UT Southwestern Medical Center

Dermatology for the Family Medicine PA!
AAPA We are Family
San Diego 2023

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Conflicts of Interest

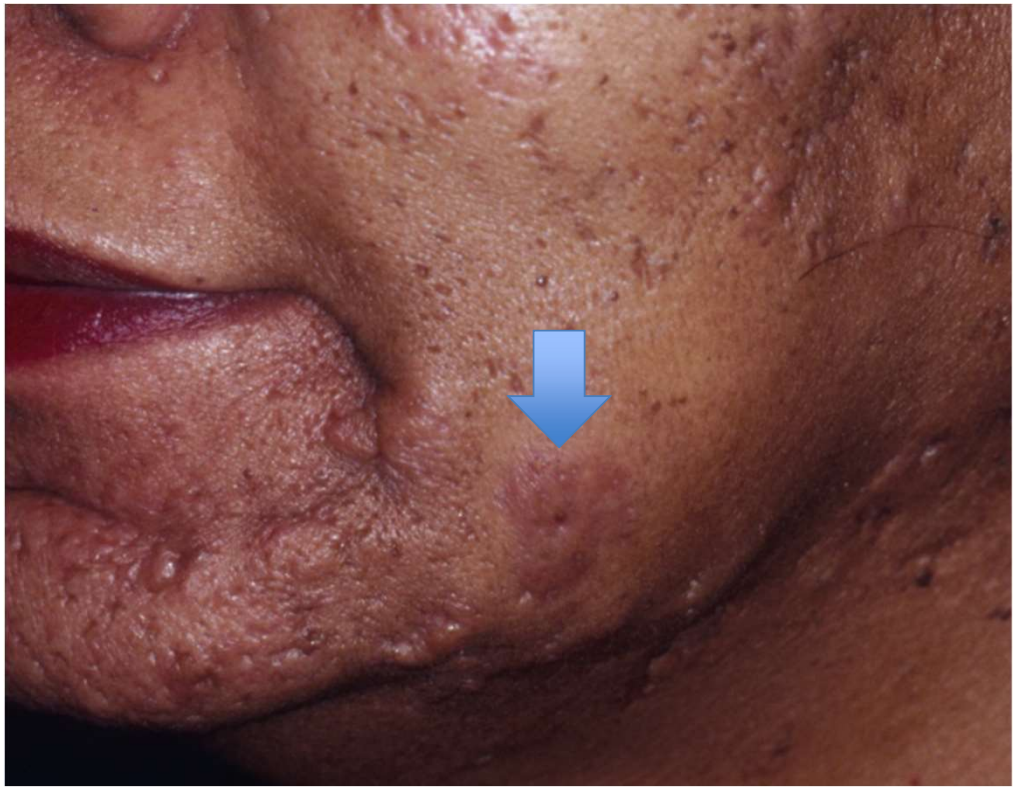
- None

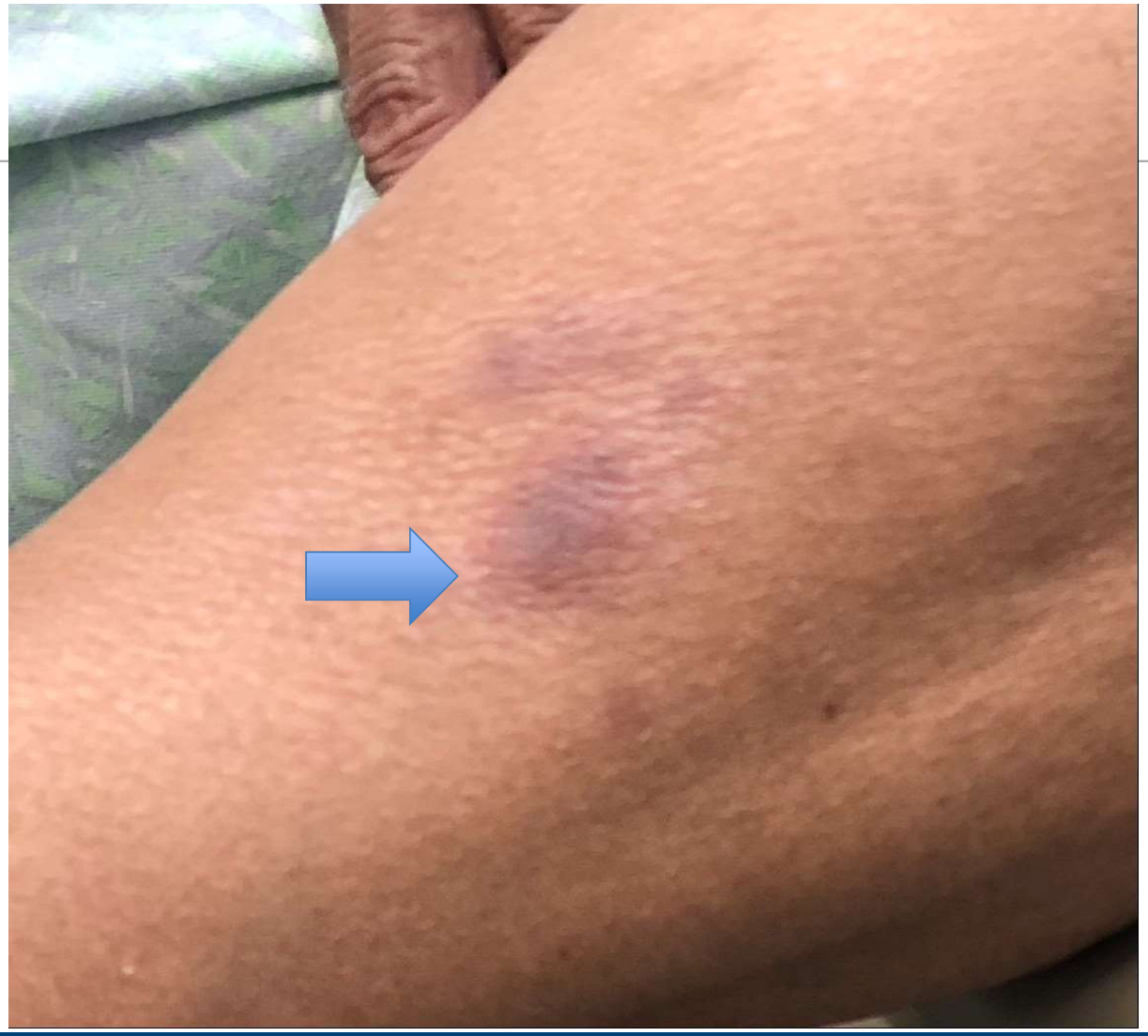
Objectives

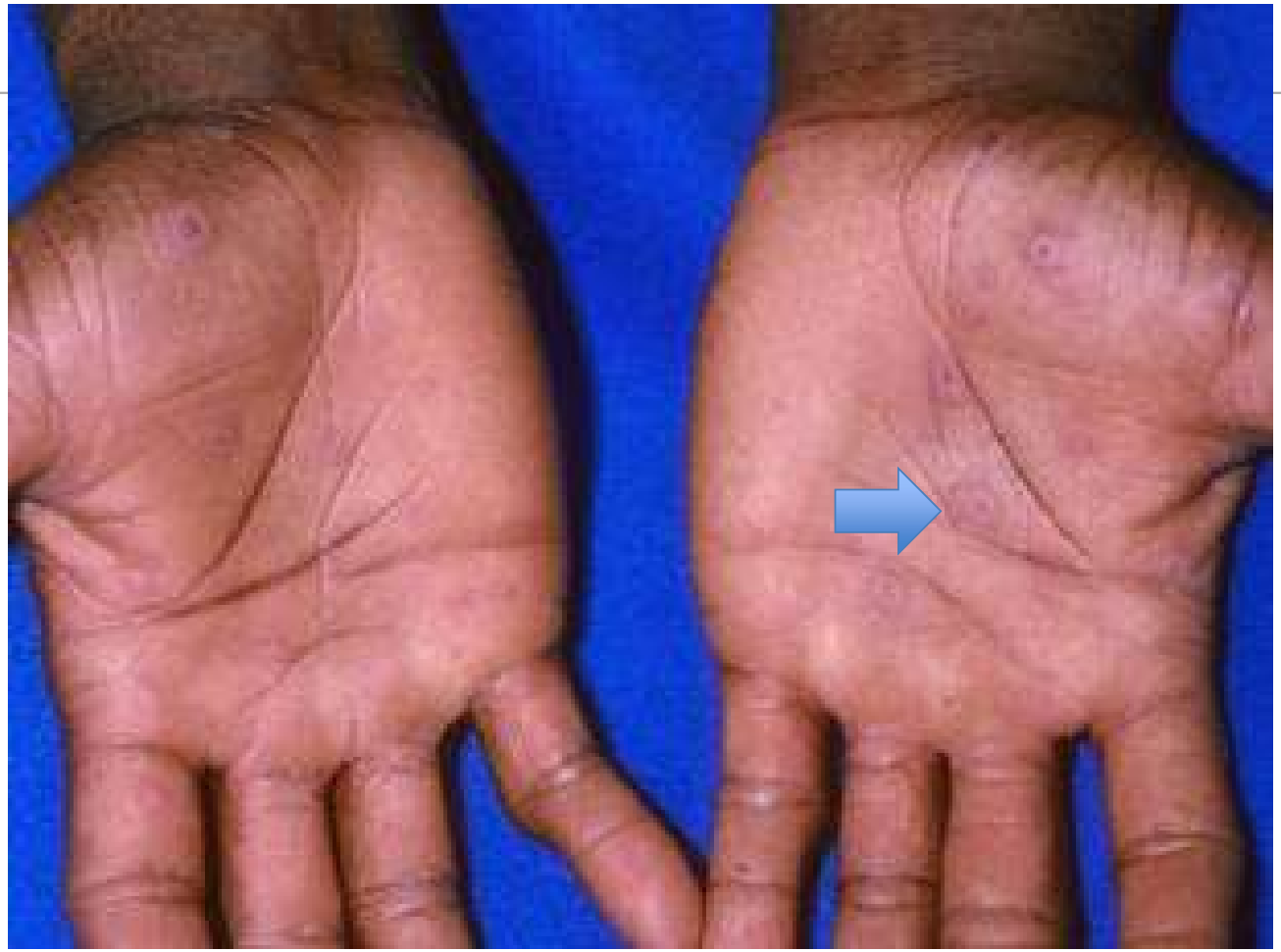
- Common skin diseases on a variety of skin types with emphasis on darker skin tones
- Practical tips about steroid cream
  - Appropriate use for topical creams
  - A word on vehicles
  - Steroid cream indications and contraindications, rashes that will not respond to steroid
  - Steroid Atrophy
- When to refer
  - Derm red flags
  - When steroids don't fix it

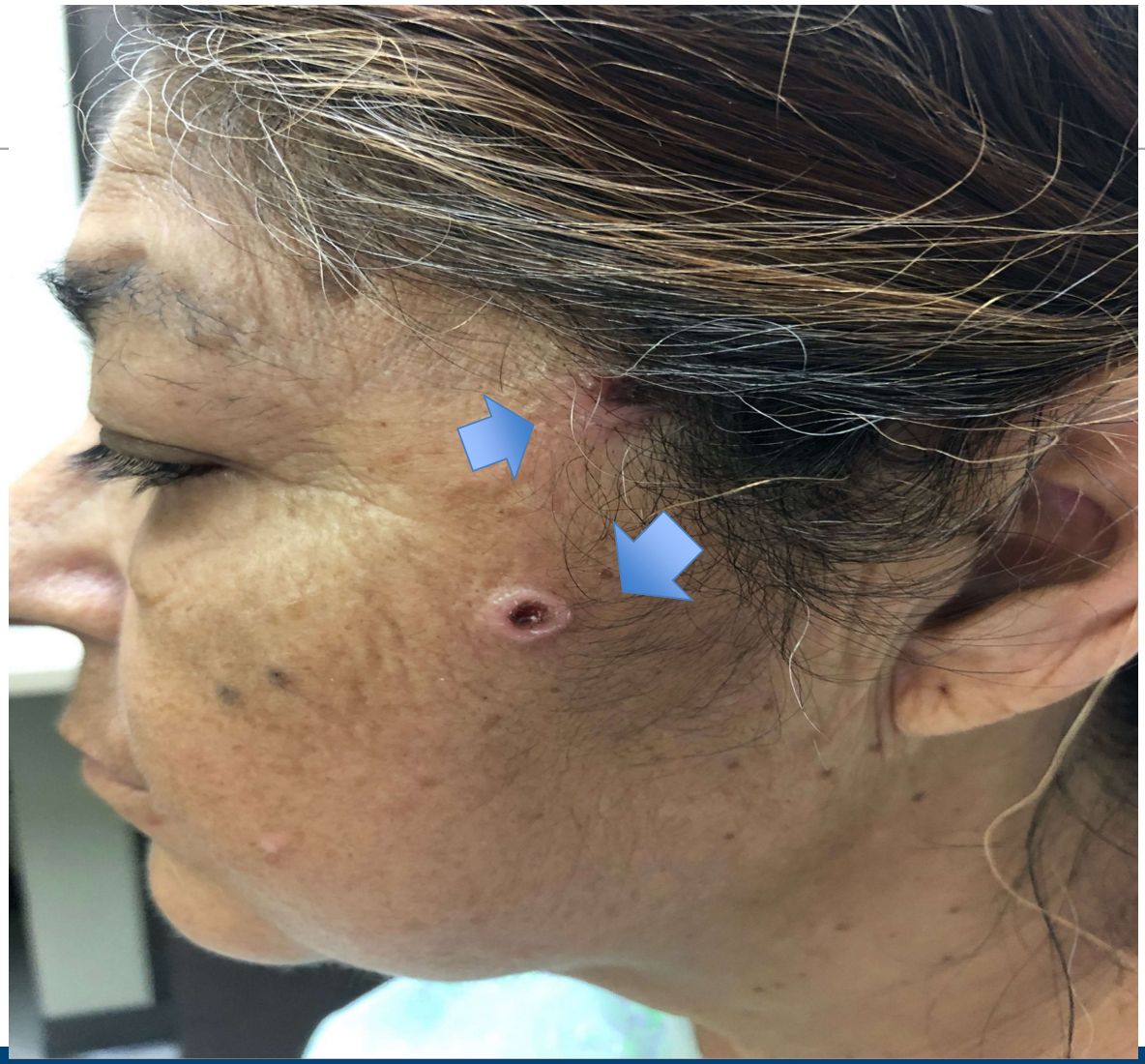
Considerations Skin of color

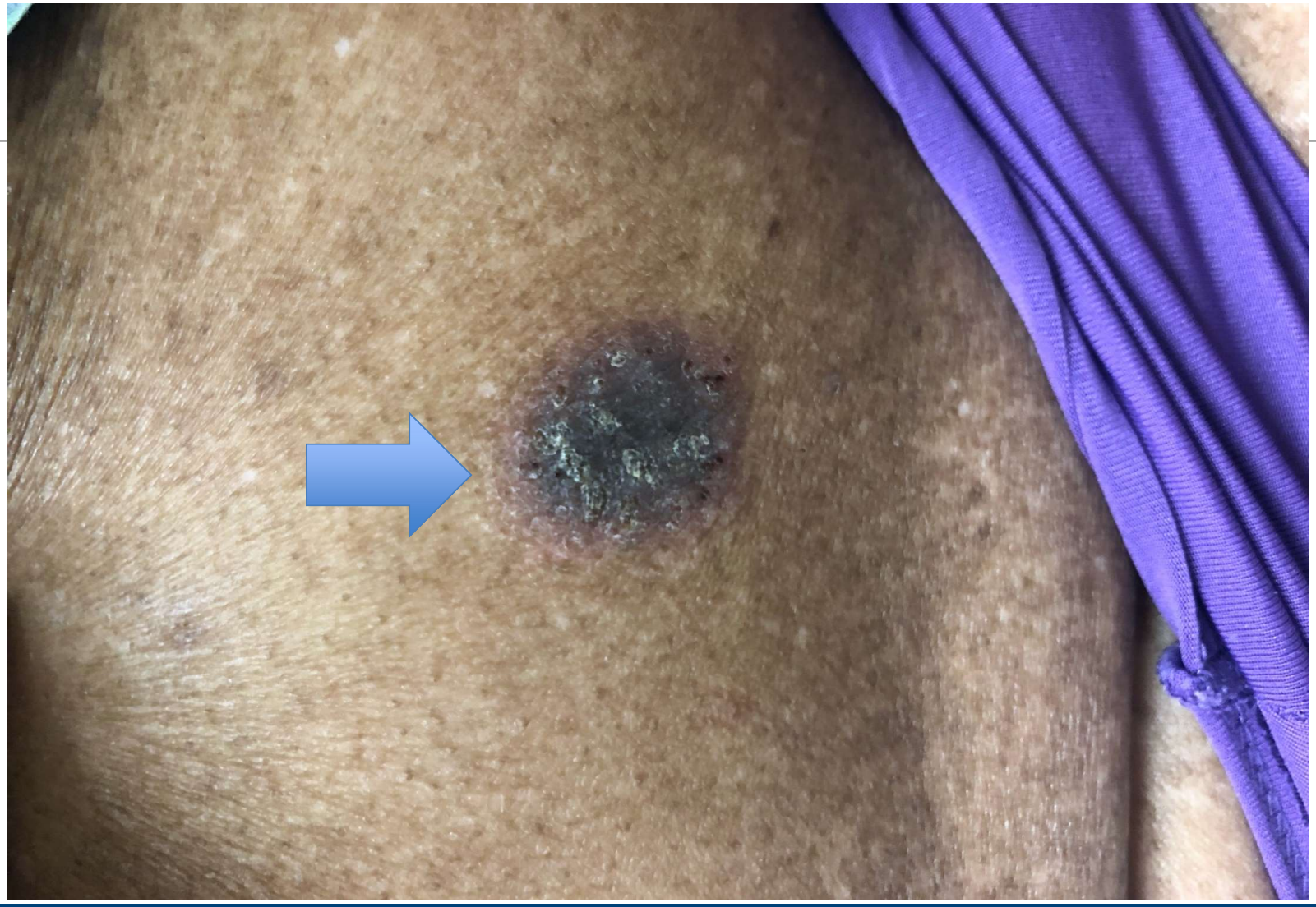
- Use context clues, erythema or redness will not be redness

