SAUSHEC Lunch and Learn Primary Care Webinar Series

Bread & Butter Dermatology: Clinical Review of Dermatologic Conditions

16 June 2021
1300 – 1400 ET

"Medically Ready Force…Ready Medical Force"

CPT Christopher A Riley, M.D., M.C.

Dr. Christopher Riley is a dermatology resident, PGY-4. He completed his Bachelor of Science in Molecular Biology with a Minor in Chemistry at Stetson University. Dr. Riley matriculated into the Virginia Commonwealth University School of Medicine followed by a transitional internship at San Antonio Military Medical Center (SAMMC). Dr. Riley has an interest in complex medical dermatology. Upon completion of dermatology residency, Dr. Riley will be stationed at Dwight D. Eisenhower Army Medical Center, Fort Gordon, Georgia as a staff dermatologist.

CPT Eddie A. Kwan, M.D., M.C.

Dr. Eddie Kwan is a dermatology resident, PGY-4. He completed his Bachelor of Science in Molecular and Cellular Biology and Anatomy and Physiology at the University of Arizona. Dr. Kwan matriculated into the Uniformed Services University of Health Sciences School of Medicine followed by a transitional internship at San Antonio Military Medical Center (SAMMC). Dr. Kwan has an interest in laser and energy-based treatment devices. Upon completion of dermatology residency, Dr. Kwan will be stationed at Brian Allgood Army Community Hospital, USAG Humphries, Korea as a staff dermatologist.

Lt Col Justin P. Bandino, M.D., F.A.A.D.

Dr. Justin Bandino is a board-certified dermatologist and board-certified dermatopathologist. After completing undergraduate studies at the United States Air Force Academy with a Bachelor of Science in Biology and a Minor in Philosophy, Dr. Bandino matriculated to the Uniformed Services University of the Health Sciences School of Medicine in Bethesda, Maryland. Dr. Bandino then completed a Transitional internship at the San Antonio Military Medical Center (SAMMC) before serving two obligatory years as a Flight Surgeon in the U.S. Air Force prior to Dermatology Residency where he served as a chief resident also at SAMMC. After Dermatology Residency, the Air Force immediately stationed Dr. Bandino at Langley Air Force Base in Virginia where he served as the solo staff dermatologist for a large base hospital practicing clinical dermatology as well as responding to inpatient, ER, and NICU consults. Dr. Bandino then completed a Dermatopathology Fellowship at the Medical University of South Carolina in Charleston, SC. Following completion of his Fellowship, Dr. Bandino returned to the military Dermatology Residency program in San Antonio as faculty and served as Academic Director, Lab Director, and Medical Director. He is currently the Program Director and an Associate Professor.

Disclosures

- Drs. Justin Bandino, Eddie Kwan, and Christopher Riley have no relevant financial or non-financial relationships to disclose relating to the content of this activity.
- The views expressed in this presentation are those of the authors and do not necessarily reflect the official policy or position of the Department of Defense or the U.S. Government.
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Learning Objectives

At the completion of this activity, participants will be able to:

1. Identify the clinical features of common eczematous, psoriasiform, and acneiform dermatoses
2. Explain cutaneous manifestations of autoimmune and connective tissue diseases
3. Differentiate various drug eruptions
4. Describe the clinical features of common immunobullous diseases
5. Recognize nodules and tumors of the skin

Outline

- Dermatitis
- Psoriasis
- Acne
- Rosacea
- Autoimmune Connective Tissue Diseases
- Drug Reactions
- Immunobullous Dermatoses
- Nodules and Tumors of the Skin

Dermatitis

- Contact Dermatitis
- Stasis Dermatitis
- Seborrheic Dermatitis

Contact Dermatitis

- Irritant Contact Dermatitis
  - 80% of contact dermatitis
  - Cytotoxic effect of irritants on skin
  - Acute
    - Acute exposure to toxic agent
    - Pain/burning > pruritus, localized erythema, vesicles, edema, +/- scale/crust, no distant spread
  - Chronic
    - Repeated exposure to mild irritants
    - Diffuse or localized ill defined scaly patches and plaques
- Examples of irritants: water, acids, alkali, metal salts, solvents, alcohols, glycols, disinfectants, detergents, cleaners, foods, body fluids

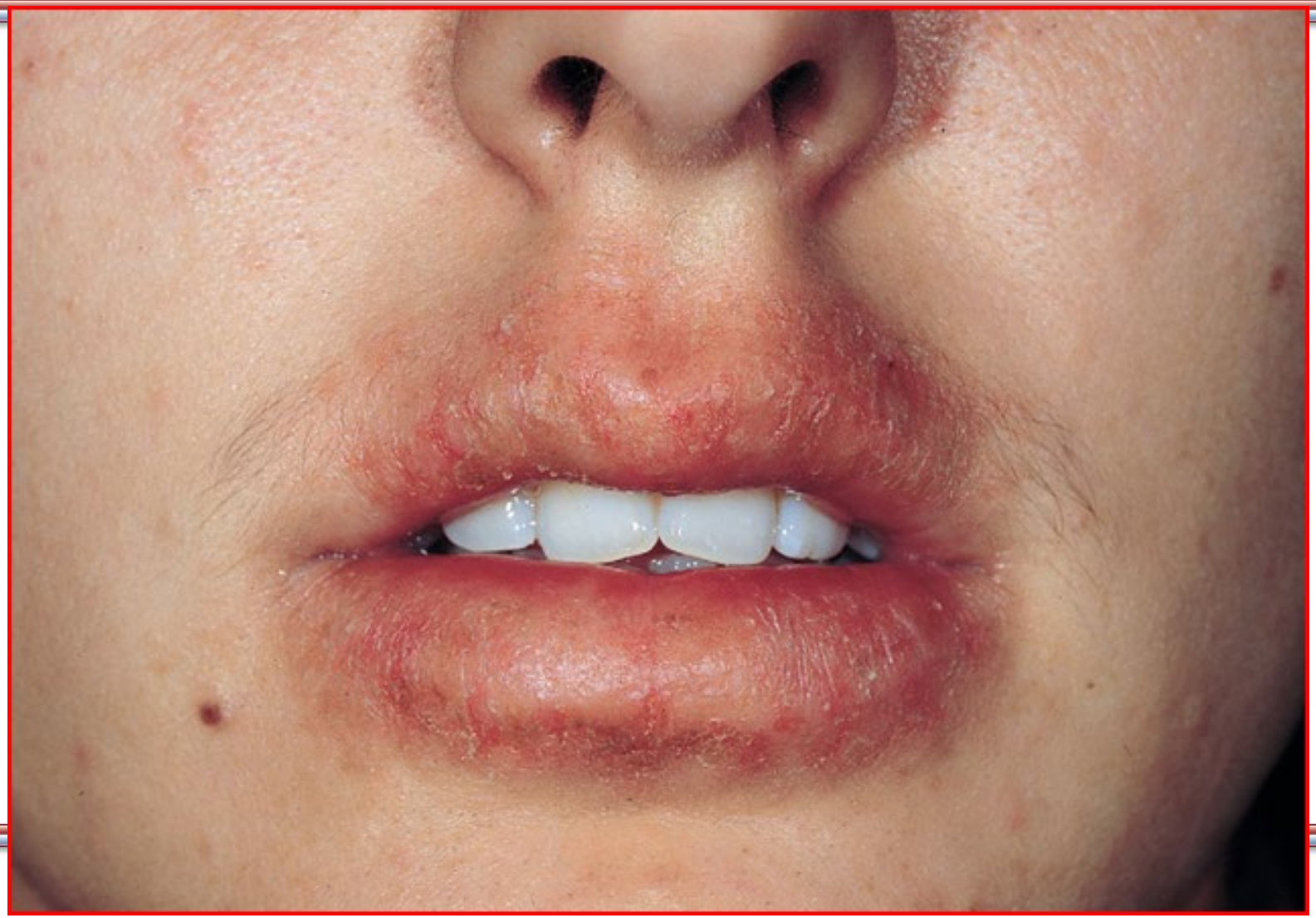
Contact Dermatitis

- Allergic
  - Delayed type IV hypersensitivity
  - 24-96 hours after exposure
    - Prior sensitization required
  - Clinical presentation: acute or chronic eczematous eruption typically localized to areas of exposure
  - **Nickel and urushiol (poison ivy) are the most common causes**
    - Neomycin, bacitracin
    - Fragrances
    - Many other causes
  - Patch testing is the gold standard for diagnosis

Contact Dermatitis

- Avoid irritants/allergens if known
- The basics
  - Gentle skin care
  - Barrier repair
  - Topical corticosteroids (potency dependent on acuity and location)
  - Topical calcineurin inhibitors
- Consider 2-4 week taper of systemic corticosteroids for widespread, acute allergic contact dermatitis
  - Avoid short courses of steroids (5-day dose pack of methylprednisolone) – increases risk of flaring after completing course
- If not responding, consider an allergy to steroids or vehicle
  - Creams most commonly
  - Consider desoximetasone [topical steroid least likely to cause allergic contact dermatitis (ACD)]

Contact Dermatitis



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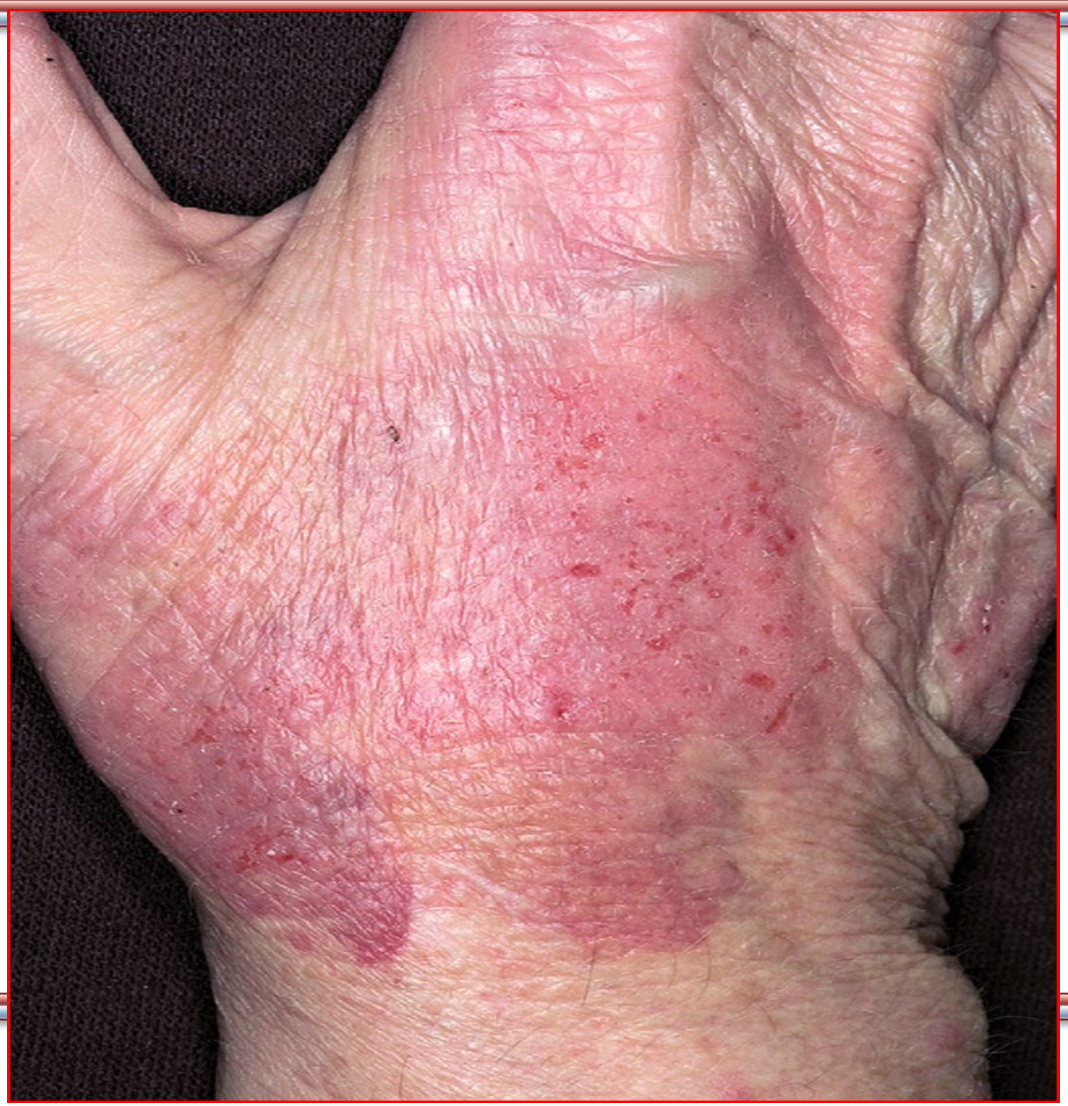
Contact Dermatitis



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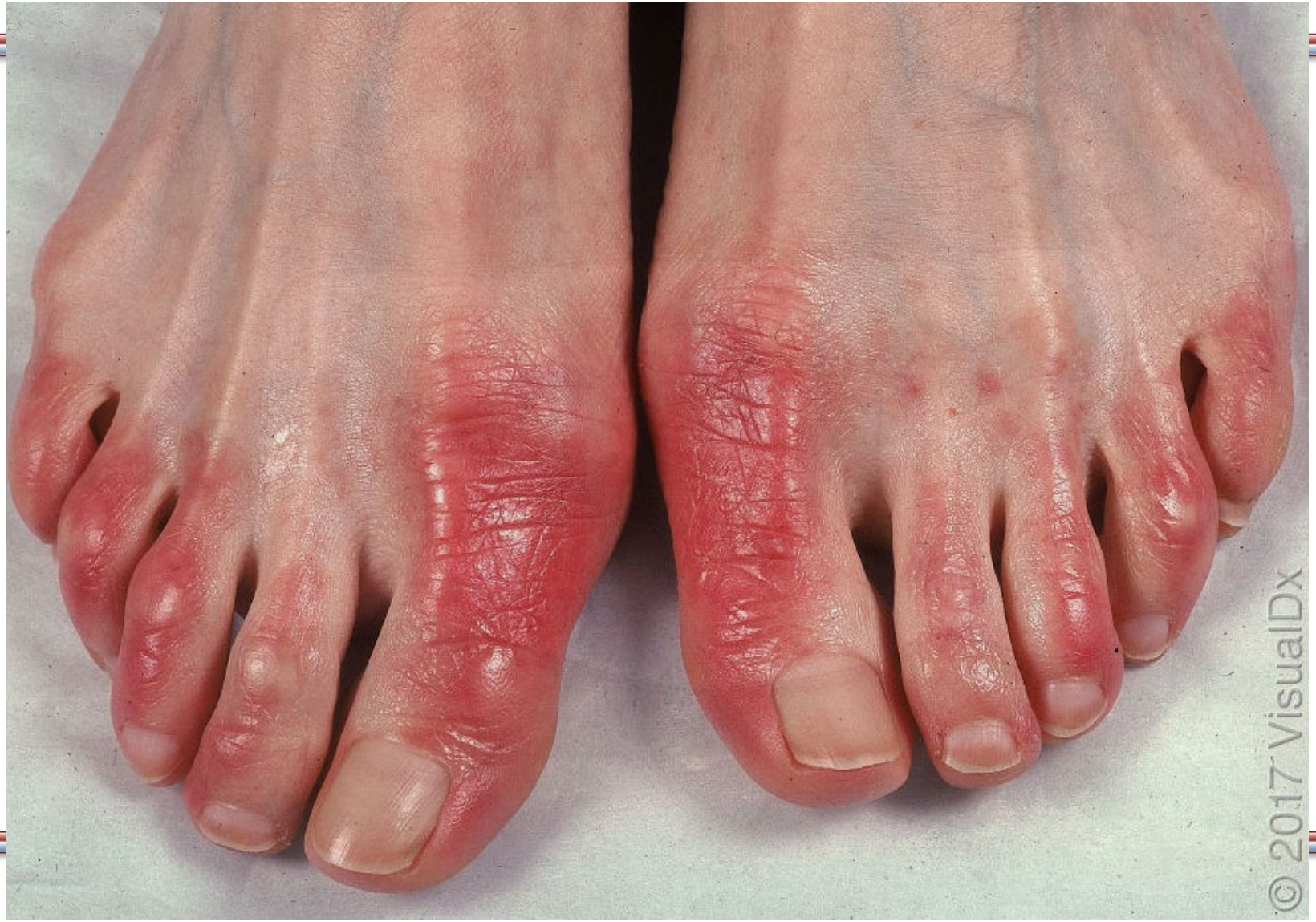
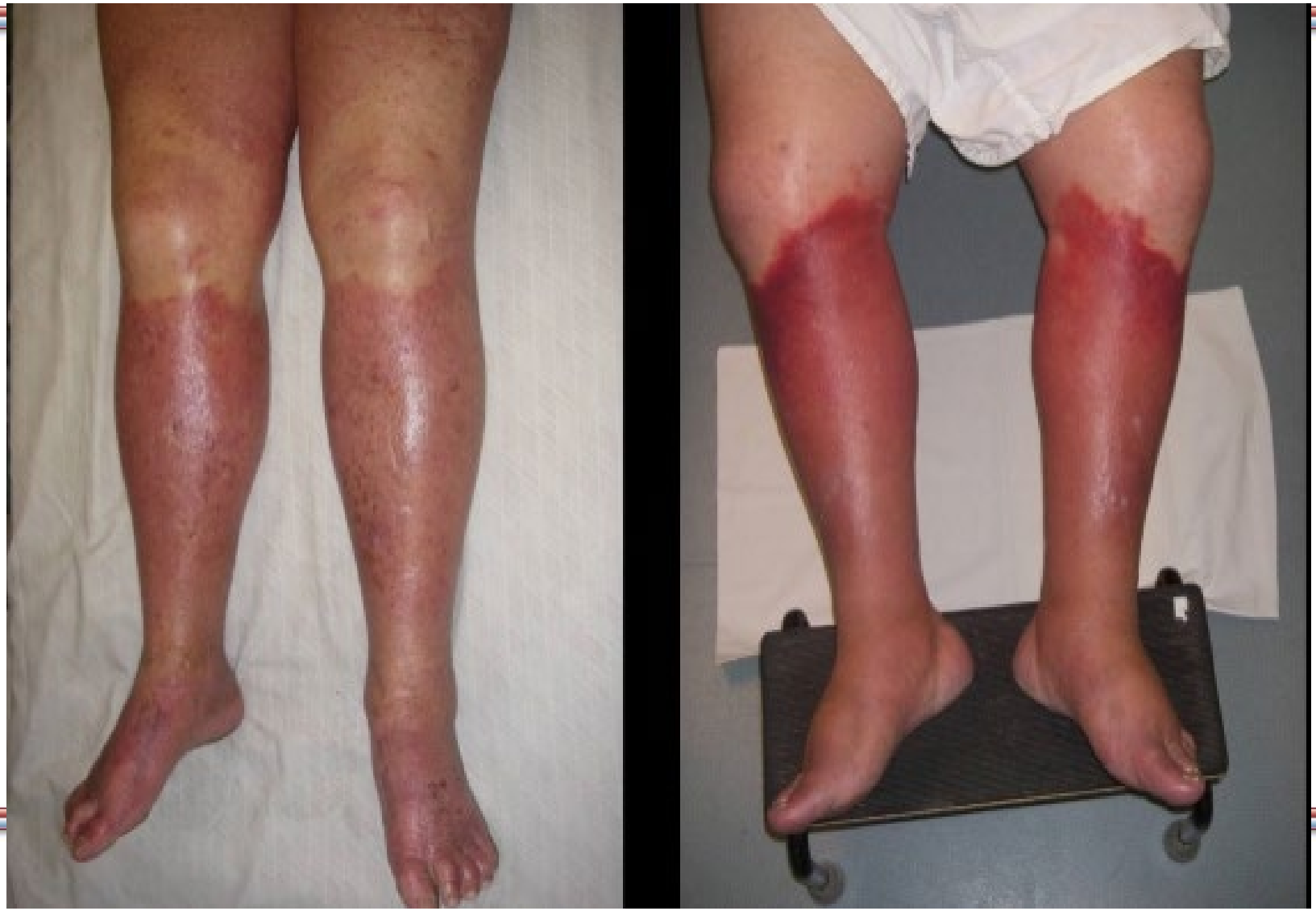


(VisualDx, 2017)

Stasis Dermatitis

- Result of chronic venous stasis
  - Other factors: ACD (from various topical applications/meds/antibiotics), irritants, superinfection
- Clinical features
  - Usually bilateral distal lower extremities (less often unilateral)
    - Often begins on medial ankle
  - Pitting edema, hemosiderin deposition (rust-colored hyperpigmented macules/patches)
  - Eczematous patches/plaques – typically chronic in nature, can see acute changes (especially with concomitant ACD or superinfection)
  - Can see associated venous stasis ulcers, lipodermatosclerosis (inverted wine-bottle shaped induration), and atrophie blanche scar-like patches/plaques

Stasis Dermatitis



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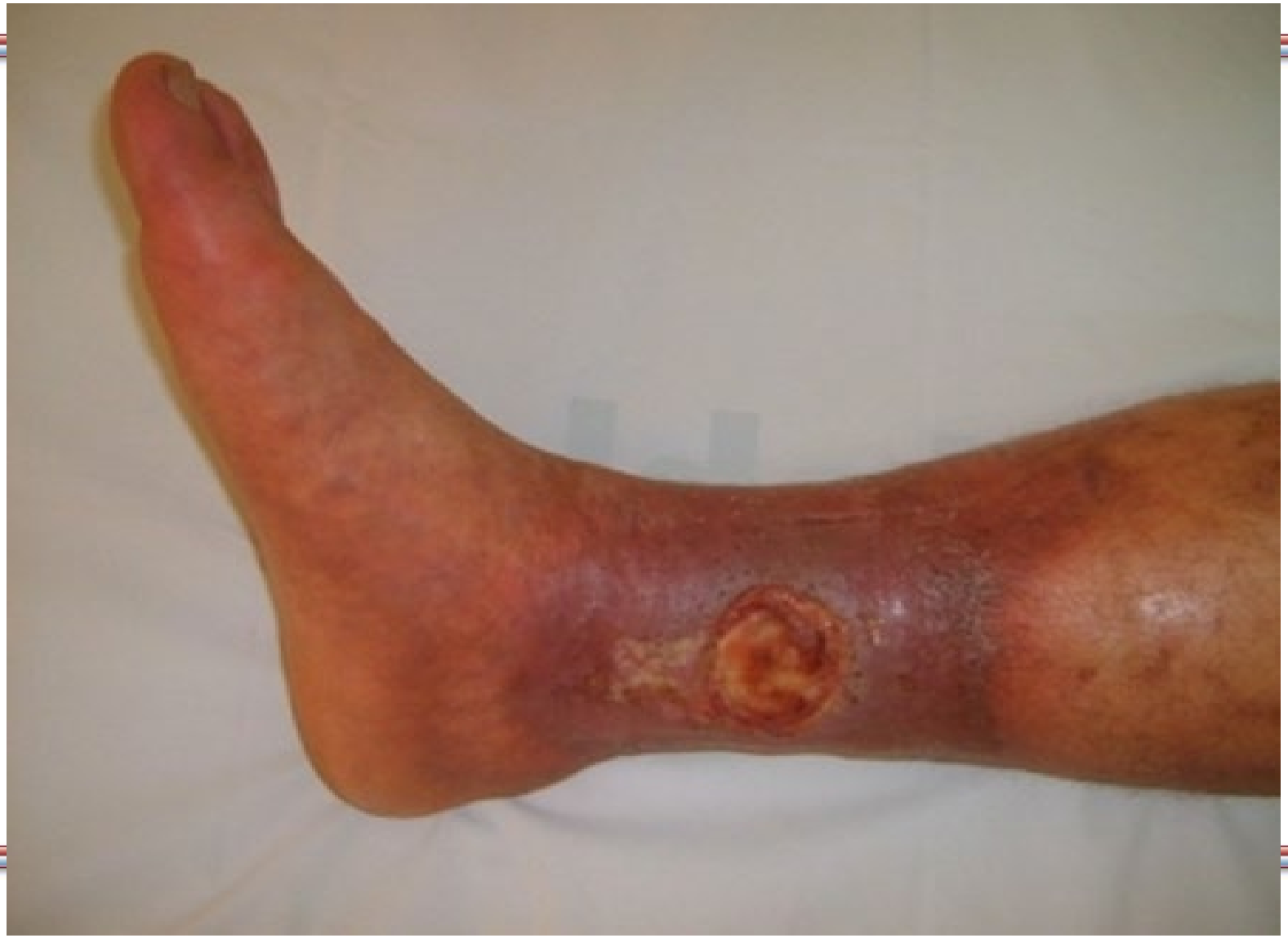
Stasis Dermatitis



