Papules, Patches and Plaques

Common Dermatologic Conditions in Primary Care

AAPA We Are Family Medicine 2024 Conference

Phoenix, Arizona

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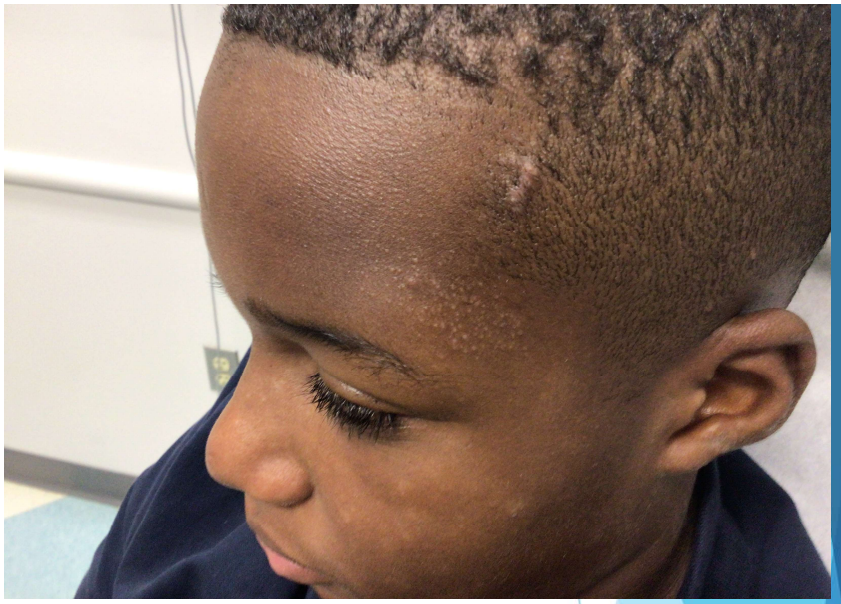
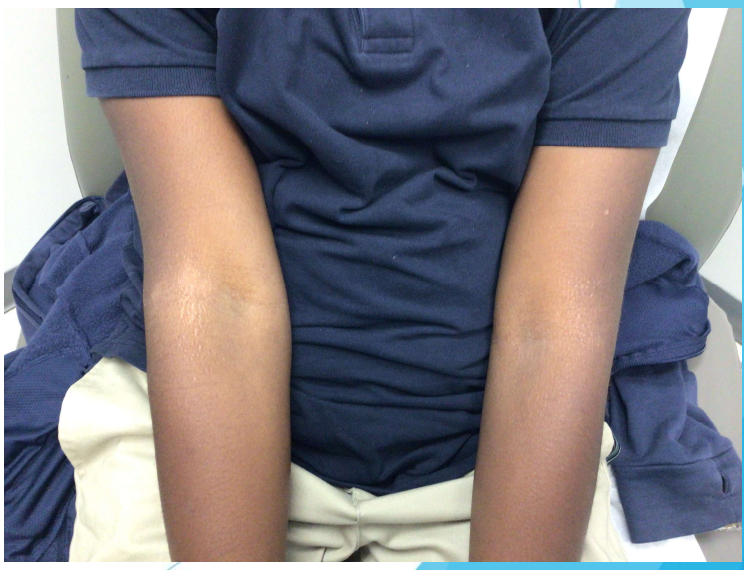
- NON-DECLARATION STATEMENT:
  - *I have no relevant relationships with ineligible companies to disclose within the past 24 months.*

Educational Objectives

- Case-based approach to the most common dermatologic conditions from a primary care perspective. The lecture will identify what the primary care provider can do and when to refer to specialist.
- At the conclusion of this session, participants should be able to:
  1. Analyze treatment approaches for common and complex skin conditions
  2. Implement updated strategies for managing atopic dermatitis, psoriasis, acne, contact dermatitis and verruca vulgaris
  3. Integrate into daily practice evidence-based recommendations on therapies for common dermatologic conditions

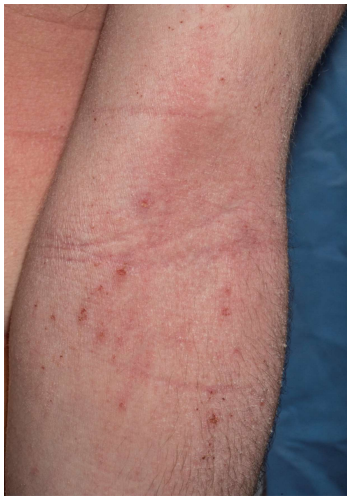
12 y/o
Presents to office with mother for evaluation of discoloration and itch

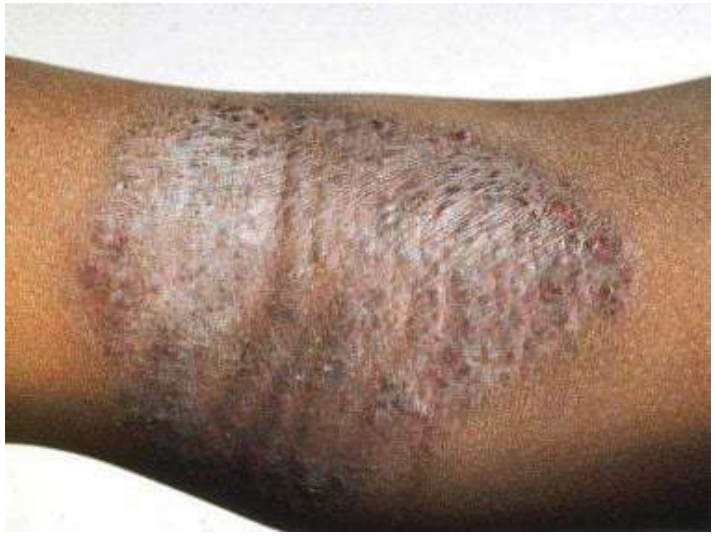


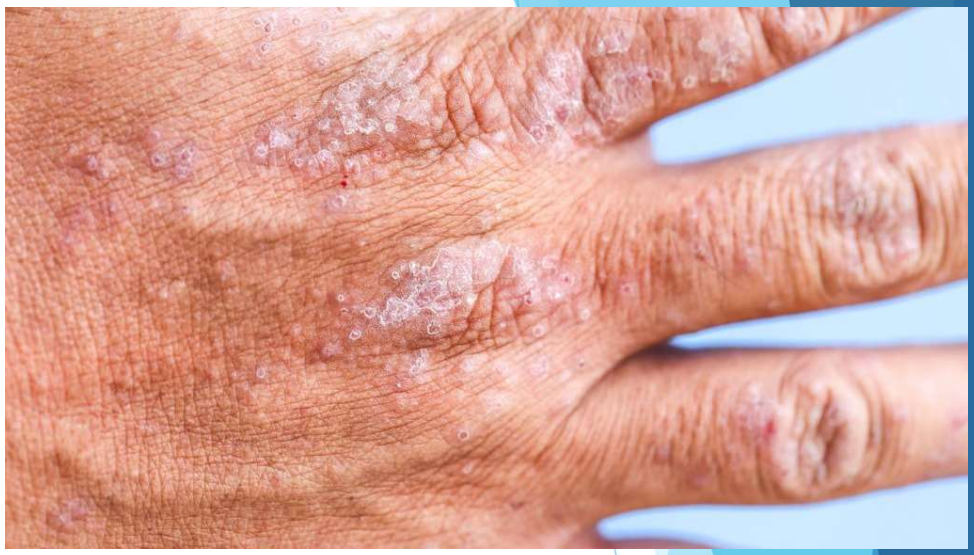


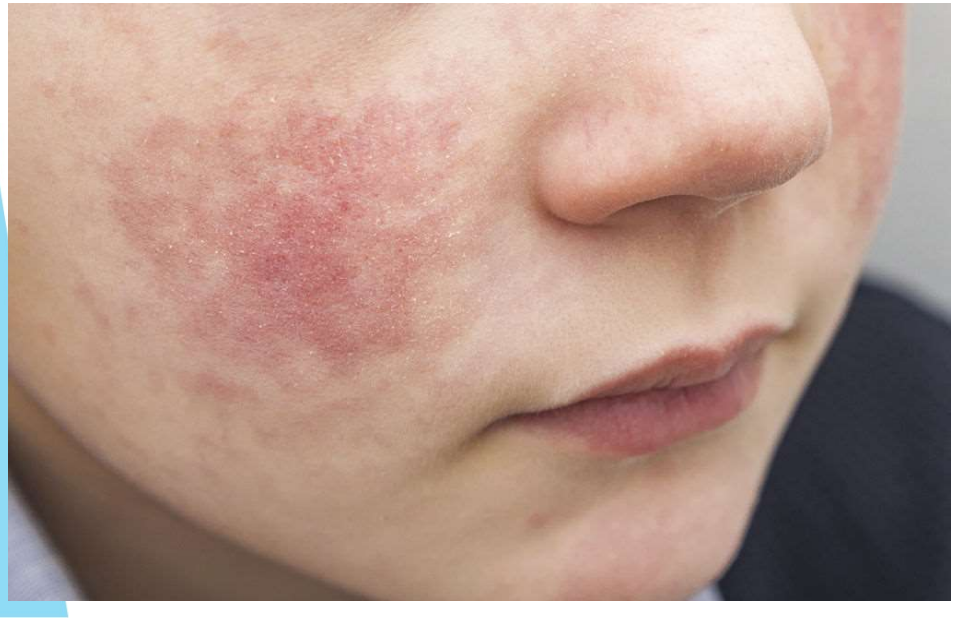
Atopic Dermatitis

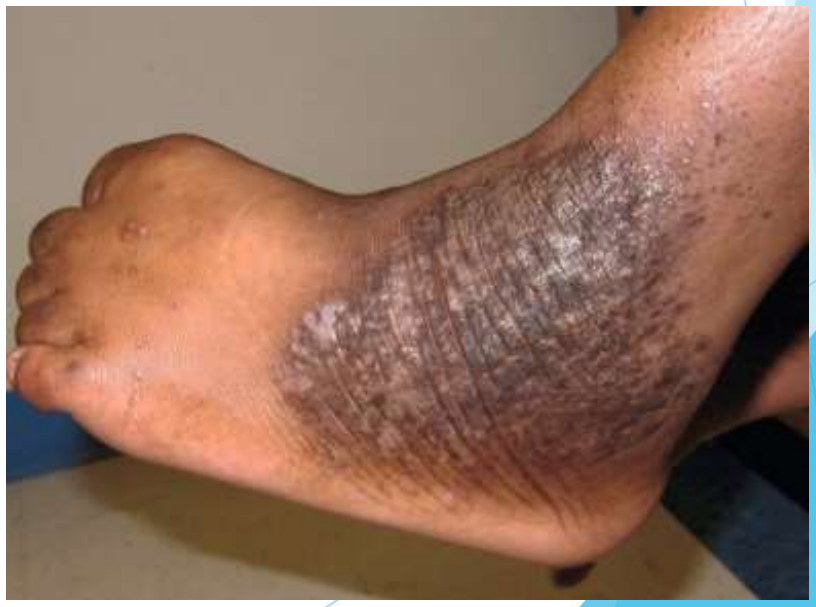
- An acute, subacute, or chronic relapsing skin disorder that usually begins in infancy and is characterized principally by dry skin and pruritis
- Common inflammatory skin disease that affects 10-25% of children and 2-10% of adults
- Characterized by intense pruritis and a chronically relapsing course
- Types of pruritic, eczematous lesions:
  - Acute: edematous, erythematous papules and plaques with +/- vesiculation, oozing and crush
  - Subacute: erythematous patches or plaques with scaling and variable crusting
  - Chronic: thickened plaques with lichenification and scaling
- DDX: Seborrheic dermatitis, contact dermatitis, psoriasis, nummular eczema, asteatotic eczema, lichen simplex chronicus, etc.