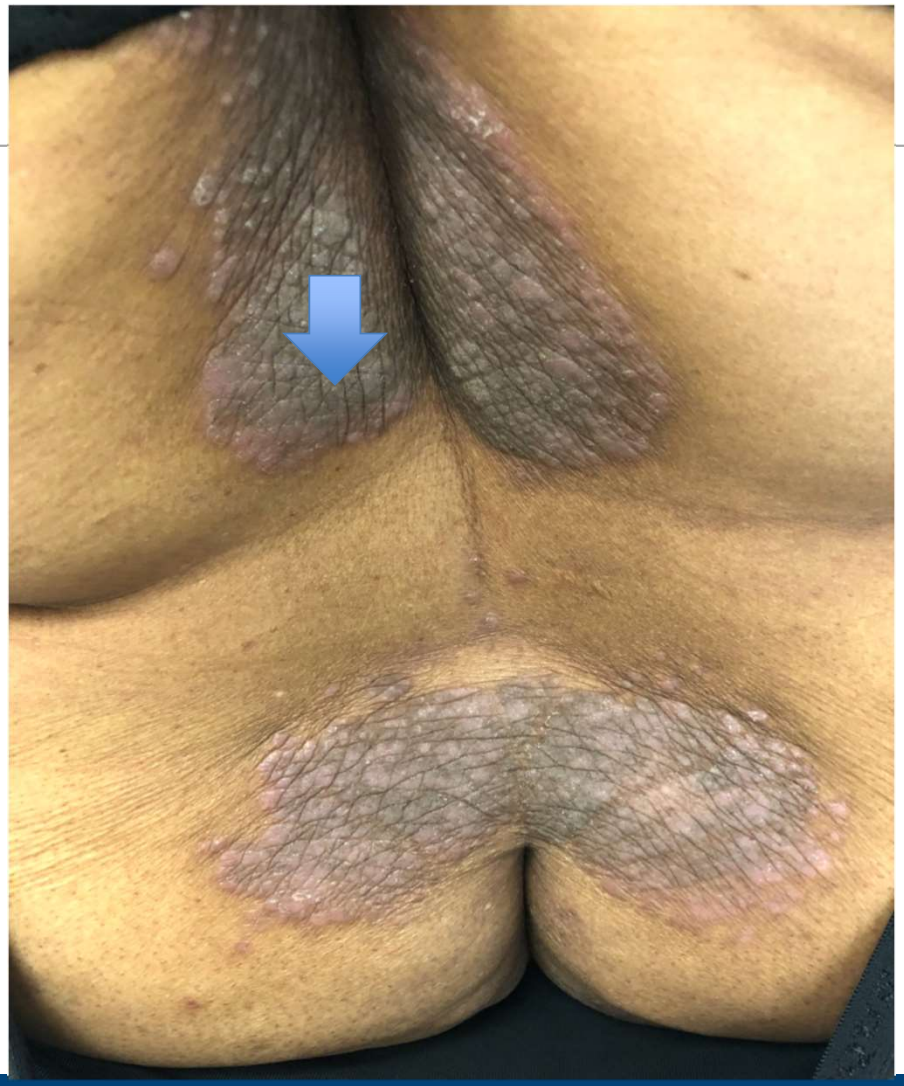


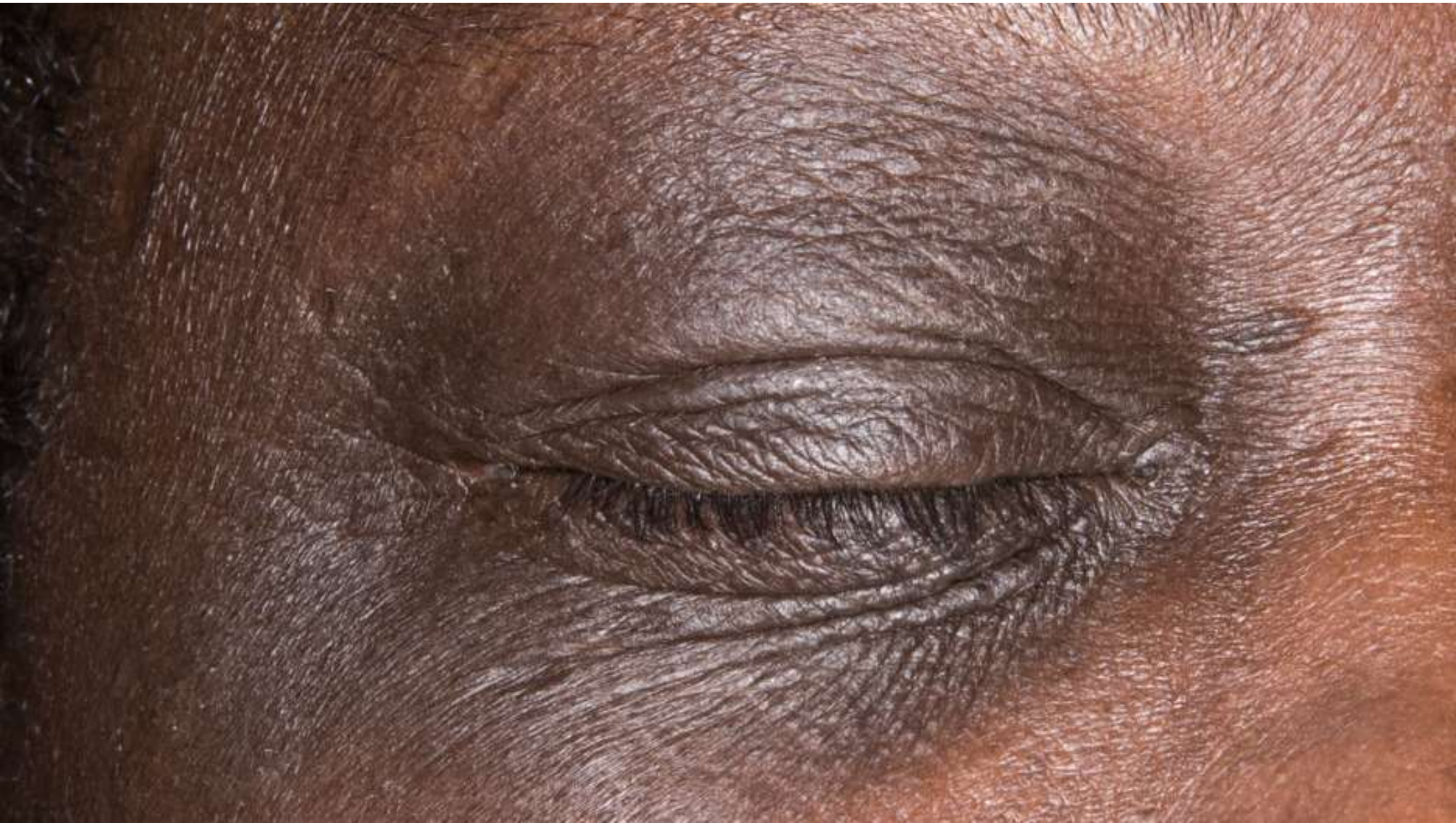






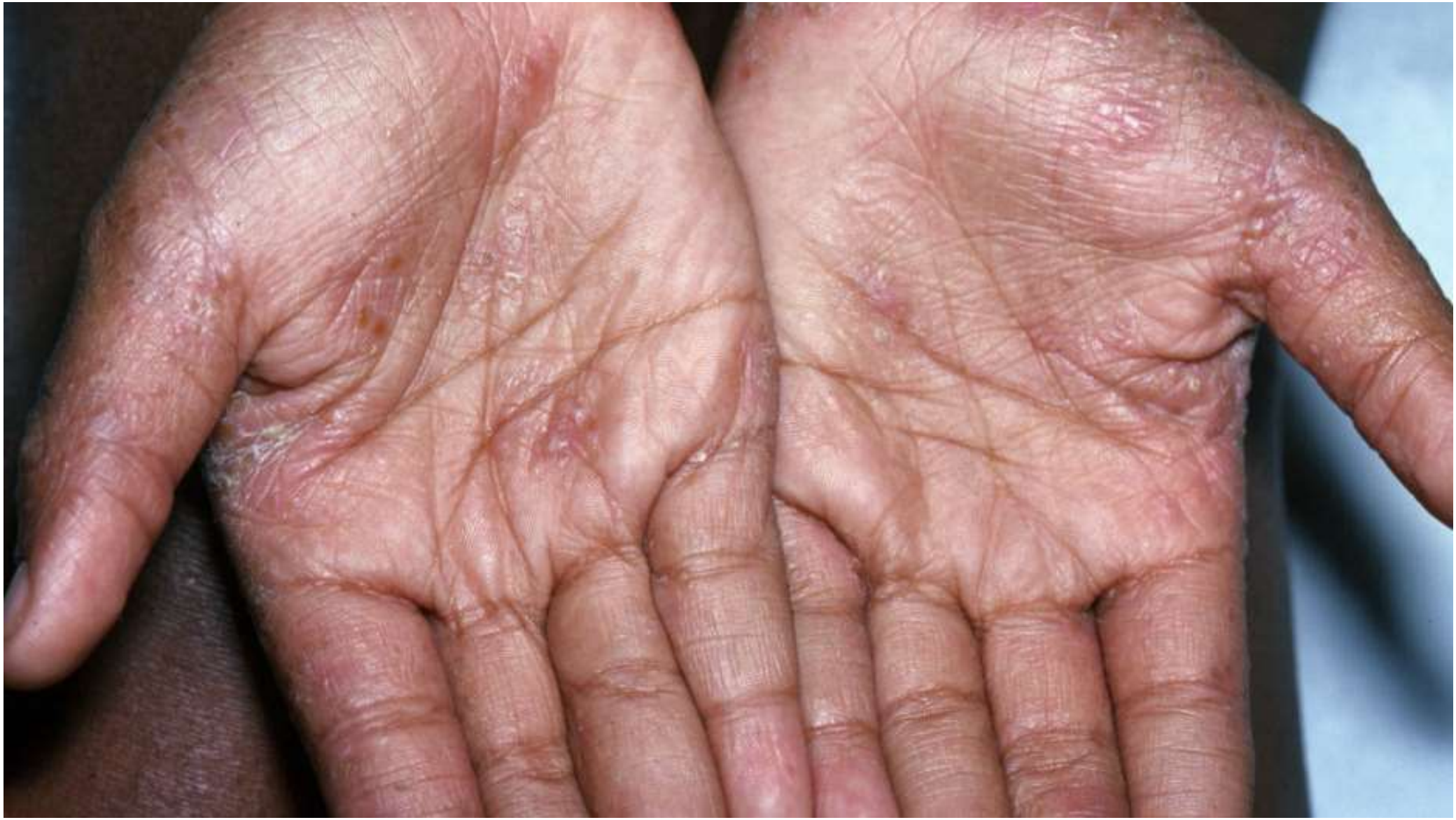






Dyshidrosis

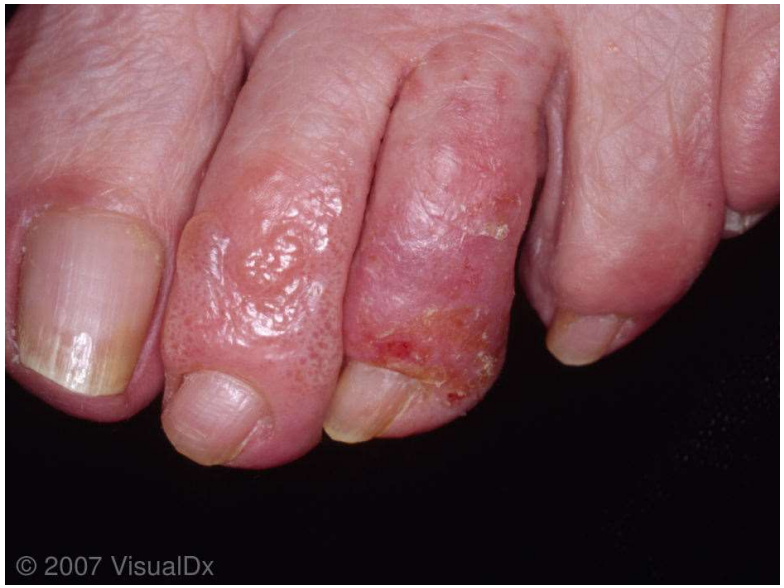
- Eczema/Atopic Dermatitis of the hands and feet
  - AKA: dyshidrotic eczema, pompholyx
- Characterized by vesicles on the sides of the fingers and/or feet
  - Small, tense, clear, fluid-filled vesicles on the lateral aspects of the digits "Tapioca"
- Differential Diagnosis includes: contact dermatitis, psoriasis, tinea, bacterial infection



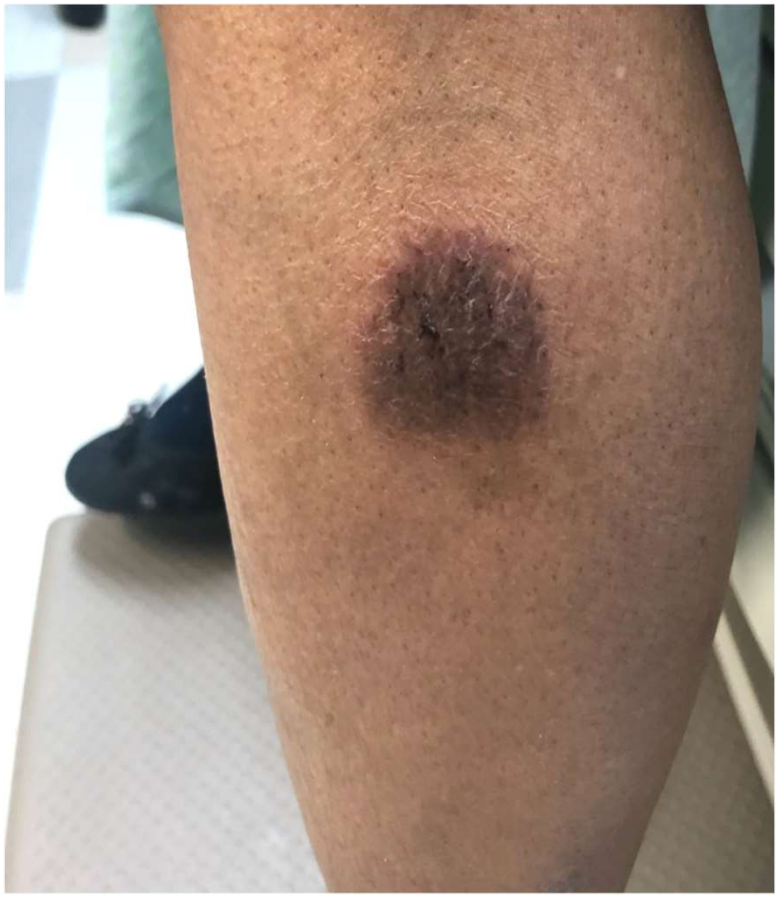
Dyshidrosis

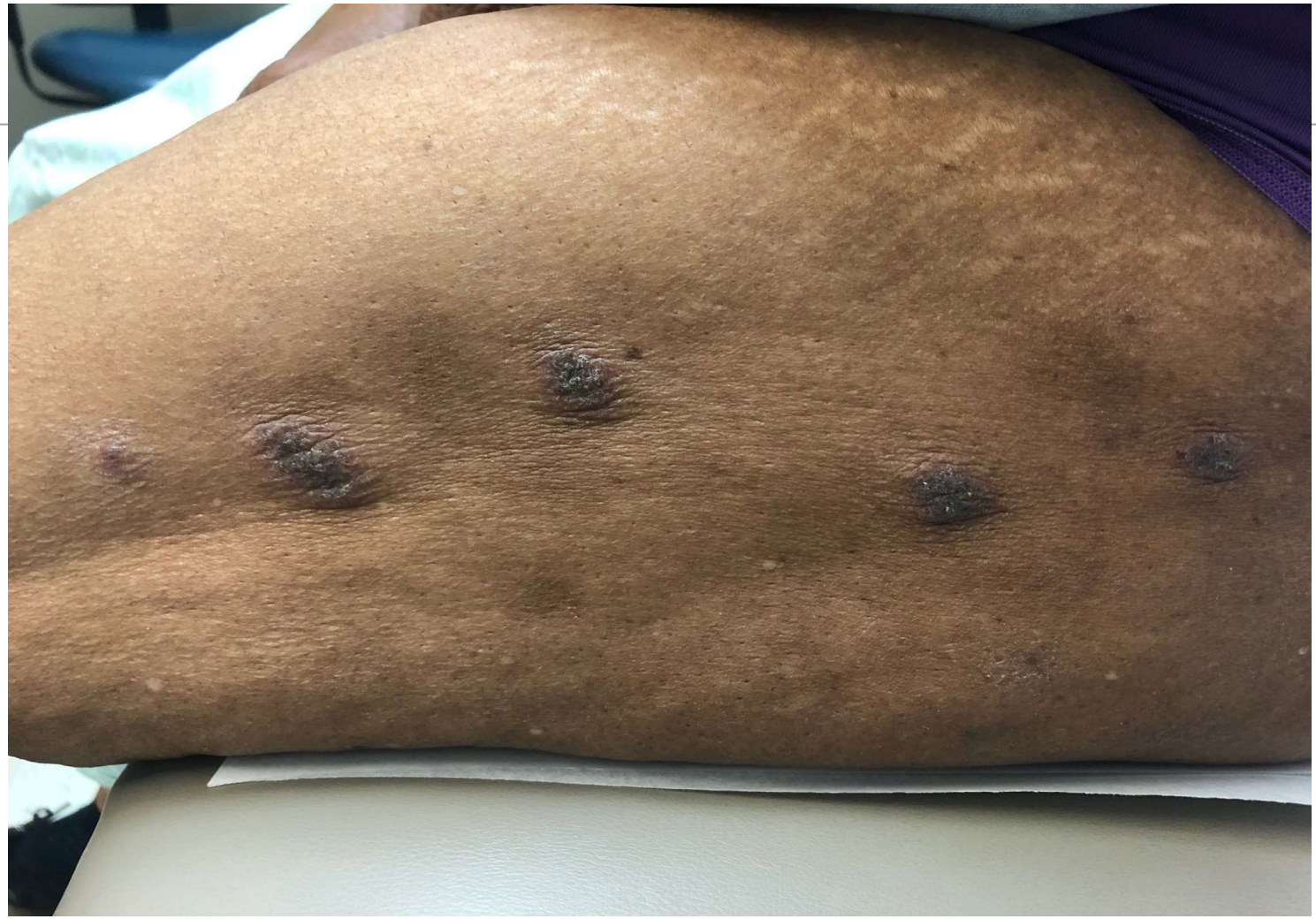
Further testing may be done to rule out other conditions:
Patch testing (allergic contact dermatitis)
KOH preparation of scrapings (dermatophyte or scabies)
Bacterial culture (bacterial infection)
Biopsy for direct immunofluorescence (bullous pemphigoid)