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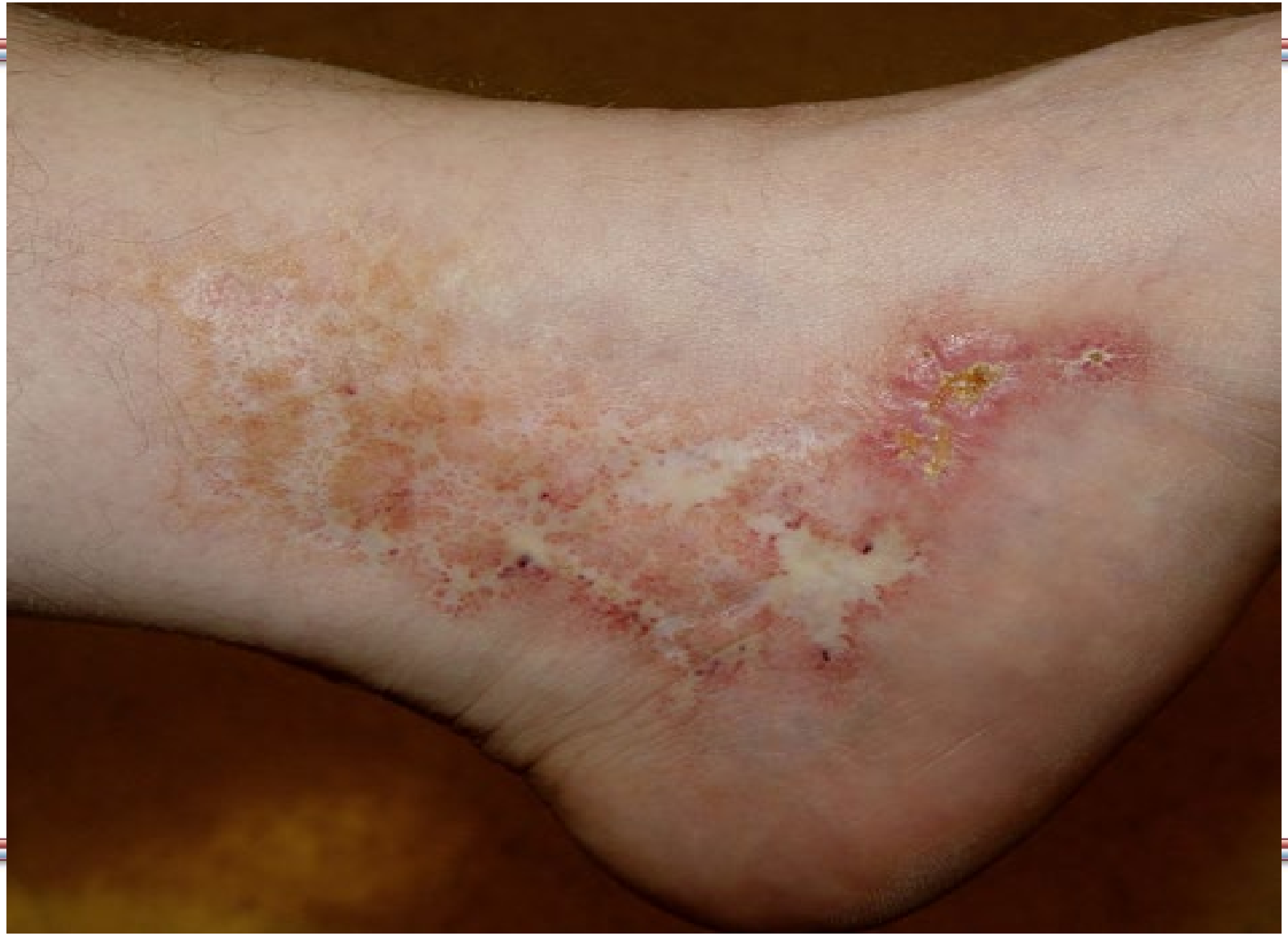
Stasis Dermatitis



(VisualDx, n.d.)

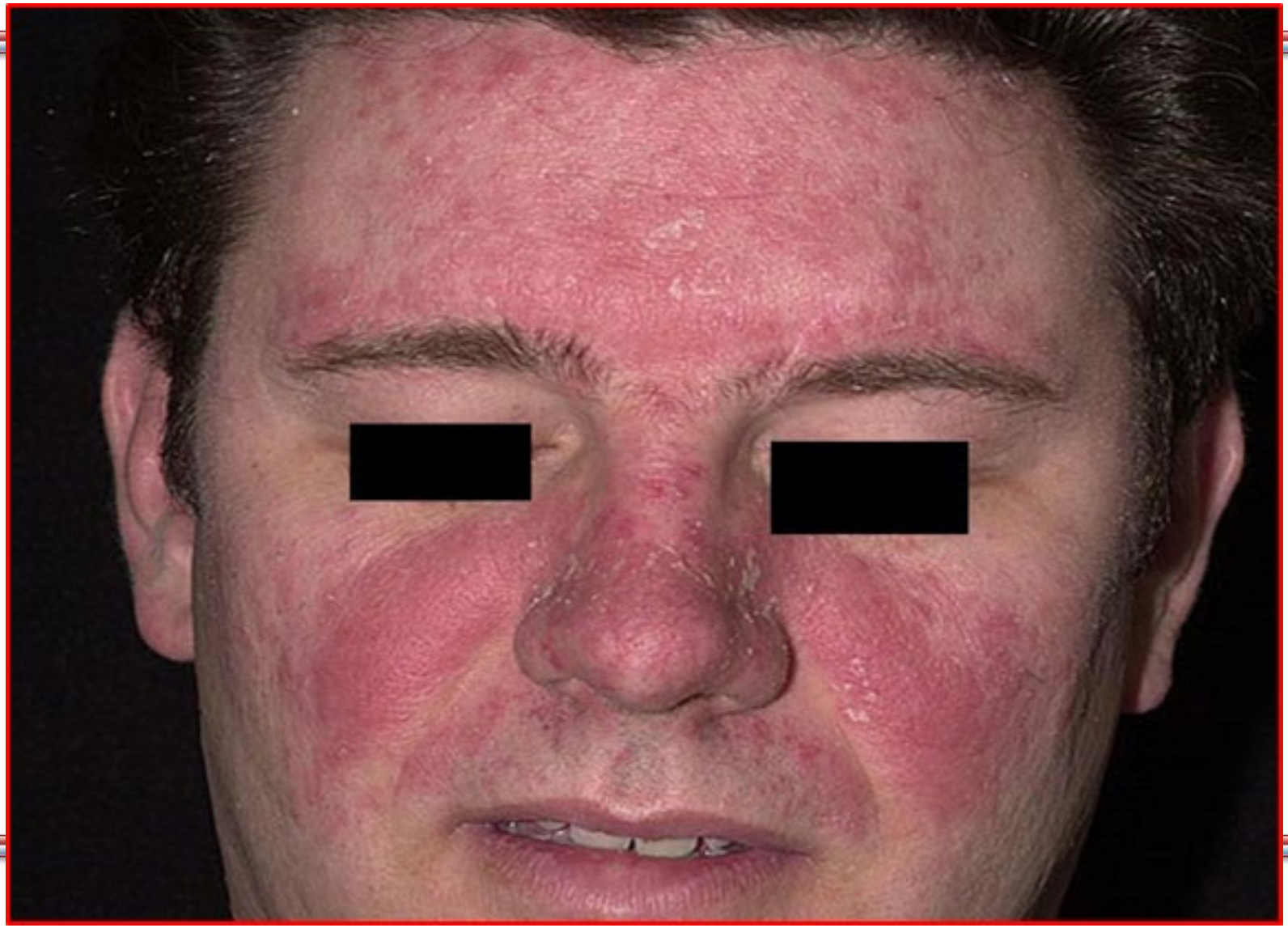
Stasis Dermatitis

- Evaluation – LE duplex ultrasound (US), ankle-brachial index (ABIs); biopsy and tissue cultures if dx unclear
- Treatment
  - Compression stockings (r/o concomitant arterial insufficiency), elevation
  - Topical corticosteroids or calcineurin inhibitors for acute inflammation
  - Avoid potential allergens (especially neomycin, bacitracin, fragrances, etc)
  - Address underlying comorbidities [hypertension (HTN), diabetes mellitus (DM), peripheral arterial disease (PAD), obesity, tobacco use, etc]

Seborrheic Dermatitis

- Affects 2-5% of population, M>F
- Pathogenesis: Malassezia (P. ovale) + active sebaceous glands + immune response
  - **Worse w/ human immunodeficiency virus (HIV), Parkinson's disease, mood disorders, and other neurologic diseases [hx of cerebrovascular accident (CVA)]**
- Well-demarcated erythematous patches or thin plaques with overlying white-yellow greasy scale; may have associated pruritus
  - Ranges from mild to severe inflammation – consider HIV with widespread and severe inflammatory seborrheic dermatitis
  - Loose dry scale of the scalp +/- underlying inflammation/erythema (dandruff)
  - May be confused or overlap with scalp psoriasis
- Loves sebaceous areas - nasolabial folds, brows, glabella, mustache, scalp, ears, chest

Seborrheic Dermatitis



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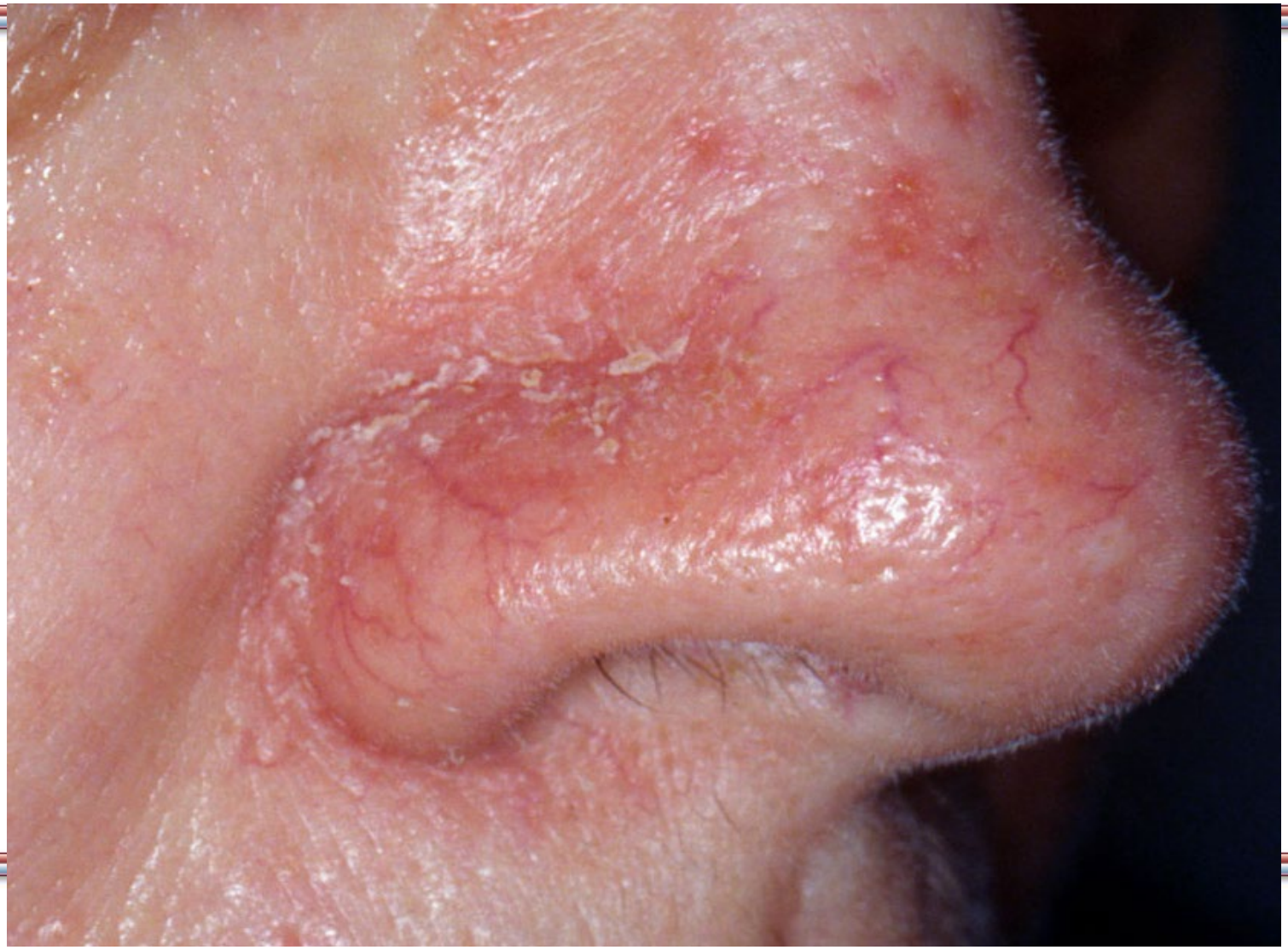

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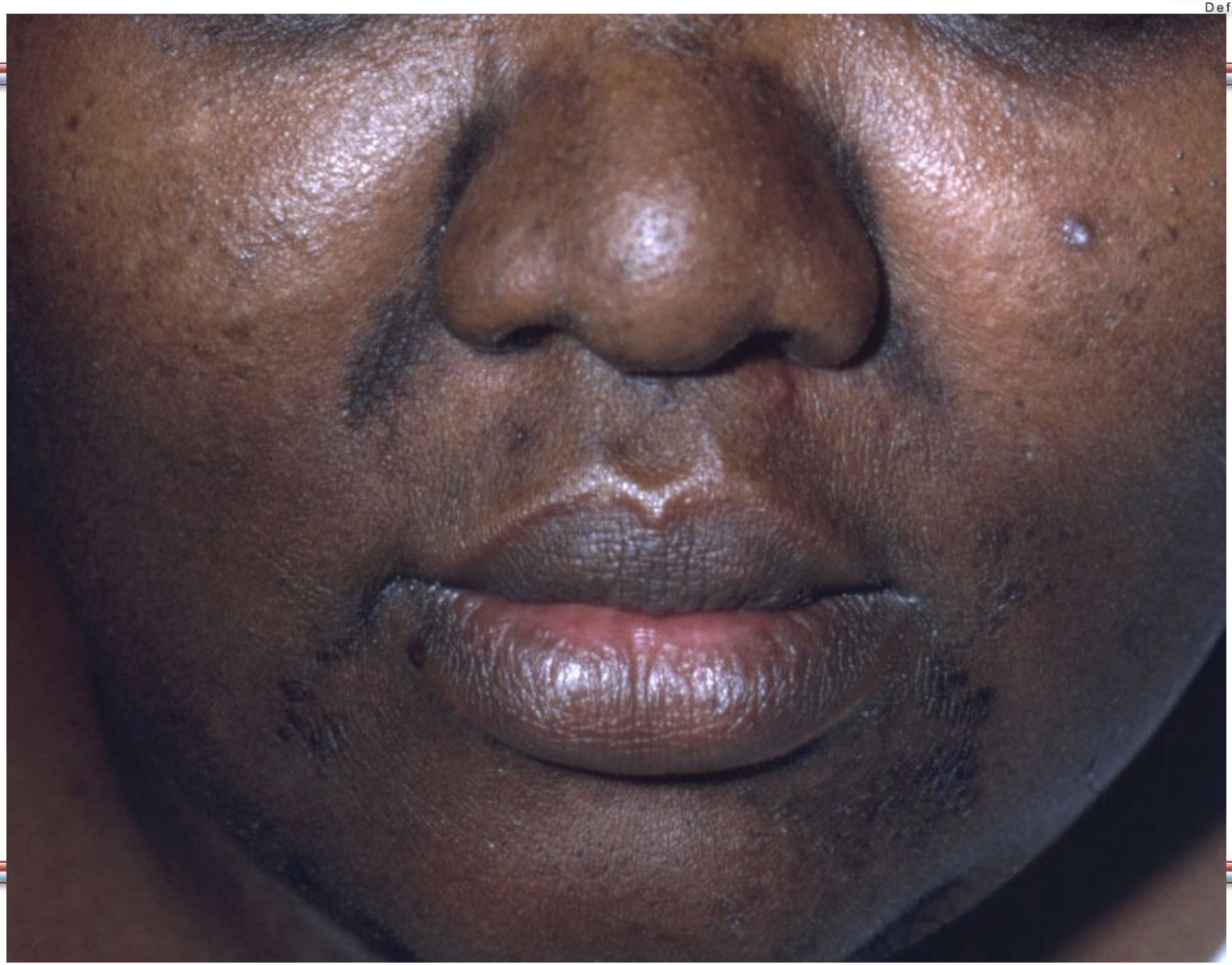

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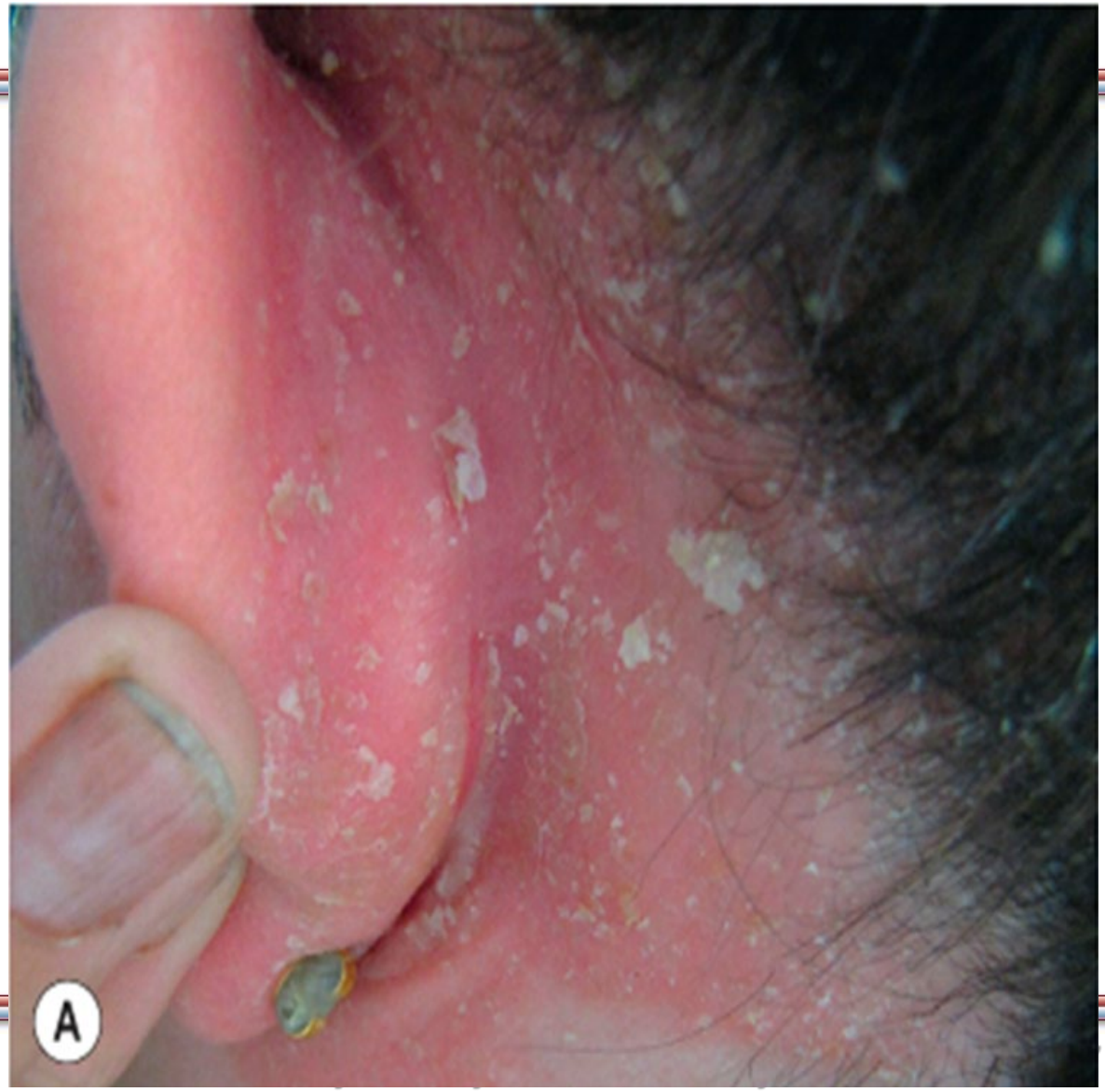
Seborrheic Dermatitis



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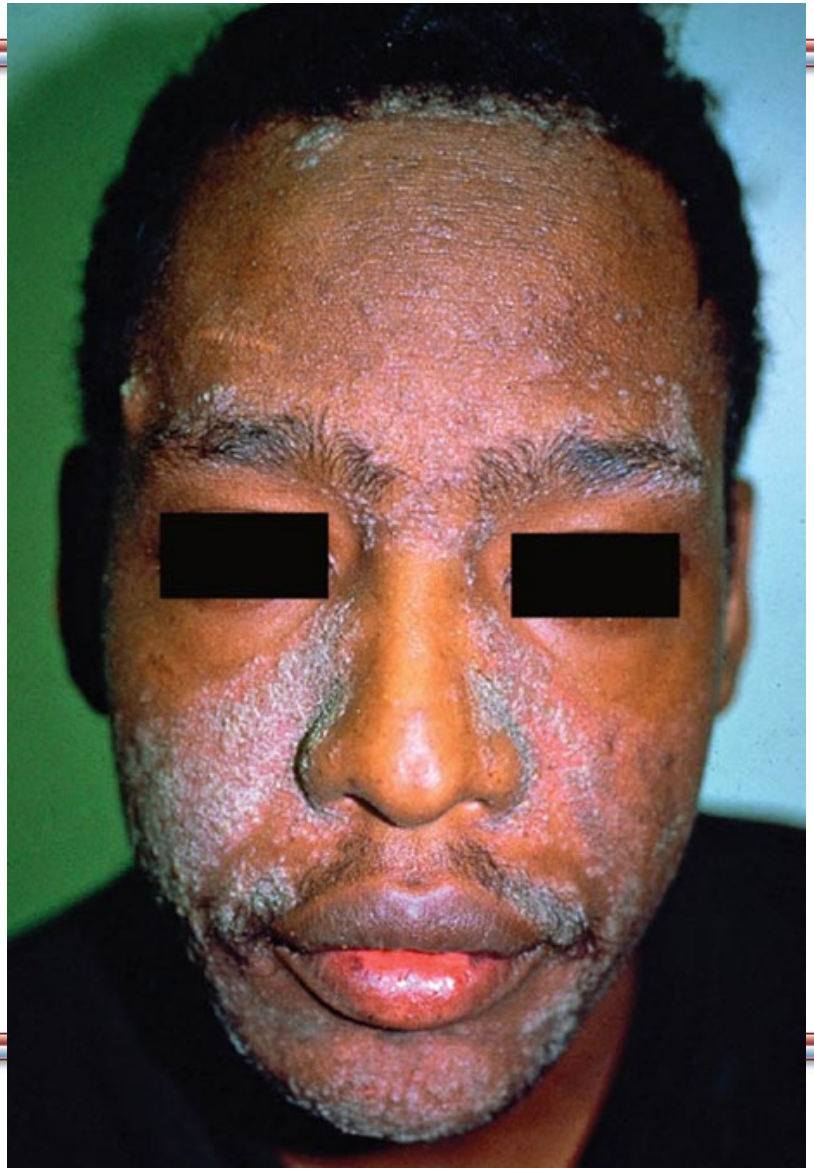
Seborrheic Dermatitis