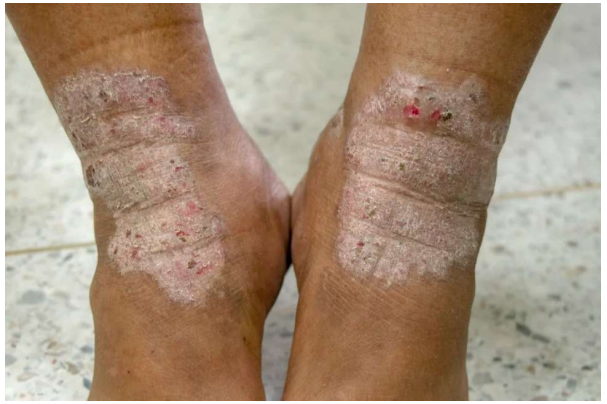


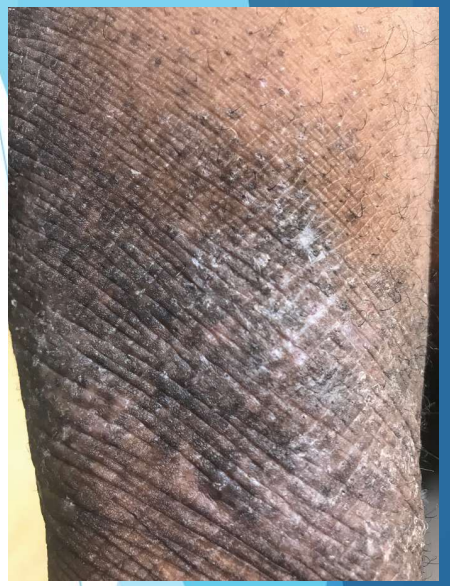


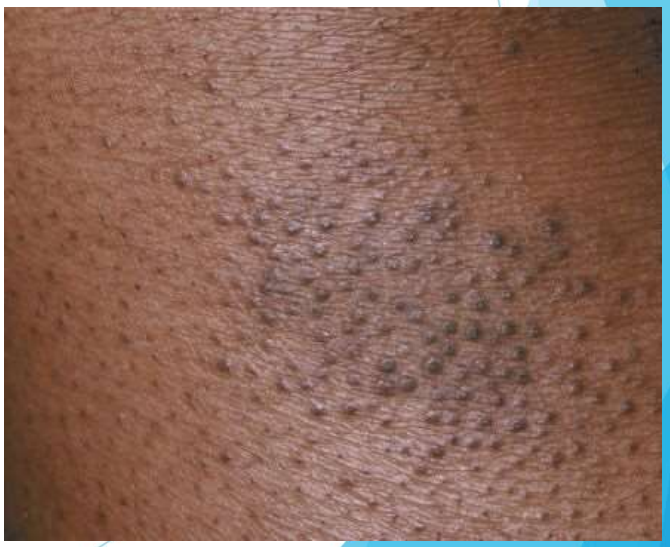


AD: Skin of Color

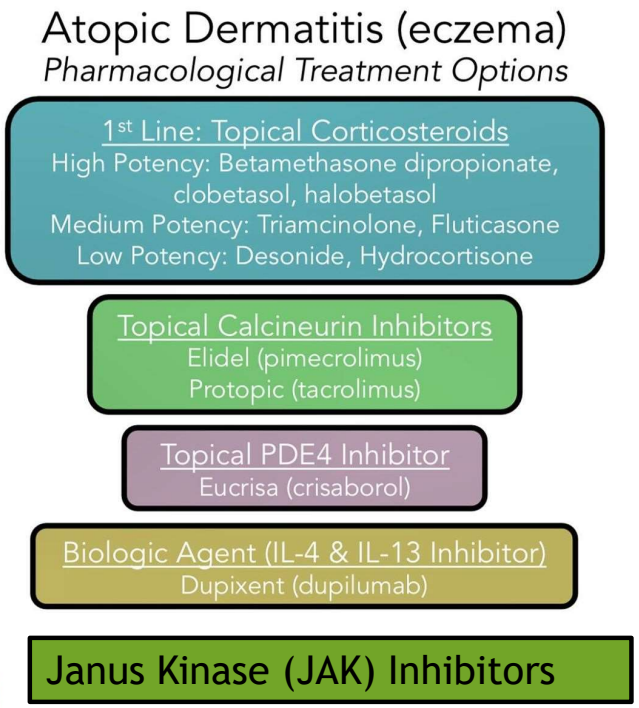
- People of African descent have the highest atopic dermatitis prevalence and severity
- Key features in people with darker skin tones:
  - Follicular prominence
  - Papular morphology
  - Prurigo nodularis
  - Hyperpigmented, violaceous-brown, or gray plaques, instead of erythematous plaques
  - Lichenification
  - Treatment resistance
- Post-inflammatory hyperpigmentation and post-inflammatory hypopigmentation may be more distressing than AD itself





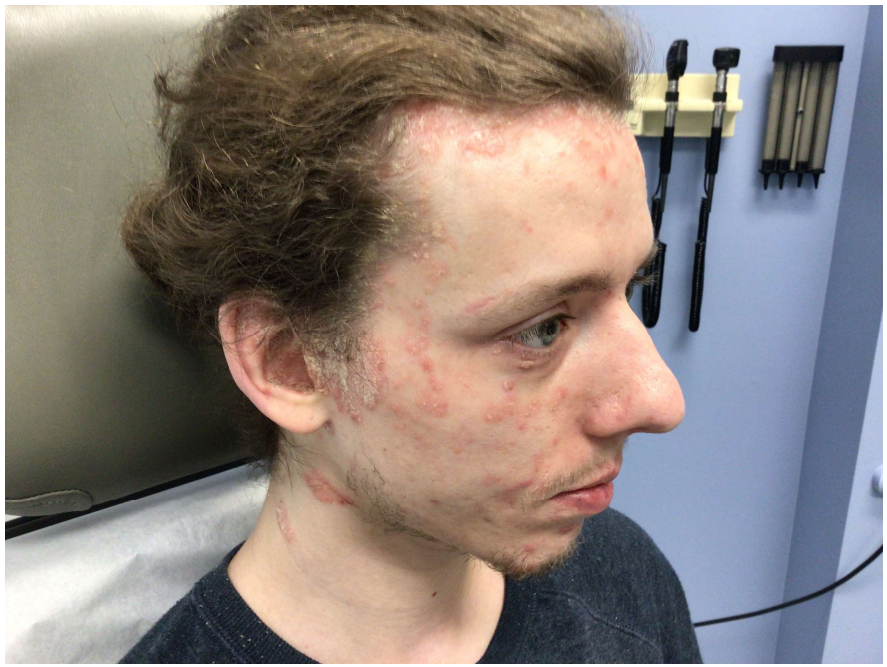


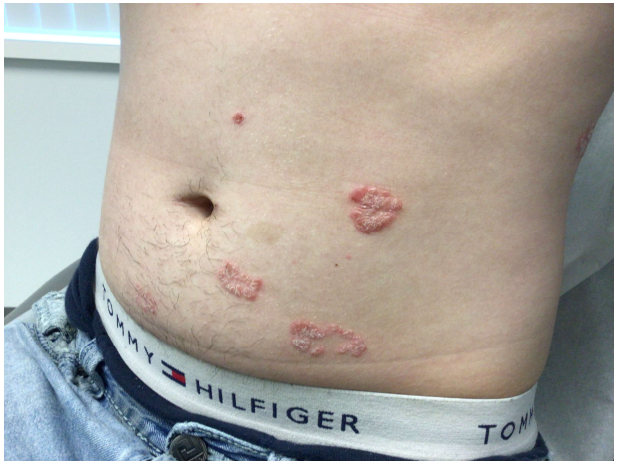
Treatment of Atopic Dermatitis

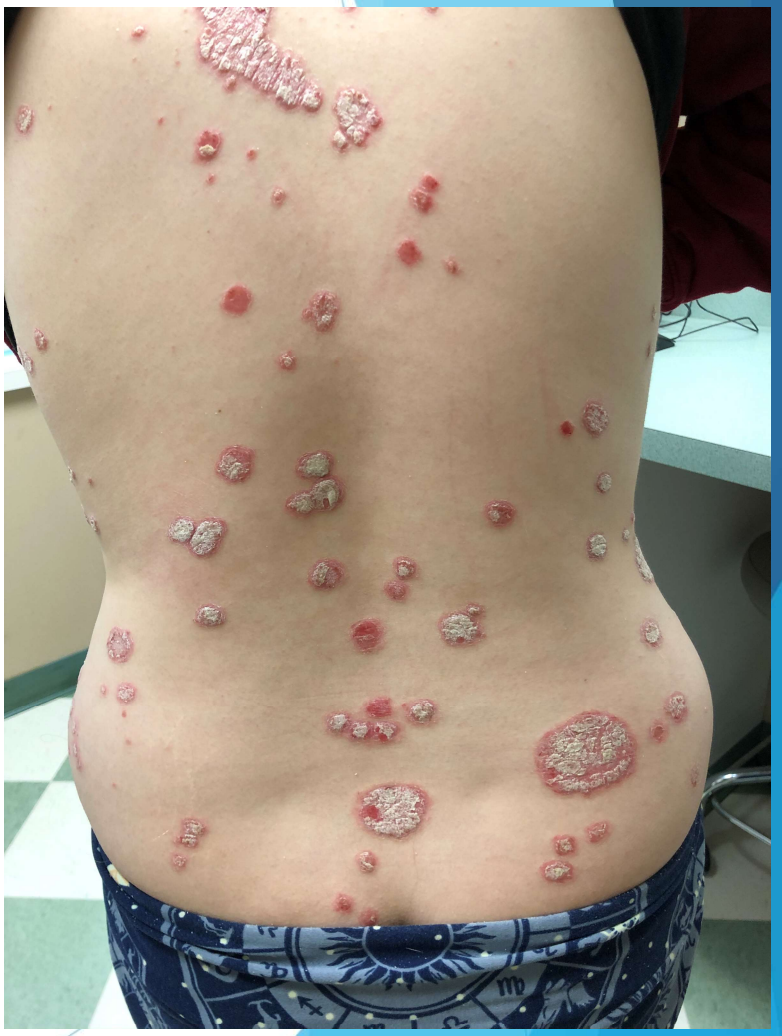


- Two major components to management of AD:
  - Treatment of active dermatitis with anti-inflammatory agent(s)
  - Maintenance designed to improve skin barrier function, control subclinical inflammation, and avoid trigger factors
- First Line
  - Topical corticosteroid of appropriate strength
    - Applied twice daily until eczema clears
    - Tap water compresses or wet wraps followed by corticosteroid application can speed improvement of acute flares
- Second Line
  - Topical calcineurin inhibitors are helpful for thinner lesions on the face and intertriginous areas

22 y/o
Presents to office with worsening rash present for 6mos





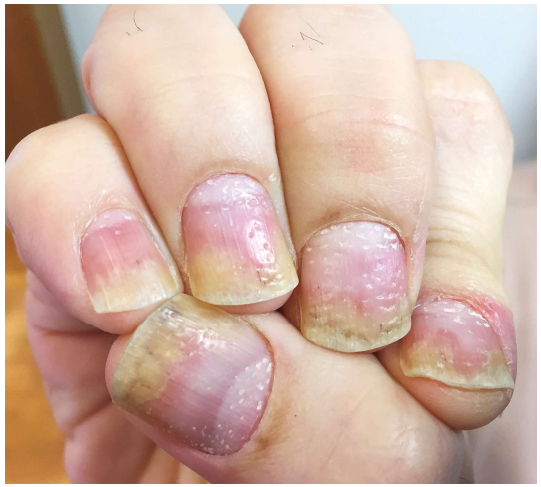


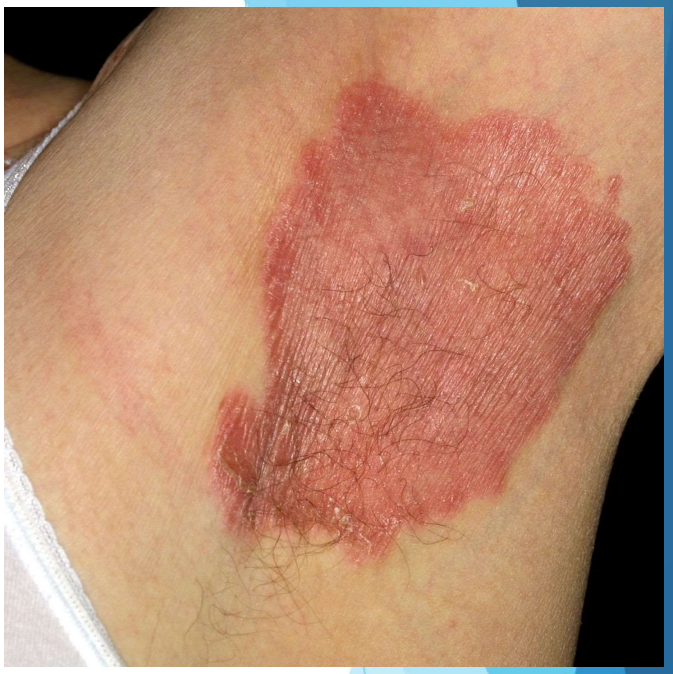
Psoriasis vulgaris

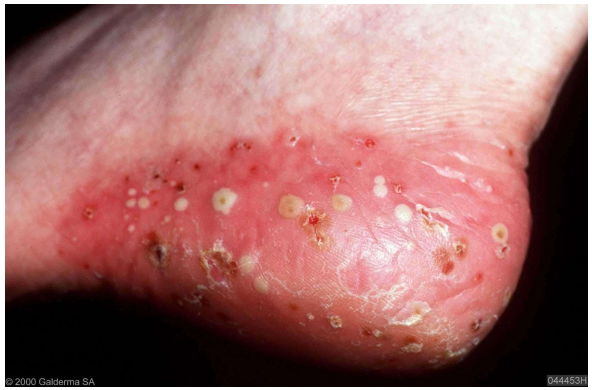
- A common, chronic, and recurrent inflammatory disease of the skin characterized by circumscribed, erythematous, dry, scaling plaques of various sizes; lesions are usually covered in silvery white scales
- Common sites: scalp, elbows, knees, nails, hands, feet, trunk (intergluteal folds)
- Keratinocytes proliferate at 28x the normal rate, leading to the characteristic pathognomic plaque lesions
- Assessing psoriasis: Patient's hand (palm + fingers + thumb) = 1% BSA
  - Mild - <3% BSA
  - Moderate – 3-10% BSA
  - Severe - >10% BSA
- DDX: Atopic dermatitis, Contact dermatitis, Seborrheic dermatitis, Tinea, Candidiasis, Pityriasis rosea, Drug eruption, Cutaneous T-cell lymphoma, Pityriasis rubra pilaris (PRP), LSC, 2° or 3° syphilis