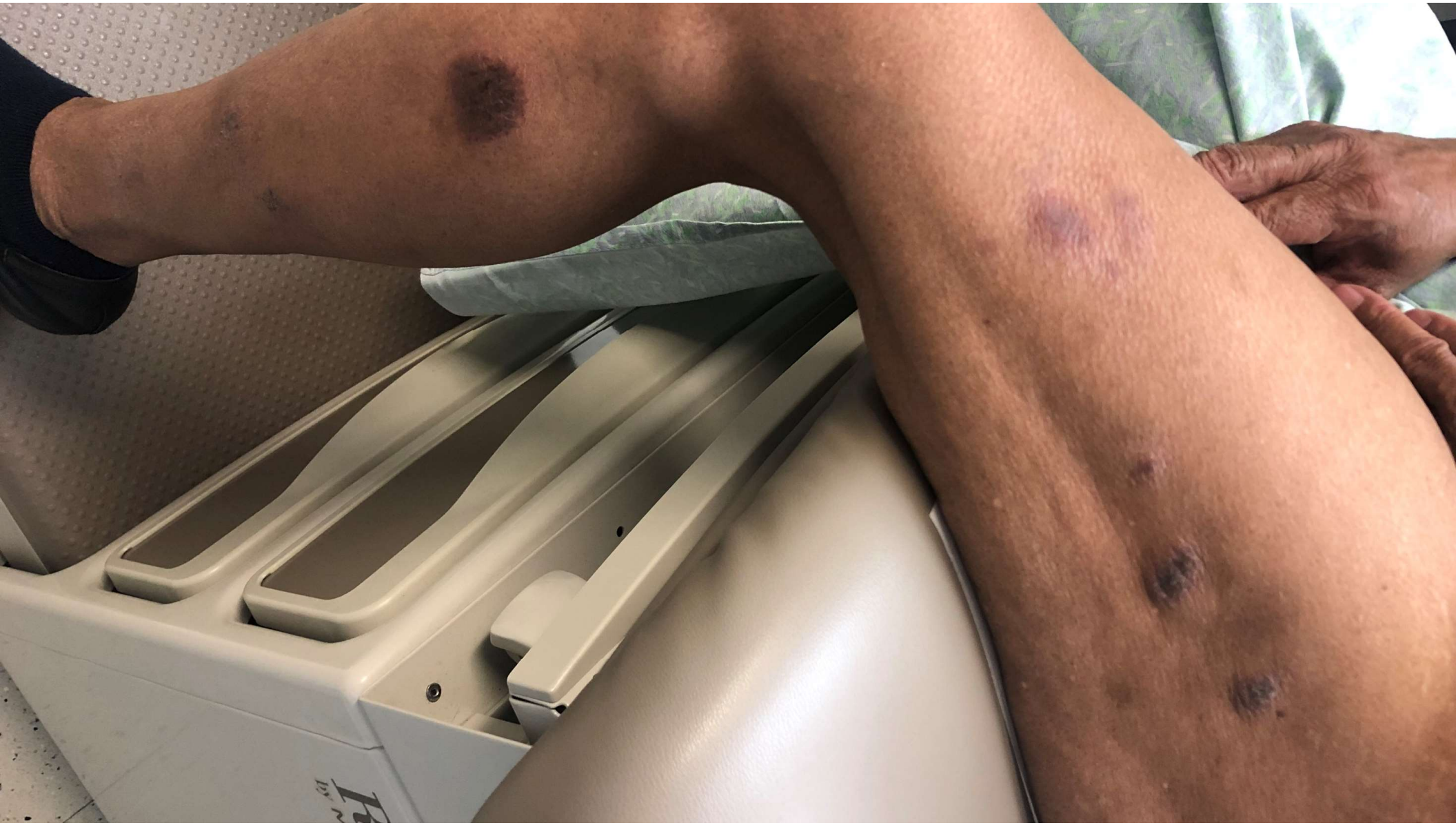
Treat like Atopic Dermatitis:
Mid to High potency Topical steroid

