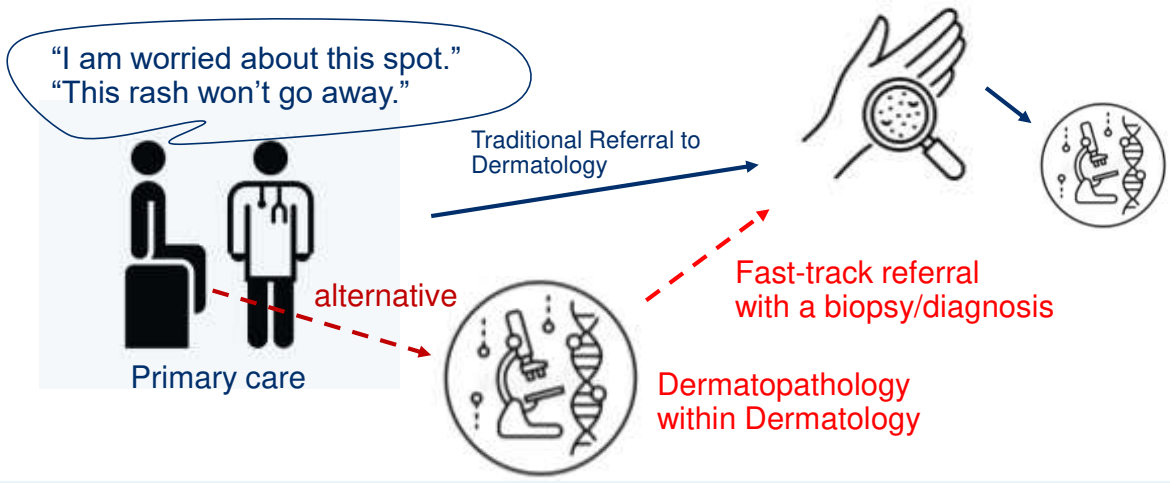

Thank you for your attention.

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Contact Dermatitis Institute (www.contactdermatitisinstitute.com