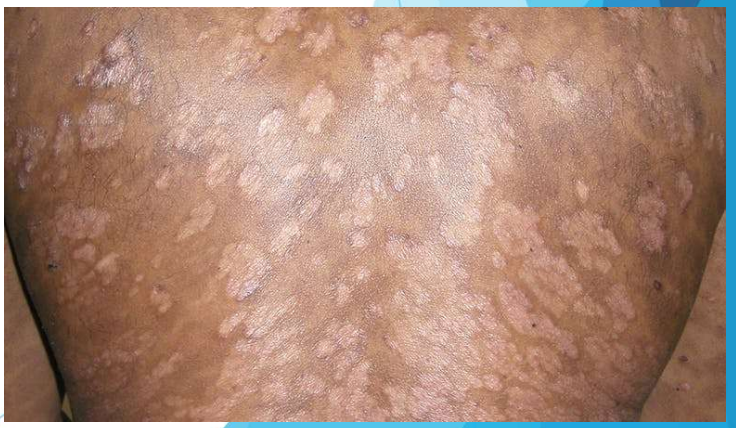
Variants of Psoriasis

- Plaque psoriasis
- Guttate psoriasis
- Pustular psoriasis (generalized, localized)
- Inverse psoriasis
- Erythrodermic psoriasis
- Nail psoriasis

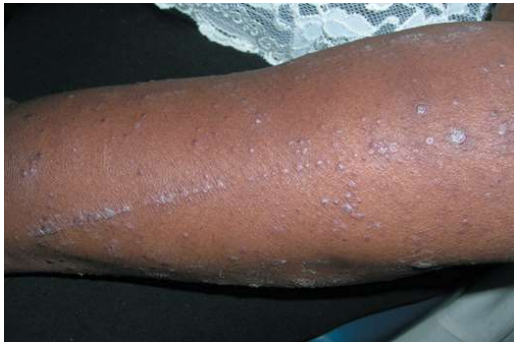


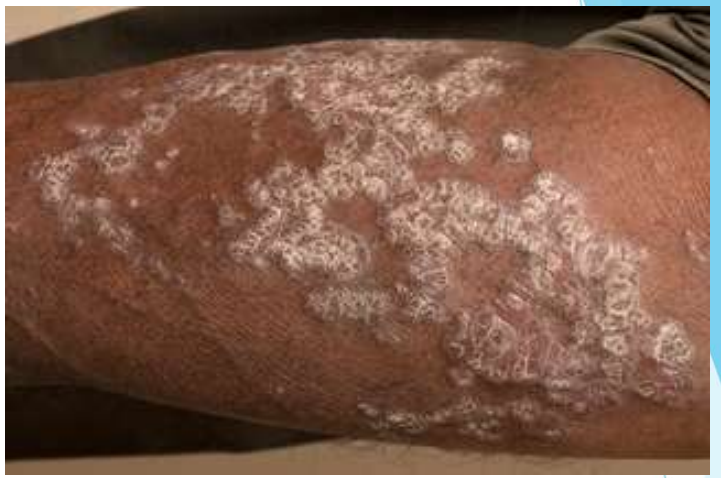


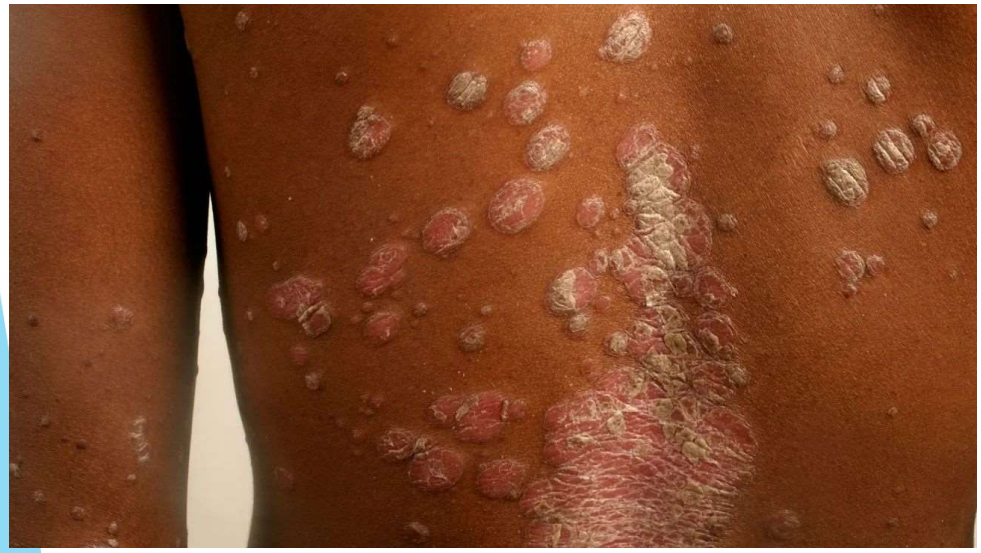




Psoriasis: Skin of Color

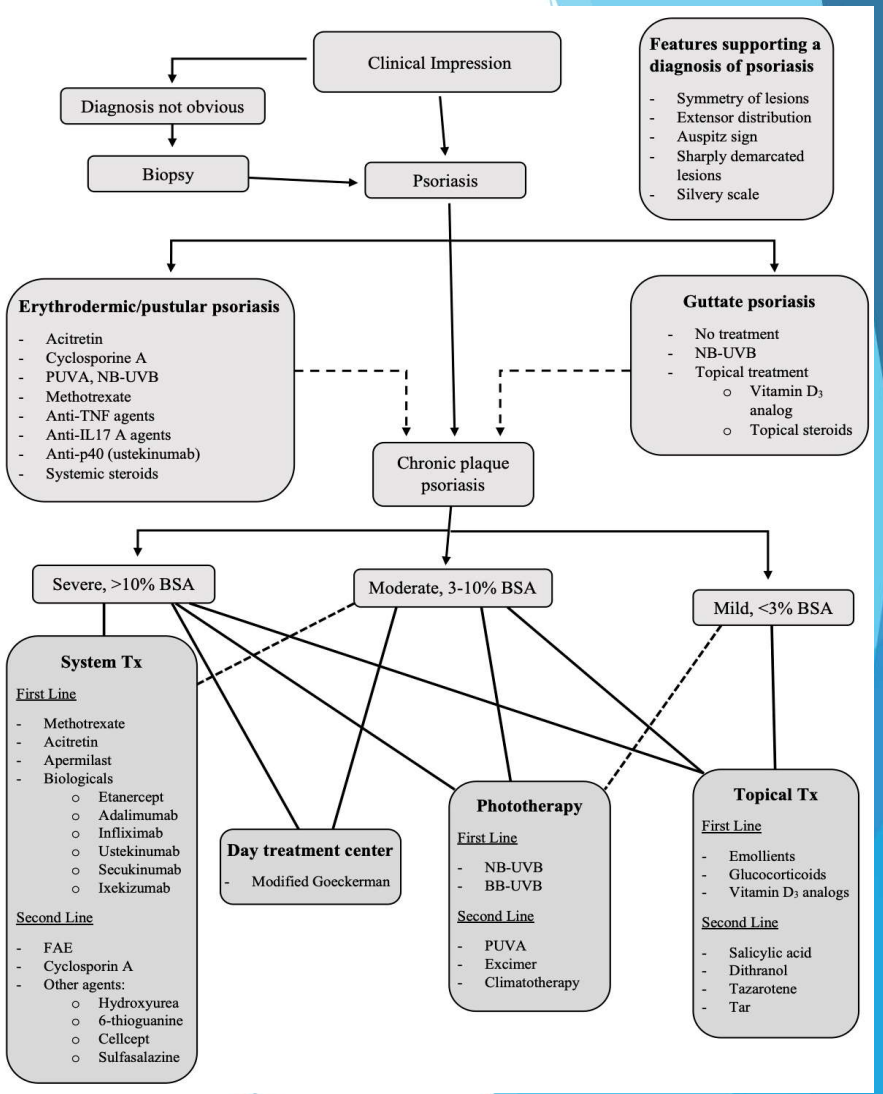






Treatment of Psoriasis

- Multiple topical, systemic and biologic therapies are available for the treatment of psoriasis as well as phototherapy
- Treatment is individualized based on extent of disease, pts perception of severity of disease and side effects potential as specific treatments
- Approximately 40% of pts are frustrated with the ineffectiveness of their current treatment



BIOLOGIC AGENT	LOADING DOSE	MAINTENANCE DOSE
Etanercept/Enbrel™	50mg subcutaneous injection twice weekly for 12 weeks	50 mg subcutaneous injection once per week
Infliximab/Remicade™	5mg/kg IV infusion administered in week 0, week 2, and week 6	5mg/kg IV infusion administered every 8 weeks (time interval can be modified and dose per kg can be increased according to the patient's response.)
Adalimumab/Humira™	80mg subcutaneous injection (2 x 40mg at the initial dose), followed by a 40mg subcutaneous injection 1 week later	40mg subcutaneous injection every 2 weeks
Certolizumab/Cimizia™	400mg subcutaneous injection (Alternative regime, for patients who weigh <90kg: 400mg initially and at week 2 and week 4)	400mg subcutaneous injection every other week (Alternative regime, 200 mg subcaneous injection every other week)
Ustekinumab/Stelera™	For patients weighing ≤100 kg: 45mg administered subcutaneously initially and 4 weeks later (For patients weighing >100kg: 90mg administered subcutaneously initially and 4 weeks later)	Patients ≤100kg: 45mg administered subcutaneously every 12 weeks (Patients >100kg: 90mg administered subcutaneously every 12 weeks)
Secukinumab/Cosentyx ™	300mg subcutaneous injection at week 0, week 1, week 2, week 3, and week 4	300mg subcutaneous injection every 4 weeks
Ixeizumab/Taltz™	160mg subcutaneous injection followed by 80mg on week 2, week 4, week 6, week 8, week 10, and week 12	80mg subcutaneous injection every 4 weeks
Brodalumab/Siliq™	210mg subcutaneous injection on week 0 , week 1, and week 2	210mg subcutaneous injection every 2 weeks
Guselkumab/Tremfya™	100mg subcutaneous injection on week 0 and week 4	100mg subcutaneous injection every 8 weeks
Tildrakizumab/Ilumya™	100mg subcutaneous injection initially and 4 weeks later	100mg subcutaneous injection every 12 weeks
Risankizumab/Skyrizi™	150mg subcutaneous injection week 0 and week 4	150 mg subcutaneous injection every 12 weeks

Biologic Therapies for Moderate to Severe Psoriasis

	TNF-α inhibitor	IL-12/IL-23 inhibitor	IL-17 inhibitor	IL-23 inhibitor
Drug	etanercept/Enbrel™	ustekinumab/Stelara™	secukinumab/Cosentyx™	guselkumab/Tremfya
	infliximab/Remicade™		ixekizumab/Taltz™	tildrakizumab/Ilumya
	adalimumab/Humira™		brodalumab/Siliq™	risankizumab/Skyrizi
	certolizumab/Cimzia™			
Baseline Monitoring	CBCD	CBCD	CBCD	CBCD
	CMP	CMP	CMP	CMP
	Hepatitis B & C serologies	Hepatitis B & C serologies	Hepatitis B & C serologies	Hepatitis B & C serologies
	HIV (provider discretion)	HIV (provider discretion)	HIV (provider discretion)	HIV (provider discretion)
	Latent TB screen (PPD, T-spot, or Quantiferon Gold)	Latent TB screen (PPD, T-spot, or Quantiferon Gold)	Evaluation for IBD prior to initiation	Latent TB screen (PPD, T-spot, or Quantiferon Gold)
			Latent TB screen (PPD, T-spot, or Quantiferon Gold)	
Ongoing Monitoring	Yearly skin cancer screening	Yearly skin cancer screening	Yearly skin cancer screening	Yearly skin cancer screening
	Yearly latent TB screening (PPD, T-spot, or Quantiferon Gold)	Yearly latent TB screening (PPD, T-spot, or Quantiferon Gold)	Exacerbation or new onset IBD	Yearly latent TB screening (PPD, T-spot, or Quantiferon Gold)
			Yearly latent TB screening (PPD, T-spot, or Quantiferon Gold)	
	infliximab: requires LFTs every 3 months until stable than every 6-12 months	CBC & CMP (provider descretion)	Siliq: assess for suicidal ideation	Follow Up: 4-6 months
	Follow Up: 4-6 months	Follow Up: 4-6 months	Follow Up: 4-6 months; Siliq requires more frequent follow up	
Adverse Events	Multiple Sclerosis - rare	Hypersensitivity reaction	Increased liver transaminases	Increased liver transaminases
	Hepatotoxicity		Small risk of IBD	
	Drug-induced reversible lupus erythematosus		Rare cases of neutropenia	
	Exacerbation or new onset of CHF		Suicidal Ideation: brodalumab	
	Cytopenia		*Candida* infection	
Contraindications	Untreated Hepatitis B infection	Untreated Hepatitis B infection	Active history IBD	Allergy to drug
	History of lymphoreticular malignancy	History of lymphoreticular malignancy	Presence of suicidal ideation: brodalumab	
	Active TB infection	Active TB infection		
	Significant CHR or pre-existing MS			
	Allergy to the drug	Allergy to the drug	Allergy to the drug	
Pregnancy/Lactation	SAFE in pregnancy and lactation	No Data	No human data	No Data

Treating patients with moderate-to-severe psoriasis vulgaris

Timothy R. Kessler, DMSc, PA-C

ABSTRACT

Psoriasis vulgaris is a common inflammatory disease of adults and children. Affected patients often are incorrectly diagnosed, undertreated, or not treated at all. The relapsing course of psoriasis negatively affects a patient's quality of life. The condition is associated with social isolation, anxiety, and depression, and can harm personal relationships and employment status. Psoriasis may have a significant psychologic and socioeconomic effect throughout a patient's life. Skin involvement is the most prominent symptom of this disease; however, understanding that psoriasis is a chronic, multisystem inflammatory disease is essential to proper treatment. Patients with mild-to-moderate psoriasis can control their disease primarily with topical medications or phototherapy. However, when used as monotherapy or combined with phototherapy, topical medication can be inadequate to treat moderate-to-severe psoriasis. Biologic agents offer treatment options with many benefits for controlling psoriasis vulgaris, whether given as monotherapy or combined with topical or systemic medications.