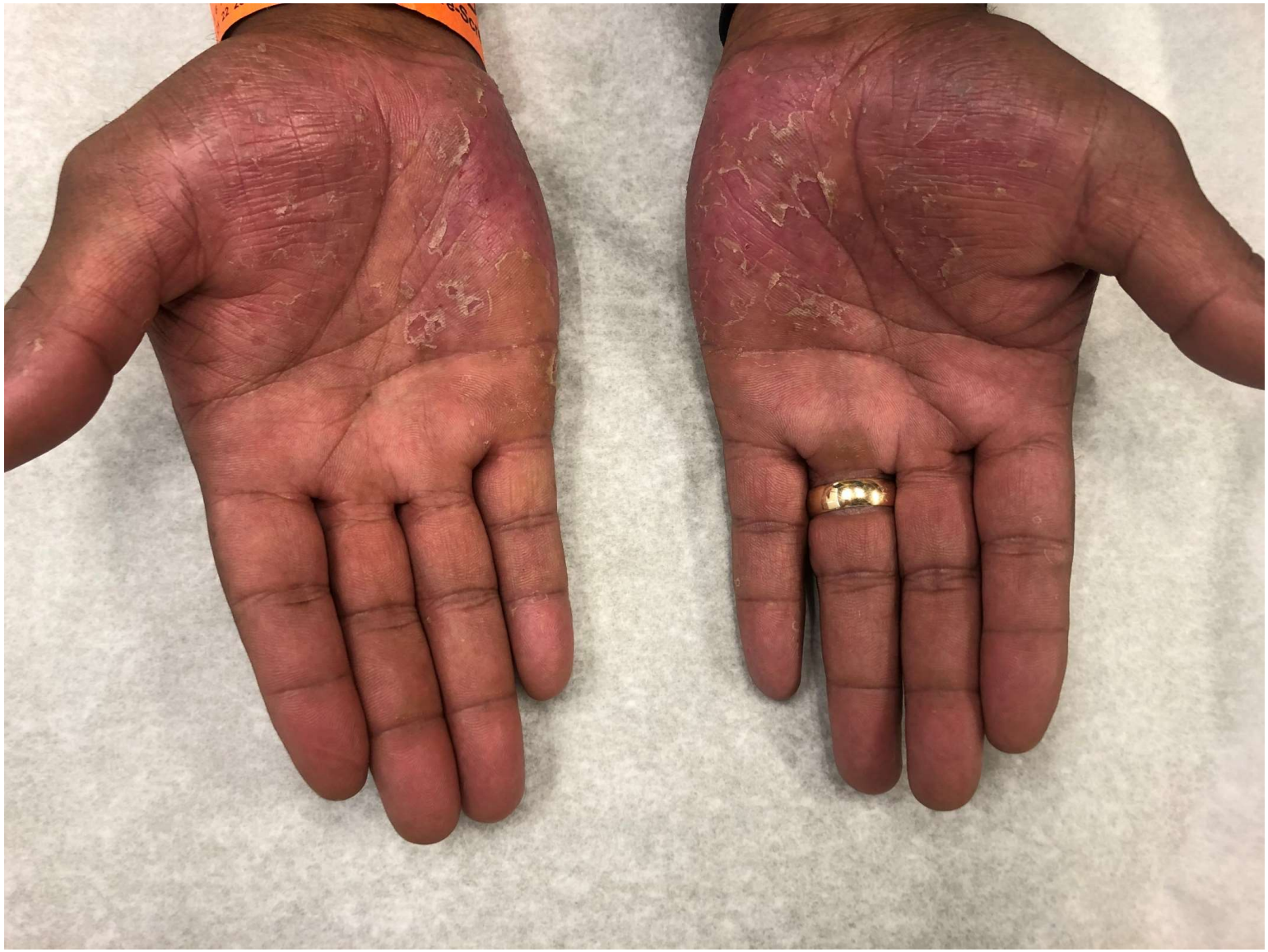


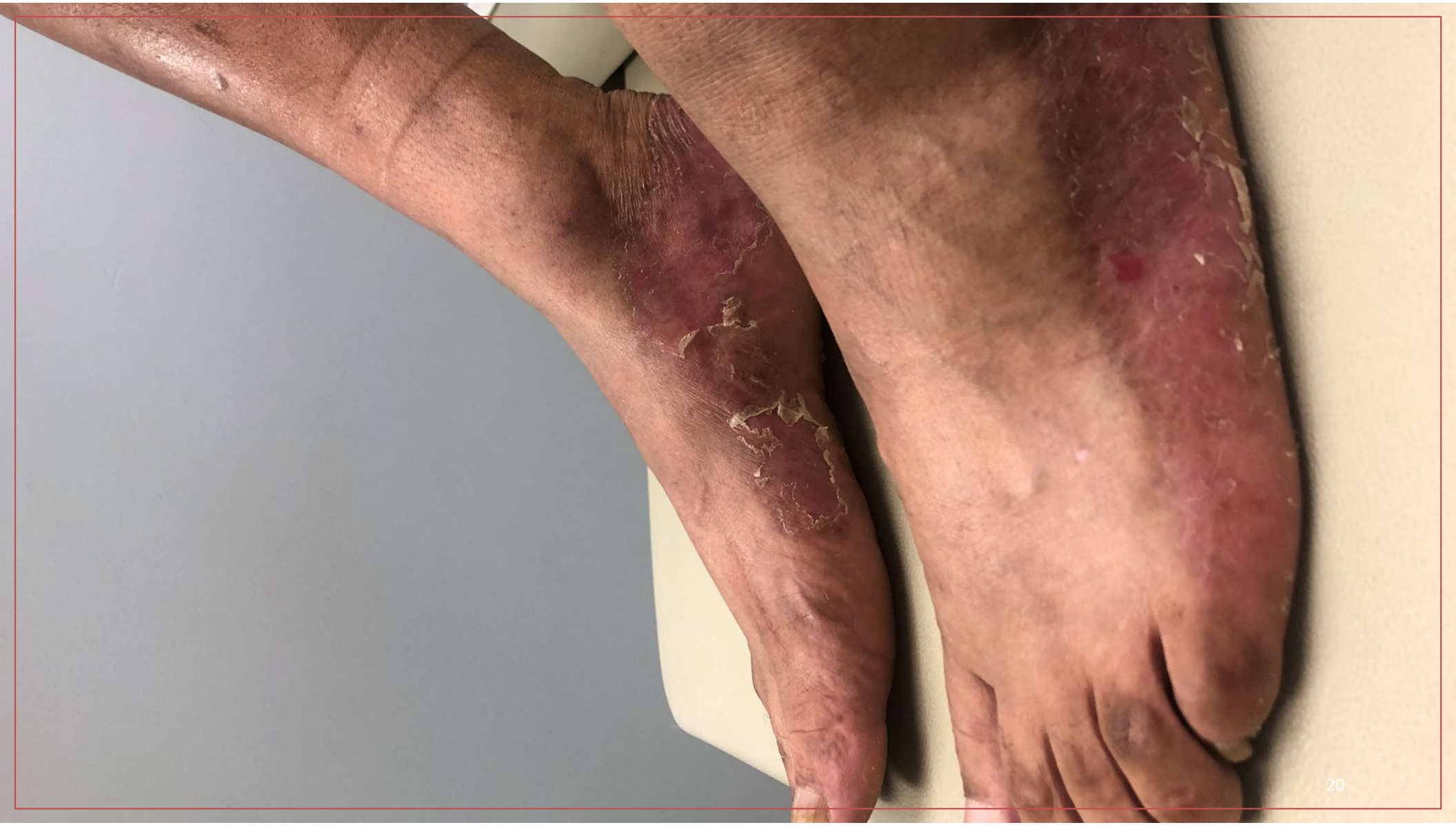
Nummular Eczema

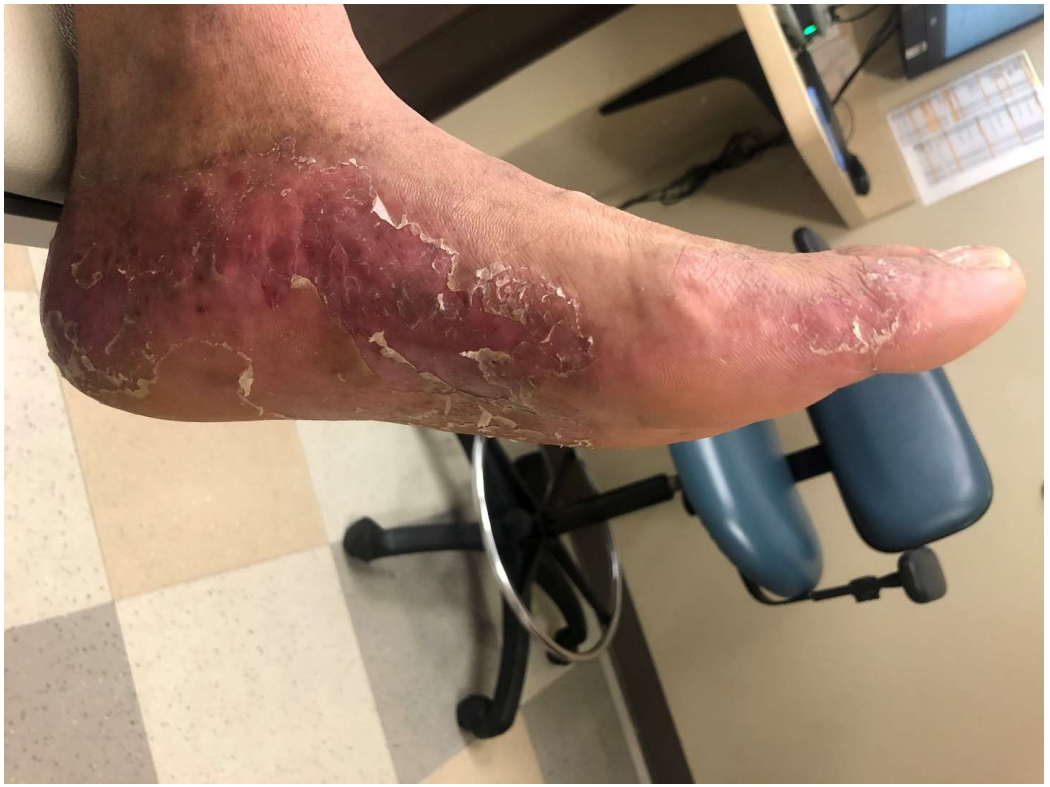


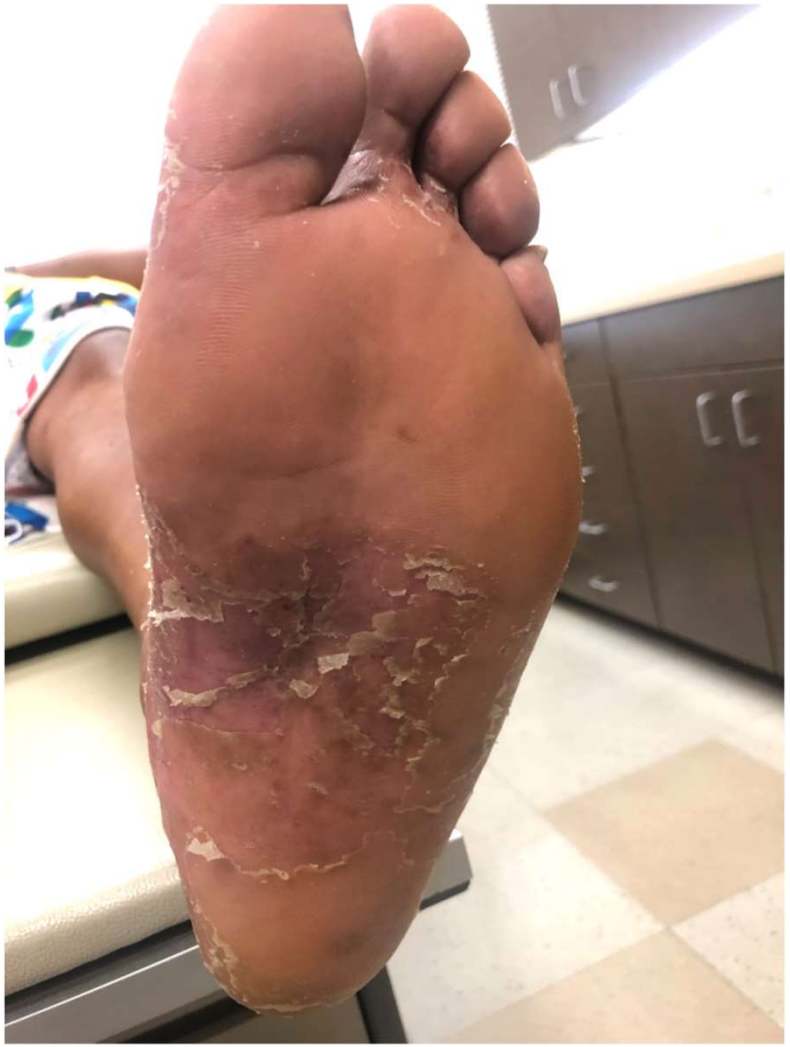


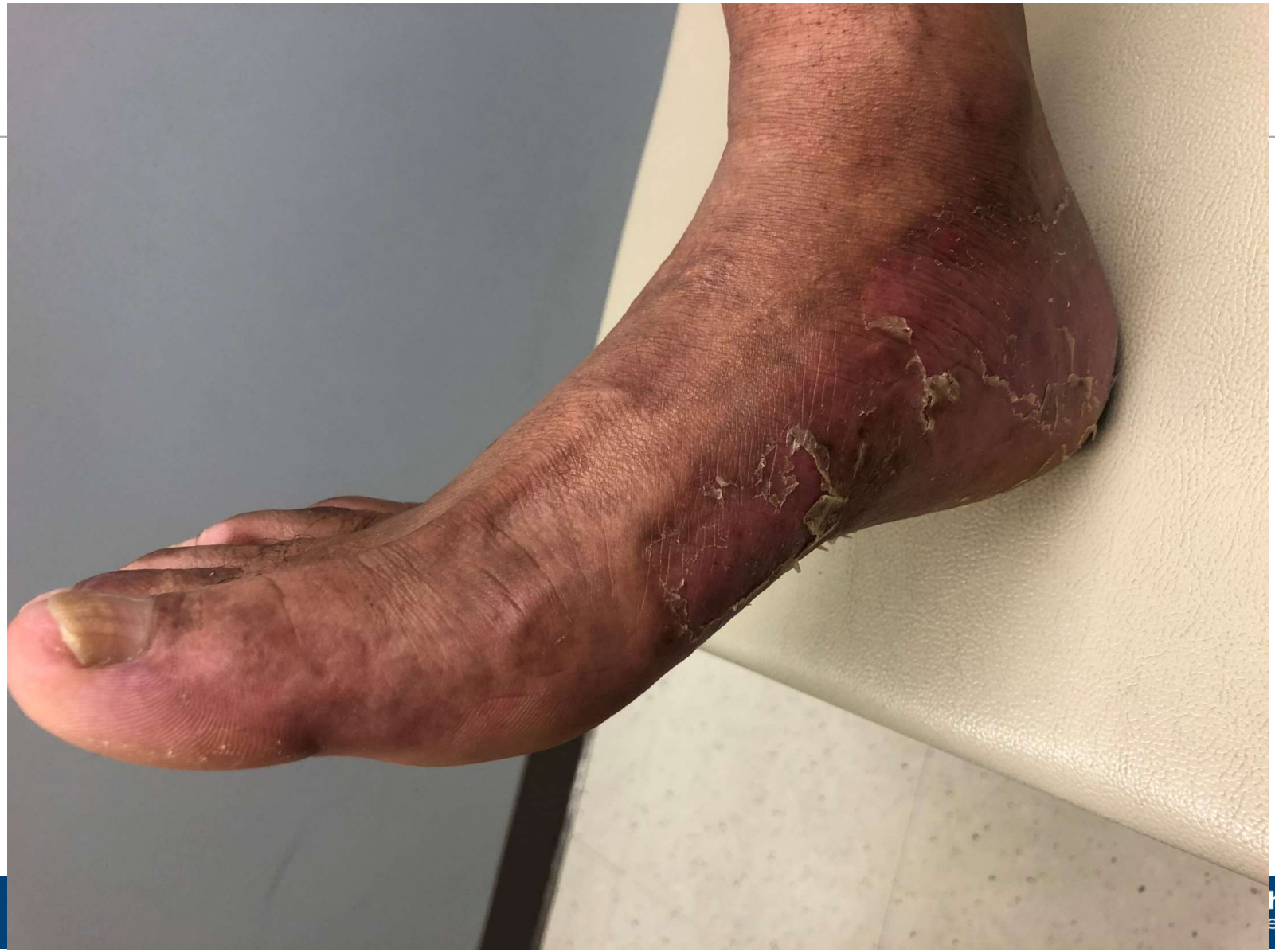












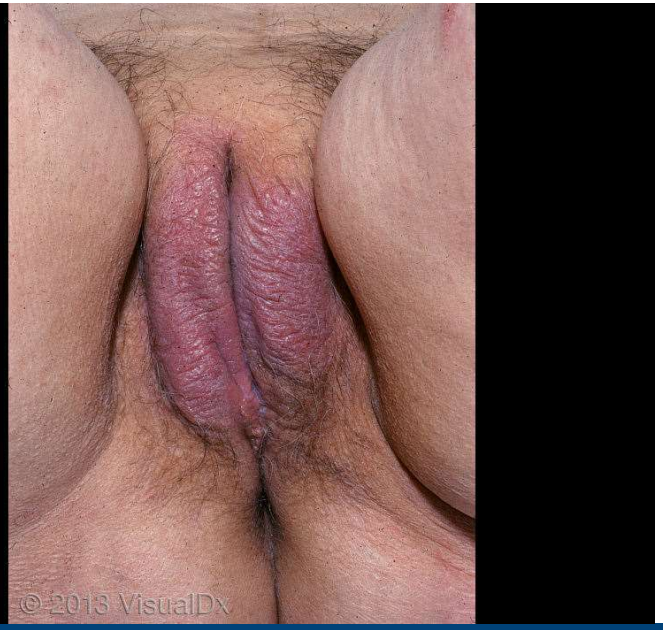
Lichen Simplex Chronicus

Caused by skin thickening in response to rubbing/friction

Hallmarks of this are:

Lichenification -> accentuation of skin tension lines

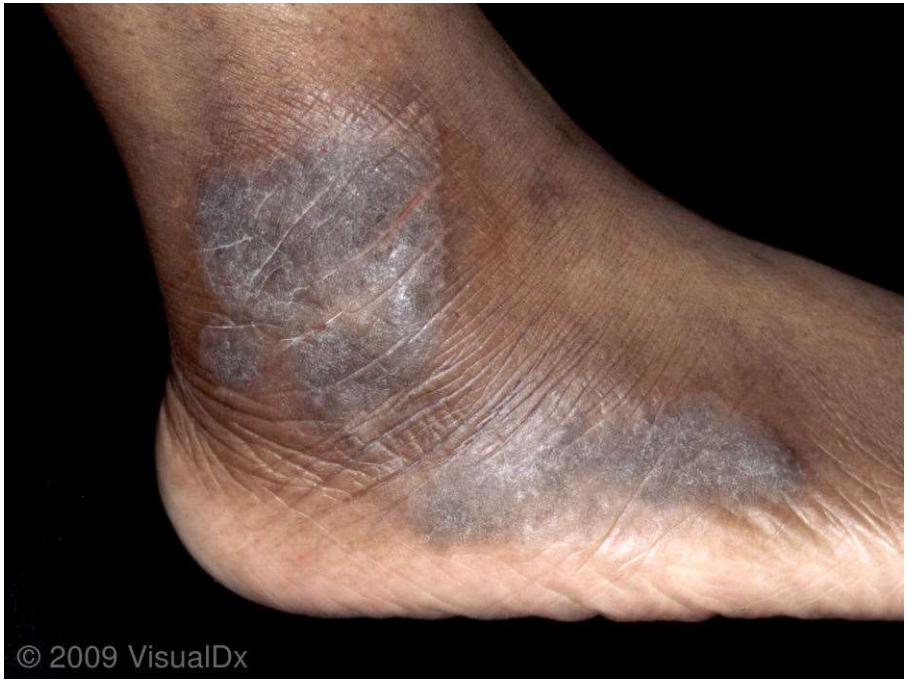
Hyperpigmentation -> dark color



Lichen Simplex Chronicus

Treatment

- Mid to High potency topical steroids twice daily for 2 weeks
- Refer to Dermatologist if this does not resolve
- Educate patient on need to stop scratching



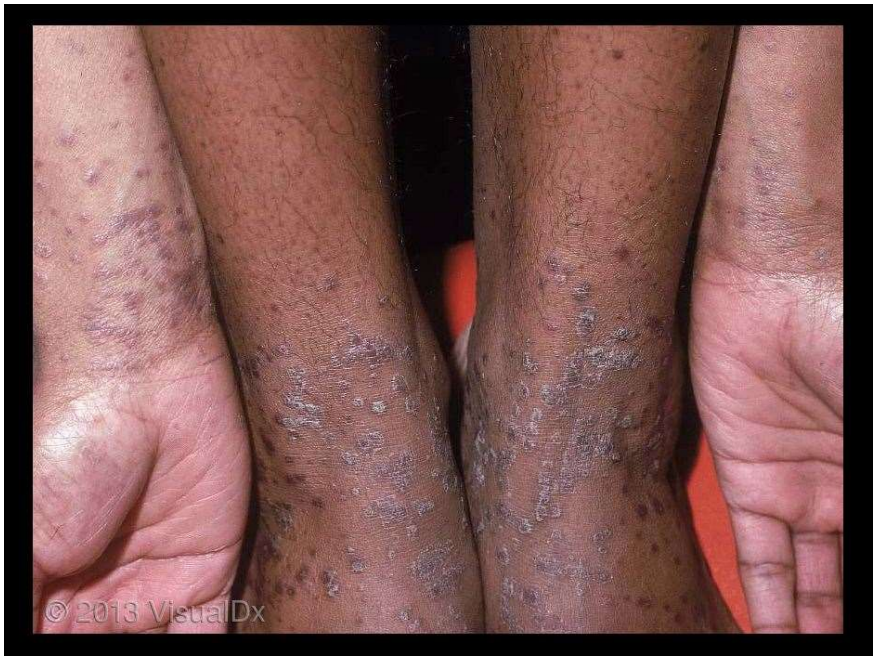
Lichen Planus

Acute and sometimes chronic inflammatory dermatosis of the skin and/or nails and mucous membranes

5 P's: Planar (flat topped), Pruritic, Polygonal, Purple (violaceous) Papules

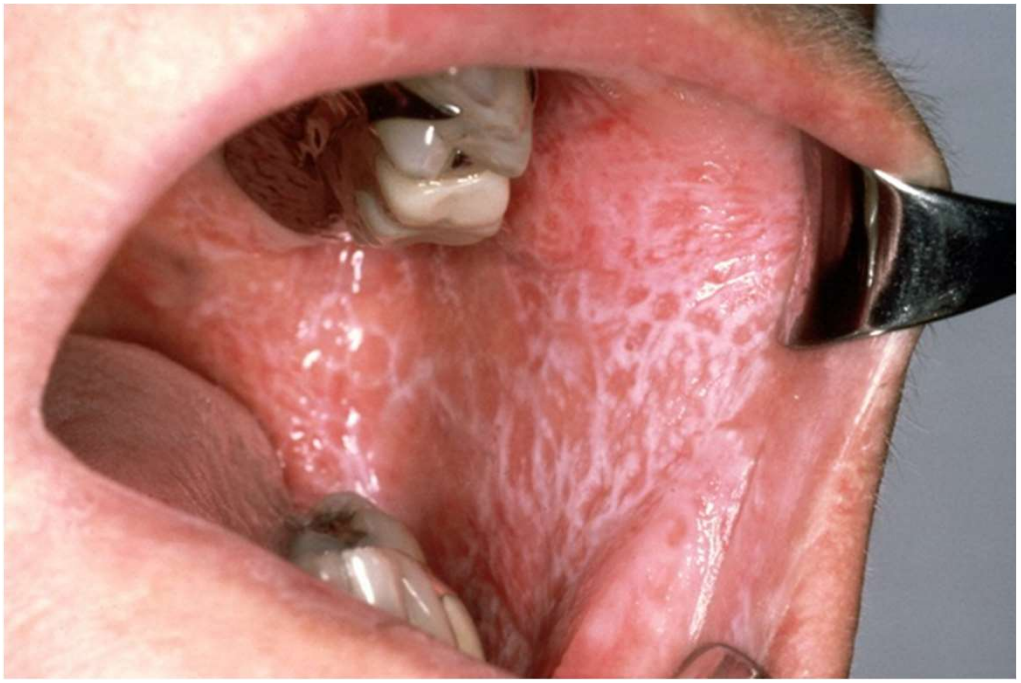
Associated with Hepatitis C infection

Typically on the flexor wrists, low back, penis/vagina, nails, lower legs

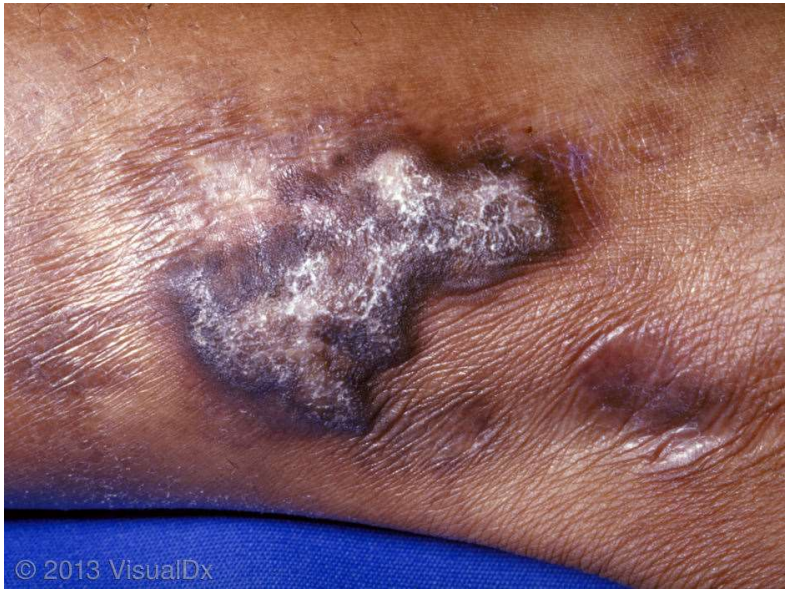


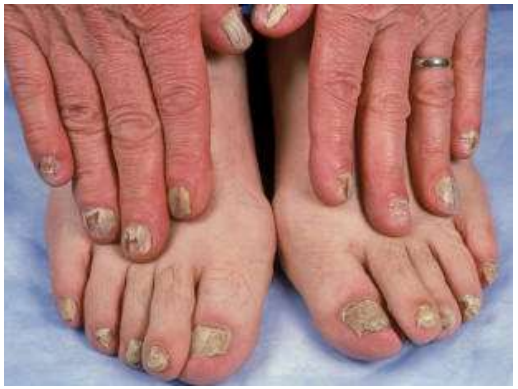
Lichen Planus – Wickham Striae

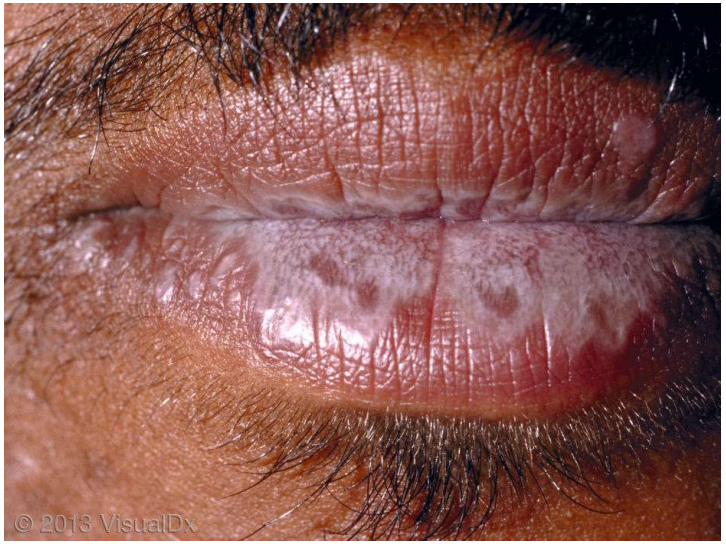
Wickham striae - Lacy white reticulated pattern, visible on mucosal surfaces on within lesion lesions



Lichen Planus







Lichen Planus Treatment:

- Topical steroids
- Oral Steroids
- Cyclosporine
- System Retinoids
- PUVA

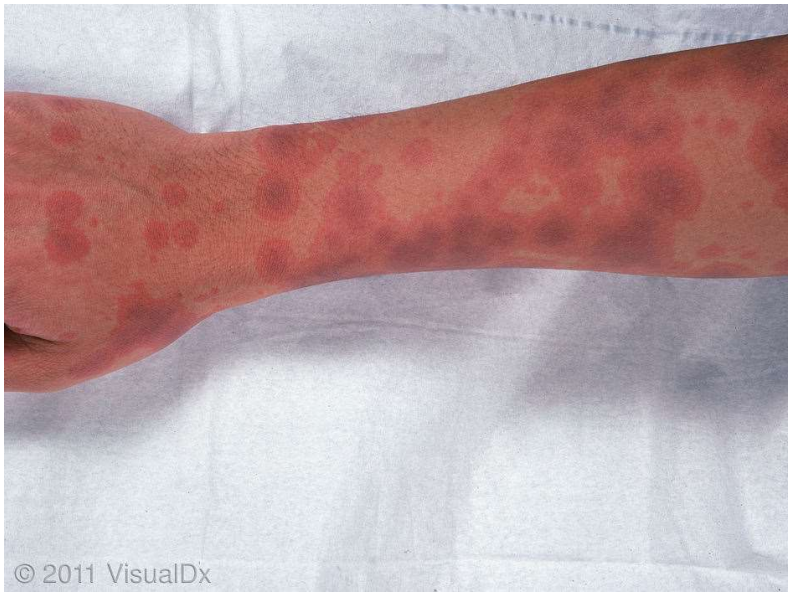
Erythema Multiforme

Self-limited Hypersensitivity reaction that presents as targetoid lesions
Previously thought to progress to SJS/TEN but this is not correct.
Occurs 24-72 hours after exposure and lasts 2 weeks
Can recur with repeated exposure