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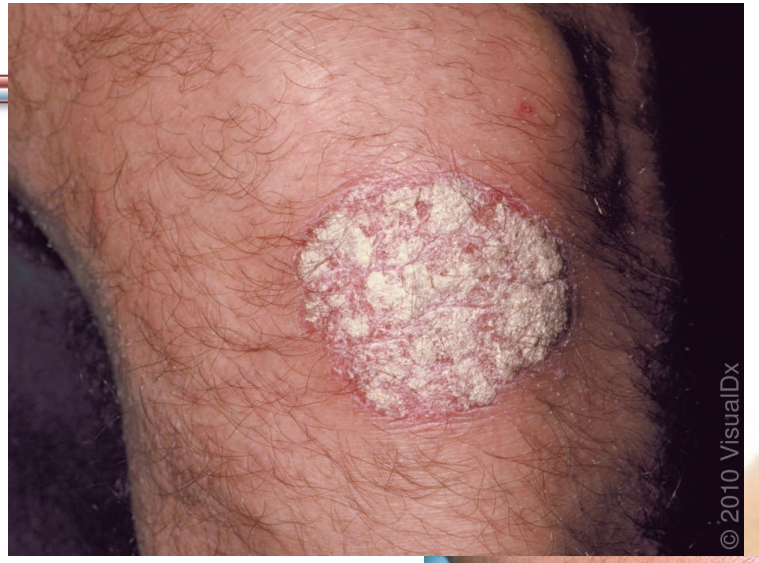
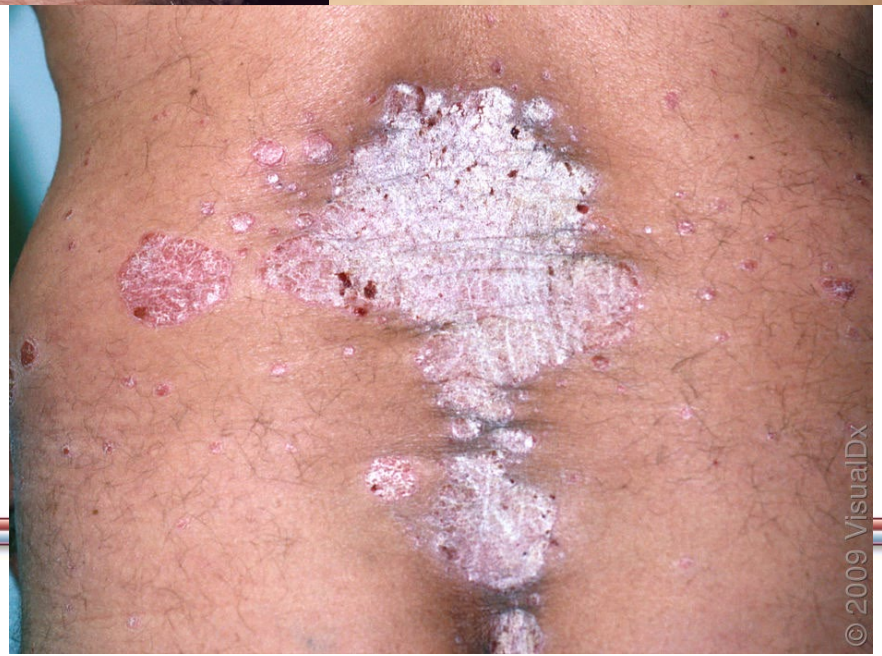
Psoriasis

- Polygenic inflammatory disease, chronic & recurrent, 2% of population
- Bimodal onset, 3rd and 6th decades
- Increased risk of CV disease, HTN, HLD, obesity, DM, renal disease
- Triggers:
  - Trauma (isomorphic, Koebner phenomenon)
  - Infection (strep pharyngitis → guttate)
  - Stress
  - **Medications - Terbinafine, interferon (IFN), nonsteroidal anti-inflammatory drugs (NSAIDs), Lithium, angiotensin-converting enzyme inhibitor (ACEI)/Antimalarial, Beta blocker, TNF-alpha inhibitors, Steroid withdrawal**
- Treatment:
  - Topicals
    - Vitamin D3 analogues (calcipotriol, calcipotriene)
    - Topical corticosteroids, topical calcineurin inhibitors
    - Tazarotene, coal tar, anthralin
  - Oral medications – acitretin, methotrexate, cyclosporine
  - Biologics (adalimumab, ustekinumab, secukinumab, etc)
  - Phototherapy [psoralen and ultraviolet light A (PUVA), narrowband ultraviolet light B (NBUVB)]

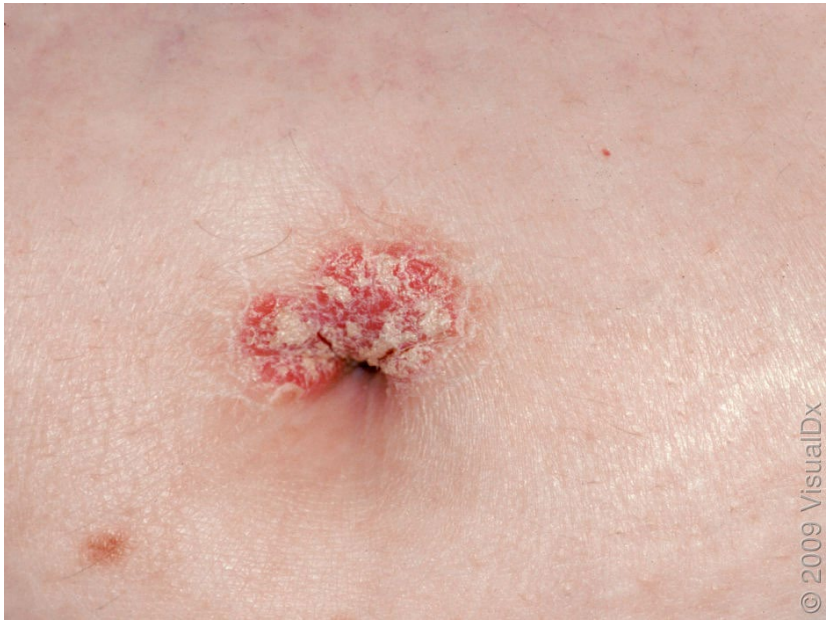
Psoriasis



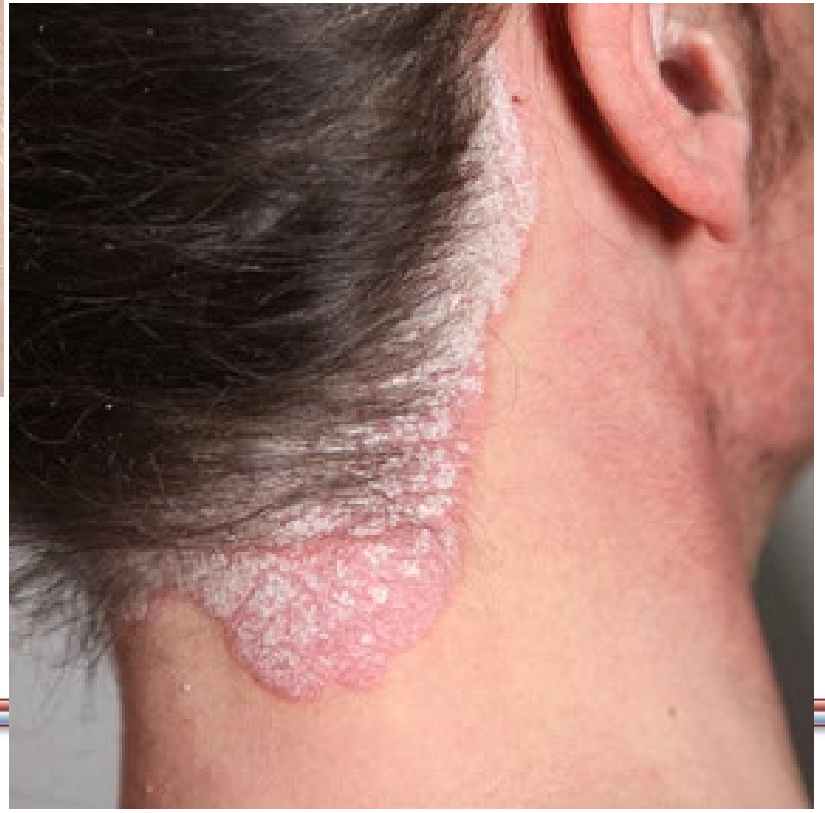


(visualDx, 2009, 2010)

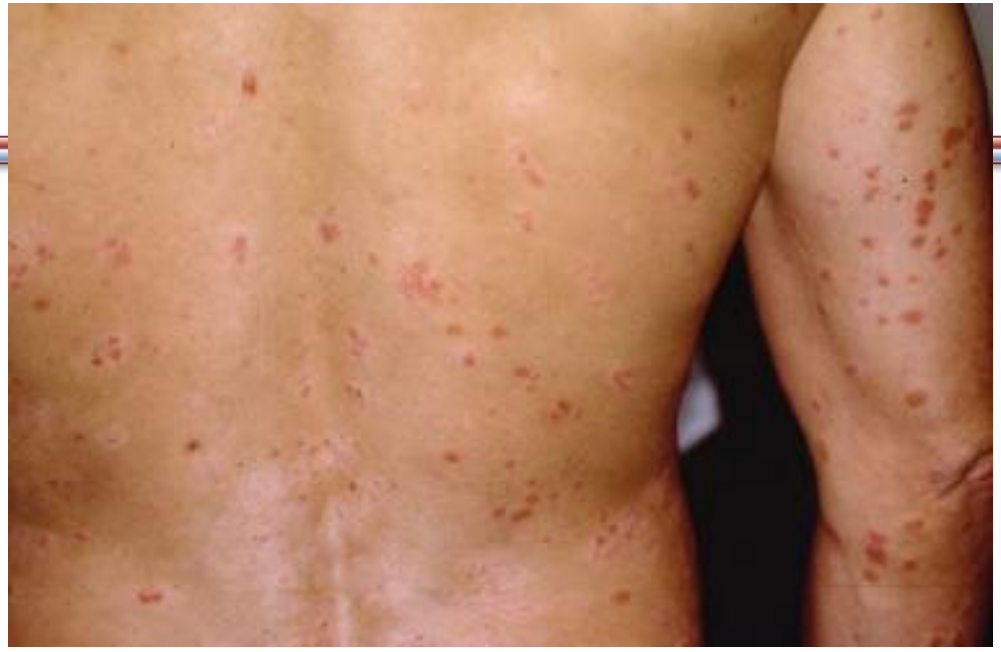
Psoriasis



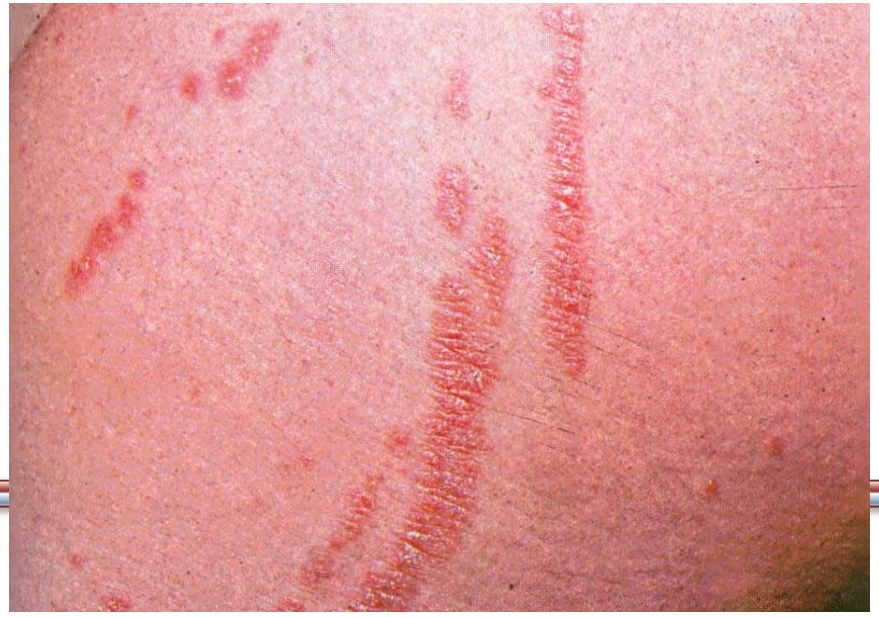
(visualDx, 2009)

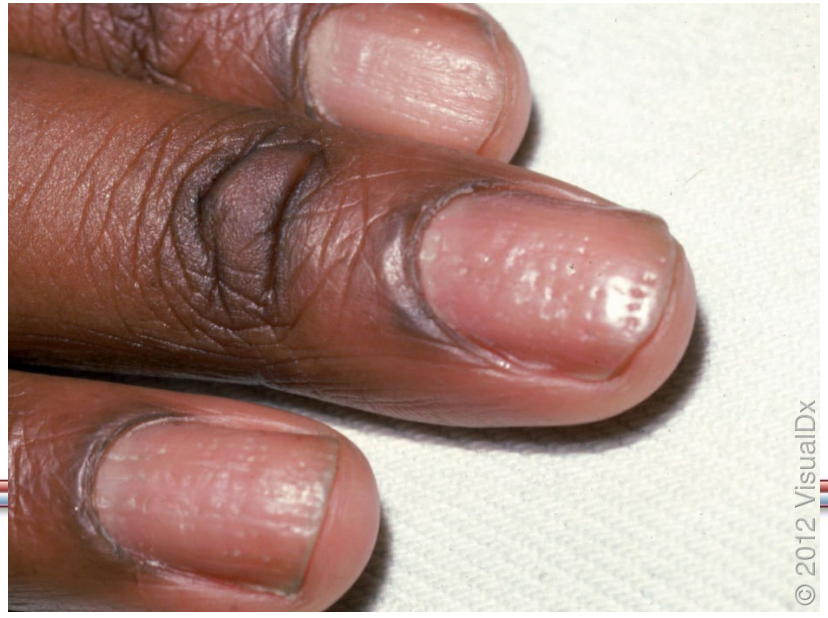


Psoriasis

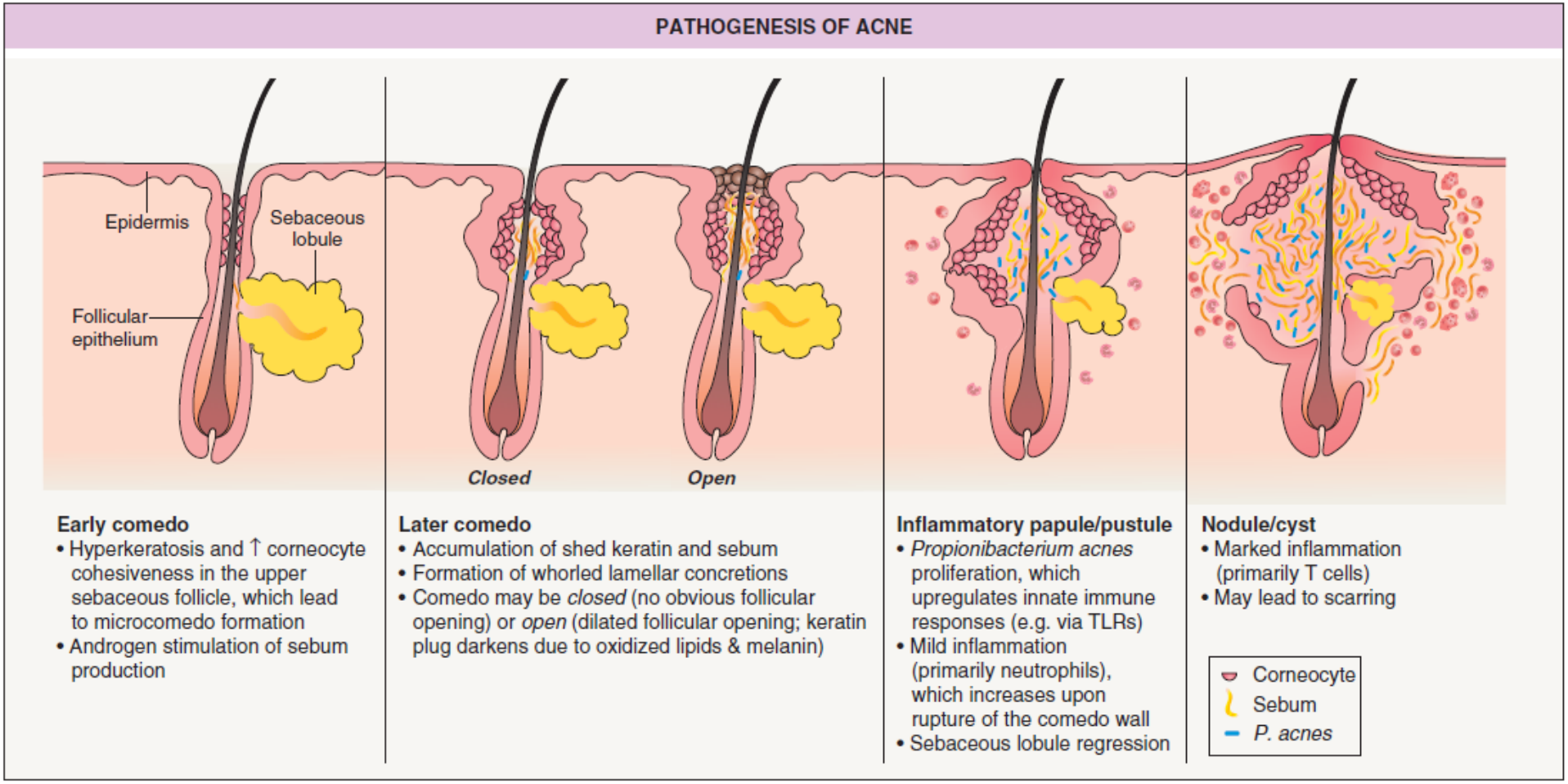


(visualDx, 2012)



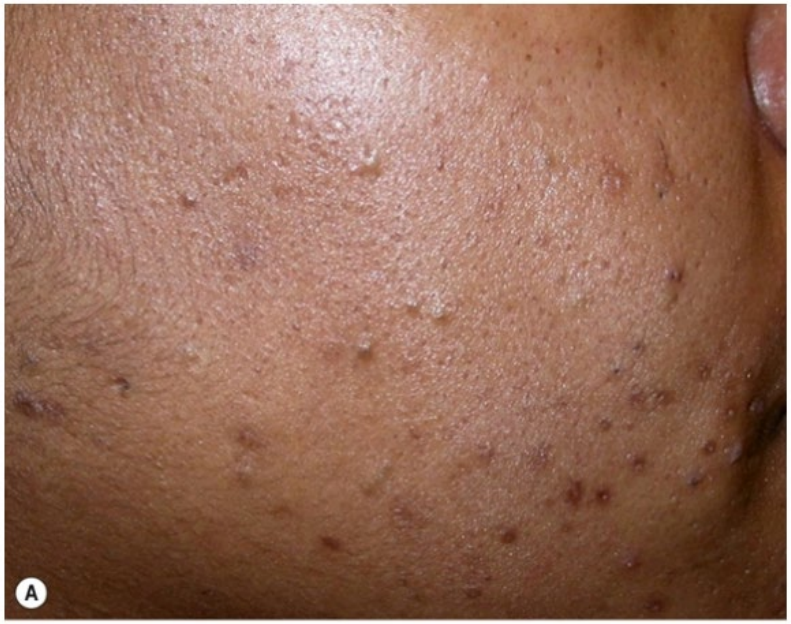


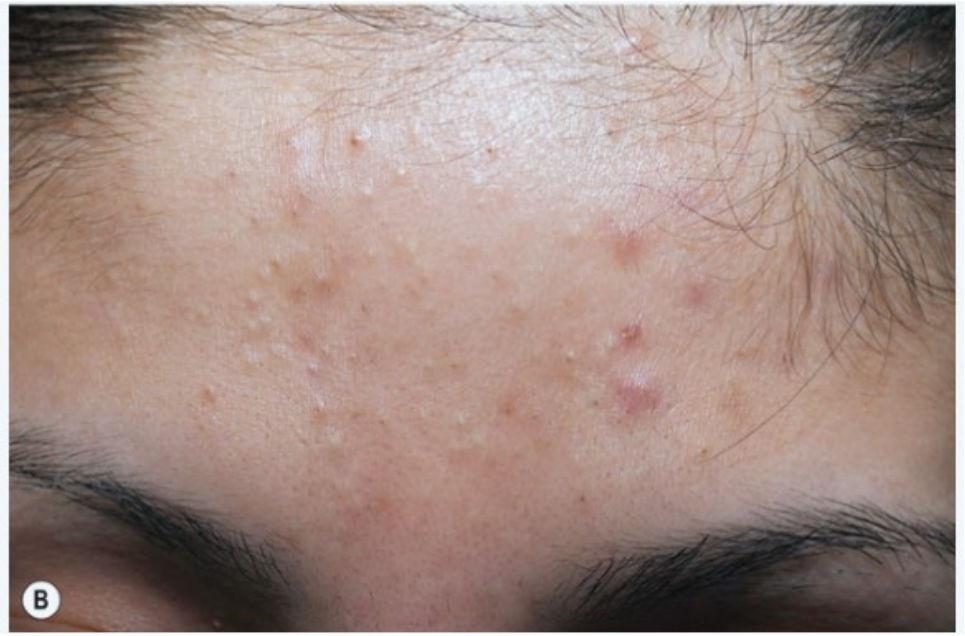
Acne



(*Dermatology*, 2018)

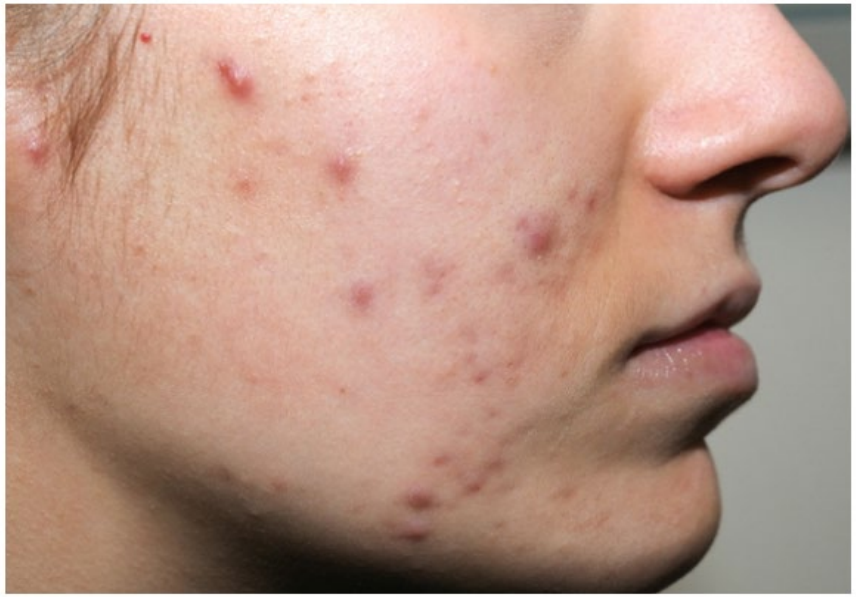
Acne

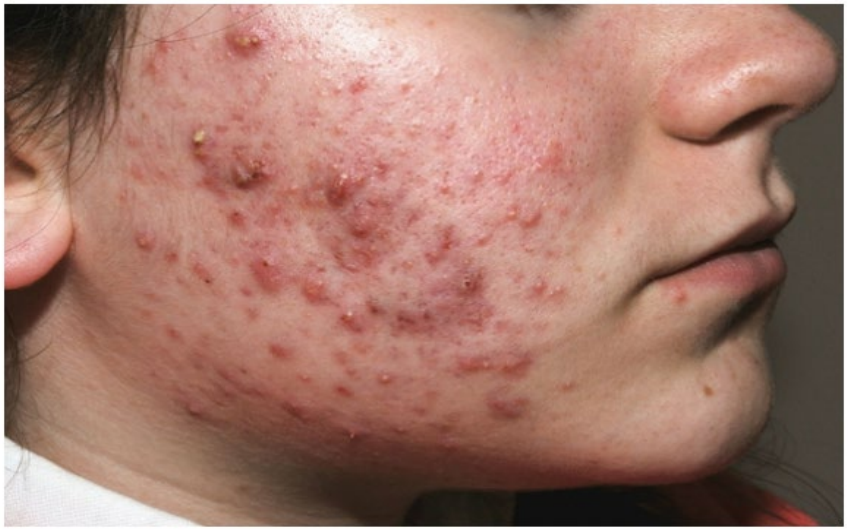


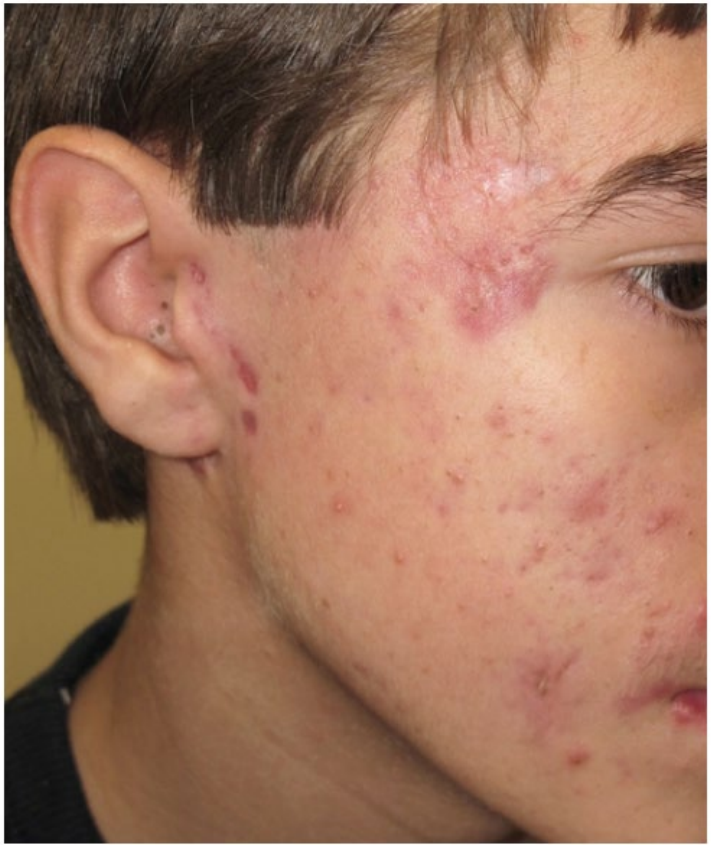


(VisualDx, n.d.)