Keywords: psoriasis vulgaris, skin disease, biologic therapy, interleukin-19, interleukin-23, inflammatory

Learning objectives

- Describe the clinical presentation and diagnosis of psoriasis vulgaris.
- Describe the treatment of moderate-to-severe psoriasis vulgaris.
- Outline the screening and maintenance process for biologic therapy and the associated adverse reactions.

Psoriasis vulgaris is a common, immune-mediated inflammatory disease characterized by inflammation of the skin, epidermal hyperplasia, risk of painful and destructive arthritis, cardiovascular morbidity, and psychologic challenges.[1] Psoriasis affects about 3.2% of the US population, but its cause remains unknown.[2] The mode of inheritance for psoriasis vulgaris is described as multifactorial, consisting of polygenetic components and environmental factors. Psoriasis may begin at any age, although it is unusual before age 10 years. It most commonly appears between ages 15 and 30 years.

Timothy R. Kessler practices in dermatology at Pinnacle-Silverton Skin Institute in Grand Blanc, Mich. The author has disclosed no potential conflicts of interest, financial or otherwise.

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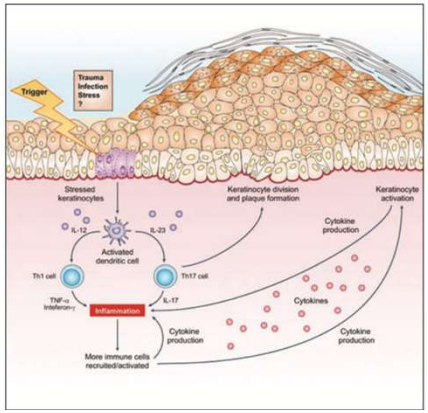


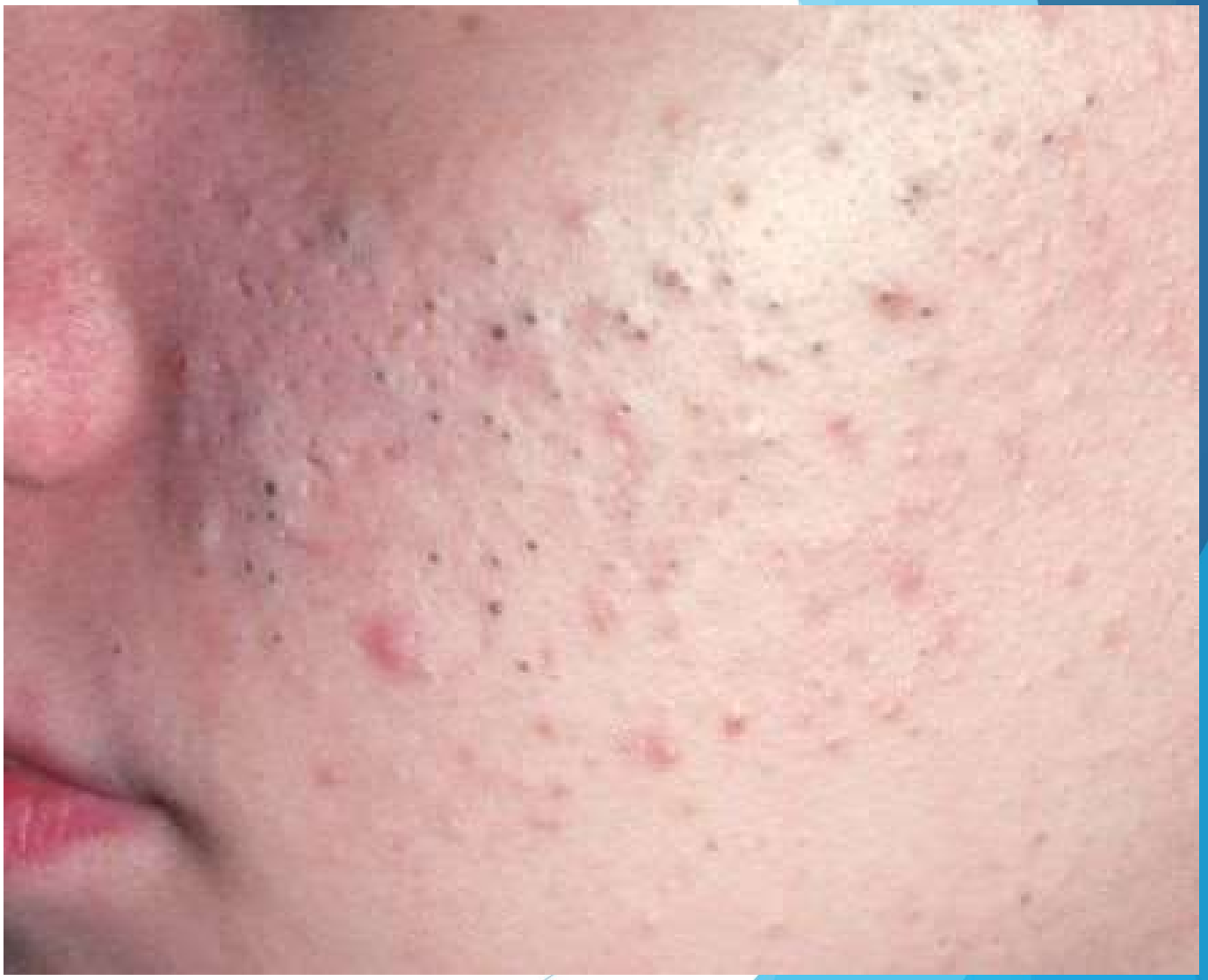
FIGURE 1. Pathogenesis of psoriasis. Stress or damage to the skin can trigger the inflammatory response. Stressed keratinocytes activate dendritic cells that pass on fragments of the microbe to T cells, which in turn release cytokines that play a part in keratinocyte activation and ongoing inflammation.

Reprinted with permission from Young M, Aldredge L, Parker P. Psoriasis for the primary care practitioner. *J Am Assoc Nurse Pract.* 2017;29(3):157-178.

PATHOGENESIS

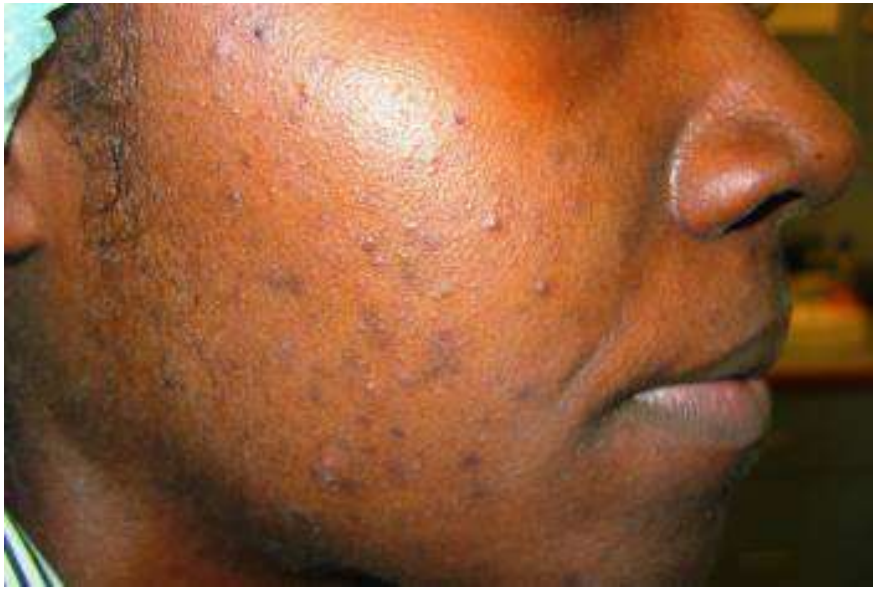
A combination of genetic and environmental factors initiates the pathogenesis of psoriasis through a complex process, prompting tumor necrosis factor-alpha (TNF-alpha) production by keratinocytes that activate dendric cells.[3] The triggered dendritic cells provide interleukin-23 (IL-23), which gives rise to helper T cell (TH17) differentiation.[4] TH17 cells produce interleukin-17A (IL-17A), promoting psoriatic skin changes (**Figure 1**).[5]

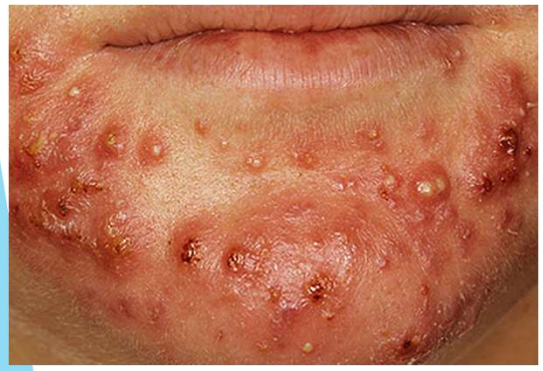
13 y/o female
Presents to office for evaluation of pimples present off and on for 6mos. Worsening.

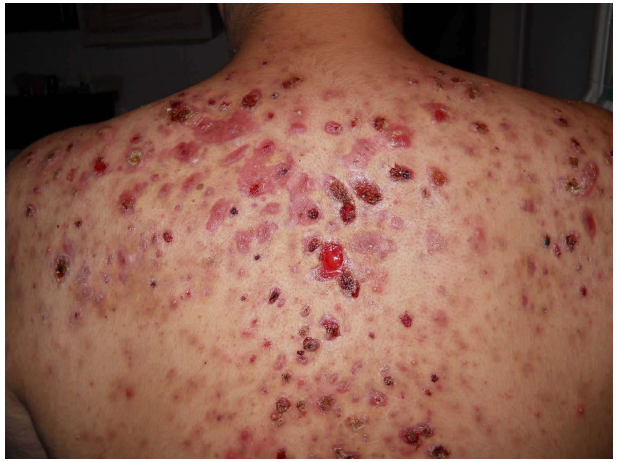


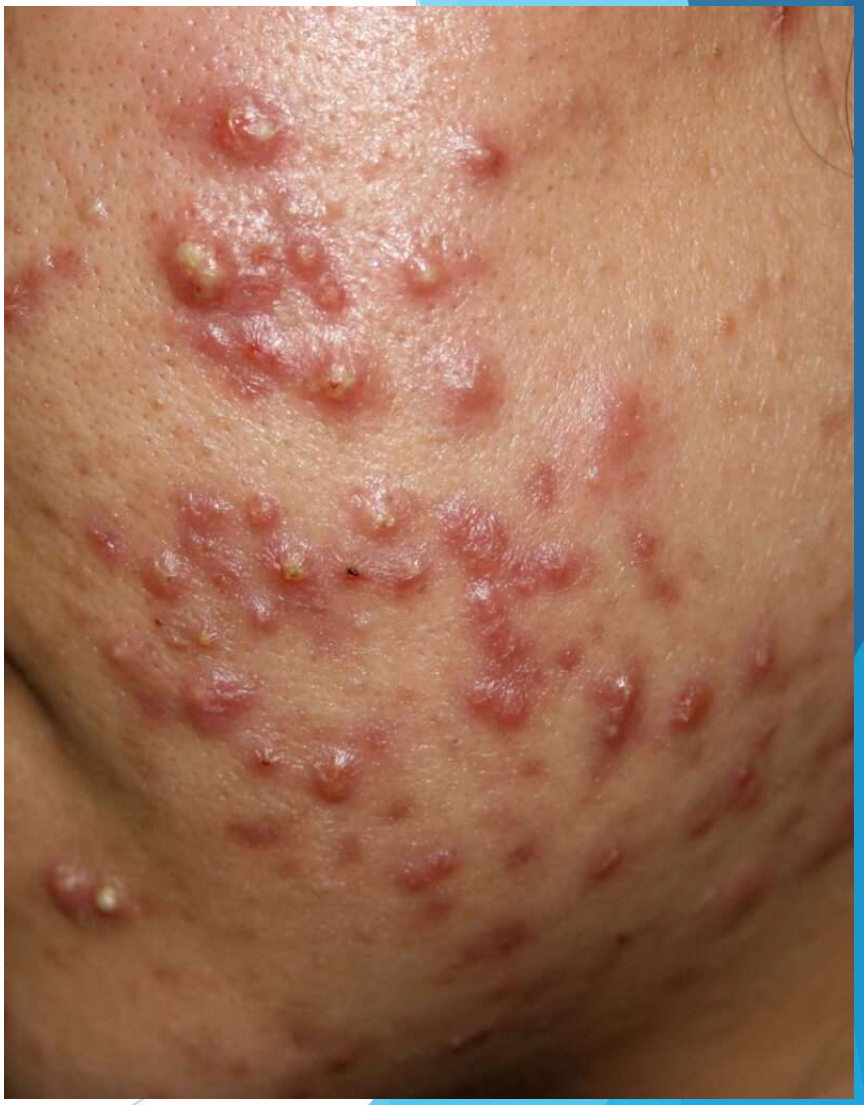
Acne vulgaris

- Disorder of pilosebaceous unit
- Occurs in
  - **85%** of people 12-24 years old
  - **15-35%** of people in 30s-40s (especially women)
- Clinical presentations range from mild comedones to severe, explosive eruptions of suppurative nodules
- Lesions favor the face, neck, upper trunk and upper arms
- DDX: Pseudoacne, Milia, Sebaceous hyperplasia, Syringoma, Flexural comedone, Dilated pore of Winer, Favre-Racouchot, Molluscum , Rosacea, Periorificial dermatitis, Folliculitis, Pseudofolliculitis barbae, Acne keloidalis nuchae, Keratosis pilaris, Follicular mucinosis