Erythema Multiforme

Two subtypes:
EM Major- Involves oral mucosa and can have systemic symptoms such as fever, arthralgias
EM Minor- skin lesions only

Caused by exposure to:
- Most Commonly HSV
- Drugs
  NSAIDs, Barbiturates, Sulphonamides, PCN, phenothiazines, anticonvulsants
- Other Viruses:
Mycoplasma pneumonia, Group A strep, Epstein-Barr



Erythema Multiforme – Treatment

Treat the underlying cause

- Antiviral for HSV -suppressive doses if EM is recurrent
- Antibiotics (Erythromycin) for Mycoplasma pneumoniae
- If it is a drug then stop the drug

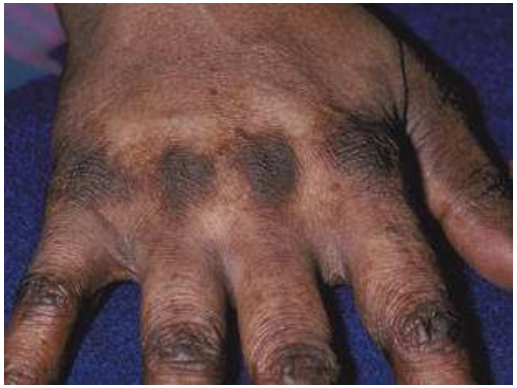
Supportive treatment

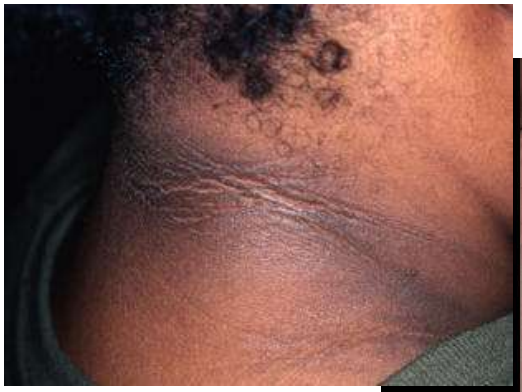
- Itch – antihistamine or topical corticosteroids
- Oral Pain – mouthwash containing local anesthetic
- Eye involvement – Refer to Ophthalmologist
- Unable to tolerate food or liquids by mouth – indication for hospitalization

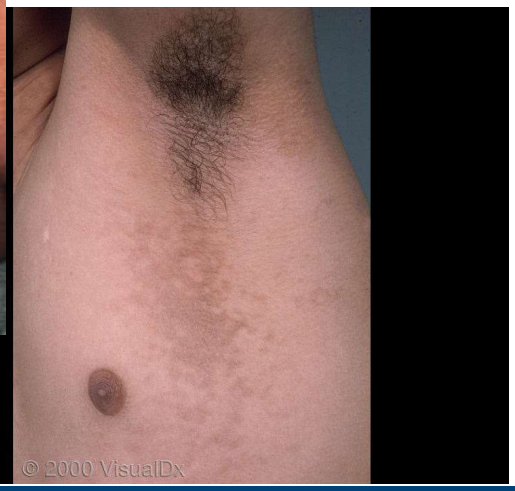
Treatment with Oral steroids is controversial

Acanthosis Nigricans

- Symmetric, dark brown hyper pigmented plaques with a velvety
- Locations: posterior neck, inguinal and inframammary folds, antecubital and popliteal fossae, and elbows and periumbilical region may also be involved. Rarer sites of involvement: knuckles, soles, eyelids, perioral
- Associated with Insulin Resistance (Test the HgA1C –lowering the HgA1C can help the hyperpigmentation

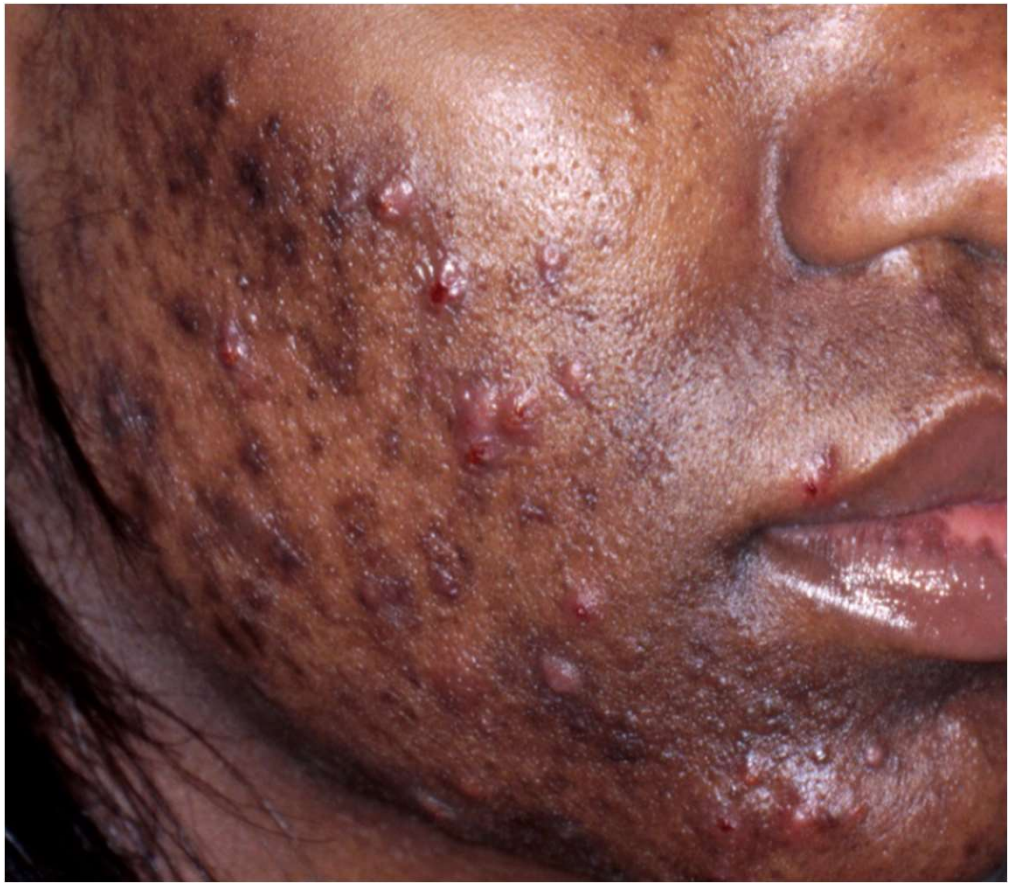


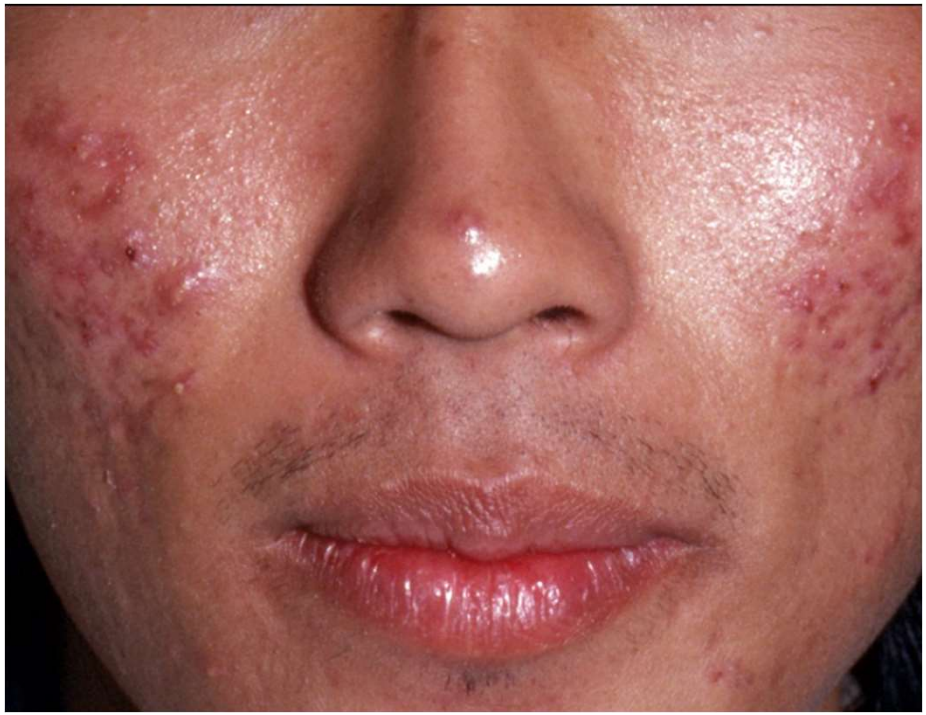


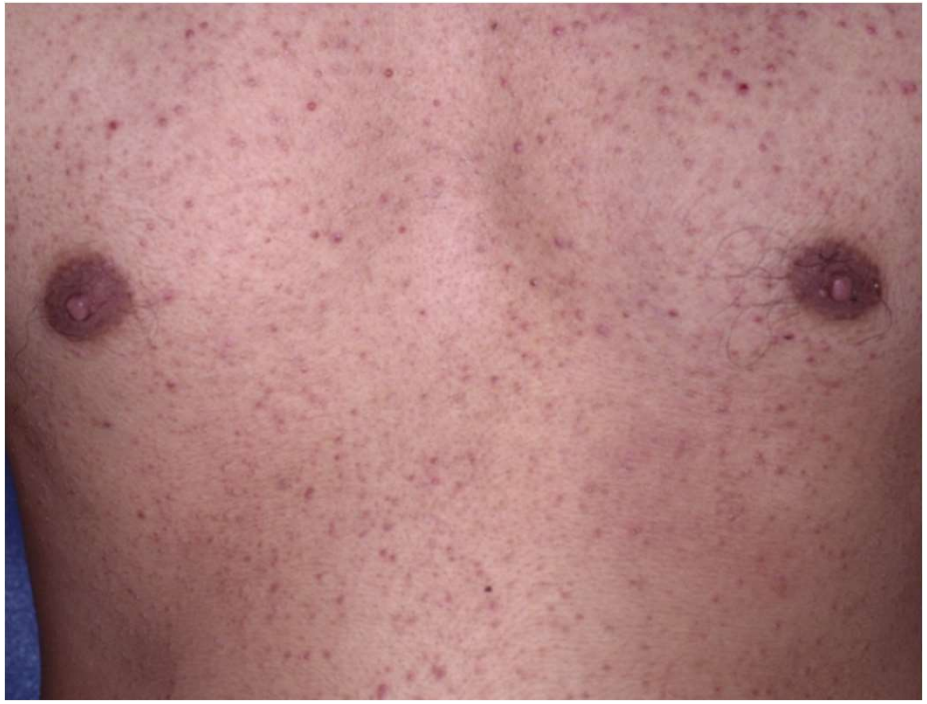


Acne Vulgaris

- acne has a variable presentation with a constellation of lesion types including open and closed comedones, papules, pustules, nodules, and cysts
- face is involved in most cases, and the trunk is affected in up to 61% of patients
- lesions can progress to scars, post inflammatory hyperpigmentation

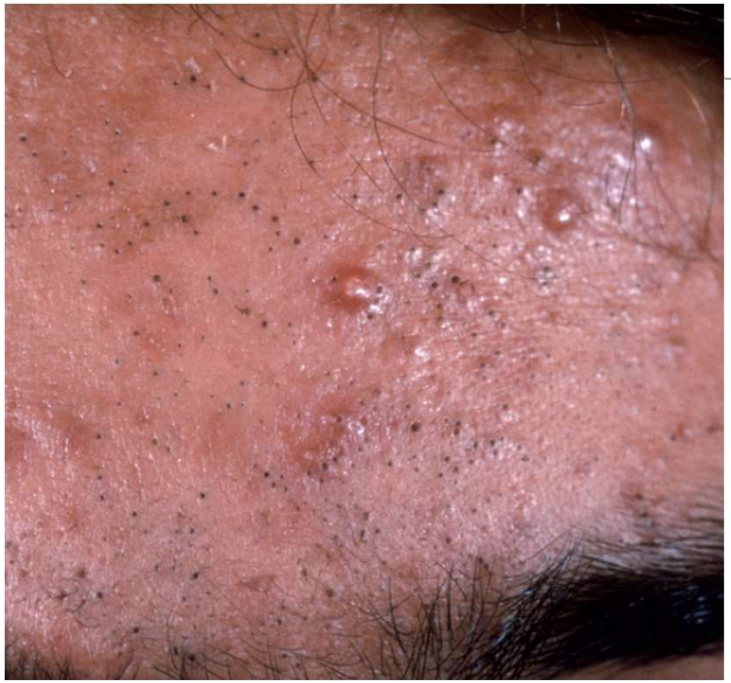


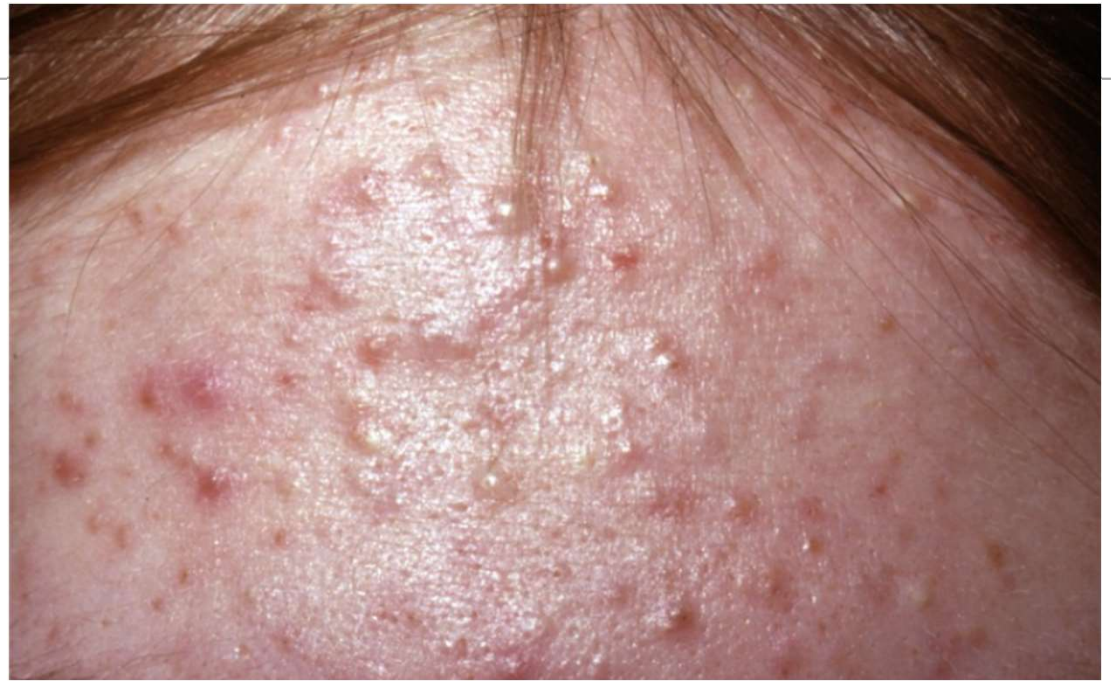




Causes of acne

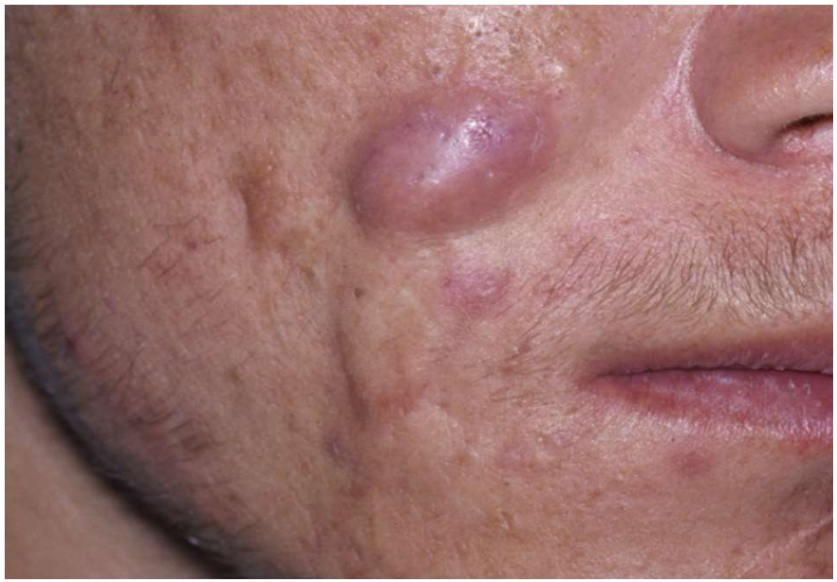
- multifactorial inflammatory disease affecting the pilosebaceous follicles of the skin
- follicular hyperkeratinization, microbial colonization with *Propionibacterium acnes*, sebum production, and complex inflammatory mechanisms involving both innate and acquired immunity.
- neuroendocrine regulatory mechanisms, diet, and genetic and nongenetic factors all may contribute to acne