Acne







(VisualDx, n.d.)

Acne

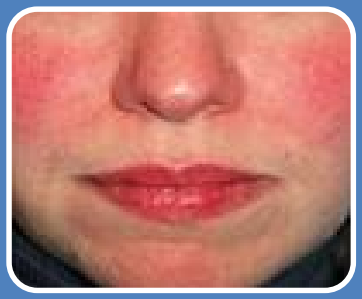
TREATMENT OF ACNE VULGARIS					
	Mild		**Moderate**		**Severe**
	Comedonal	**Papular/pustular**	**Papular/pustular**	**Nodular**	**Conglobata/fulminans**
First line	Topical retinoid	BPO ± topical antibiotic Topical retinoid + topical antimicrobial(s)*	Oral antibiotic† + topical retinoid ± BPO Topical retinoid + BPO ± topical antibiotic	Oral antibiotic† + topical retinoid ± BPO	Oral isotretinoin (may require concurrent oral corticosteroid, esp. for acne fulminans)
Second line	Alternative topical retinoid Azelaic acid Salicylic acid	Alternative topical retinoid and/or topical antimicrobial Azelaic acid Salicylic acid Topical dapsone	Alternative oral antibiotic‡ + alt. topical retinoid ± BPO/azelaic acid	Oral isotretinoin Alternative oral antibiotic‡ + alt. topical retinoid ± BPO/azelaic acid	Oral antibiotic (± high dose)+ topical retinoid + BPO Oral dapsone
Options for female patients			Oral contraceptive/antiandrogen	Oral contraceptive/antiandrogen	Oral contraceptive/antiandrogen
Procedural options	Comedo extraction		Comedo extraction	Comedo extraction Intralesional corticosteroid	Intralesional corticosteroid
Refractory to treatment		Exclude Gram-negative folliculitis	Exclude Gram-negative folliculitis		
			Female patient: exclude adrenal or ovarian dysfunction Exclude use of anabolic steroid or other acne-exacerbating medications		
Maintenance			Topical retinoid ± BPO	Topical retinoid ± BPO	Topical retinoid ± BPO

(Bolognia et al., 2018)

Rosacea

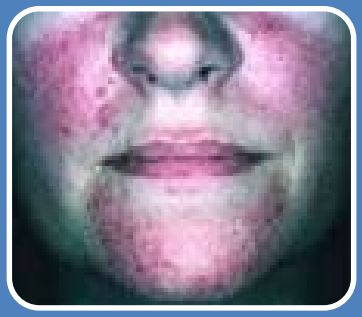
- Chronic inflammatory skin disorder
  - Neurovascular dysregulation
  - Aberrant innate immune response
  - ***Demodex folliculorum***
- Clinical Features
  - Persistent facial erythema
  - Inflammatory papules and pustules
  - Telangiectasias
  - Flushing
  - Ocular inflammation
  - Phymatous changes

Rosacea – Subtypes



Erythematotelangiectatic (vascular)

- Flushing and persistent erythema

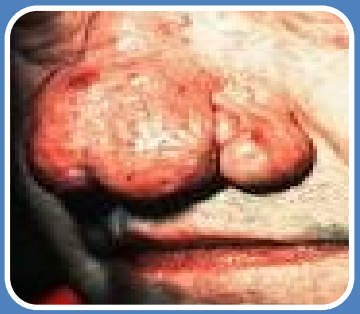


Papulopustular (inflammatory)

- Persistent erythema with papules and pustules

(VisualDx, n.d.)

Rosacea – Subtypes



Glandular (Phymatous)

- Thickened skin, nodules, on nose, chin, forehead, cheeks

Ocular

- Foreign body sensation in eye, burning, stinging, photosensitivity

(VisualDx, n.d.)

Rosacea

- Treatment
  - Identifying triggers
  - Basic skin care
    - Avoid irritants
    - Moisturizer
    - Sunscreen
  - Metronidazole cream
  - Azelaic acid
  - Sulfur sulfacetamide cleanser
  - Topical minocycline (Zilxi)
  - Topical ivermectin (Soolantra)
  - Oral antibiotics
    - Doxycycline 100mg BID
      - Submicrobial dosing → 20mg BID

Rosacea

- Topical adrenergic agonists
  - Brimonidine gel (Mirvaso)
  - Oxymetazoline cream (Rhofade)
  - Afrin
- Lasers
  - Pulsed dye laser (PDL)
  - Intense pulsed light (IPL)

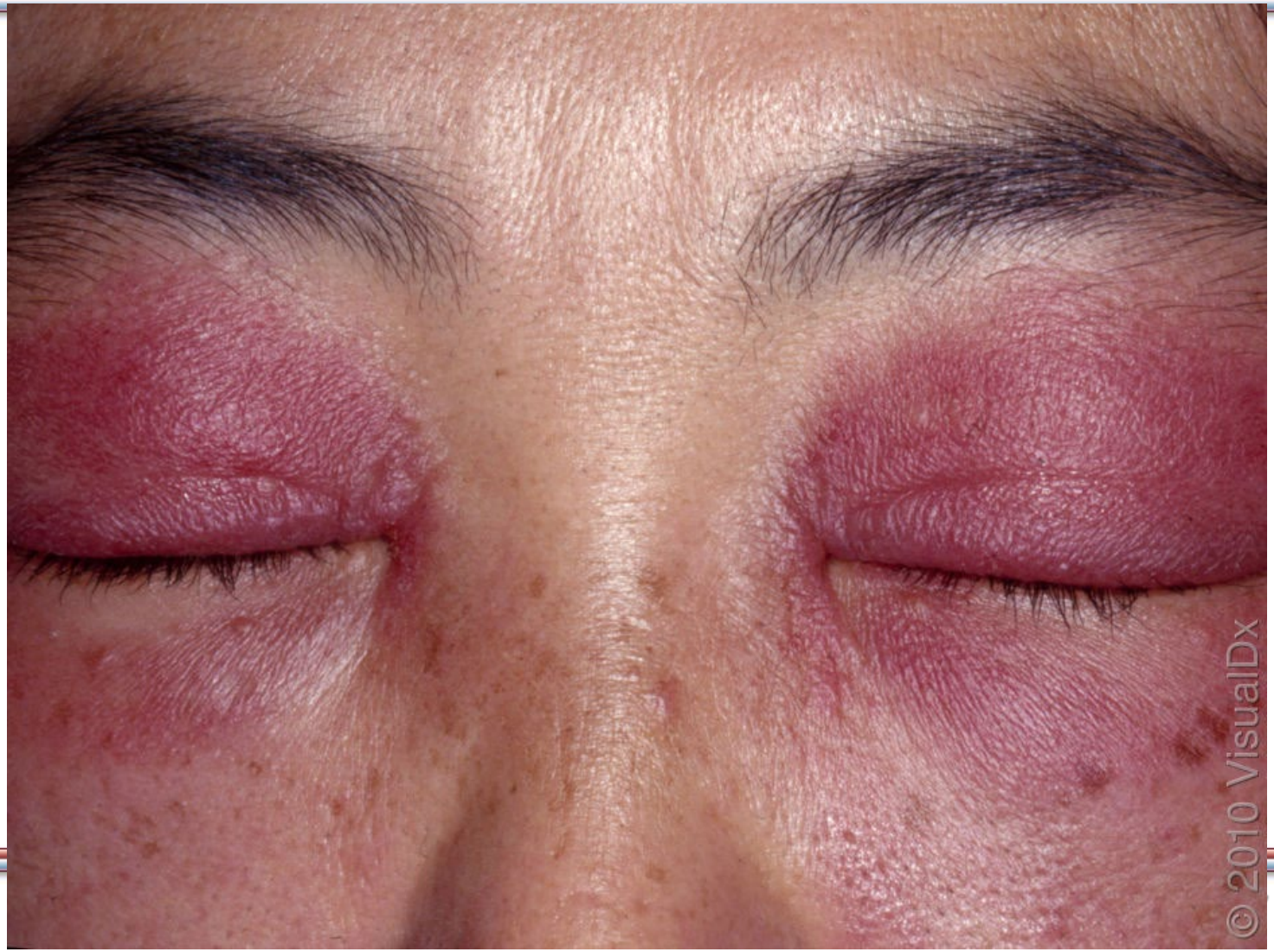
Autoimmune Connective Tissue Disease

- Dermatomyositis
- Lupus

Dermatomyositis

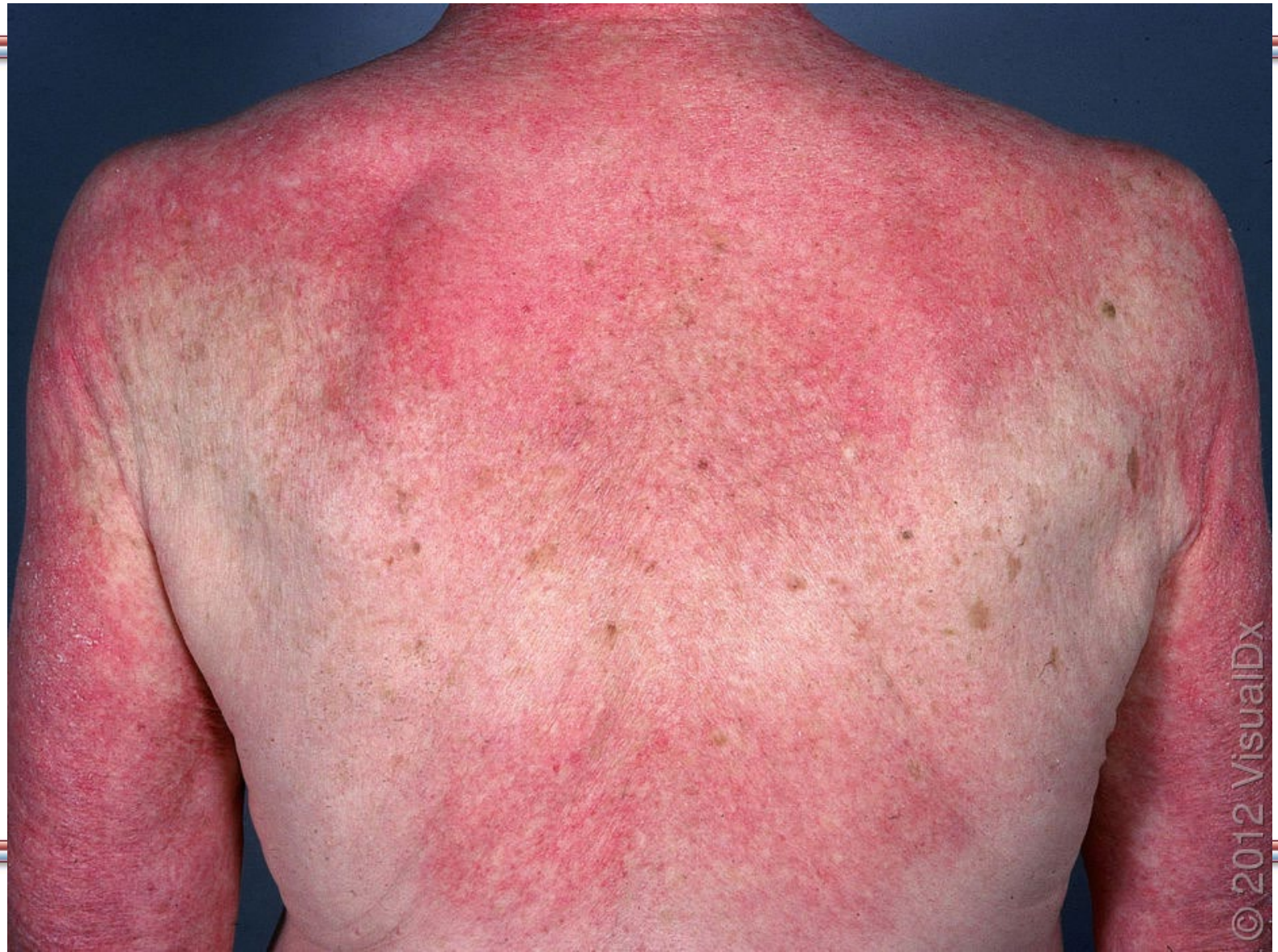
- Cutaneous findings – atrophic dermal papules of DM/Gottron papules (pathognomonic), symmetric confluent macular erythema (periorbital, cheeks, shawl sign), ragged cuticles, mechanic's hands
- Muscular findings – symmetric proximal muscle weakness (extensors > flexures), generally lacks muscle pain; may see esophageal, diaphragmatic, and cardiac dysfunction
  - Amyopathic forms exist
- Antibody associations:
  - ANA + in ~40%
  - **Anti-p155/140 (TIF-1-gamma) – a/w malignancy and amyopathic DM**
    - Ovarian and GI cancers are overrepresented; most cancer detected within 1-2 years of DM dx
  - Anti-CADM-140 (MDA5) – a/w amyopathic + rapidly progressive ILD DM
  - Anti-Jo1 – a/w anti-synthetase syndrome (mechanic's hands, severe ILD)
  - Anti-Mi-2 – a/w classic DM findings, good prognosis
  - Anti-SRP – a/w cardiac involvement

Dermatomyositis



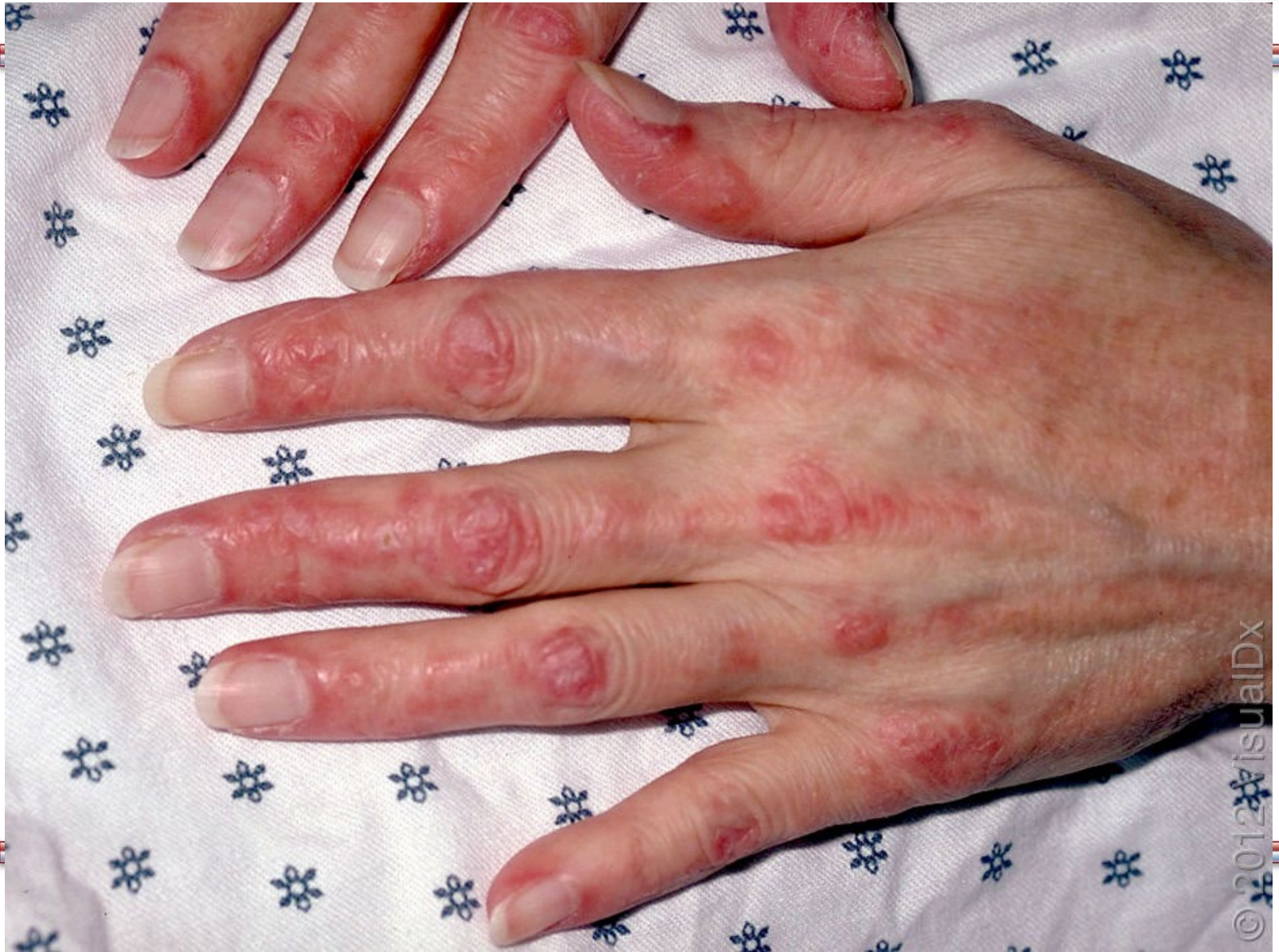
(VisualDx, 2010)

Dermatomyositis



(VisualDx, 2012)

Dermatomyositis

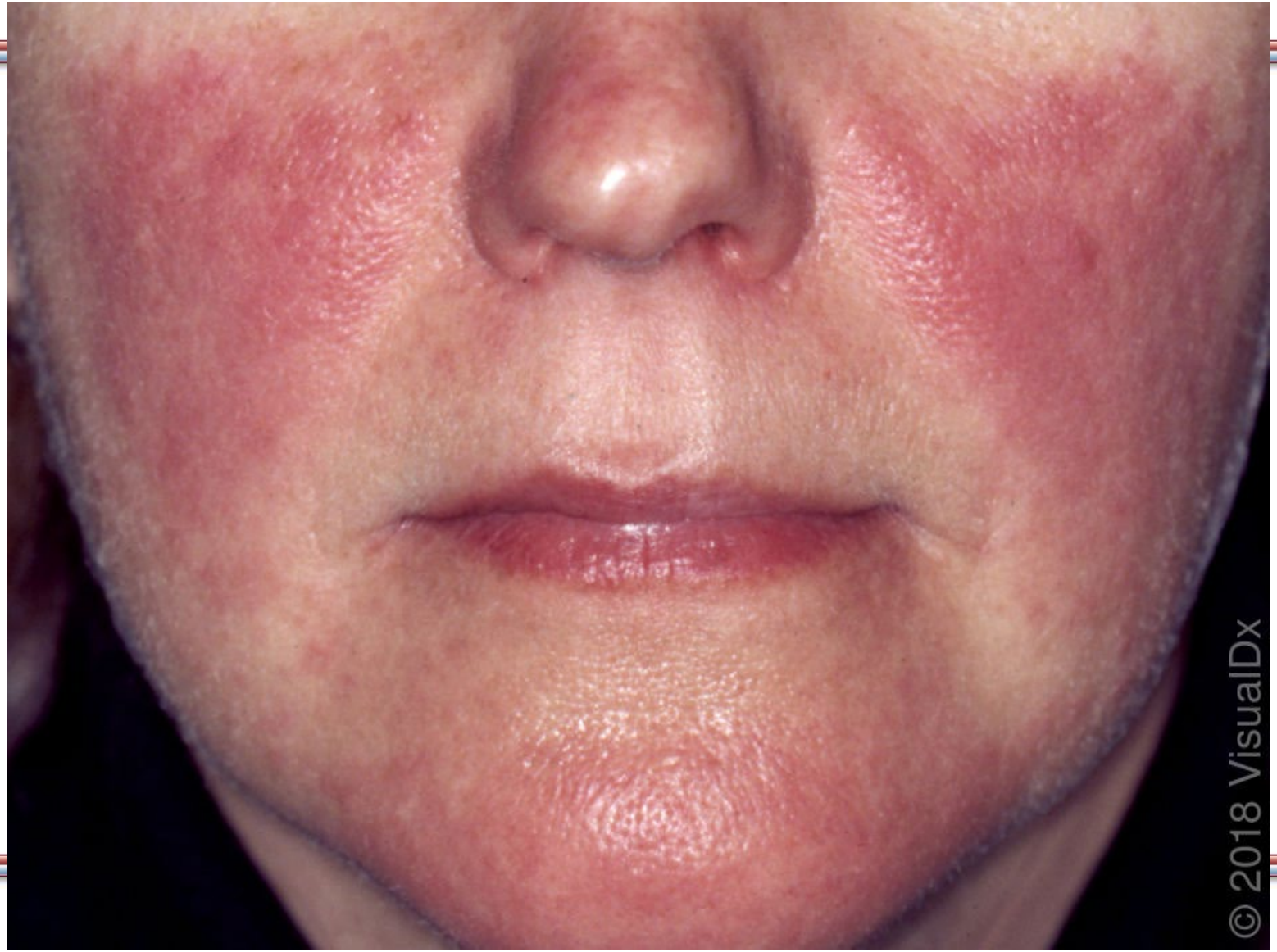


(VisualDx, 2012)