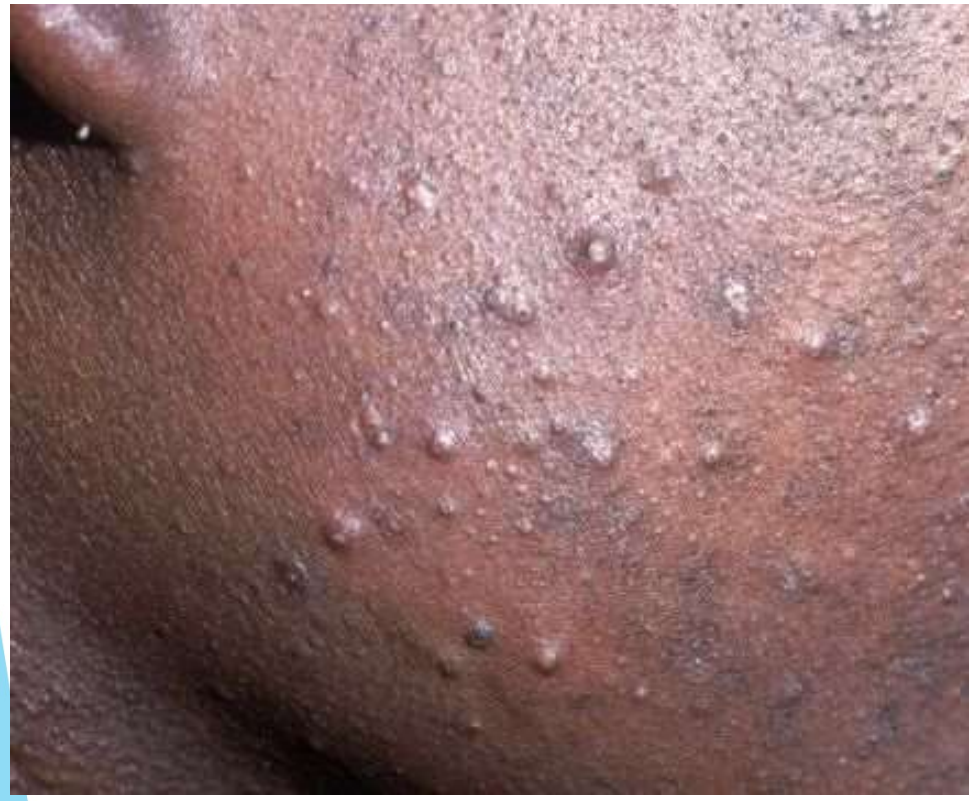


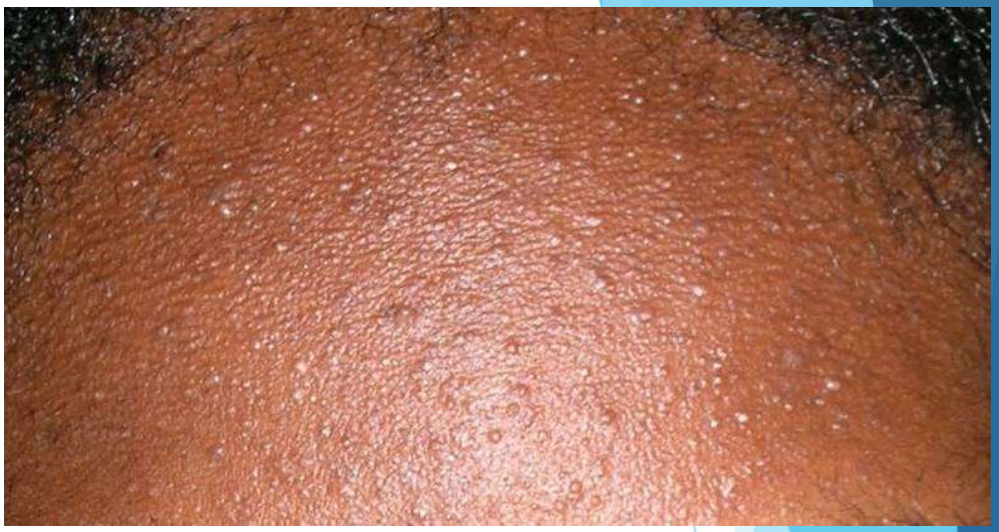


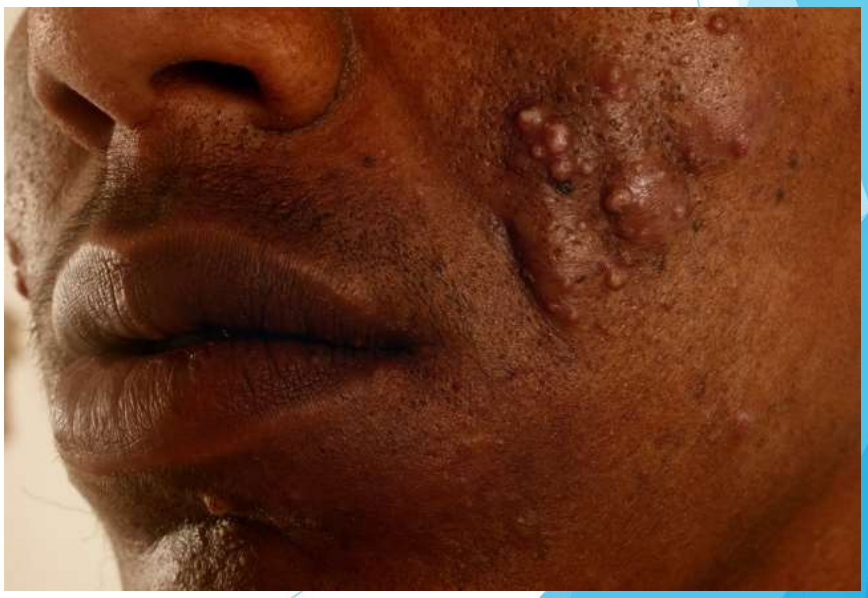




AV: Skin of Color







Treatment of **MILD** Acne Vulgaris

Comedonal

- First Line
  - Topical retinoid (tretinoin, adapalene, tazarotene, trafarotene)
- Second Line
  - Alt. topical retinoid
  - Azelaic acid
  - Salicylic acid
  - Clascoterone
- Procedural options
  - Comedo extraction
- Maintenance
  - Topical retinoid

Mostly inflammatory

- First Line
  - Topical antimicrobial (BPO and/or antibiotic clindamycin, erythromycin, sodium sulfacetamide/sulfur) + topical retinoid
- Second Line
  - Alt. topical retinoid + antimicrobial
  - Azelaic acid
  - Topical dapsone
  - Clascotersone
- Maintenance
  - Topical retinoid ± BPO

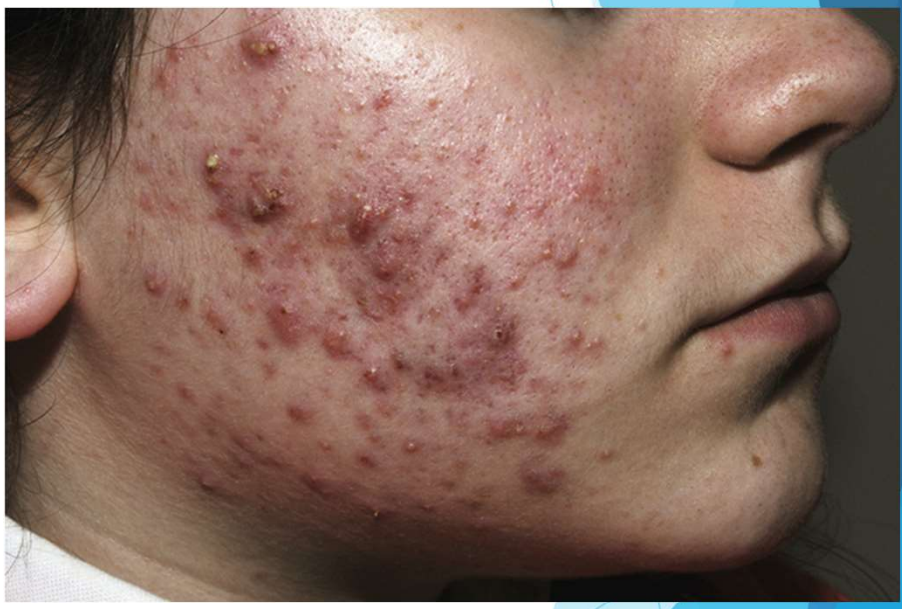
Treatment of **MODERATE** Acne Vulgaris

- First line
  - Oral antibiotic + topical retinoid ± BPO
- Second Line
  - Alt. oral antibiotic + alt. topical retinoid ±BPO/azelaic acid
  - Oral isotretinoin (if nodular, scarring or recalcitrant)
- Options for females
  - OCP/antiandrogen
- Procedural options
  - Comedo extraction
  - Intralesional steroid (2-5mg/ml)
- Maintenance
  - Topical retinoid ± BPO

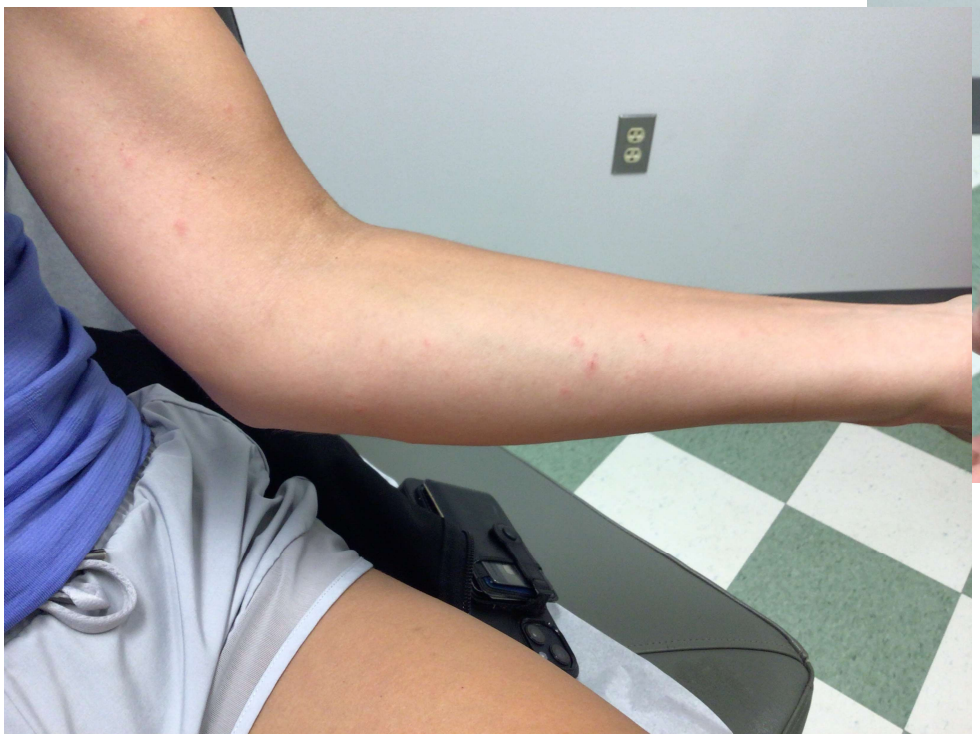
- Oral Antibiotics
  - First Line
  - Tetracycline derivatives
    - Doxycycline
    - Minocycline
    - Sarecycline
  - Alternative
    - Azithromycin
    - Trimethoprim-sulfamethoxazole
- OCPs
  - Ortho Tri-Cyclen®, Yaz®/Loryna®/Nikki®/Melamisa®/Lo-Zumandimine®, Estrostep®
- Antiandrogens
  - spironolactone