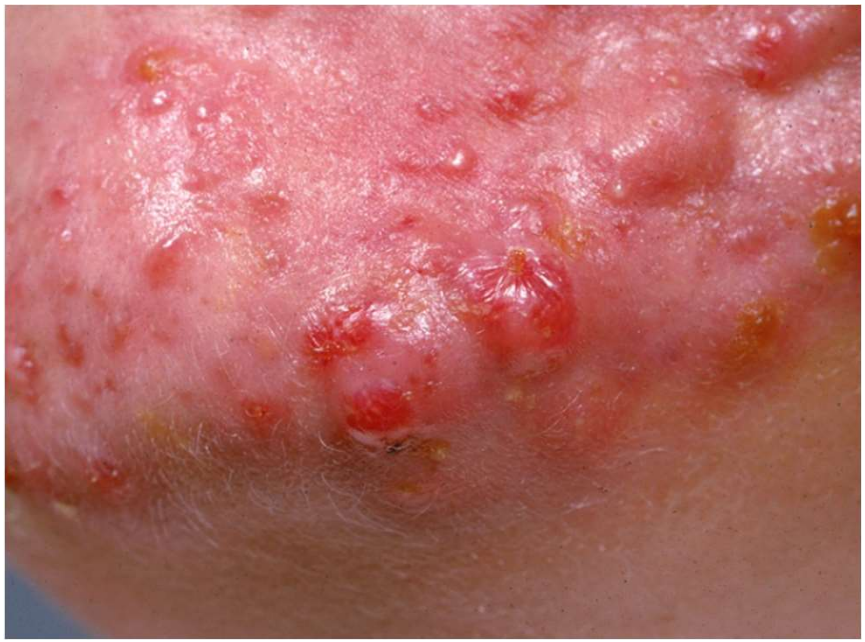




Types of Acne

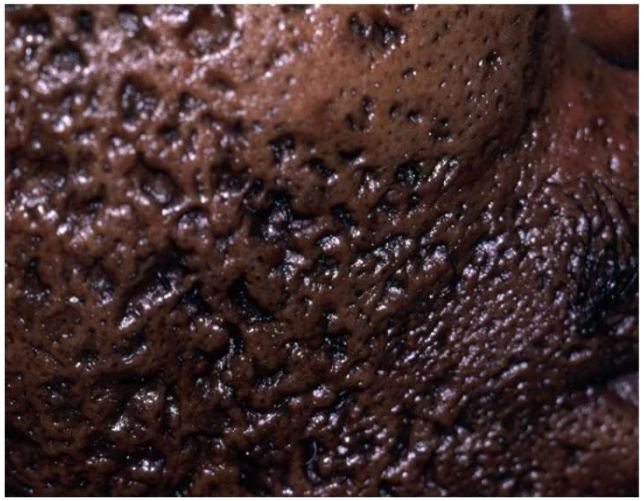
- Nodulocystic

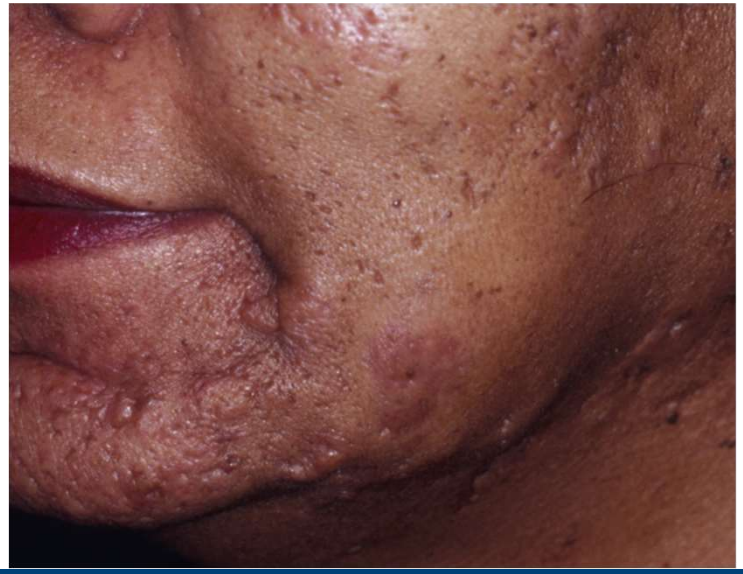


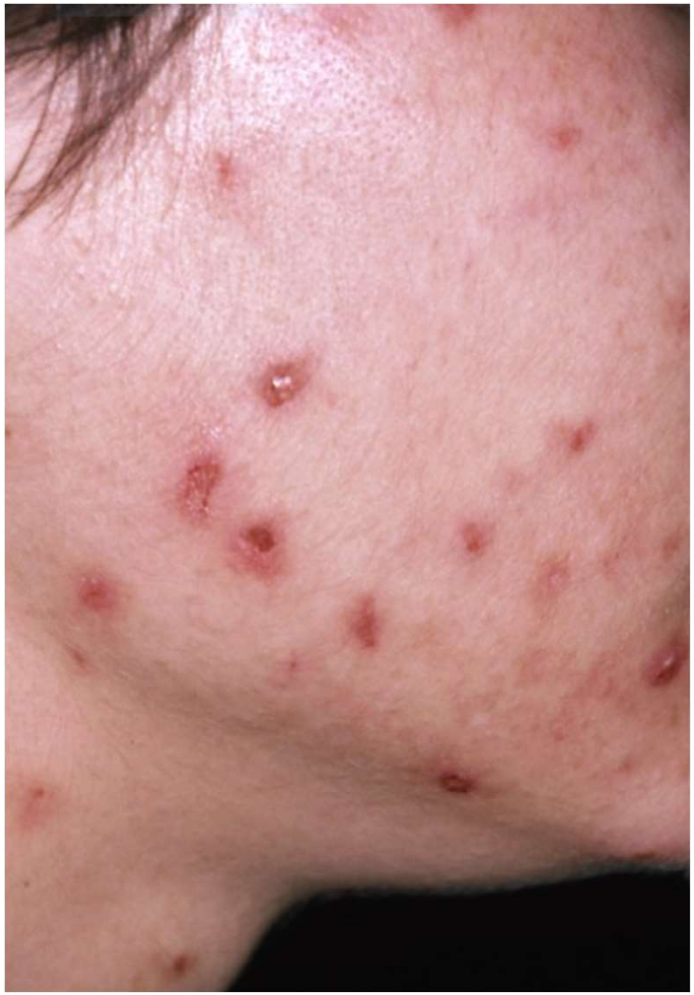


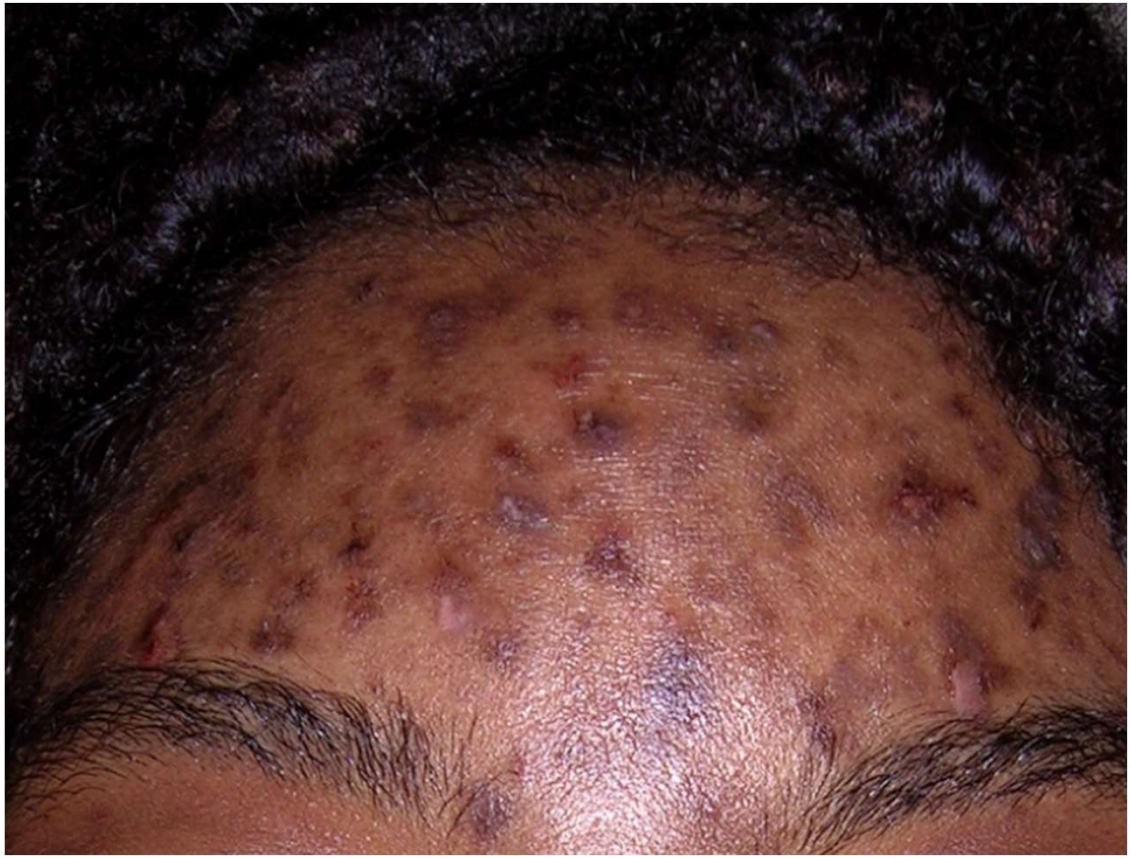
Types of Acne

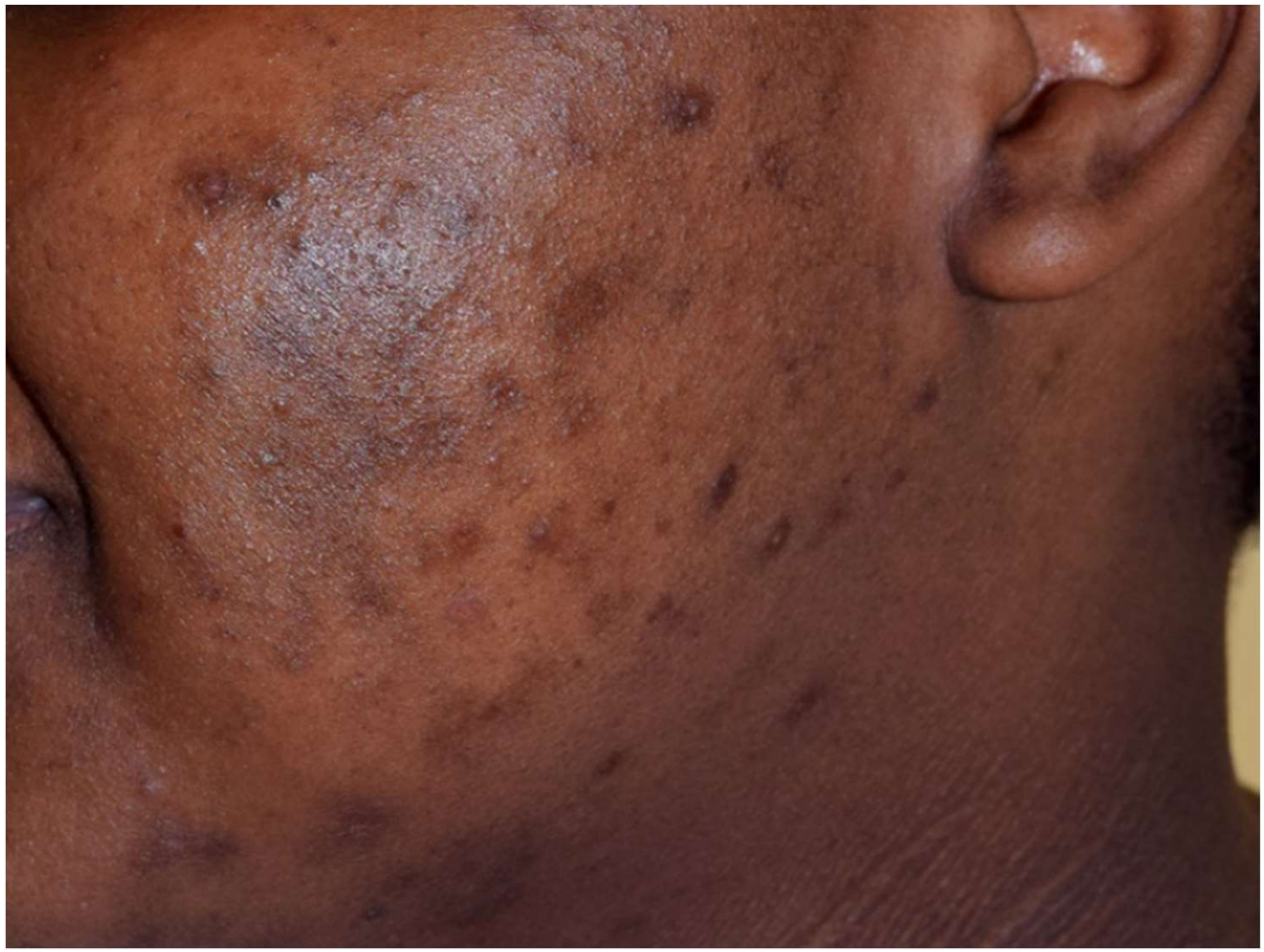
- Scarring





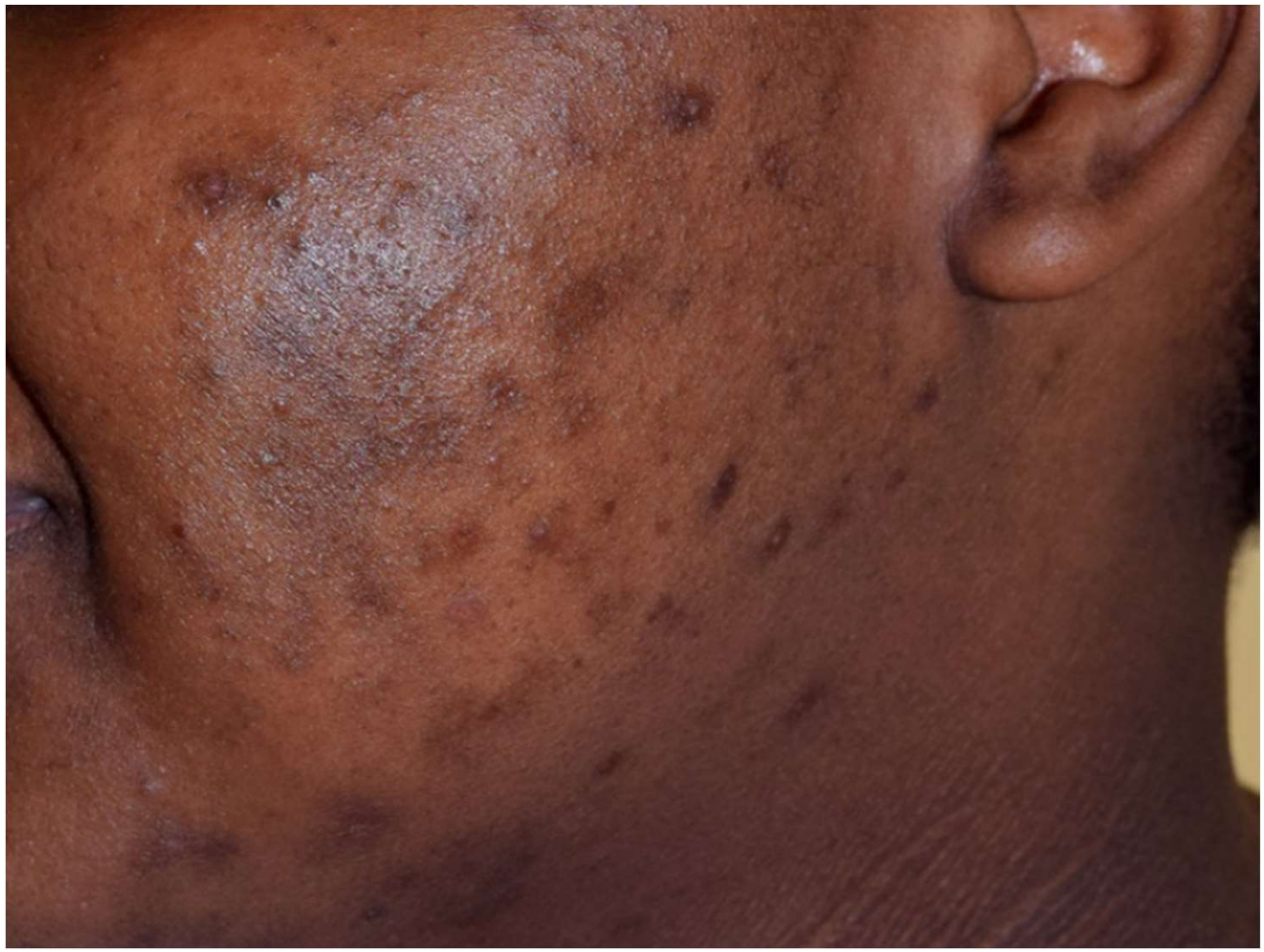






Special considerations skin of color

- Generally, darker skin reacts to injury or insult with localized melanin deposition, resulting in uneven skin tones, but even pale skin can have long-lasting dark or red spots after resolution of an acne lesion
- PIH is a common occurrence in patients with acne, particularly in those with darker skin and those who excoriate their lesions.
- PIH often has a prolonged duration and can be more bothersome than active acne lesions for the patient.