Lupus

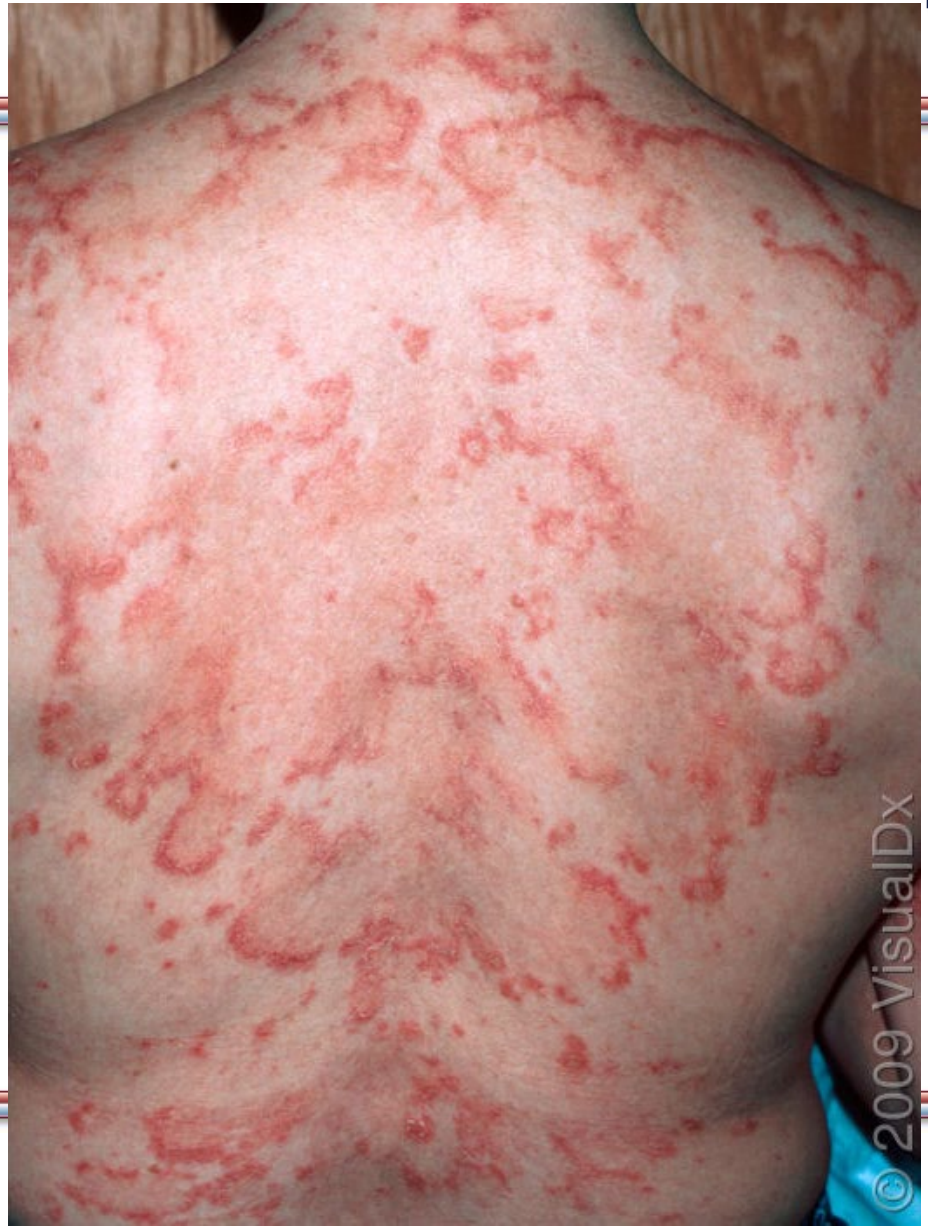
- Discoid lupus
  - **5-20% risk of progression to systemic lupus erythematosus (SLE) [however, 40-70% of patients with SLE have discoid lupus erythematosus (DLE)]**
  - +/- antinuclear antibody (ANA) – more common with widespread disease
  - Rx: topical/intralesional steroids +/- antimalarials; may require other immunosuppressive meds if recalcitrant
- Subacute Cutaneous Lupus
  - 30-50% risk of progression to SLE
  - Drug-induced – hydrochlorothiazide (HCTZ) (most common), terbinafine, griseofulvin, calcium channel blockers (CCBs)
  - Anti-Ro/SS-A (75-90%), ANA (60-80%)
  - Rx: sun protection and antimalarials; may require other immunosuppressive meds if recalcitrant
- Acute Cutaneous Lupus
  - Highly associated with SLE
  - Rx depends on systemic involvement

Lupus



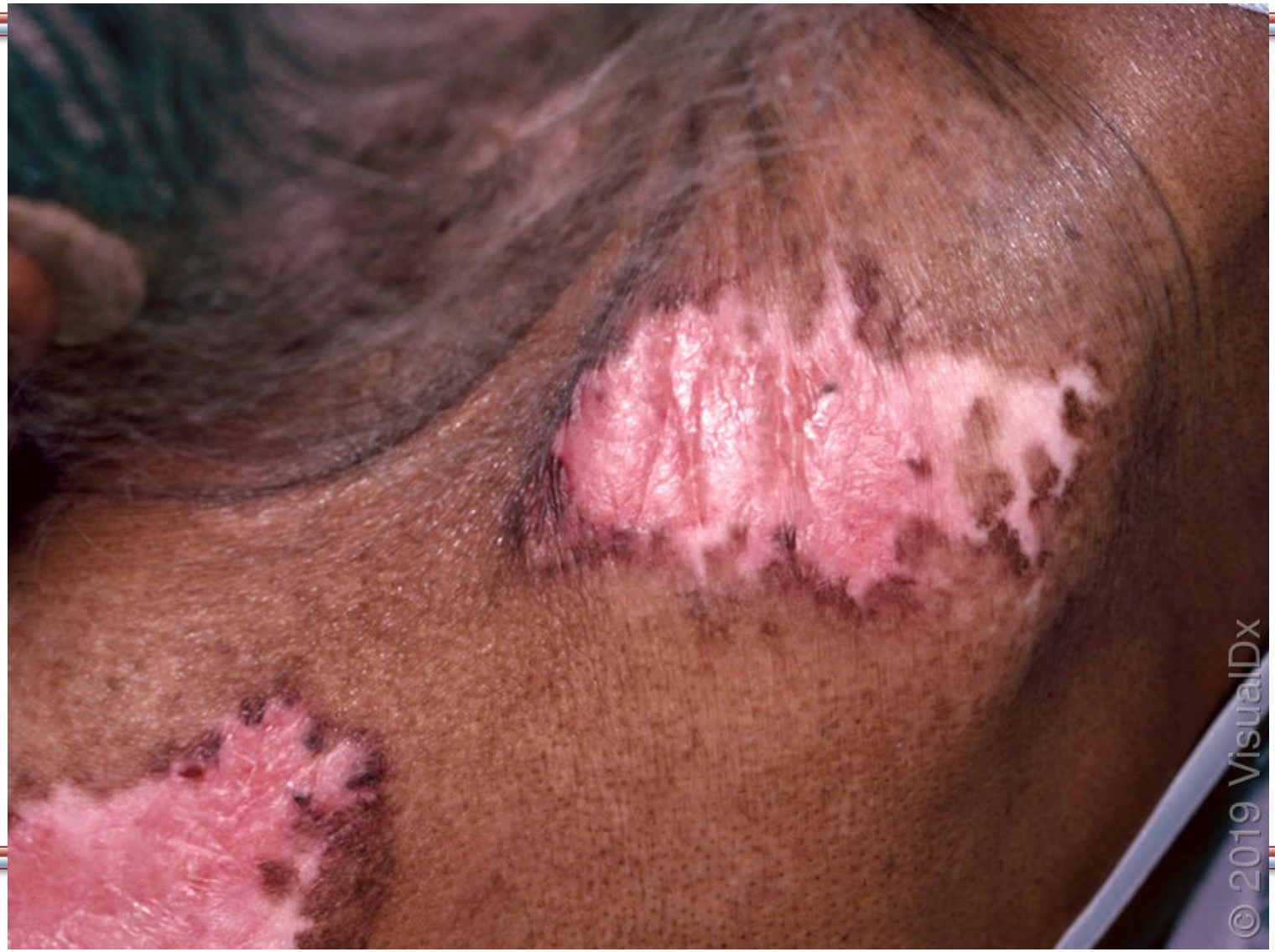
(VisualDx, 2018)

Lupus



(VisualDx, 2009)

Lupus



(VisualDx, 2019)

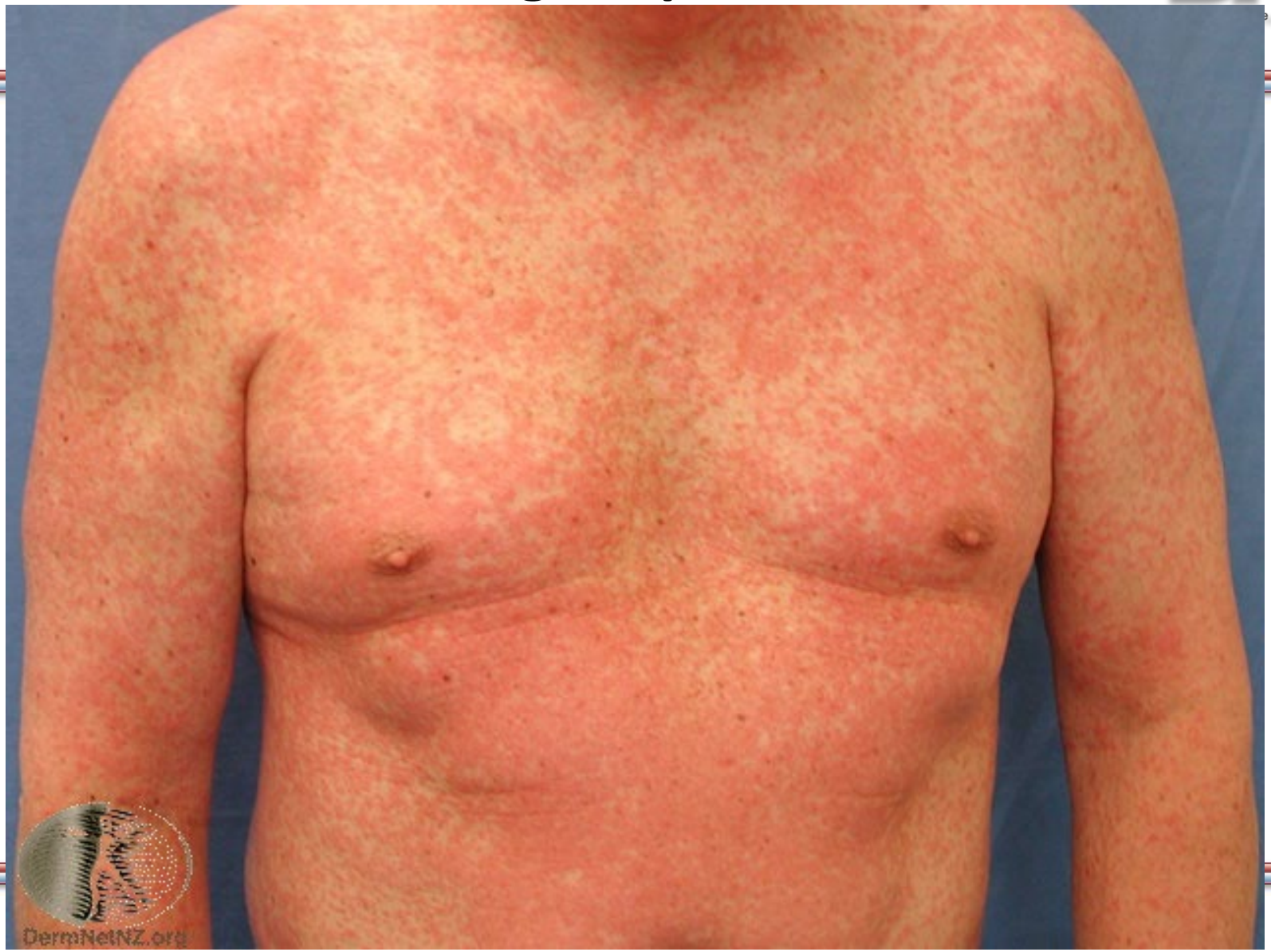
Drug Eruptions

- Morbilliform Drug Eruption (Exanthematous Drug Eruption)
- Acute Generalized Exanthematous Pustulosis (AGEP)
- Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)
- Fixed Drug Eruption (FDE)
- Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN)

Morbilliform Drug Eruption

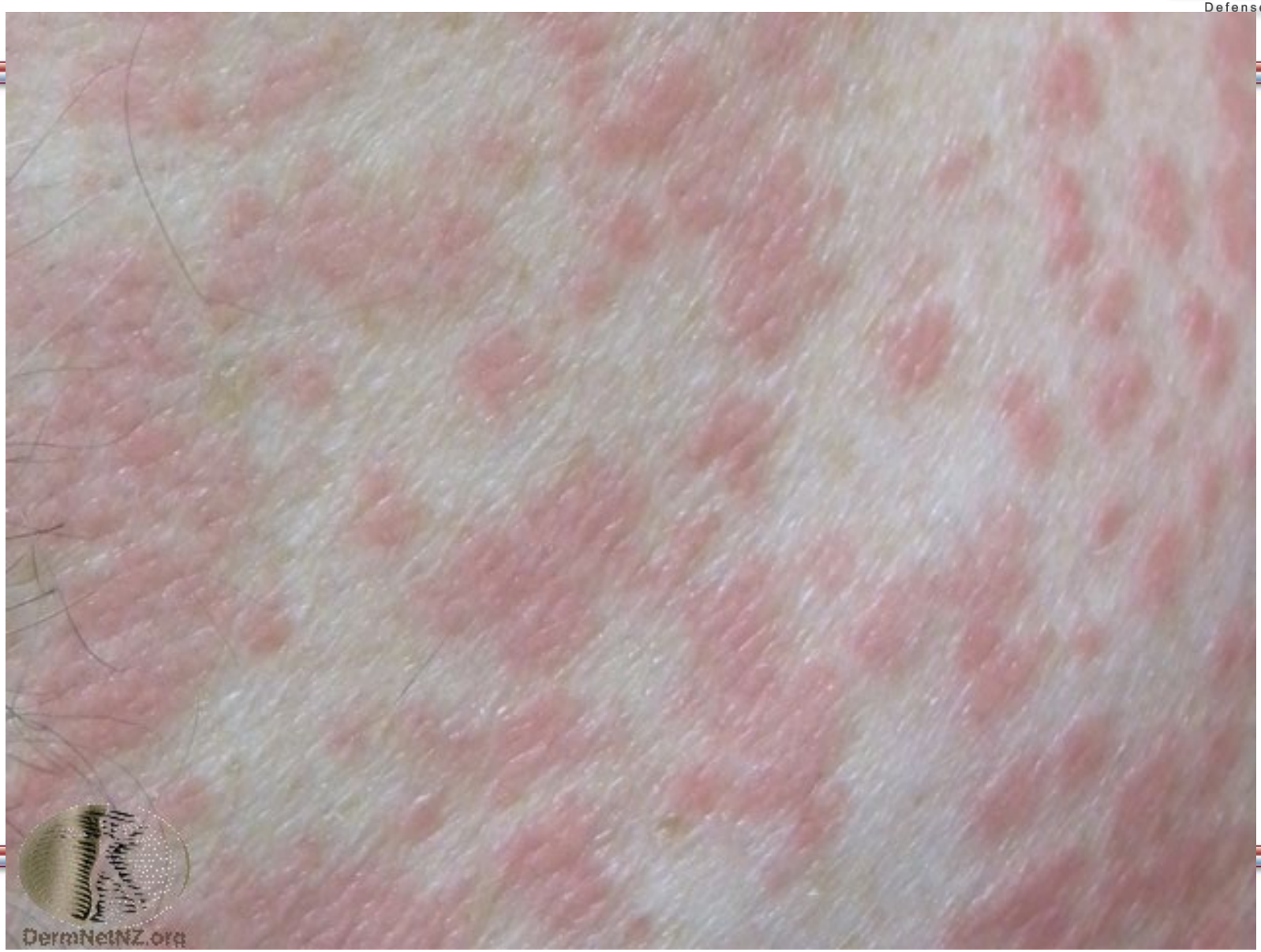
- Most common medication induced drug reaction
  - Penicillin (PCN), sulfa, allopurinol, anti-epileptics, NSAIDS, many others
- Onset 7-10 days of starting medication
- Clinical features:
  - Symmetrically distributed on trunk and extremities, may be pruritic
  - Erythematous or pink to red macules and papules, can eventually become confluent
  - Lesions typically blanch, dependent areas may be purpuric
- Treatment
  - Self limited
  - Discontinue treatment if possible
    - Can ”treat through"
  - Topical corticosteroids

Morbilliform Drug Eruption



(DermNetNZ.org, n.d.)

Morbilliform Drug Eruption

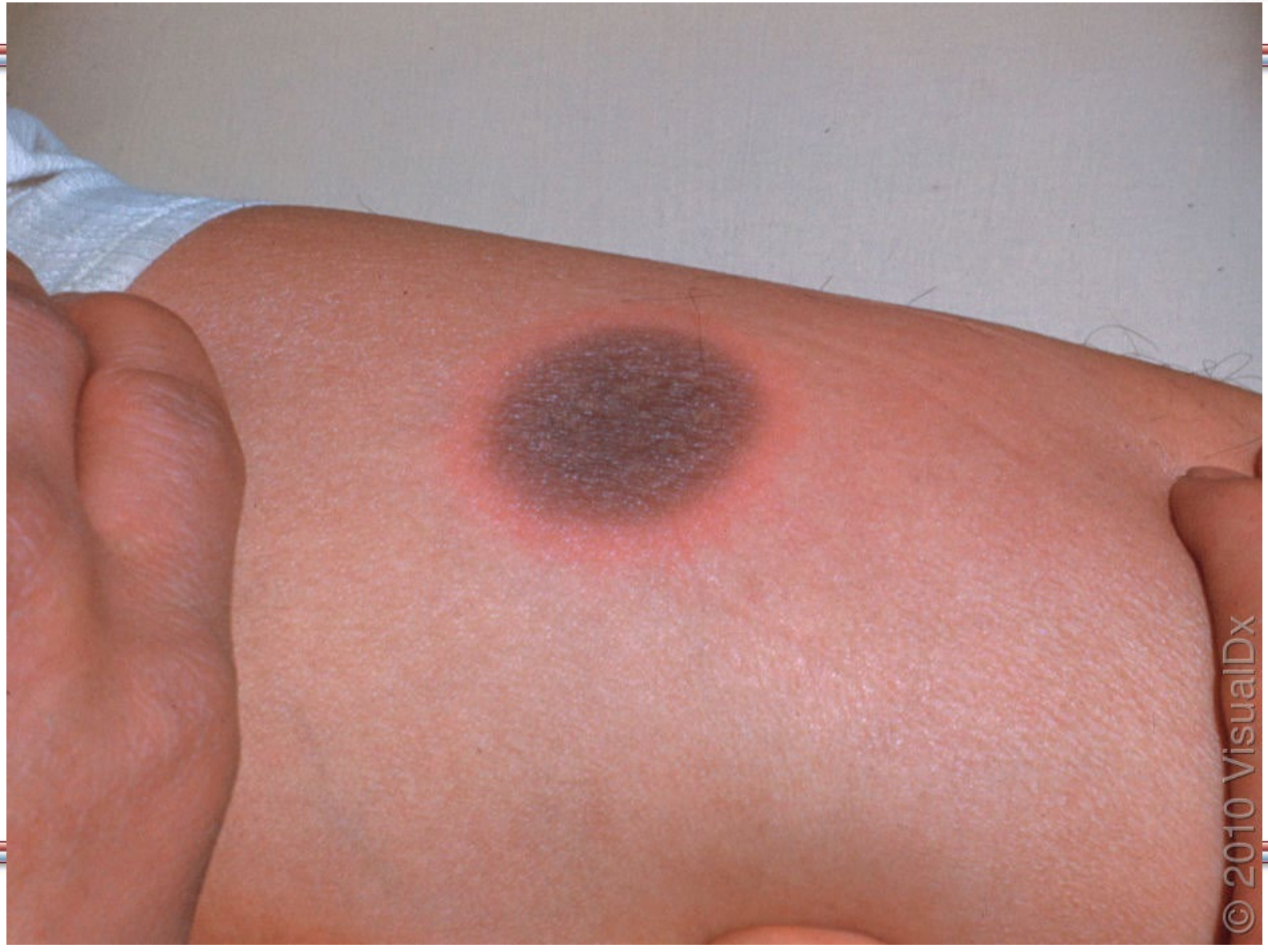


(DermNetNZ.org, n.d.)