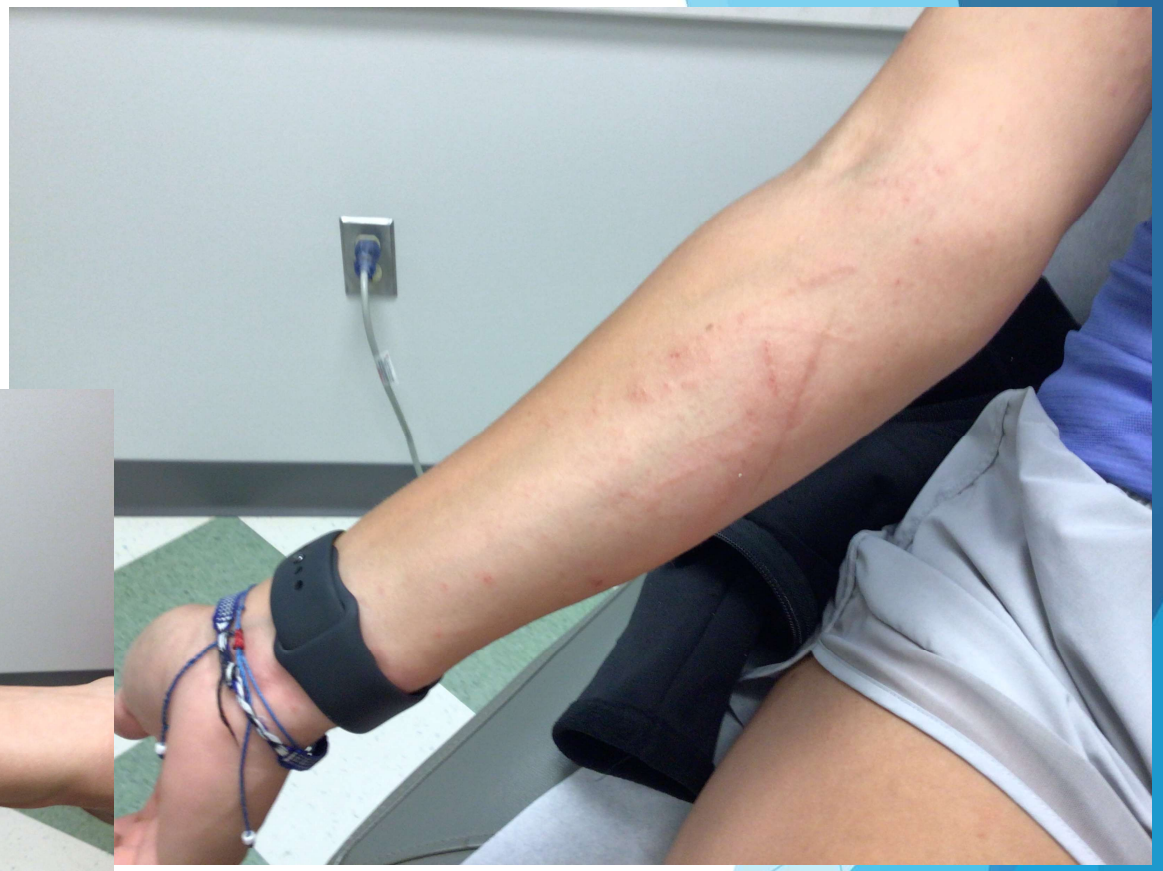
Treatment of **SEVERE** Acne Vulgaris

- First line
  - Oral isotretinoin (+oral steroid for acne fulminans)
- Second Line
  - Oral dapsone
  - High-dose oral antibiotic + topical retinoid + BPO
- Options for females
  - OCP/antiandrogen
- Procedural options
  - Comedo extraction
  - Intralesional steroid (2-5mg/ml)
- Refractory to treatment
  - Exclude gram-negative folliculitis
  - Female patient: exclude adrenal or ovarian dysfunction
  - Exclude use of anabolic steroid or other acne-exacerbating drugs
  - Compliance
- Maintenance
  - Topical retinoid ± BPO



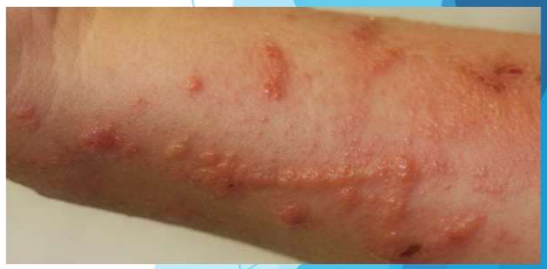
36 y/o
Presents to office with worsening rash present for 2ds. +itch



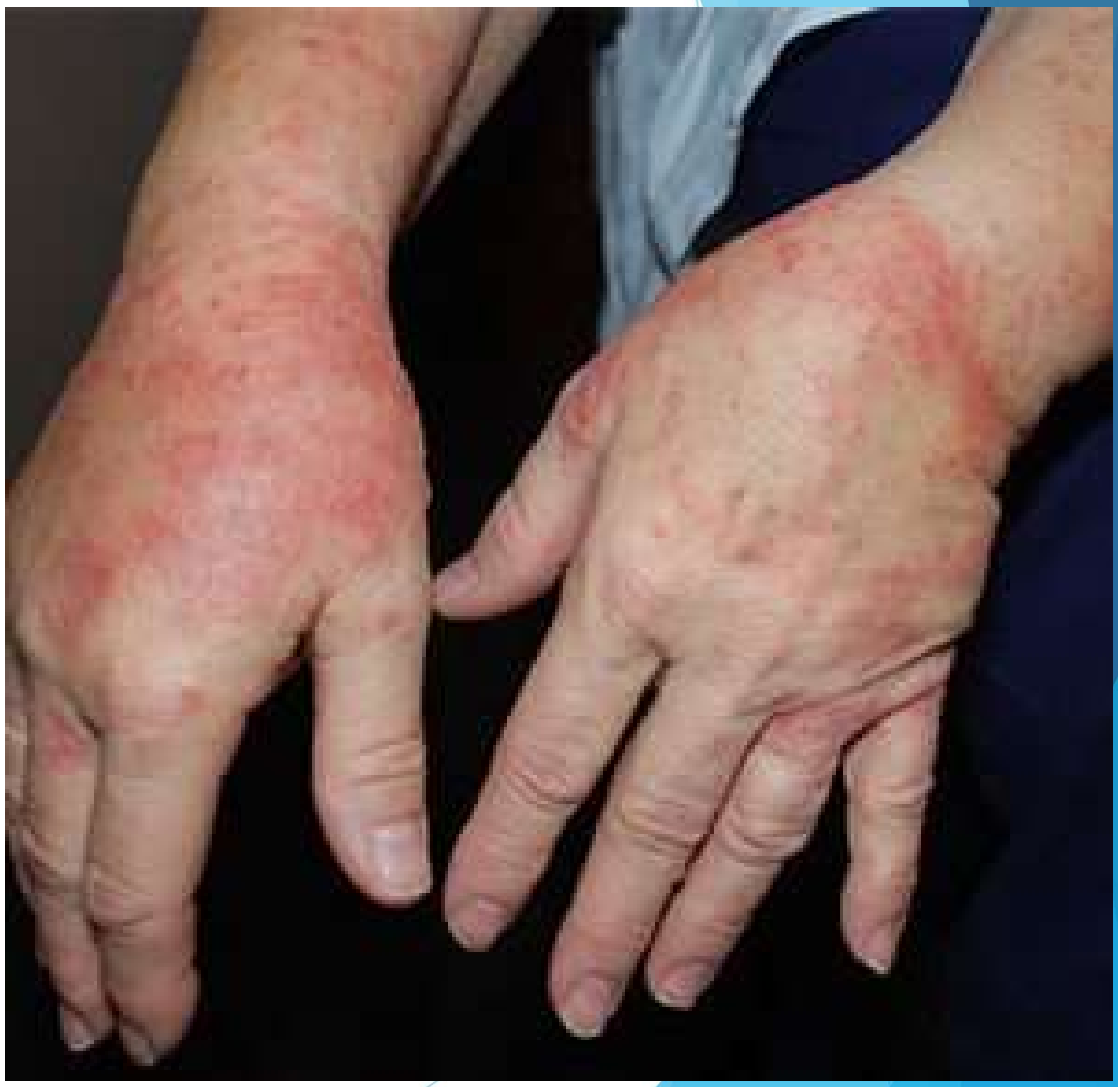


Allergic Contact dermatitis

- Plants containing Urushiol
- Allergen-laden smoke can create systemic contact dermatitis
- Sensitization occurs after initial exposure and may last 2-3 weeks
- Treatment should extend for a minimum of 2 weeks to prevent the rebound phenomenon from occurring
- DDX: ICD, Atopic dermatitis, Stasis dermatitis, Seborrheic dermatitis, Rosacea, Dermatophyte infection
- Treatment
  - Topical and/or systemic corticosteroids can be utilized acutely to resolve the inflammatory process associated with the reaction
  - Patient Education
    - Educate and caution subsequent exposure to allergen and potential for increased severity of reaction

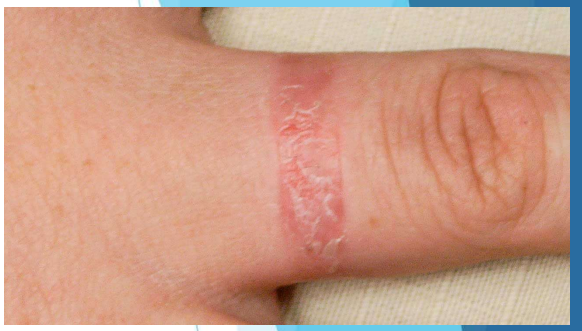


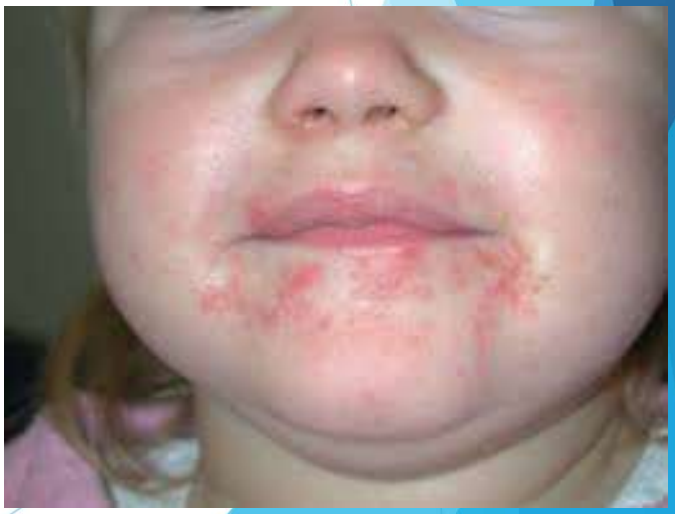
57 y/o
Presents to office with worsening rash on hands present for 4mos.



Irritant Contact Dermatitis

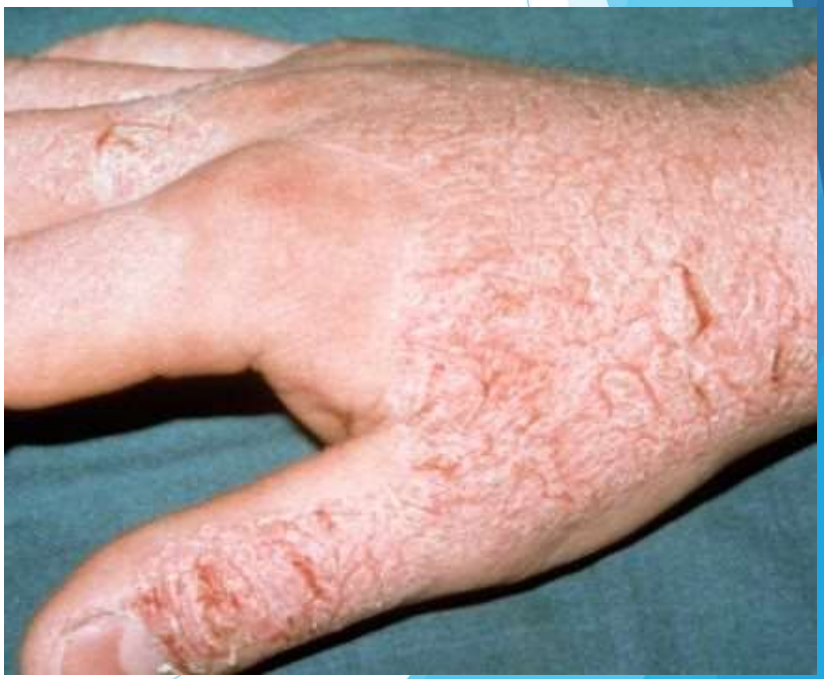
- A localized cutaneous, inflammatory reaction secondary to direct exposure to a topical external agent
- Non allergic, occurring without associated immunological mediation
- Do not require previous exposure to the agent
- Reaction typically begins within a few minutes or hours of exposure
- Everyone is susceptible and severity of reaction is dependent upon individual sensitivity, amount of irritant exposed to and length of exposure
- Most common cause of contact dermatitis
  - Accounts for approximately 80% of cases
- Acute and chronic phases
  - Acute
    - Erythema, edema, and vesiculation followed by erosions and scaling
  - Chronic
    - Erythema, fissures, and scale which is often thick
- Commonly affects hands
- Common cause of cheilitis (lip-licking)
- May be secondary of an occupational exposure





Treatment of Contact Dermatitis

- **AVOIDANCE OF IRRITANT!**
- Topical corticosteroids help treat symptoms of the associated insult