Special considerations: Skin of color

- study of Middle Eastern acne patients, more than half (56.4%) were primarily concerned with uneven skin tone
- Asian population (324 persons from 7 countries), Abad-Casintahan et al found PIH in 60% of acne
- PIH ranges in color from light brown to grey or black; dark purple lesions may be an early form of PIH.

M. Chandra, J. Levitt, C.A. Pensabene **Hydroquinone therapy for post-inflammatory hyperpigmentation secondary to acne: not just prescribable by dermatologists** Acta Derm Venereol, 92 (2012), pp. 232-235

Treatment of post inflammatory hyperpigmentation (PIH)

- sun protection
- treatment of underlying acne

Table III. Actions of agents used to treat postinflammatory hyperpigmentation

Agent	Mechanism
Retinoids	Increase keratinocyte turnover and remove pigmentation, inhibit tyrosinase, and reduce pigment transfer
Hydroquinone	Inhibition of melanogenesis via reduction in active tyrosinase
Kojic acid	Inactivates tyrosinase by chelating copper atoms
Azelaic acid	Selectively influences hyperactive and abnormal melanocytes, prevents tyrosine-tyrosinase binding
Flavonoids (aloesin from aloe vera plants, stilbene derivatives such as resveratrol, licorice extracts)	Inhibit tyrosinase activity at distal portions of the melanogenic pathway
Antioxidants/Redox agents (beta carotene and vitamin C and E)	Prevent oxidative damage to skin, scavenge reactive oxygen species, inhibit second messengers that stimulate melanogenesis, interact with copper at active site of tyrosinase
Niacinamide	Interrupts melanosome transfer from melanocyte to keratinocyte
Alpha hydroxy acids, salicylic acid, linoleic acid	Accelerate skin turnover, dispersing melanin; linoleic acid also reduces tyrosinase activity
Arbutin	Structural homolog for tyrosinase (competitive inhibitor), inhibits melanosome maturation

H.A. Gollnick, A. Abanmi, M. Al-Enezi, *et al.* **Management of acne in the Middle East** J Eur Acad Dermatol Venereol, 31 (Suppl 7) (2017), pp. 4-35

Additional treatments for PIH

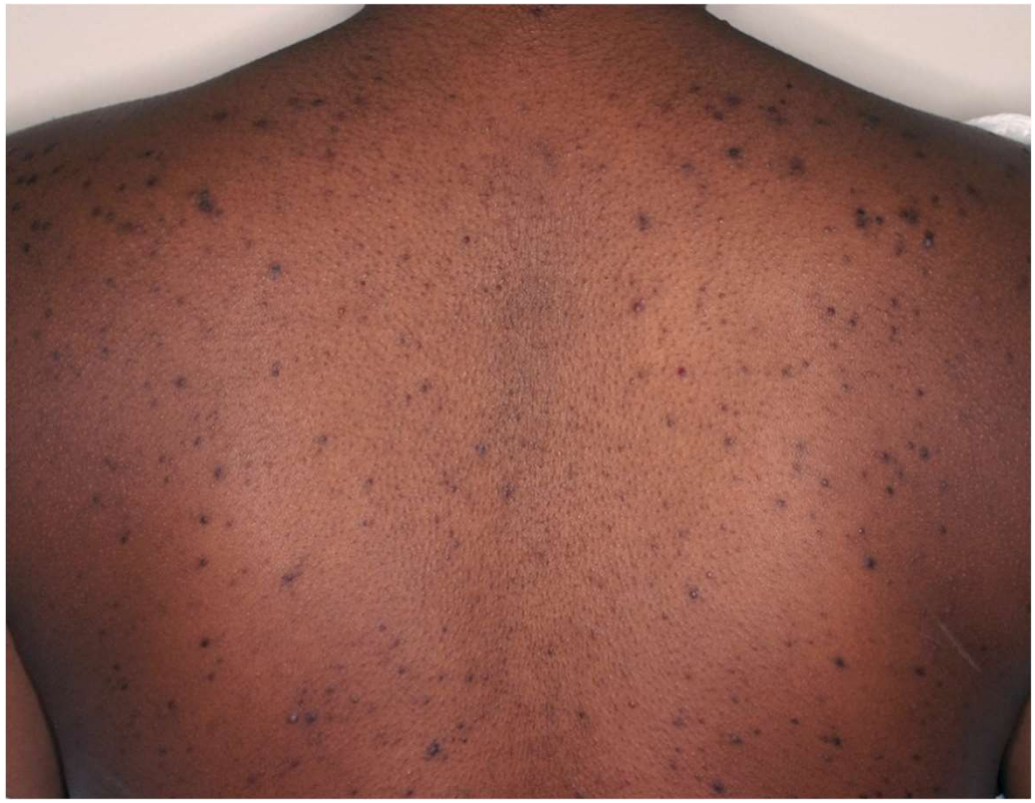
Chemical peels like salicylic acid peels

Lasers – Intense pulse light

Improving insulin resistance through diet and lifestyle can have a positive impact on both acne and the propensity for PIH.

Pityrosporum folliculitis

- Caused by Malassezia (normal flora) grows with antibiotic use or immunosuppression
- In cases of acne unresponsive to typical treatments
- Clinical presentation: truncal involvement or monomorphic appearance
- Itraconazole 200 mg daily for 1-2 weeks, or
- Fluconazole 100-200 mg daily for 1-3 weeks
- Recurrence is common



Rubenstein RM, Malerich SA. Malassezia (pityrosporum) folliculitis. *J Clin Aesthet Dermatol*. 2014;7(3):37-41.

Practical tips about steroid cream

Practical tips about steroid cream

- A word on vehicles
- Appropriate use for topical creams
- Steroid cream indications and contraindications, rashes that will not respond to steroid

Vehicles – foam/spray, solution, lotion, cream, ointment

Level of potency all else being equal

foam -->solution-->lotion-->cream -->ointment

Topicals- steroids, low-med-hi

Mild – Brand Name	Generic Name
Aclovate Cream/Ointment, 0.05%	Alclometasone dipropionate
Derma-Smoothe/FS Oil, 0.01%	Fluocinolone acetonide
Desonate Gel, 0.05%	Desonide
Synalar Cream/Solution, 0.01%	Fluocinolone acetonide
Verdeso Foam, 0.05%	Desonide

Least Potent – Brand Name	Generic Name
Cetacort Lotion, 0.5%/1%	Hydrocortisone
Cortaid Cream/Spray/Ointment	Hydrocortisone
Hytone Cream/Lotion, 1%/2.5%	Hydrocortisone
Micort-HC Cream, 2%/2.5%	Hydrocortisone
Nutracort Lotion, 1%/2.5%	Hydrocortisone
Synacort Cream, 1%/2.5%	Hydrocortisone

Mid-Strength – Brand Name	Generic Name
Cordran Ointment, 0.05%	Flurandrenolide
Elocon Cream, 0.1%	Mometasone furoate
Kenalog Cream/Spray, 0.1%	Triamcinolone acetonide
Synalar Ointment, 0.03%	Fluocinolone acetonide
Topicort LP Cream, 0.05%	Desoximetasone
Topicort LP Ointment, 0.05%	Desoximetasone
Westcort Ointment, 0.2%	Hydrocortisone valerate

Lower Mid-Strength – Brand Name	Generic Name
Capex Shampoo, 0.01%	Fluocinolone acetonide
Cordran Cream, 0.05%	Flurandrenolide
Cutivate Cream/Lotion, 0.05%	Fluticasone propionate
DermAtop Cream, 0.1%	Prednicarbate
DesOwen Lotion, 0.05%	Desonide
Locoid Cream/Lotion/Ointment/Solution, 0.1%	Hydrocortisone
Pandel Cream, 0.1%	Hydrocortisone
Synalar Cream, 0.03%/0.01%	Fluocinolone acetonide
Westcort Cream, 0.2%	Hydrocortisone valerate

Superpotent – Brand Name	Generic Name
Clobex Lotion/Spray/Shampoo, 0.05%	Clobetasol propionate
Cordran Tape, 4mcg/sq. cm.	Flurandrenolide
Cormax Cream/Solution, 0.05%	Clobetasol propionate
Diprolene Ointment, 0.05%	Betamethasone dipropionate
Lexette Foam, 0.05%	Halobetasol propionate
Olux E Foam, 0.05%	Clobetasol propionate
Olux Foam, 0.05%	Clobetasol propionate
Psorcon Ointment, 0.05%	Diflorasone diacetate
Psorcon E Ointment, 0.05%	Diflorasone diacetate
Temovate Cream/Ointment/Solution, 0.05%	Clobetasol propionate
Topicort Topical Spray, 0.25%	Desoximetasone
Ultravate Cream/Ointment, 0.05%	Halobetasol propionate
Ultravate Lotion, 0.05%	Halobetasol propionate
Vanos Cream, 0.1%	Fluocinonide

Potent to Superpotent – Brand Name	Generic Name
Bryhali Lotion, 0.01%	Halobetasol propionate
Doubrii Lotion, 0.01%/0.045%	Halobetasol propionate/tazarotene

Practical clinical practice

- Low potency –Triamcinolone 0.025% cream (for use on the face or body fold areas)
- Mid potency - Triamcinolone 0.1% cream (for use on the body)
- High Potency – Clobetasol 0.05% cream (for use on thick plaques)

Steroid cream indications

- Indications:
  - Eczemas (itchy skin)
  - Contact dermatitis
  - Poison ivy
  - Psoriasis
  - Hand dermatitis
  - Lichen planus

Rashes that will not respond to steroid

- Infection
  - Tinea
  - Candida
  - Bacterial
  - Yeast –Malassezia
  - Demodex
  - Scabies
- Cutaneous lymphoma
- Cancer
- Rosacea/perioral dermatitis

Steroid cream cautions

- Long term continuous use
- Intertriginous skin
- Thin skin
  - Eyelids, children's skin

Steroid atrophy

- Telangiectases
- Thinning of the skin
- Striae
- Hypopigmentation (Skin of color)

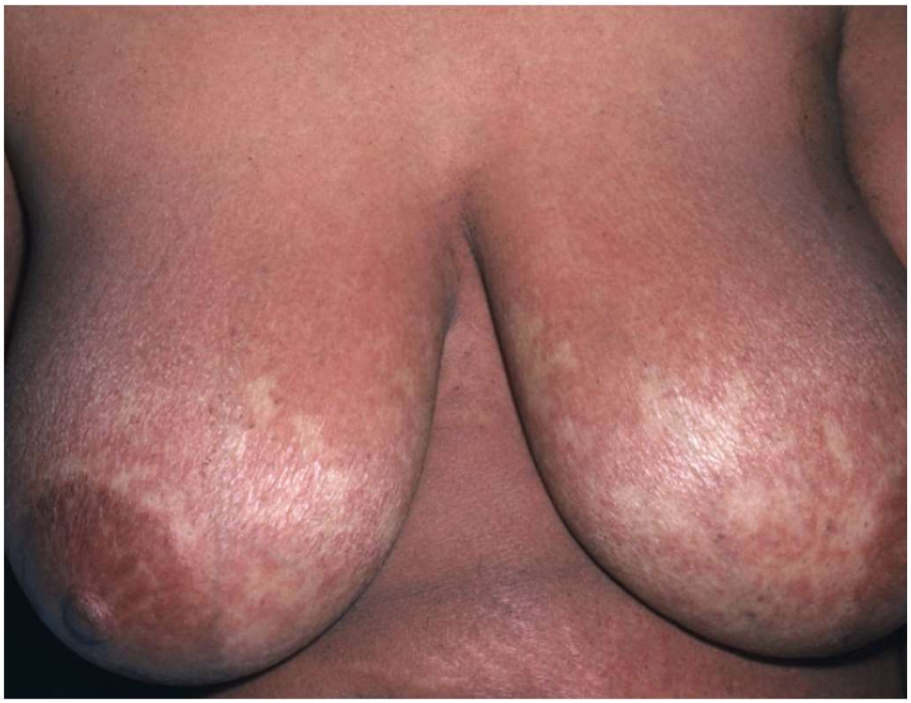
When to refer

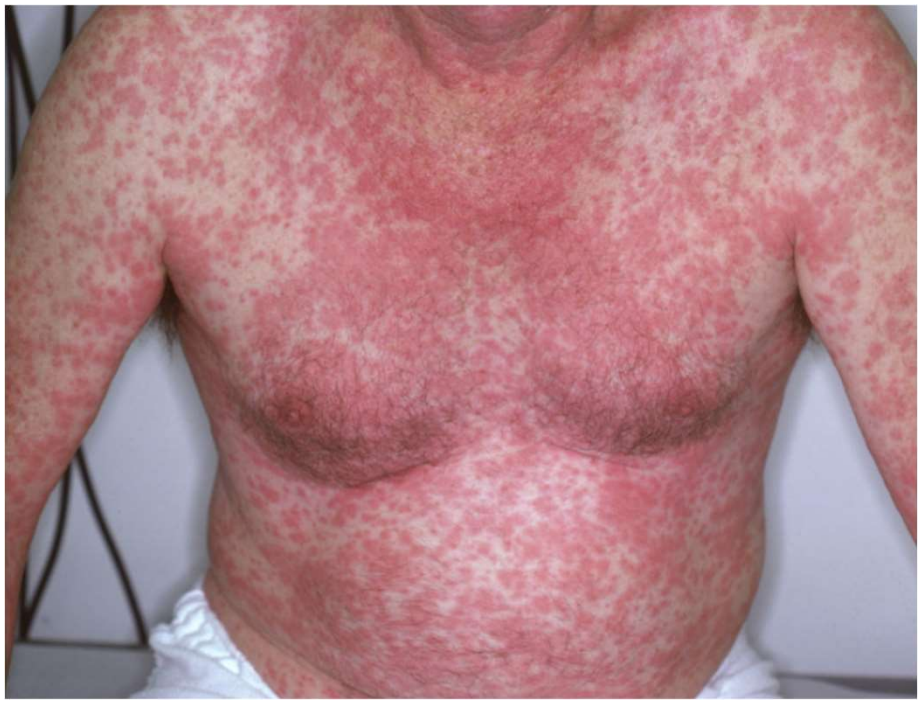
Derm red flags

When steroids don't fix it

Dermatologic Red Flags:

- Rash+ fever (infectious vs. inflammatory)
- Mucosal membrane involvement
- Evidence of blistering or desquamation
- Systemic instability: tachycardia, hypotension, renal failure, etc
- Skin pain out of proportion
- Erythroderma, patient shaking/inability to regulate body temperature





Morbilliform drug

Morbilliform drug

Erythematous macules and papules start on the chest and spread outwards to arms and back and legs, symmetric

History of new medication started 2-8 weeks previously

Itching, most patients are afebrile, a low-grade fever may occur in more severe reactions

Morbilliform drug

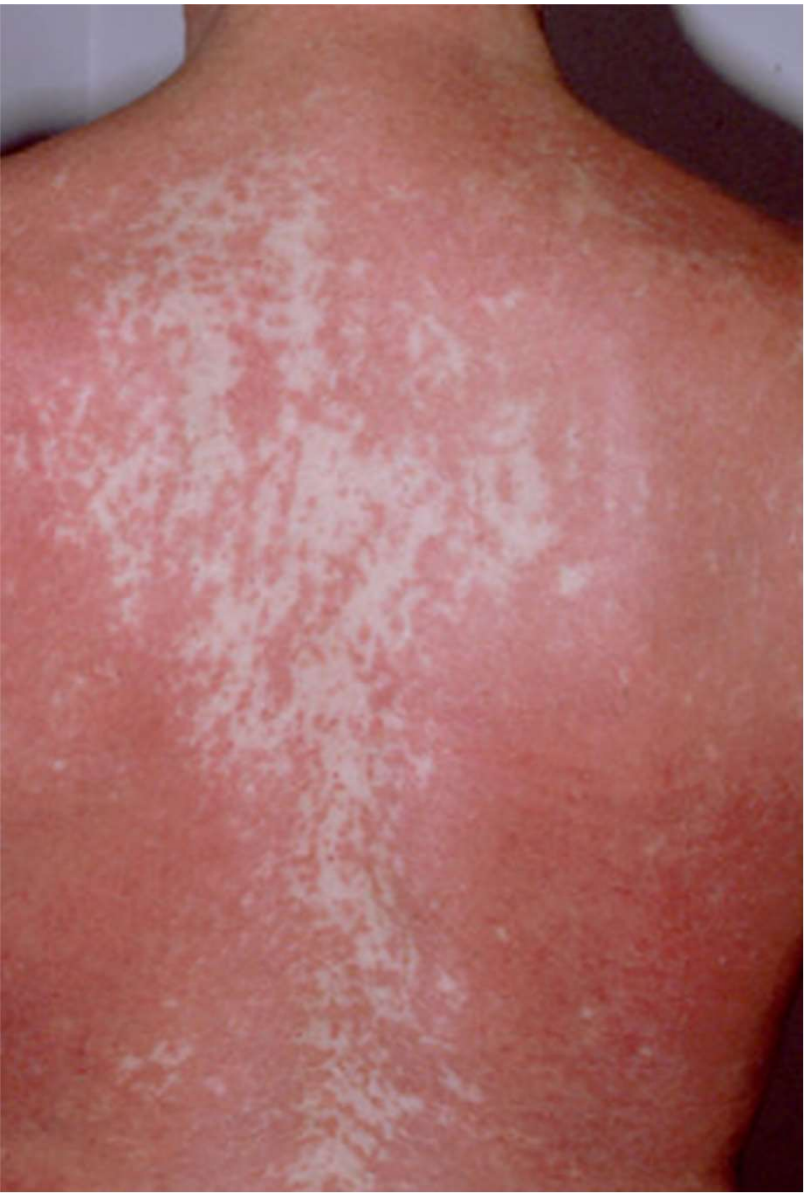
- most commonly seen with the use of antibiotics (penicillins and sulfas), allopurinol, phenytoin, barbiturates, chlorpromazine, carbamazepine, gold, d-penicillamine, captopril, naproxen, and piroxicam, but many other drug culprits have been reported, including chemotherapeutic, biologic, and immunotherapeutic (checkpoint inhibitor) agents

Morbilliform drug – work up

- A punch biopsy usually shows nonspecific perivascular mononuclear cells. Eosinophils in the biopsy are suggestive of a drug-induced eruption.
- Liver function tests – Look for elevated alanine transaminase (ALT), although aspartate transaminase (AST) and alkaline phosphatase can also be elevated. Liver function test abnormalities can persist for months despite adequate therapy.
- CBC with differential – Look for the presence of leukocytosis with eosinophilia and atypical lymphocytes.
- Urinalysis and renal function tests – Look for elevated blood urea nitrogen (BUN) and creatinine, proteinuria, and hematuria.