Fixed Drug Eruption (FDE)

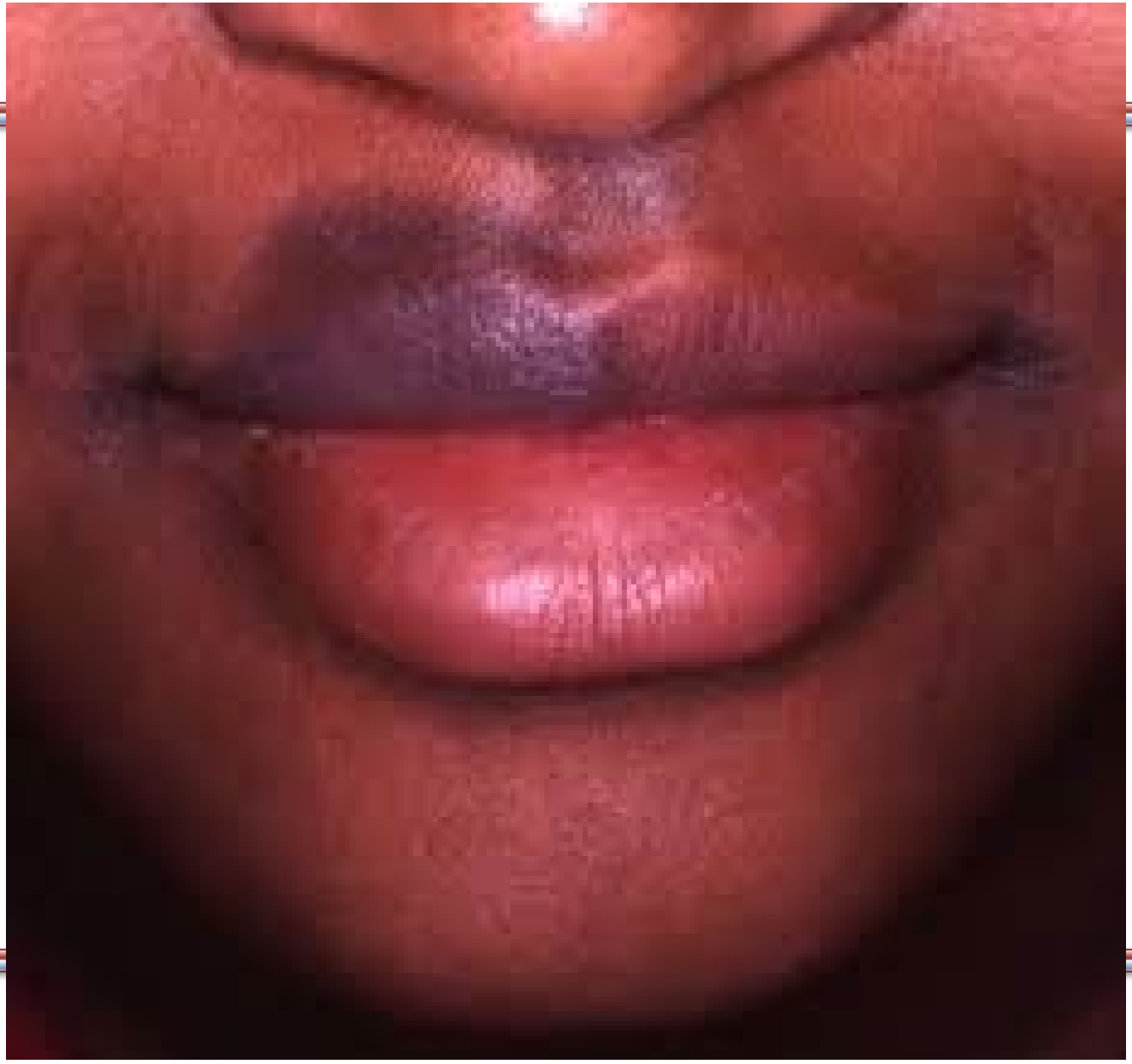
- Occurs up to two weeks after 1st exposure, within 24 hours with subsequent exposure
- Causes:
  - NSAIDS - often on lips, MC naproxen
  - Sulfonamides - genital FDE
  - Pseudoephedrine – non-pigmented
  - Others
- Clinical features:
  - Isolated round or oval dusky to violaceous plaques
  - Recurs in the same place
  - Can be generalized and/or develop bullae
  - Leave post inflammatory hyperpigmentation
  - No systemic symptoms
- Treatment:
  - Remove offending agent
  - Topical corticosteroids

Fixed Drug Eruption (FDE)



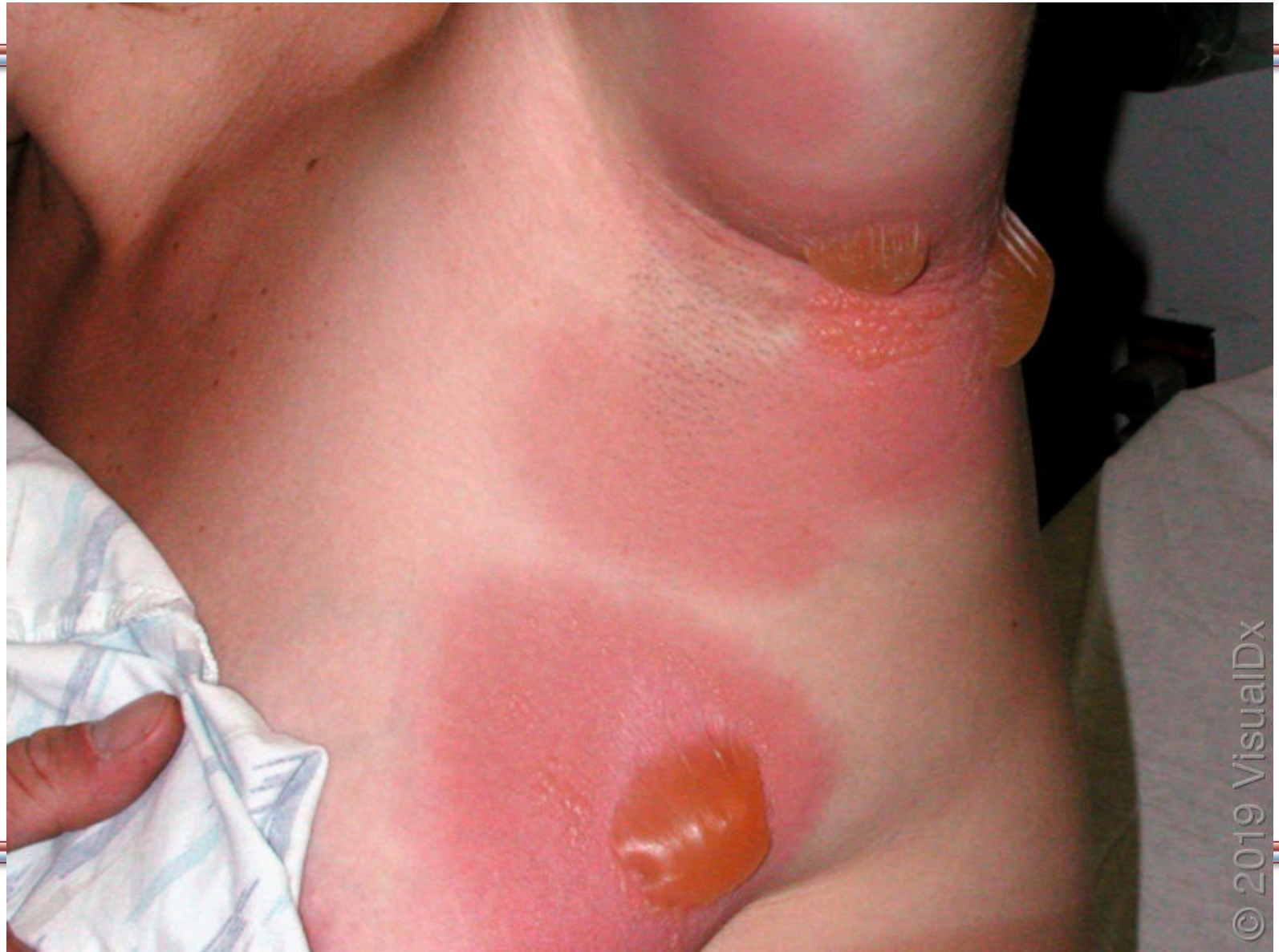
(VisualDx, 2010)

Fixed Drug Eruption (FDE)



(VisualDx, n.d.)

Fixed Drug Eruption (FDE)

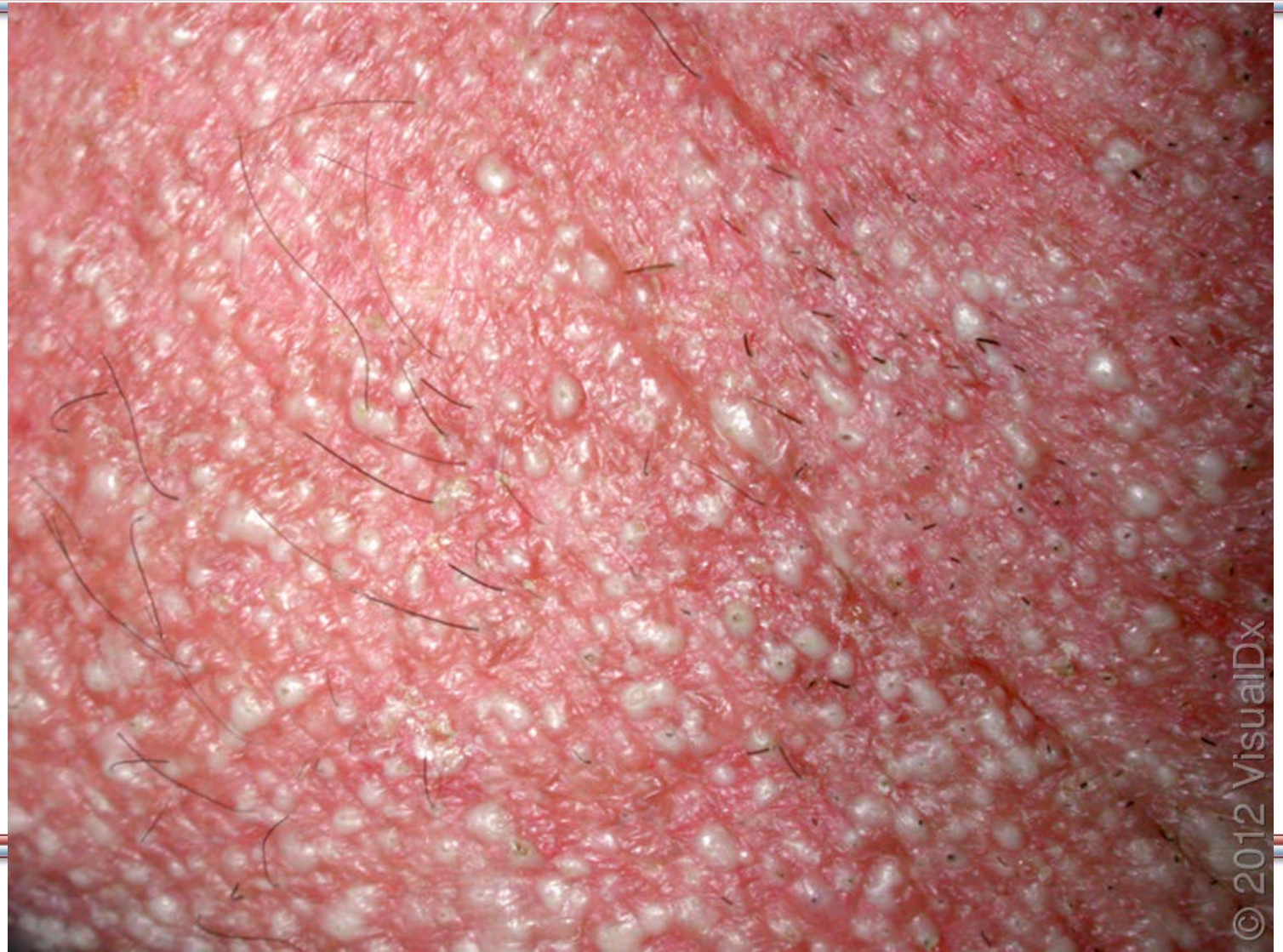


(VisualDx, 2019)

Acute Generalized Exanthematous Pustolosis (AGEP)

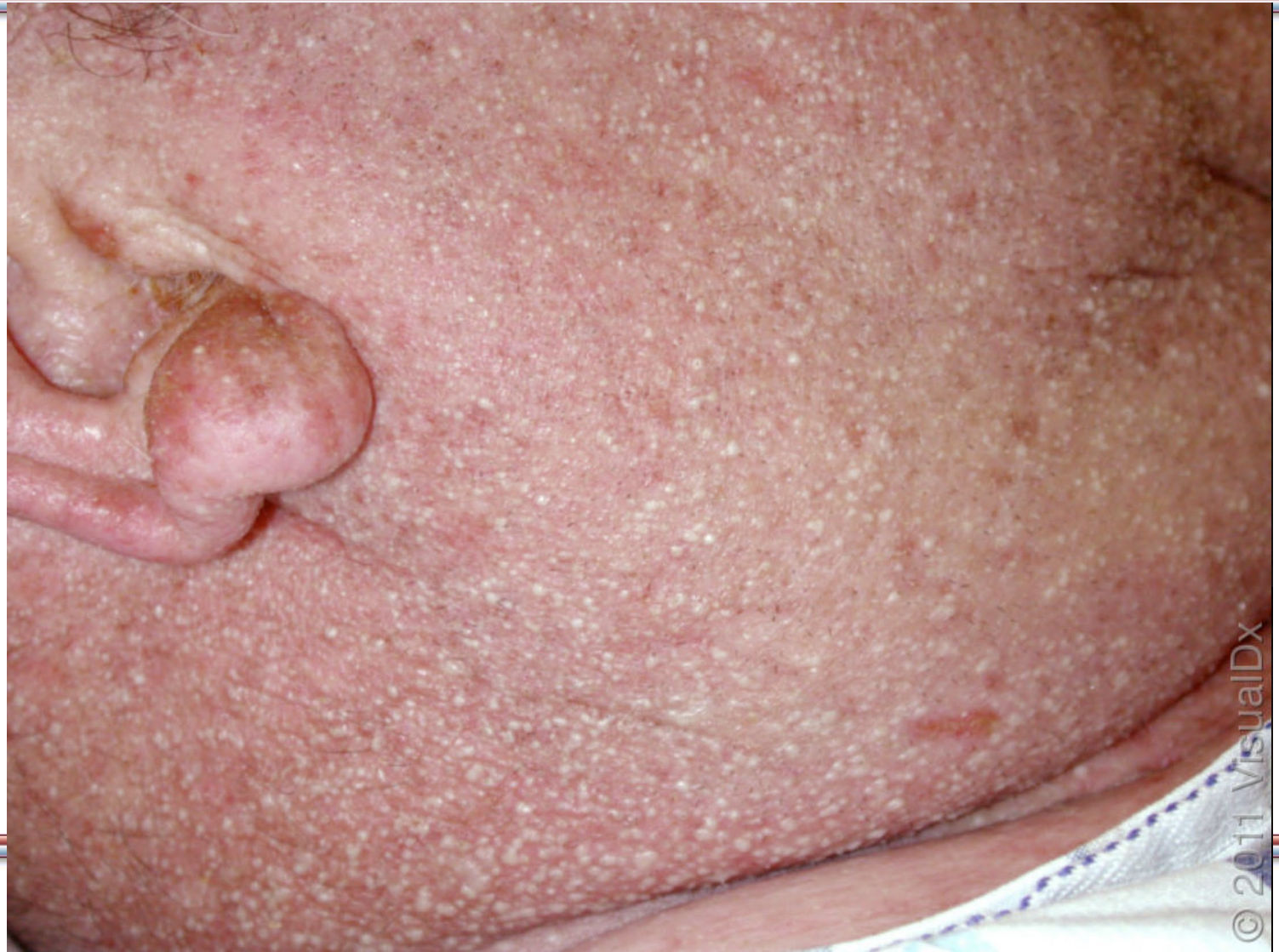
- **Febrile drug reaction, usually within <4 days of starting medication**
- Causes:
  - Medications: beta-lactams, macrolides, CCB's (diltiazem), antimalarials, terbinafine, carbamazepine, acetaminophen, others
- Clinical features:
  - Punctate non-follicular sterile pustules on background of edematous erythema
    - Face and intertriginous, may spread to trunk and extremities
    - May have edema of face and hands
  - Possible mucous membrane involvement
  - Few will have systemic symptoms
  - Typically resolves within 1-2 weeks with desquamation
- Treatment:
  - Discontinue medication
  - Topical corticosteroids

Acute Generalized Exanthematous Pustolosis (AGEP)



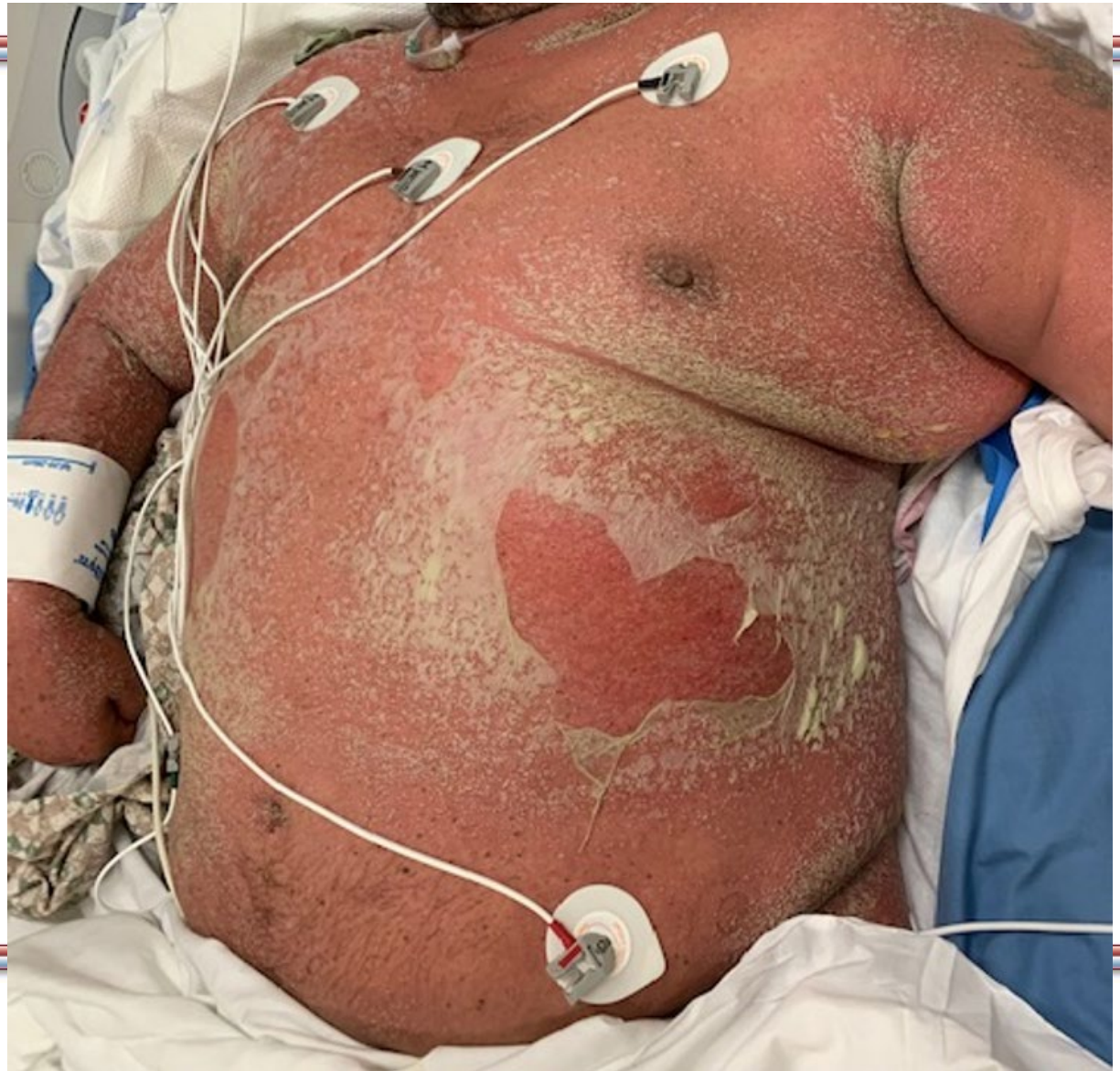
(VisualDx, 2012)

Acute Generalized Exanthematous Pustolosis (AGEP)



(VisualDx, 2011)

Acute Generalized Exanthematous Pustolosis (AGEP)

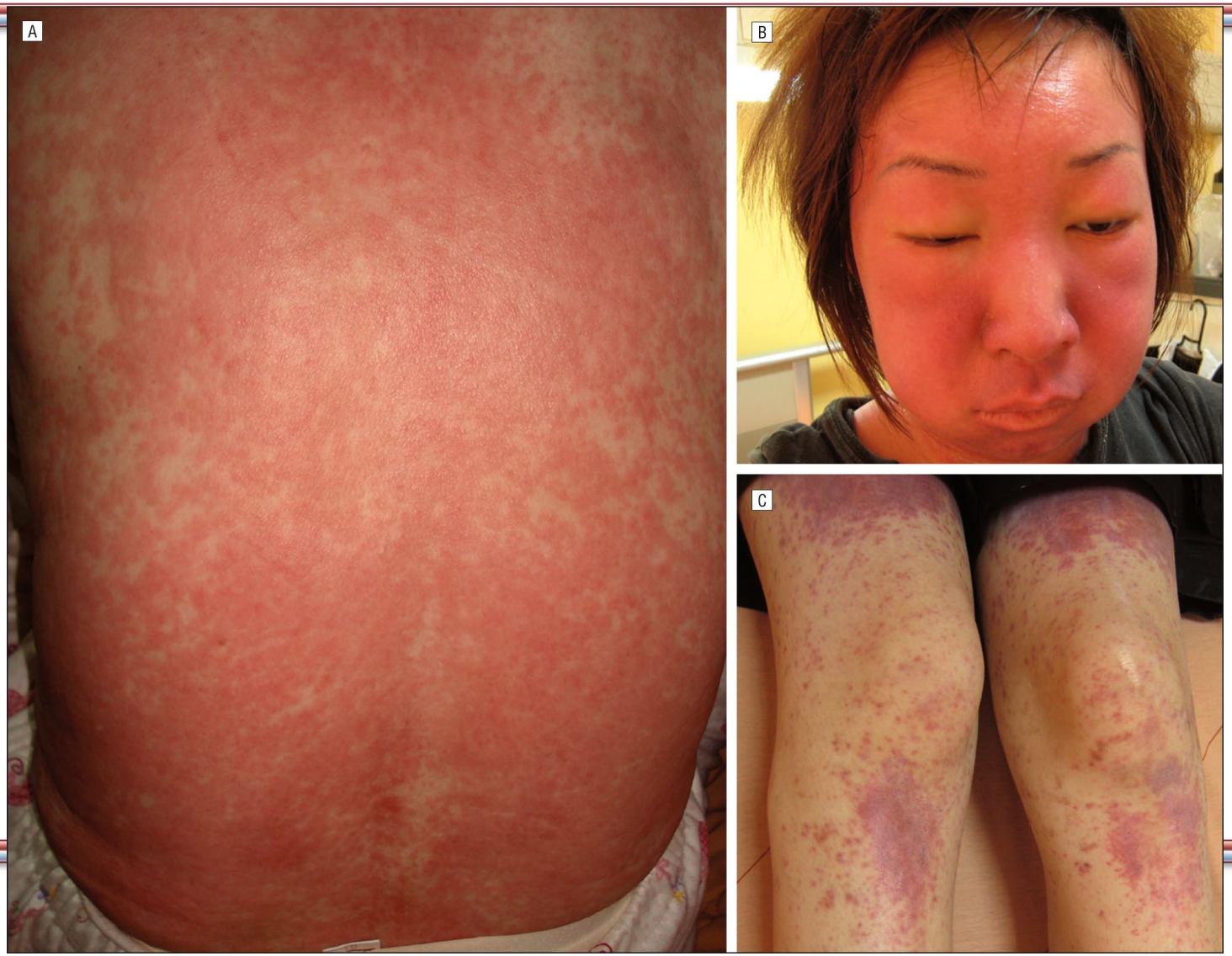


(VisualDx, n.d.)

Drug Reaction with Eosinophlia and Systemic Symptoms (DRESS)

- Rash 2-6 weeks after starting drug
  - Medications: Allopurinol (HLA-B5801), sulfa, phenobarbital, carbamazepine, phenytoin, lamotrigine, dapsone, abacavir (HLA-5701), others
- Clinical features:
  - Morbilliform eruption with perifollicular accentuation
    - Usually upper trunk and extremities
    - Facial edema
    - Limited mucosal involvement
  - Fever, leukocyte adhesion deficiency (LAD)
  - Internal organ involvement – liver, kidney, cardiac, lung, CNS
  - Late sequelae: autoimmune thyroiditis, DM, syndrome of inappropriate antidiuretic hormone secretion (SIADH), SLE
    - Check thyroid stimulating hormone (TSH) for several months after
- Treatment:
  - Oral steroids (1-2 months); can relapse if tapered too quickly
  - Monitor for systemic involvement [urinalysis (UA), electrocardiogram (EKG), liver function tests (LFTs), chest x-ray (CXR)]

Drug Reaction with Eosinophlia and Systemic Symptoms (DRESS)



(VisualDx, n.d.)