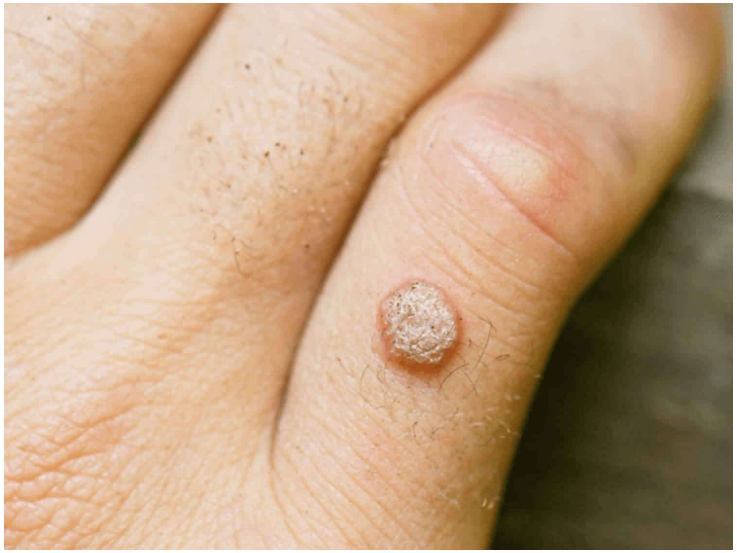


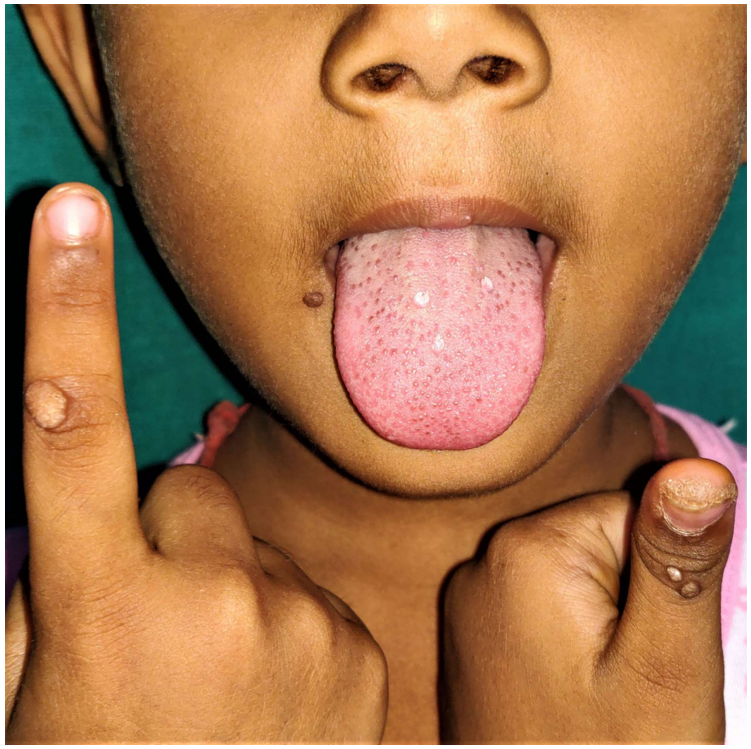
34 y/o

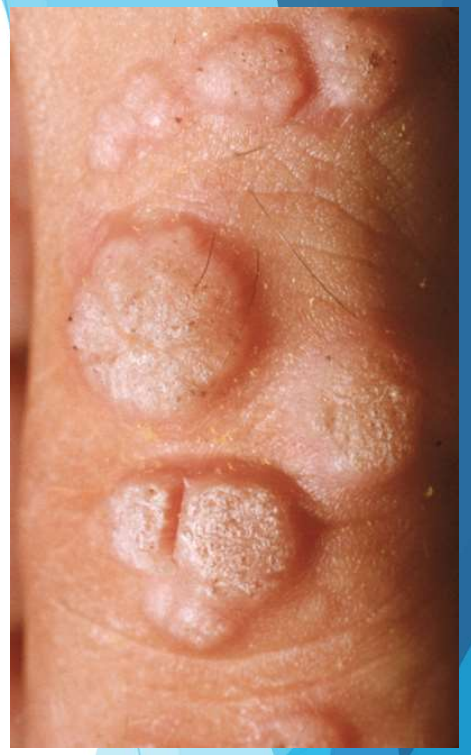
Presents to office with bump on forehead present for 6wks.

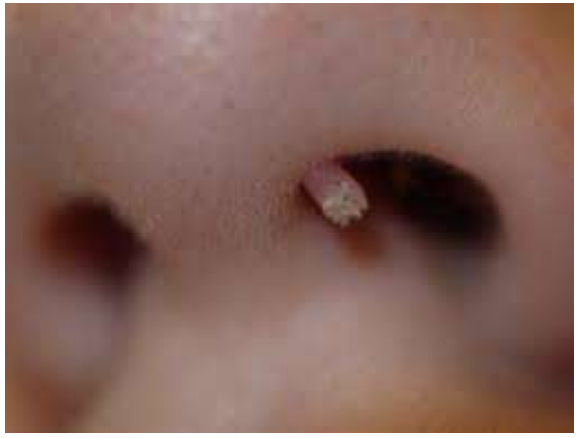
Verruca Vulgaris

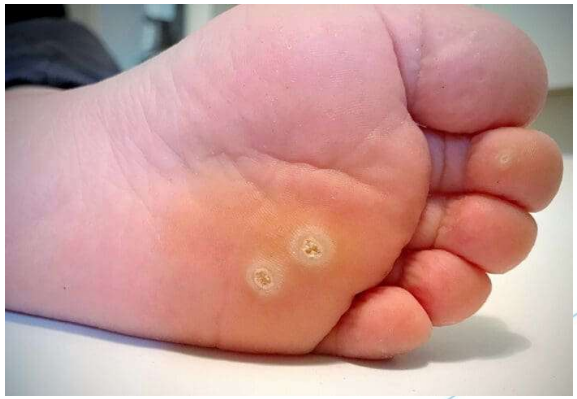
- Human papillomavirus (HPV) transmitted via person-to-person contact or contact with contaminated surface
- Prevalence of 20% in schoolchildren
- A third or more self-regress within 1-2yrs
- Any site, commonly found on the fingers, dorsal hands, and/or sites prone to trauma
- Clinical presentation: hyperkeratotic, exophytic or dome-shaped papules or plaques with punctate black dots (hemorrhage in the stratum corneum)
- DDX: SK, AK, cutaneous horn, SCC, trichilemmoma, Spitz nevus

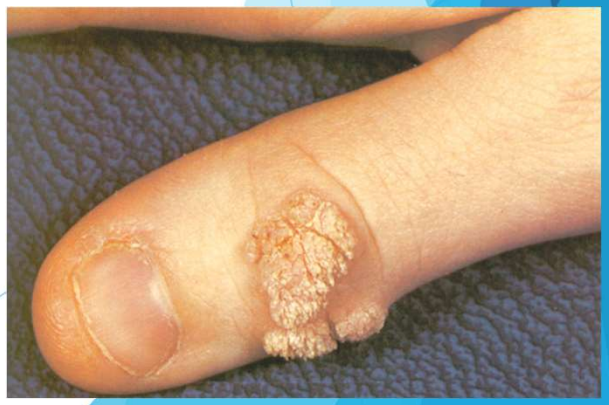












Treatment of VV

- Provider and patient/parent decide on treatment based on:
  - Number, morphology and distribution of warts
  - Tolerability of and anxiety regarding provider-applied therapies
  - Motivation and ability to use pt/parent-administrated therapies

Single or Few Warts

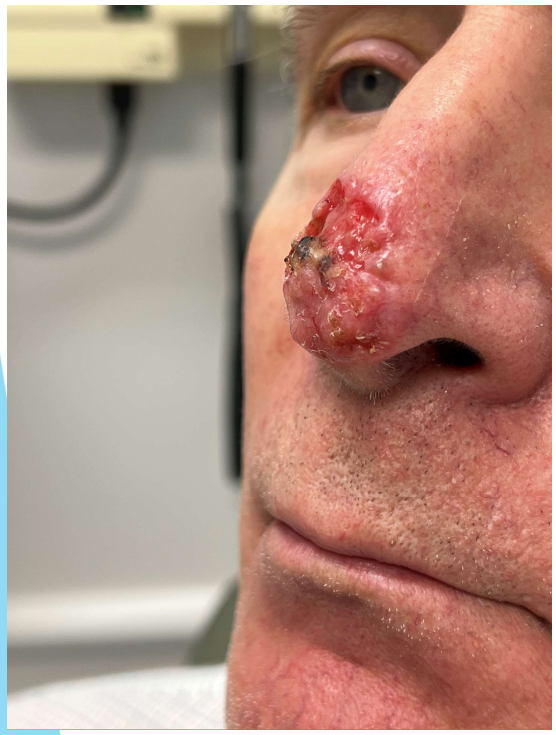
- PROVIDER-ADMINISTERED TX
  - Cryotherapy
  - Curettage + Electrodesiccation
  - Cantharidin

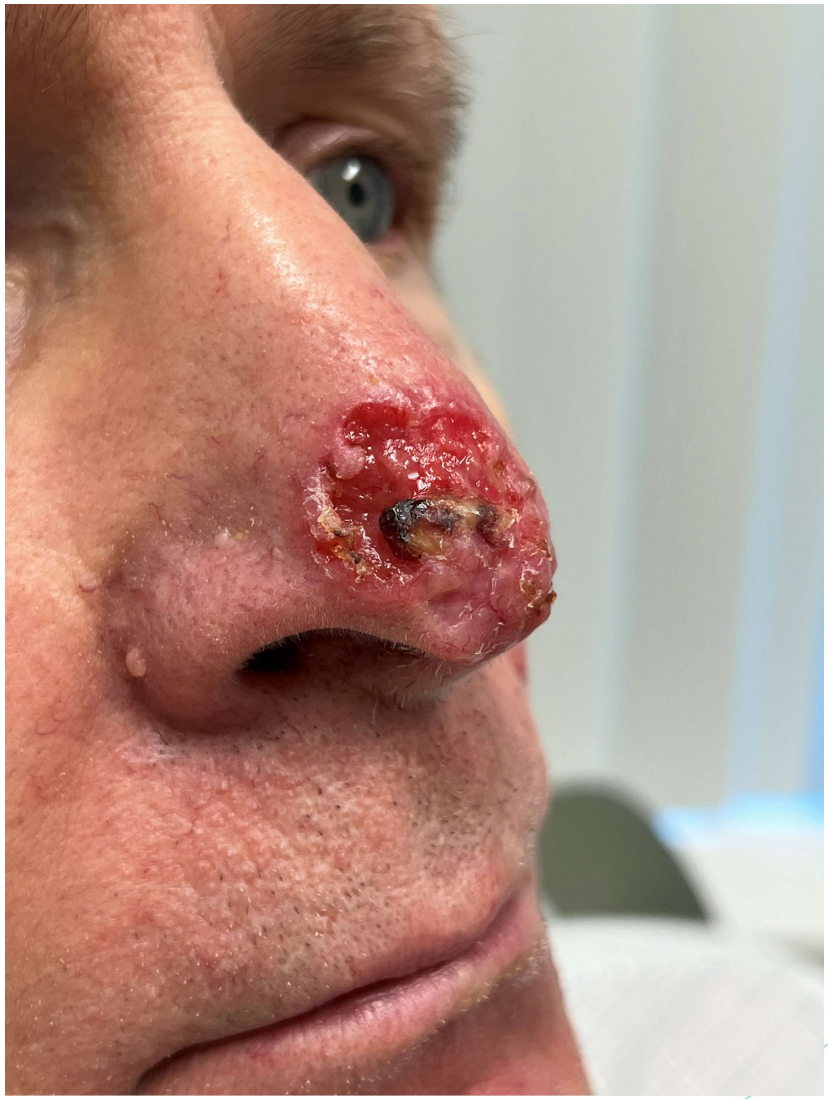
- PT/PARENT-ADMINISTERED TX
  - Salicylic acid
  - Imiquimod ± occlusion, salicylic acid
  - 5-fluorouracil ± occlusion, salicylic acid

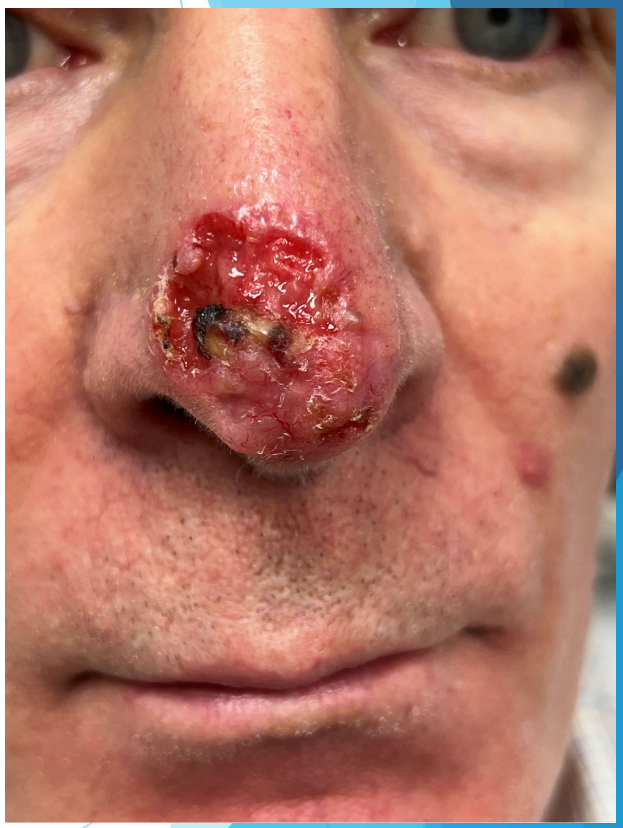
Many or Recalcitrant Warts

- PROVIDER-ADMINISTERED TX
  - Cryotherapy
  - Intralesional immunotherapy with skin test antigens (*Candida*)
  - Topical immunotherapy with DPCP or SADBE
  - Intralesional cidofovir
  - Intralesional 5-fluorouracil
  - Intralesional bleomycin
  - Laser treatment