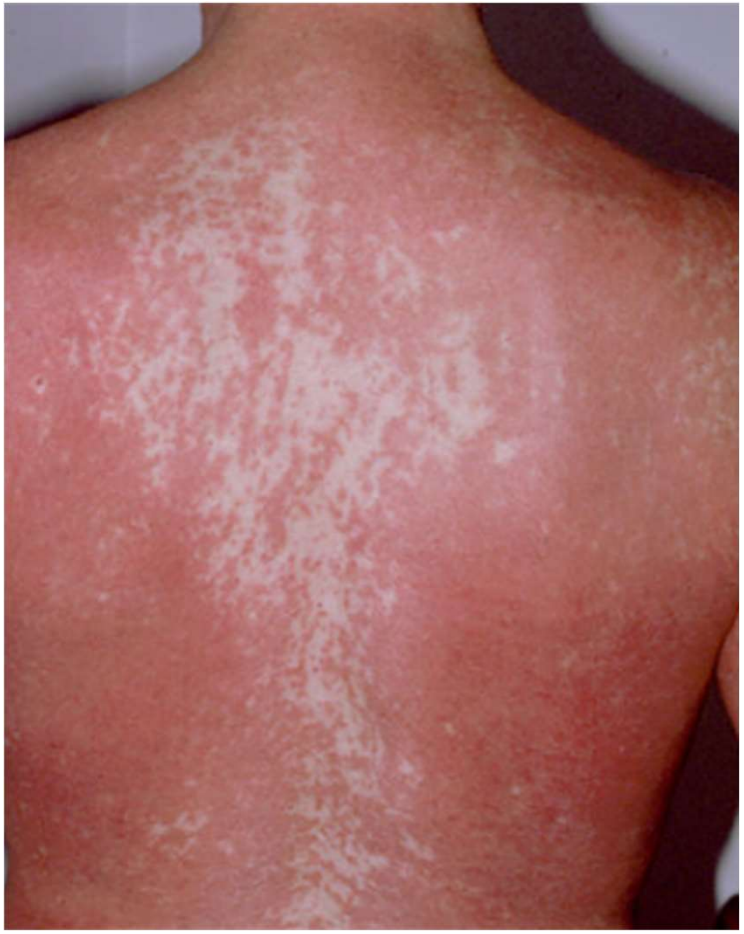
Morbilliform drug – Treatment

- Discontinue the offending agent(s).
- Antihistamines for itching (hydroxyzine 50 mg every 4-6 hours, cetirizine 10-20 mg twice daily, or fexofenadine 180 mg daily).
- Systemic corticosteroids are usually of little benefit. Medium- or high-potency topical steroids (such as betamethasone dipropionate 0.05% cream twice daily or triamcinolone acetonide 0.1% cream twice daily, for up to 14 days) may alleviate pruritus in some patients.
- **Note:** It may take an additional 7-14 days after stopping the medication before the eruption completely resolves.



DRESS (Drug reaction with eosinophilia and system symptoms)

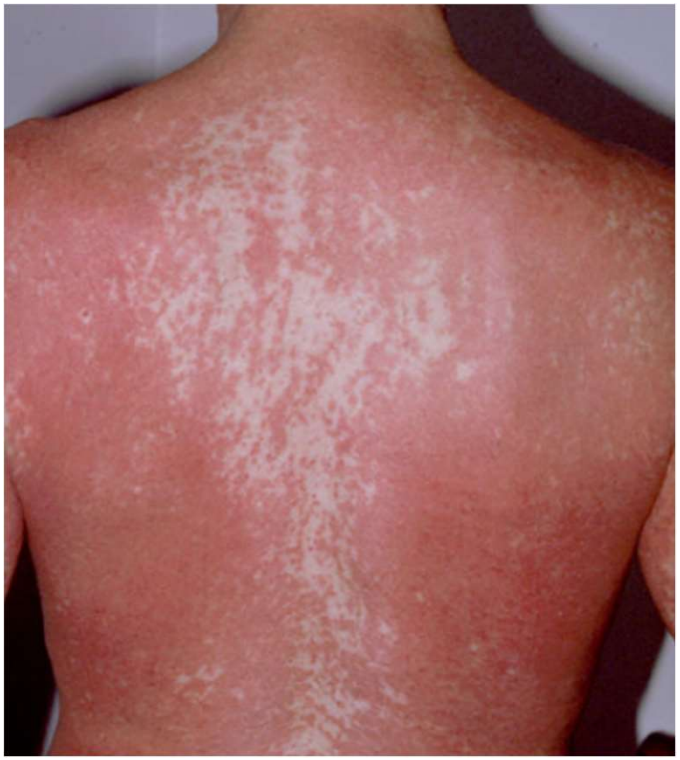
- a serious multisystem drug reaction
- fever, rash, eosinophilia, and internal organ involvement
- occur between 2-8 weeks after starting a new medication but may develop months later
- Additional clinical findings include pharyngitis, lymphadenopathy, and facial and hand edema, while internal organ involvement most commonly affects the liver and hematologic and renal systems



DRESS (Drug reaction with eosinophilia and system symptoms)

Common Inciting Drugs

- Anticonvulsants
  - phenytoin, carbamazepine, phenobarbital, and lamotrigine
- Minocycline, allopurinol, azathioprine, metronidazole, dapsone, antiretroviral agents (eg, abacavir), clopidogrel, and ticlopidine, sulfonamides, and nonsteroidal anti-inflammatory drugs (NSAIDs)



DRESS (Drug reaction with eosinophilia and system symptoms)

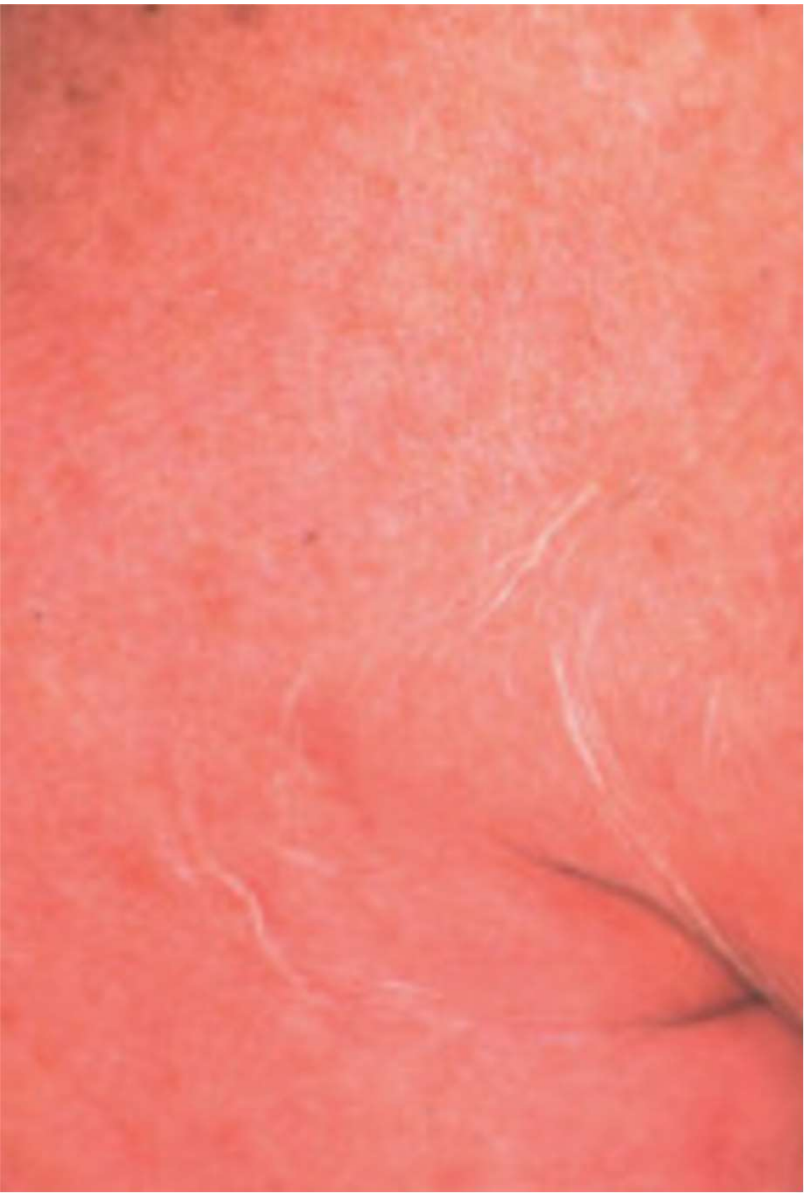
- It is important to identify any internal organ involvement.

  Laboratory studies:

- Liver function tests – Look for elevated alanine transaminase (ALT), although aspartate transaminase (AST) and alkaline phosphatase can also be elevated. Liver function test abnormalities can persist for months despite adequate therapy.
- CBC with differential – Look for the presence of leukocytosis with eosinophilia and atypical lymphocytes.
- Urinalysis and renal function tests – Look for elevated blood urea nitrogen (BUN) and creatinine, proteinuria, and hematuria.
- Chest x-ray and/or ECG can be ordered if symptoms of cough or chest discomfort are present.

DRESS (Drug reaction with eosinophilia and system symptoms)

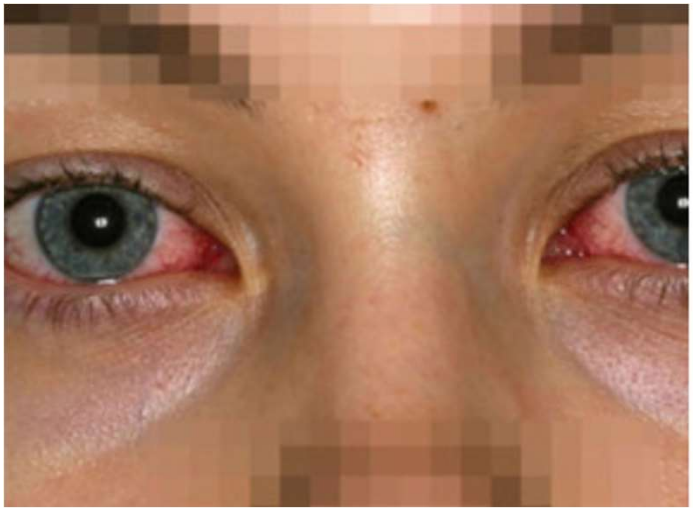
- inciting drug must be identified and immediately stopped
- May warrant hospitalization
  - temperature regulation, nutrition, and fluid and electrolyte balance
- Oral rechallenge tests and skin testing may be harmful and are therefore not recommended
- increased risk for becoming hypothyroid. This usually occurs 4-12 weeks after the reaction.
- dose of 1-2 mg/kg prednisone daily, slow taper 4-6 weeks relapses may occur with more rapid tapering



Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)

- Variants of the same condition
- Serious **drug reaction** of the skin and mucous membranes. Starts with flu-like symptoms, followed by a painful rash that spreads and blisters. Then the top layer of affected skin dies, sheds and begins to heal after several days.
- SJS - Skin detachment < 10% of body surface area (BSA)
- TEN- Detachment > 30% of BSA

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)



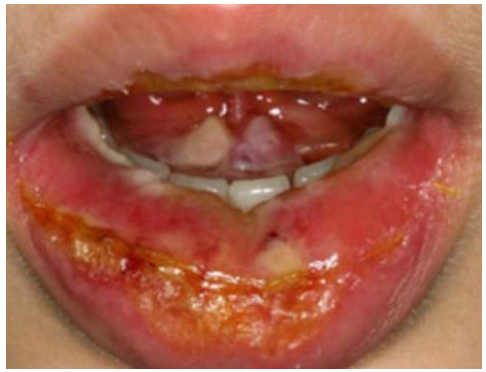
- Develops within the first week of antibiotic therapy but up to 2 months after starting an anticonvulsant. For most drugs, the onset is within a few days up to 1 month.
- Before the rash appears, there is usually a prodromal illness of several days duration resembling an upper respiratory tract infection or 'flu-like illness. Symptoms may include:
- Fever > 39 C
- Sore throat, difficulty swallowing
- Runny nose and cough
- Sore red eyes, conjunctivitis
- General aches and pains.
- There is then an abrupt onset of a tender/painful red skin rash starting on the trunk and extending rapidly over hours to days onto the face and limbs (but rarely affecting the scalp, palms or soles). The maximum extent is usually reached by four days.

SJS/TEN Offending medications

- The drugs that most commonly cause SJS/TEN are antibiotics in 40%. Other drugs include:
- Sulfonamides: cotrimoxazole
- Beta-lactam: penicillins, cephalosporins
- Anti-convulsants: lamotrigine, carbamazepine, phenytoin, phenobarbitone
- Allopurinol
- Paracetamol/acetaminophen
- Nevirapine (non-nucleoside reverse transcriptase inhibitor)
- Nonsteroidal anti-inflammatory drugs (NSAIDs) (oxicam type mainly).

Care of a patient with SJS/TEN requires:

- Cessation of suspected causative drug(s) — the patient is less likely to die, and complications are less if the culprit drug is on or before the day that blisters/erosions appear
- Hospital admission — preferably immediately to an intensive care and burns unit with specialist nursing care, as this improves survival, reduces infection and shortens hospital stay
- Consider fluidised air bed
- Nutritional and fluid replacement (crystalloid) by intravenous and nasogastric routes — reviewed and adjusted daily
- Temperature maintenance — as body temperature regulation is impaired, the patient should be in a warm room (30–32C)
- Pain relief — as pain can be extreme
- Sterile handling and reverse isolation procedures.
- **Skin care**
- Examine daily for the extent of detachment and infection (take swabs for bacterial culture).
- Topical antiseptics can be used (eg, silver nitrate, chlorhexidine [but not silver sulfadiazine as it is a sulfa drug])
- Dressings such as gauze with petrolatum, non-adherent nanocrystalline-containing silver gauze or biosynthetic skin substitutes such as Biobrane® can reduce pain.
- Avoid using adhesive tapes and unnecessary removal of dead skin; leave the blister roof as a ‘biological dressing’.



Care of a patient with SJS/TEN requires:

- **Eye care**
- Daily assessment by an ophthalmologist
- Frequent eye drops/ointments (antiseptics, antibiotic, corticosteroid)
- **Mouth care**
- Topical oral anaesthetic
- **Genital care**
- If ulcerated, prevent vaginal adhesions using intravaginal steroid ointment, soft vaginal dilators.
- **Lung care**
- Consider aerosols, bronchial aspiration, physiotherapy
- May require intubation and mechanical ventilation if trachea and bronchi are involved
- **Urinary care**
- Catheter because of genital involvement and immobility
- Culture urine for bacterial infection
- **General**
- Psychiatric support for extreme anxiety and emotional lability
- Physiotherapy to maintain joint movement and reduce the risk of pneumonia
- Regular assessment for staphylococcal or gram negative infection
- The appropriate antibiotic should be given if an infection develops; prophylactic antibiotics are not recommended and may even increase the risk of sepsis
- Consider heparin to prevent thromboembolism (blood clots).

How can SJS/TEN be prevented?

- People who have survived SJS/TEN must be educated to avoid taking the causative drug or structurally related medicines as SJS/TEN may recur. Cross-reactions can occur between:
- The anticonvulsants carbamazepine, phenytoin, lamotrigine and phenobarbital
- Beta-lactam antibiotics penicillin, cephalosporin and carbapenem
- Nonsteroidal anti-inflammatory drugs
- Sulfonamides: sulfamethoxazole, sulfadiazine, sulfapyridine.
- In the future, we may be able to predict who is at risk of SJS/TEN using genetic screening.
- Allopurinol should be prescribed for good indications (eg, gout with hyperuricemia) and commenced at a low dose (100 mg/day), as SJS/TEN is more likely at doses > 200 mg/day.

Dermatologic Red Flags:

- Rash+ fever (infectious vs. inflammatory)
- Mucosal membrane involvement
- Evidence of blistering or desquamation
- Systemic instability: tachycardia, hypotension, renal failure, etc
- Skin pain out of proportion
- Erythroderma, patient shaking/inability to regulate body temperature

When Steroids don't fix it

- Diagnosis may be in question
- The Clinical treatment is not adequate by the determination of the provider or patient
- Need for additional therapies like phototherapy, isotretinoin, Biologic treatments that the PCP provider does not have access to expertise to monitor adverse effects

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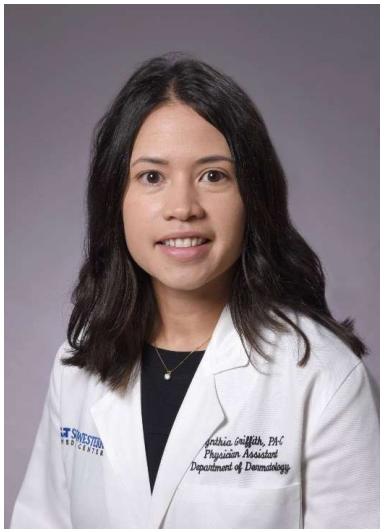

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Questions and discussion