Stevens-Johnson Syndrome (SJS) / Toxic Epidermal Necrolysis (TEN)

- Rare, life threatening mucocutaneous reaction almost always medication related
- Medications - sulfa, PCN, allopurinol, anticonvulsants, NSAIDS, antiretrovirals, others
  - Eruption starts 1-3 weeks after starting treatment
- Clinical features
  - Initial lesions are flat, atypical targets (two zones)
    - Necrotic center starts to detach to form flaccid bullae
    - Nikolsky sign, Asboe Hanson sign
  - Mucosal erosions and pain are prominent features
  - SJS <10% BSA, SJS/TEN overlap 10-30% BSA, TEN >30% BSA
  - Complications: symblepharon, synechiae, scar, eruptive nevi, phimosis, nail dystrophy, alopecia, blindness

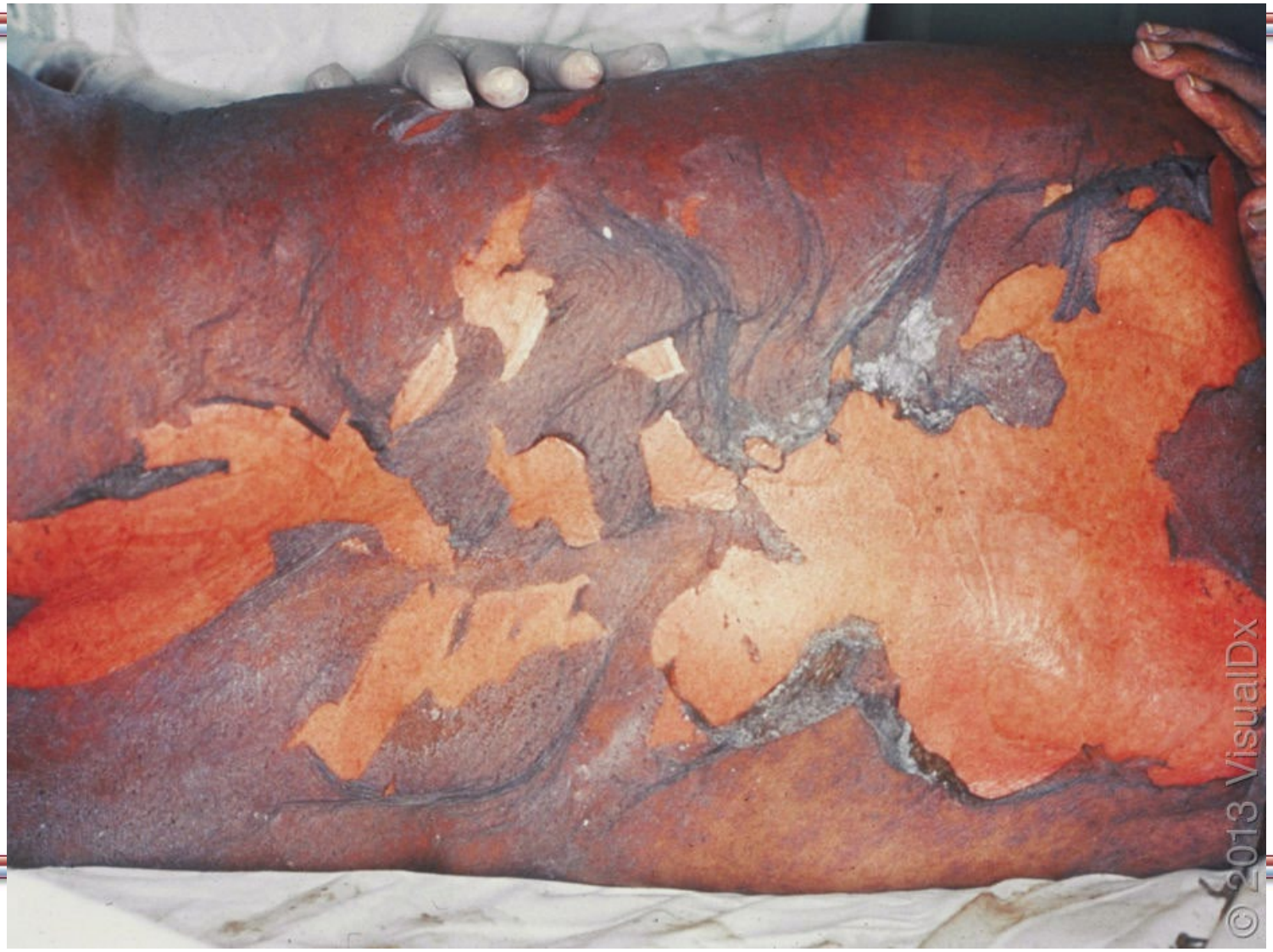
SJS/TEN

- SCORTEN - predicts mortality
- Treatment:
  - Remove offending agent
  - Transfer to higher level of care (BICU)
  - Supportive care
  - Consult gynecology/urology/ophthalmology if indicated
  - Medications (controversial):
    - Cyclosporine
    - Corticosteroids
    - Intravenous immune globulin (IVIG)
    - Tumor necrosis factor alpha (TNFa) inhibitors
    - Plasmapheresis

Stevens-Johnson Syndrome (SJS) / Toxic Epidermal Necrolysis (TEN)

(VisualDx, n.d.)

Stevens-Johnson Syndrome (SJS) / Toxic Epidermal Necrolysis (TEN)



(VisualDx, 2013)

Immunobullous

- Bullous Pemphigoid
- Pemphigus Vulgaris

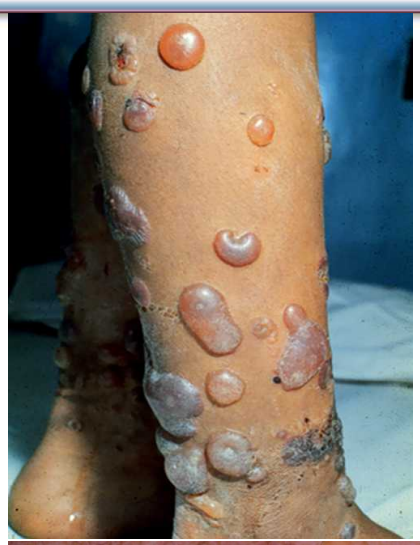
Bullous Pemphigoid

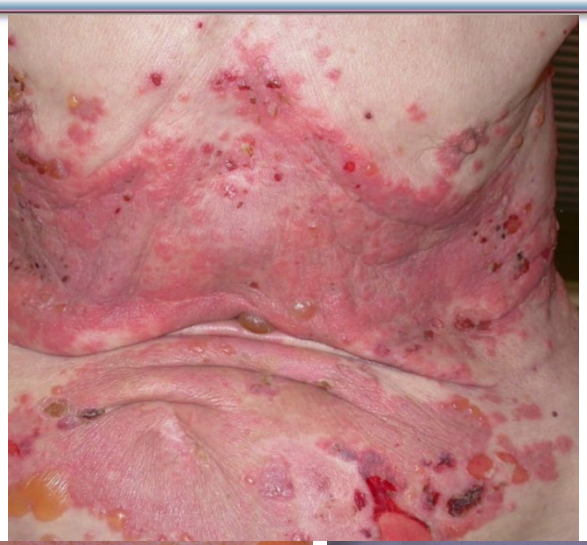
- Most common autoimmune blistering disease
- Elderly age group commonly affected; low mortality
- Due to autoantibodies directed against hemidesmosomal proteins in the betamethasone (BMZ)
  - BP180 (BPAG2) is primary antigenic target in BP
  - BP230 (BPAG1) antibodies due to epitope spreading
- Drug-induced pemphigus (usually younger age)
  - **Furosemide (#1),** ACE-inhibitors, cephalosporins, B-lactams, D-penicillamine, others
- Clinical
  - Early (non-bullous) – polymorphic; often see urticarial plaques on the trunk, abdomen, and flexural extremities; severe pruritus is common
  - Bullous – tense bullae arising on an erythematous and urticarial background
  - Oral involvement in 10-30%

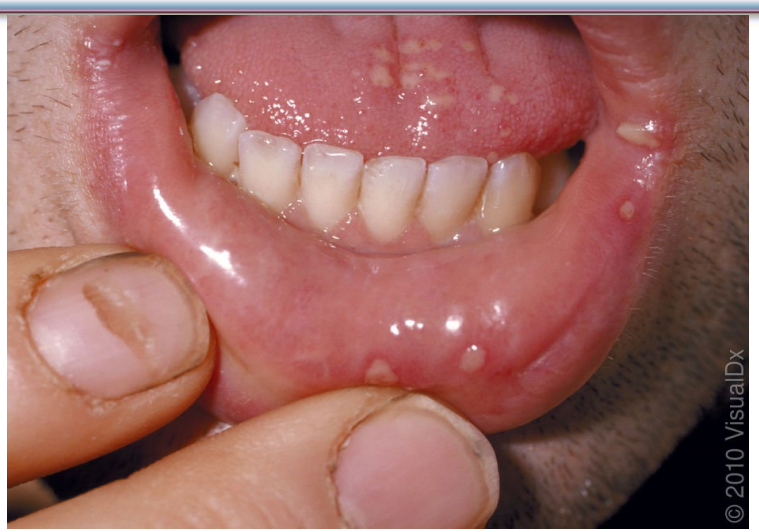
Bullous Pemphigoid

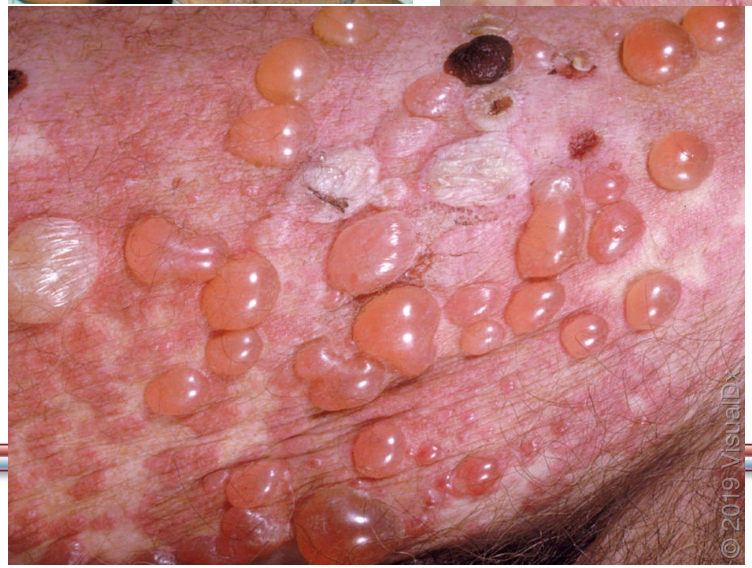
- Work-up
  - Biopsies for H/E and DIF (linear)
  - Indirect immunofluorescence (IIF) – serum test for circulating anti-BMZ antibodies
  - Enzyme-linked immunosorbent assay (ELISA) – for detecting antibodies to BP180/230 – correlates strongly with disease activity
- Treatment
  - 1st Line: systemic steroids + steroid-sparing immunosuppressives [mycophenolate mofetil (MMF), methotrexate (MTX), azathioprine, rituximab (RTX), cyclophosphamide]
  - Alt 1st Line: widespread superpotent topical steroids
  - Other: tetracycline (TCN) + nicotinamide for mild disease, dapsone for mucosal predominant disease, rituximab for recalcitrant cases, IVIG, plasma exchange

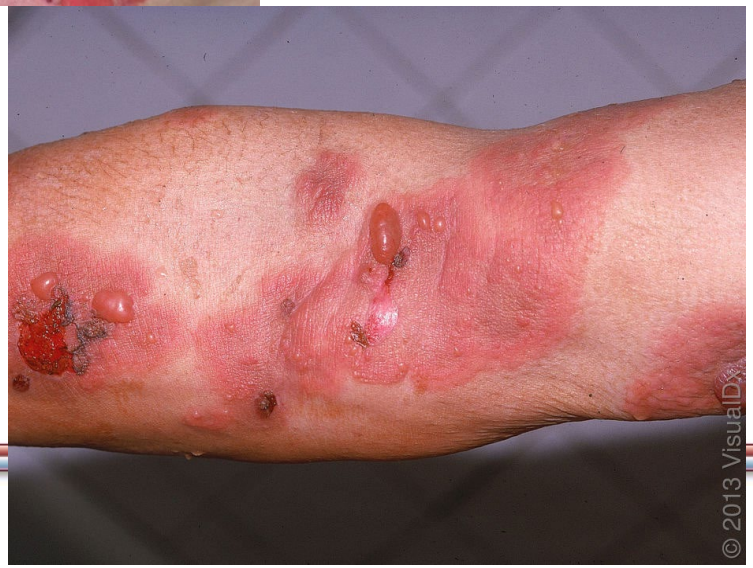
Bullous Pemphigoid











(VisualDx, 2010, 2013, 2019)

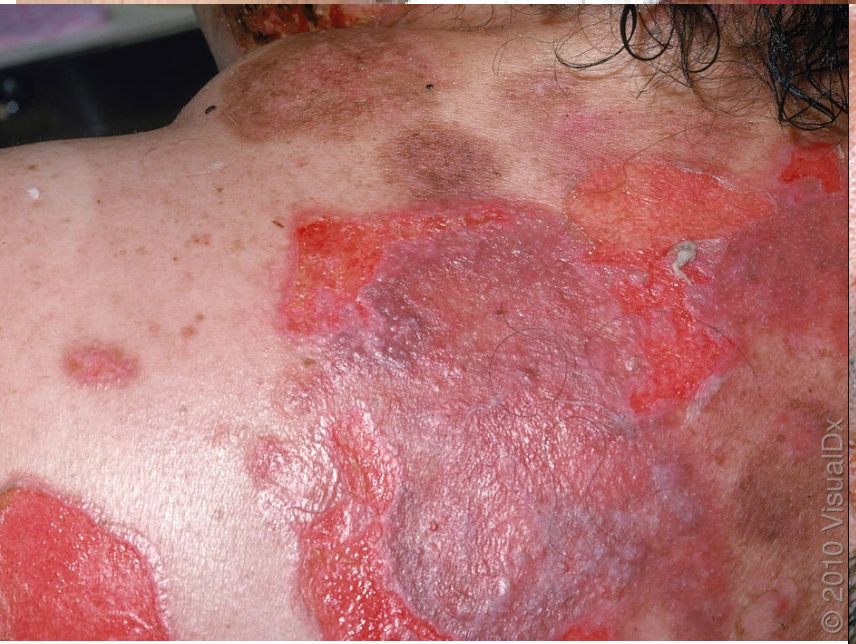
Pemphigus Vulgaris (PV)

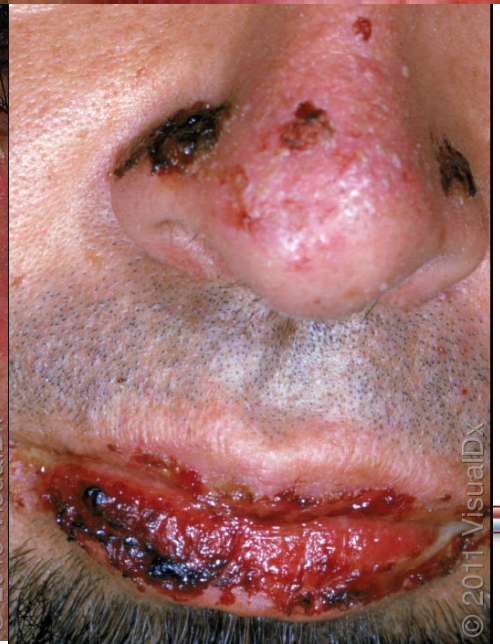
- Younger age group than BP – 50-60s
- Associated with myasthenia gravis, thymoma, and autoimmune thyroiditis
- Due to autoantibodies directed against desmosomal proteins (intraepidermal)
  - Desmoglein 3 – mucosal dominant PV
  - Desmoglein 1 and 3 – mucocutaneous PV
- Clinical features
  - ALL patients have painful oral erosions; other mucosal sites – esophagus, conjunctiva, nasal mucosa, vagina, penis, anus
  - Skin involvement in 50% - painful flaccid bullae and erosions with + Nikolsky sign; heals without scarring
    - If widespread – can result in death from fluid imbalance or secondary infection

Pemphigus Vulgaris

- Work-up
  - Biopsies for H/E and DIF (chicken wire)
  - IIF – serum test for circulating Abs – correlates w/ disease activity
  - ELISA – for detecting antibodies to Dsg 1, 3 – correlates w/ disease activity and can distinguish from pemphigus subtypes
- Treatment
  - 1st Line: systemic steroids + steroid-sparing immunosuppressives (MMF, MTX, azathioprine, cyclophosphamide)
  - Other: TCN + nicotinamide for very mild disease; plasmapheresis for rapid control of severe disease; IVIG or rituximab for recalcitrant disease

Pemphigus Vulgaris





(VisualDx, 2010, 2011, n.d.)

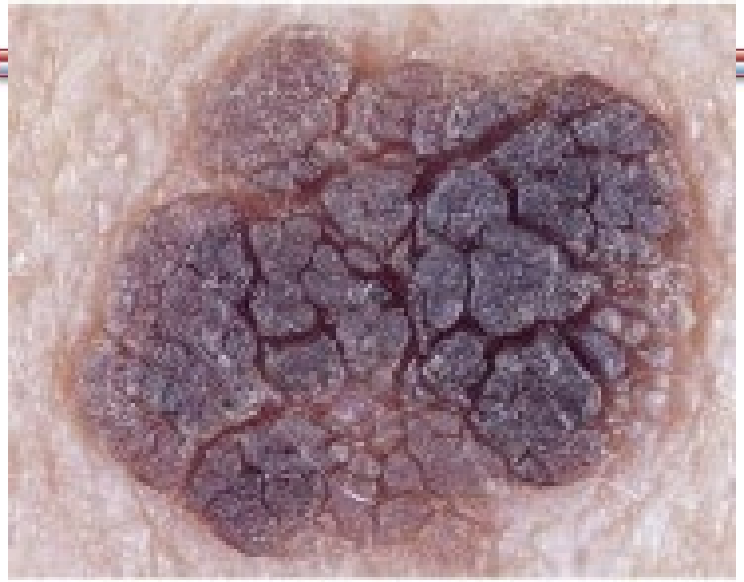
Nodules and Tumors of the Skin

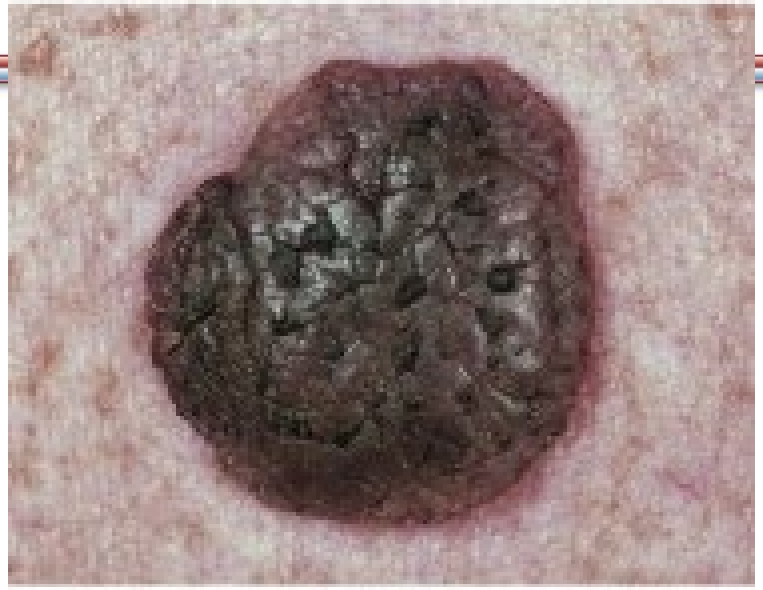
- Seborrheic keratosis
- Dermatofibroma
- Actinic Keratosis
- Squamous Cell Carcinoma
- Basal Cell Carcinoma
- Melanoma

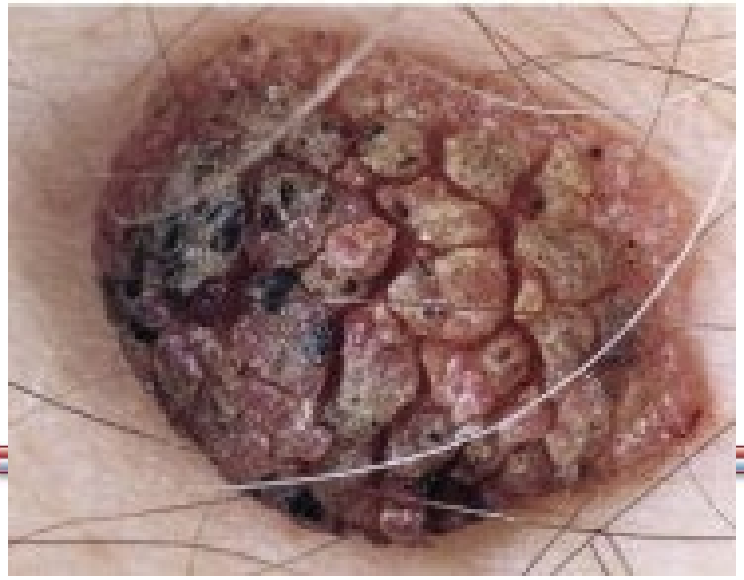
Seborrheic Keratosis (SK)

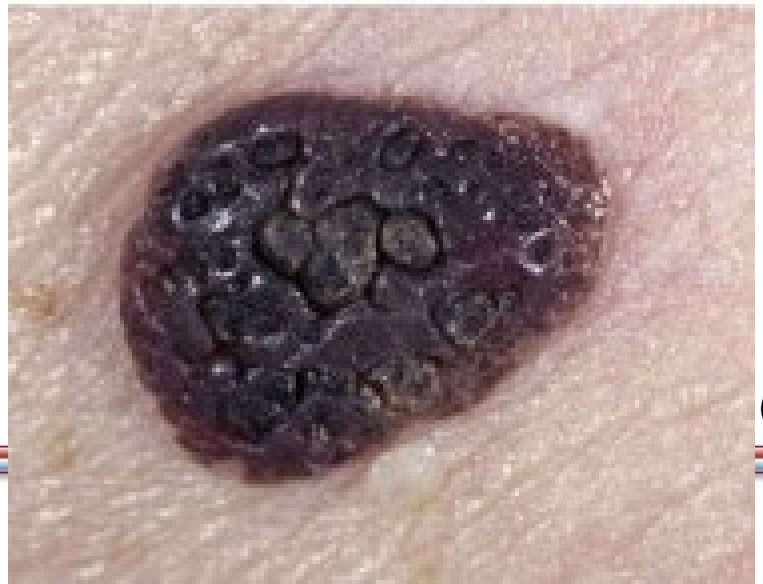
- Benign lesion associated with sun exposure, onset 4th decade
- Clinical features:
  - Well circumscribed papule or plaque with smooth or rough surface
  - Can be tan, brown, or black in color
  - Waxy "stuck on" appearing
  - Found in hair bearing areas
  - Sign of Leser-Trelat → eruptive SKs associated with internal malignancy (GI, lung)
- Treatment:
  - Cryotherapy
  - Shave biopsy
  - Curettage
  - Electrosurgery

Seborrheic Keratosis (SK)







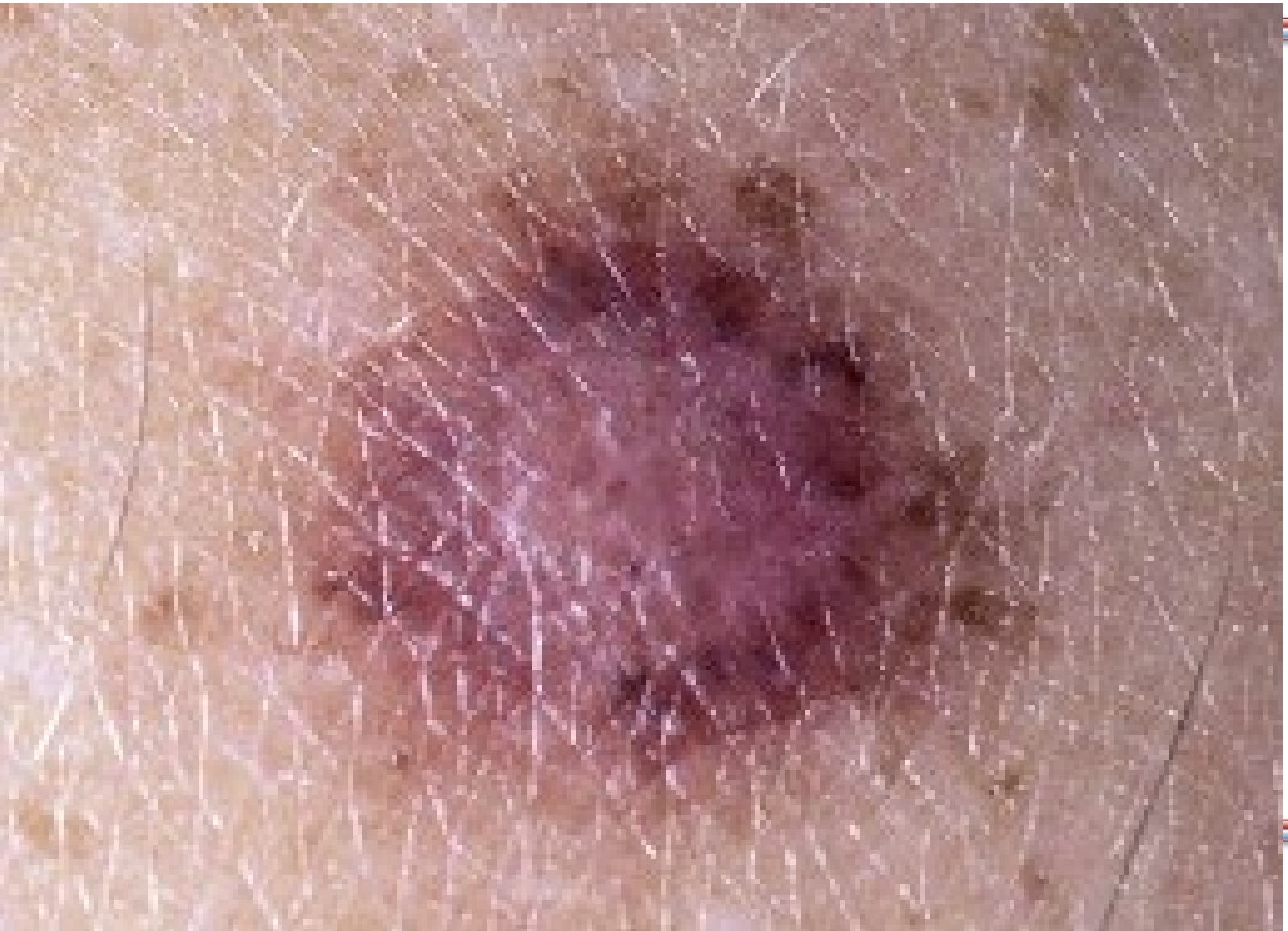


(VisualDx, n.d.)