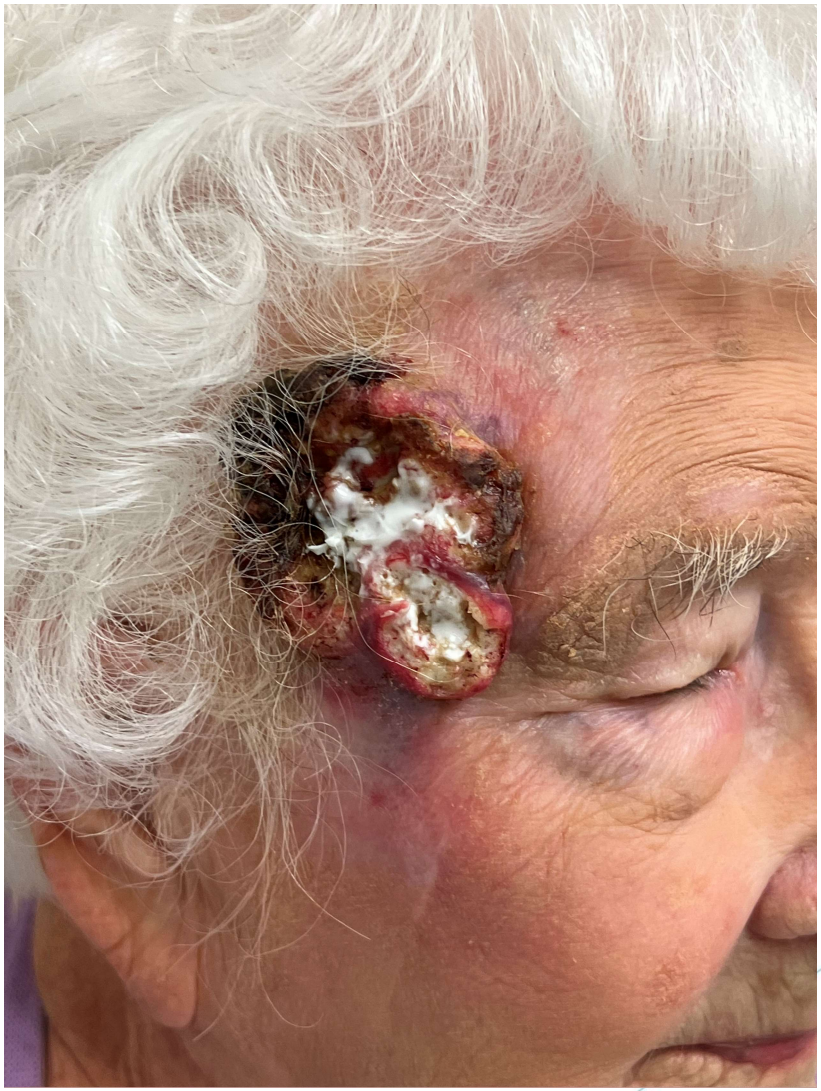
55 y/o
Presents to office with worsening, non-healing rash present for 6wks. No improvement with abx



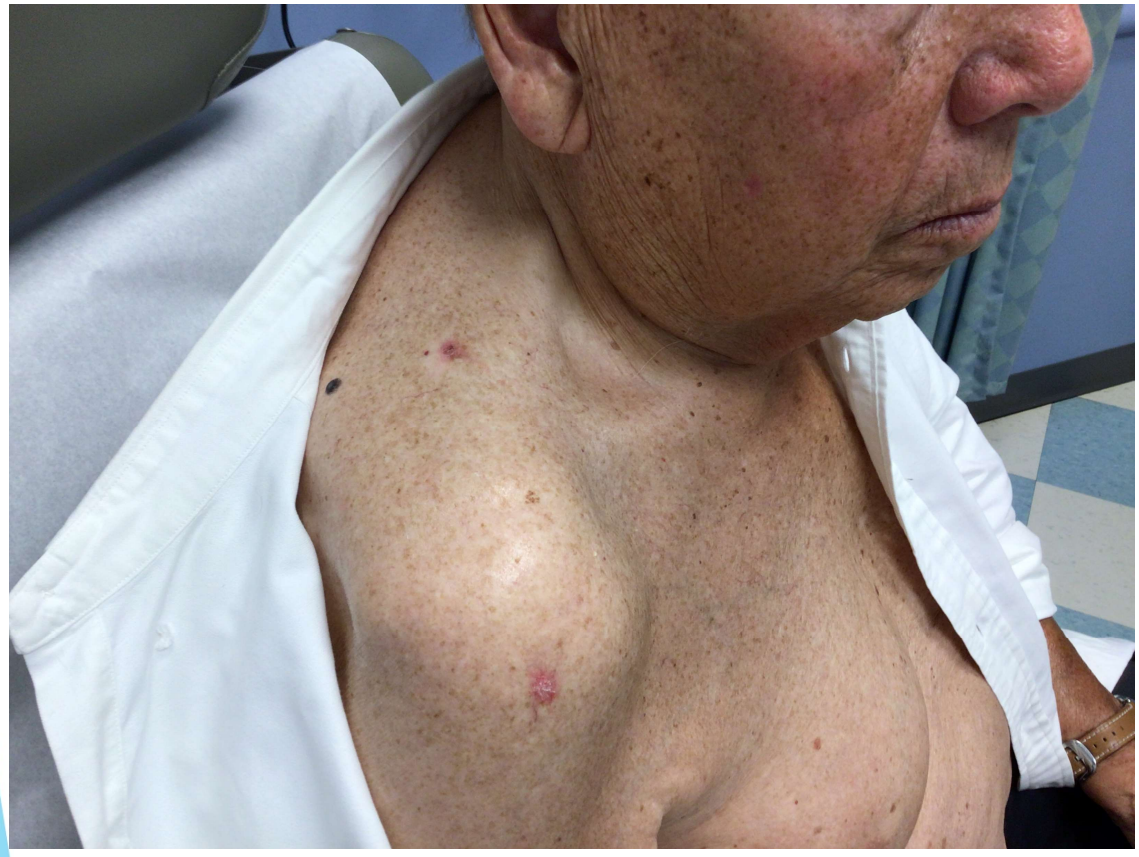


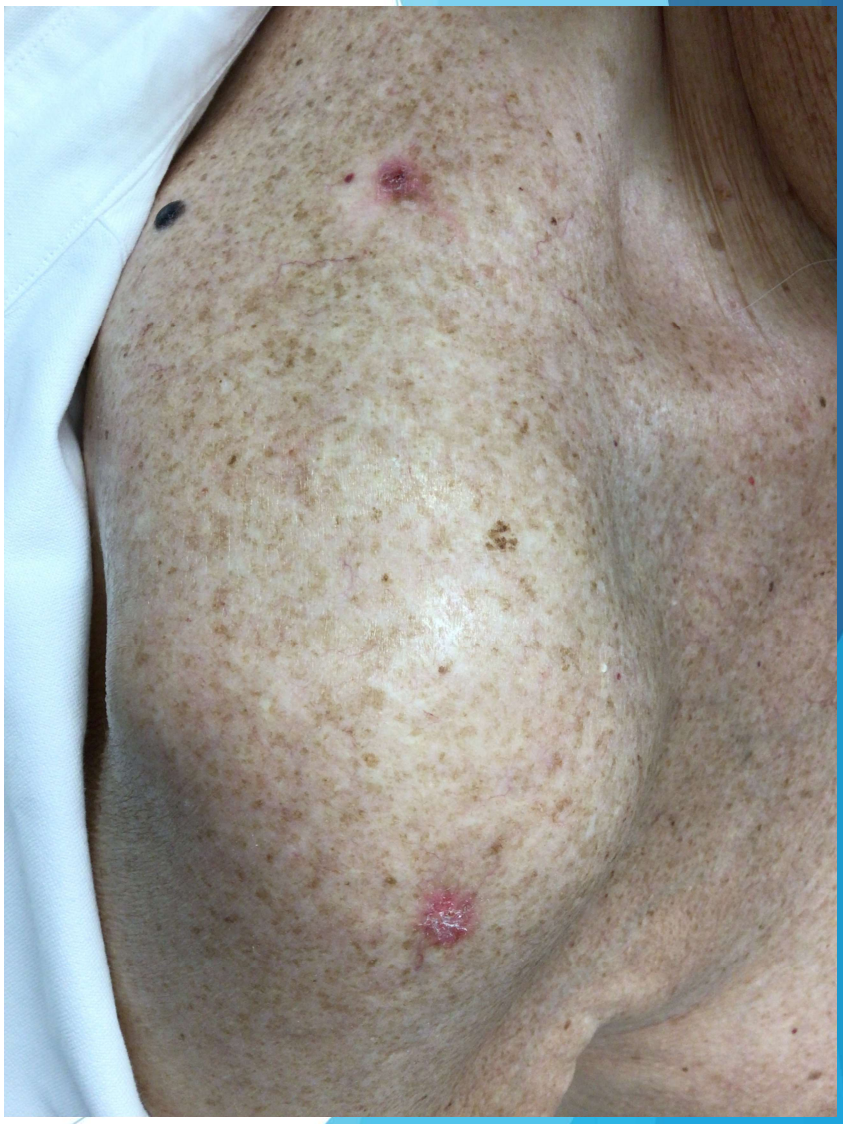


89 y/o
Presents to office with worsening, non-healing rash present for 4wks. No improvement with Silvadene cream and cephalexin.

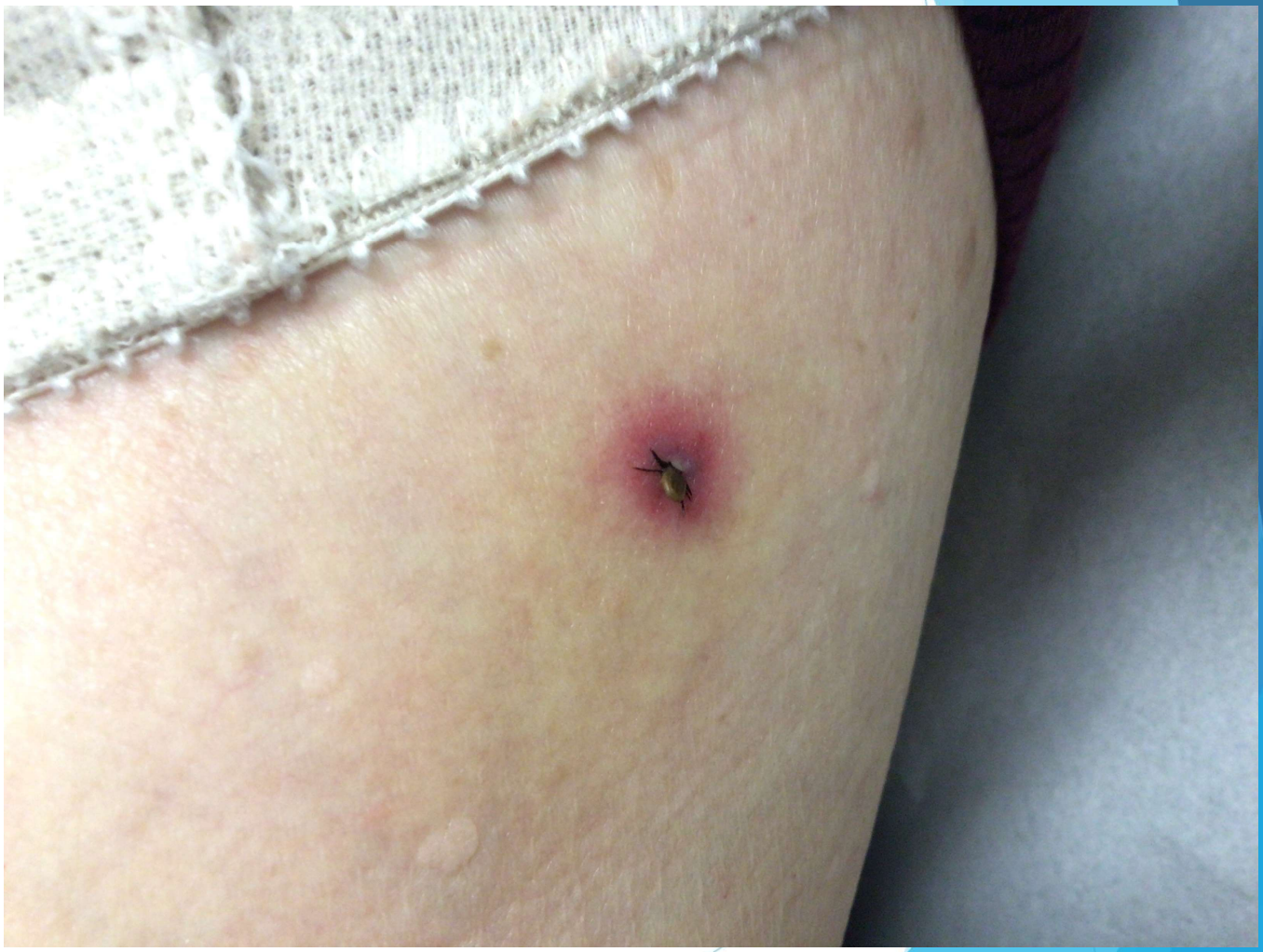


85 y/o
Presents to office with 2 nonhealing sores present for ~6mos





72 y/o female
Presents to office with nonhealing sore present for 6ds. +painful Not responding to cephalexin



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Questions??

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