Dermatofibroma

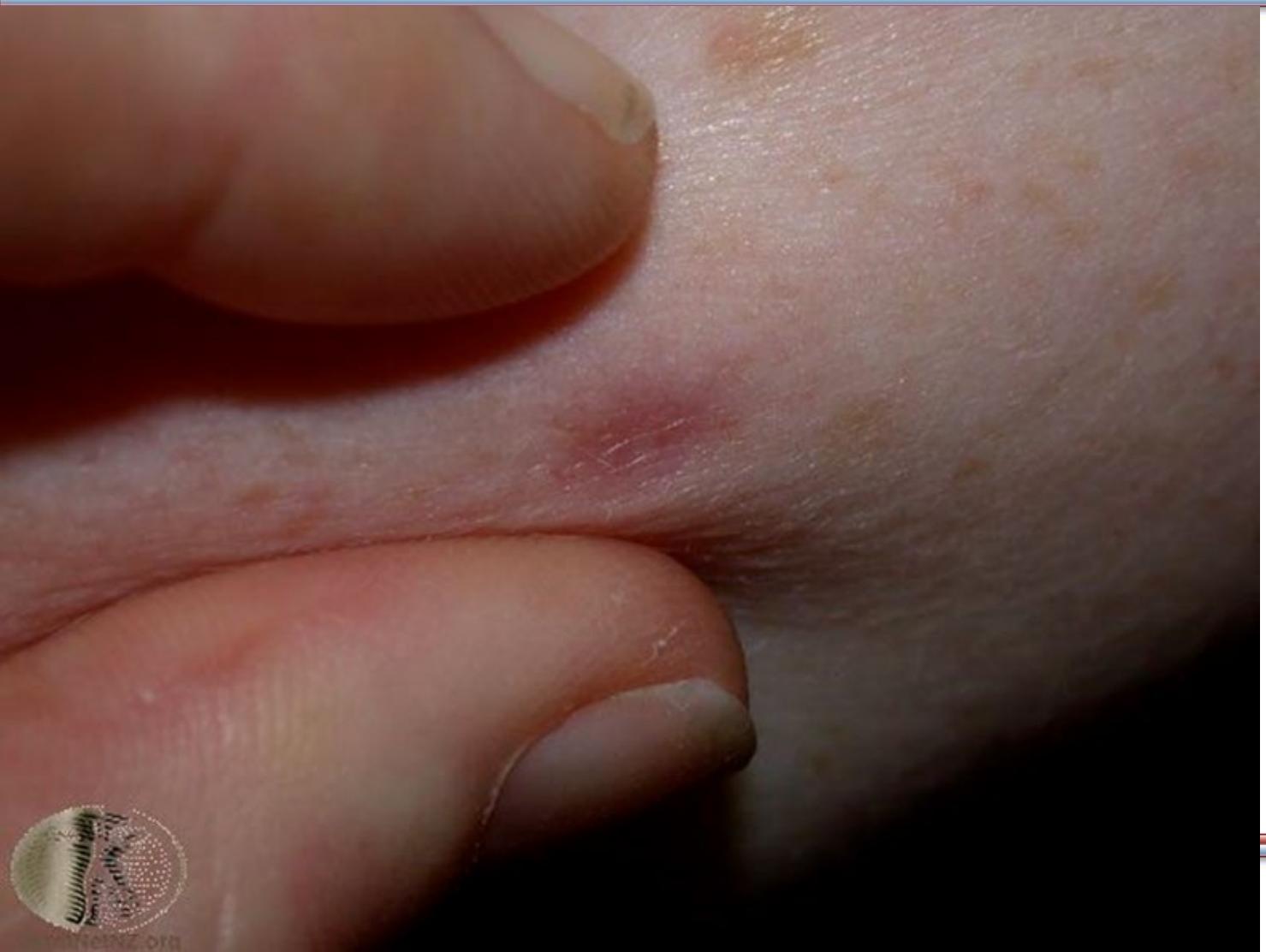
- Benign lesion favoring adults, most commonly located on lower extremities
- Unknown etiology, could be related to trauma or arthropod bites
- Clinical features:
  - Presents as solitary or few lesions
  - White scar like center with a pigmented periphery
  - ”Dimple” sign when pinched
  - Can be pruritic
  - Multiple DFs → immunosuppression or SLE
- Treatment:
  - Reassurance
  - Biopsy/excision
  - Cryotherapy

Dermatofibroma



(VisualDx, n.d.)

Dermatofibroma

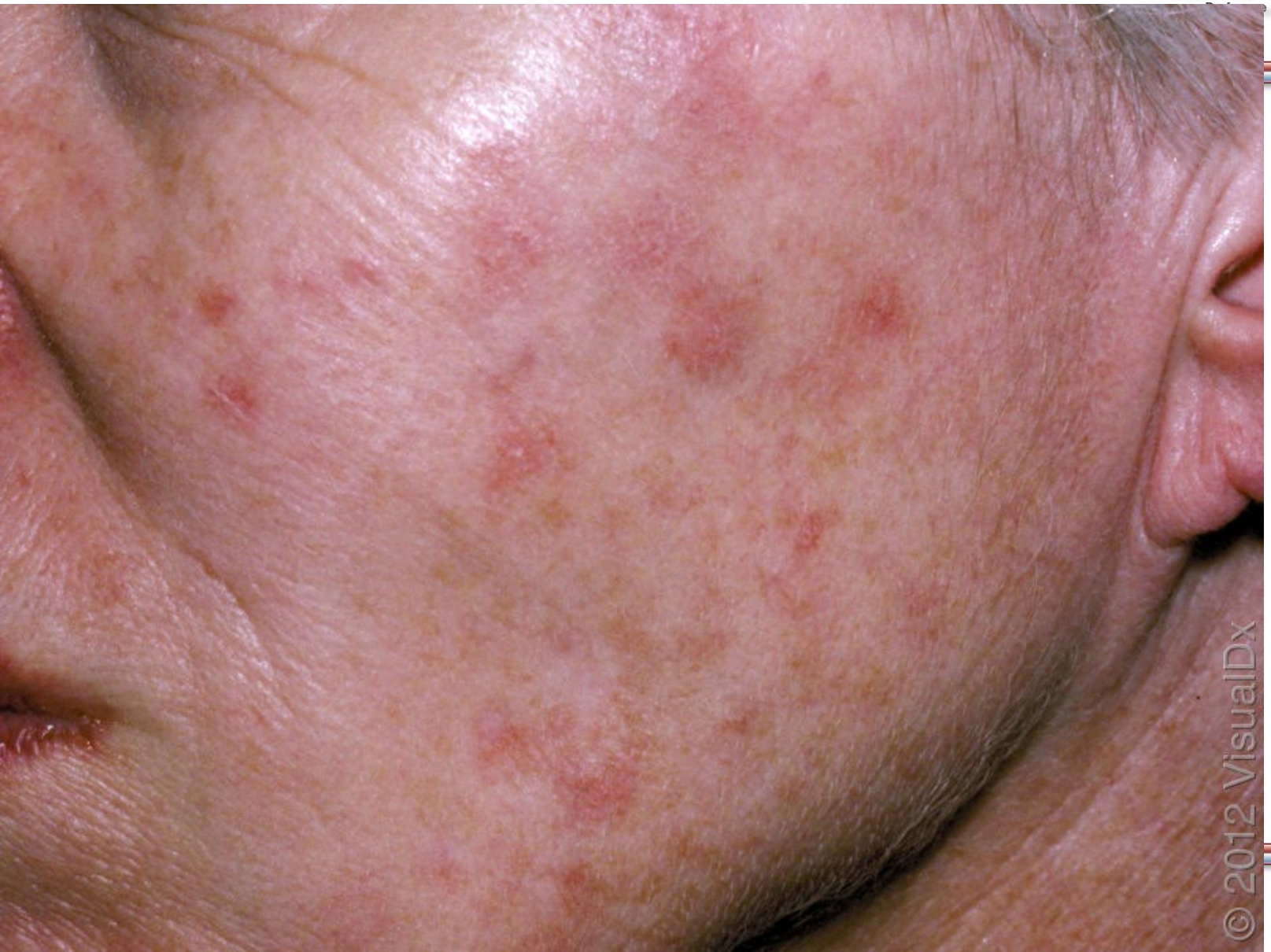


(DermNetNZ.org, n.d.)

Actinic Keratosis

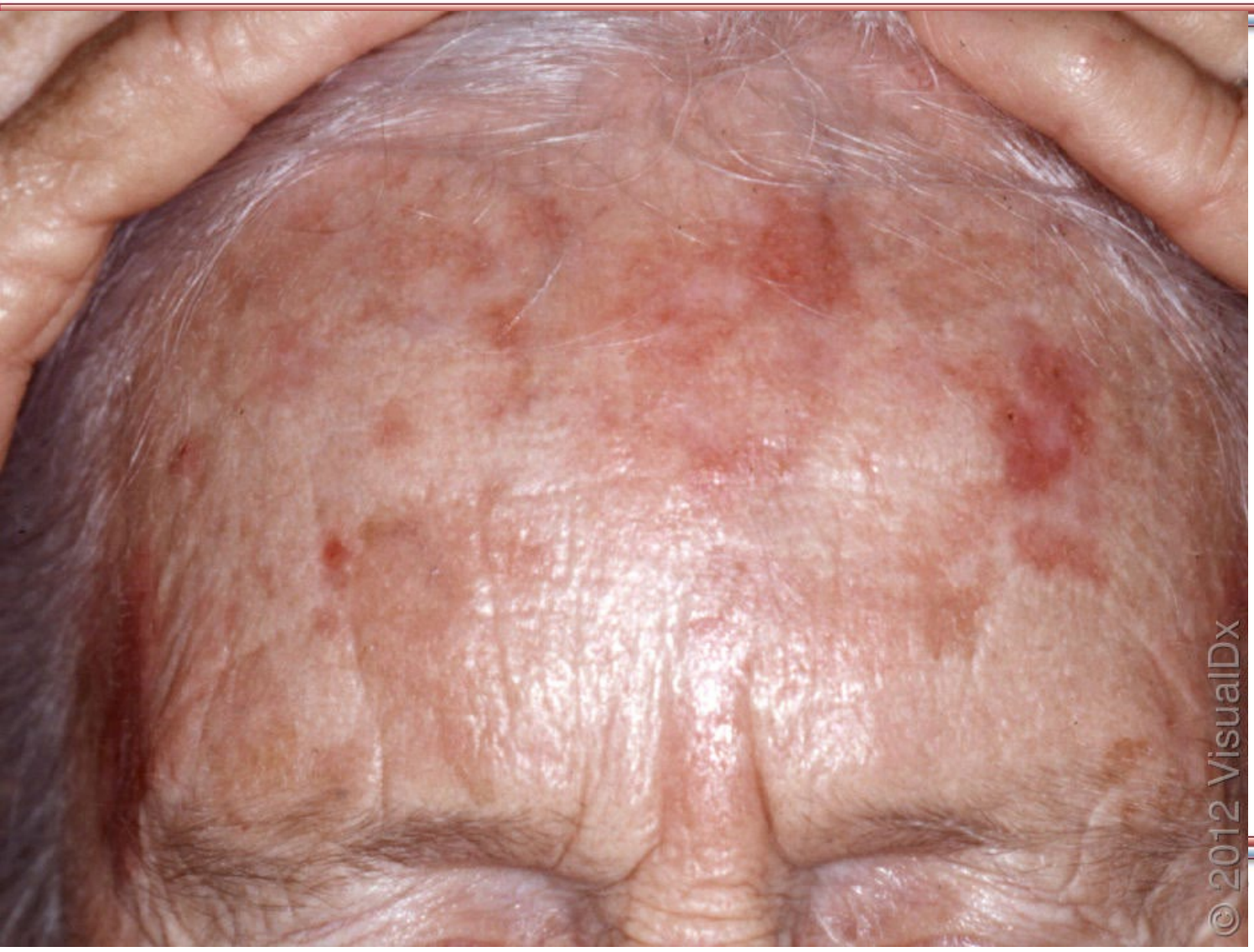
- Precancerous lesions found on sun-exposed areas of the body
- Frequency increases with age and cumulative lifetime sun exposure
- Clinical features:
  - Ill-defined erythematous macules or papules with hard, gritty scale
  - Usually asymptomatic, but can be painful
- Potential to evolve into squamous cell carcinoma
- Treatment:
  - Sun protection
  - Individual lesions
    - Cryotherapy
  - Multiple lesions
    - Cryotherapy
    - 5-Fluorouracil cream
    - Imiquimod cream
    - Photodynamic Therapy

Actinic Keratosis



(VisualDx, 2012)

Actinic Keratosis



(VisualDx, 2012)

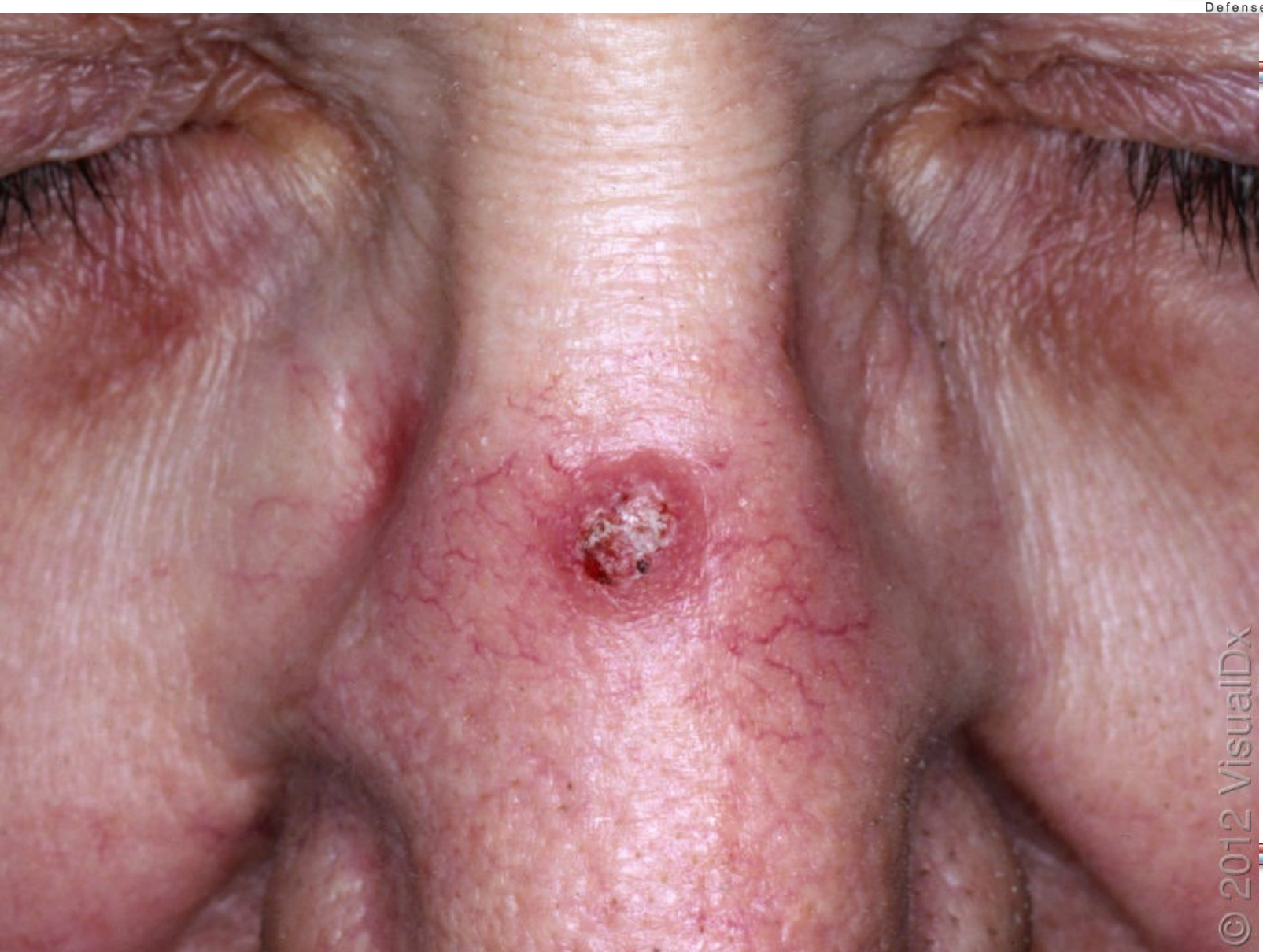
Squamous Cell Carcinoma

- Risk Factors:
  - Male, older age, skin type
  - Extensive sun exposure, tanning bed usage, ionizing radiation
  - Human papilloma virus (HPV) (16,18, 5, 8)
  - Immunosuppression (depends on degree of suppression needed i.e. cardiac > renal)
  - Occupational Exposures (eg Arsenic)
- Clinical features:
  - Hyperkeratotic papule or plaque
  - Induration
  - Ulceration, hemorrhagic crust

Squamous Cell Carcinoma

- Treatment
  - Excision/Mohs micrographic surgery **
  - Electrodessication and curettage (ED&C) **
  - XRT, cryotherapy, photodynamic therapy (PDT)
  - Topicals
  - Intralesional therapy
  - Systemic chemotherapy
- 30-50% of pts with nonmelanoma skin cancer (NMSC) will develop another within 5 yrs
  - 10-fold higher risk than general population

Squamous Cell Carcinoma



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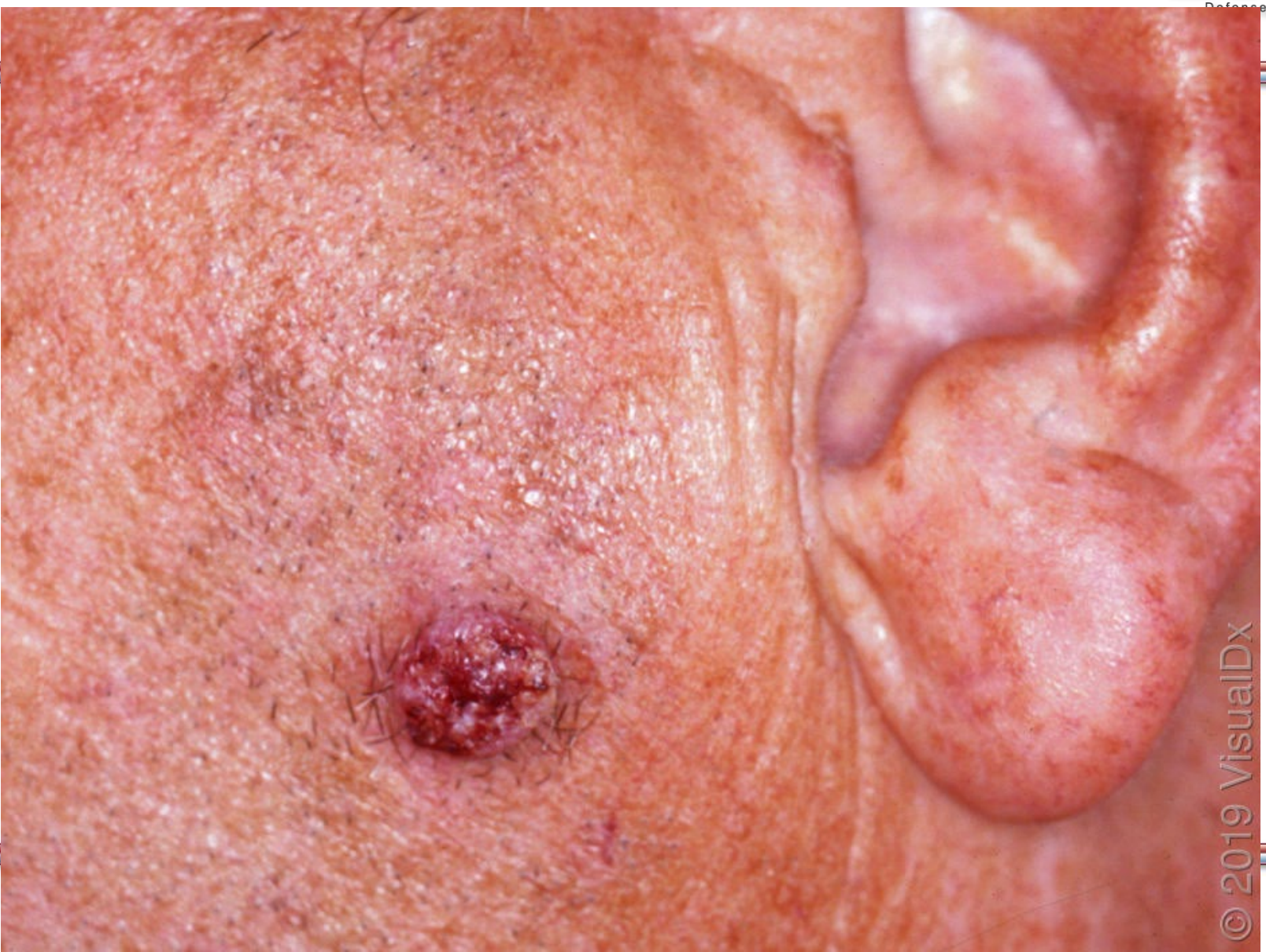
Squamous Cell Carcinoma



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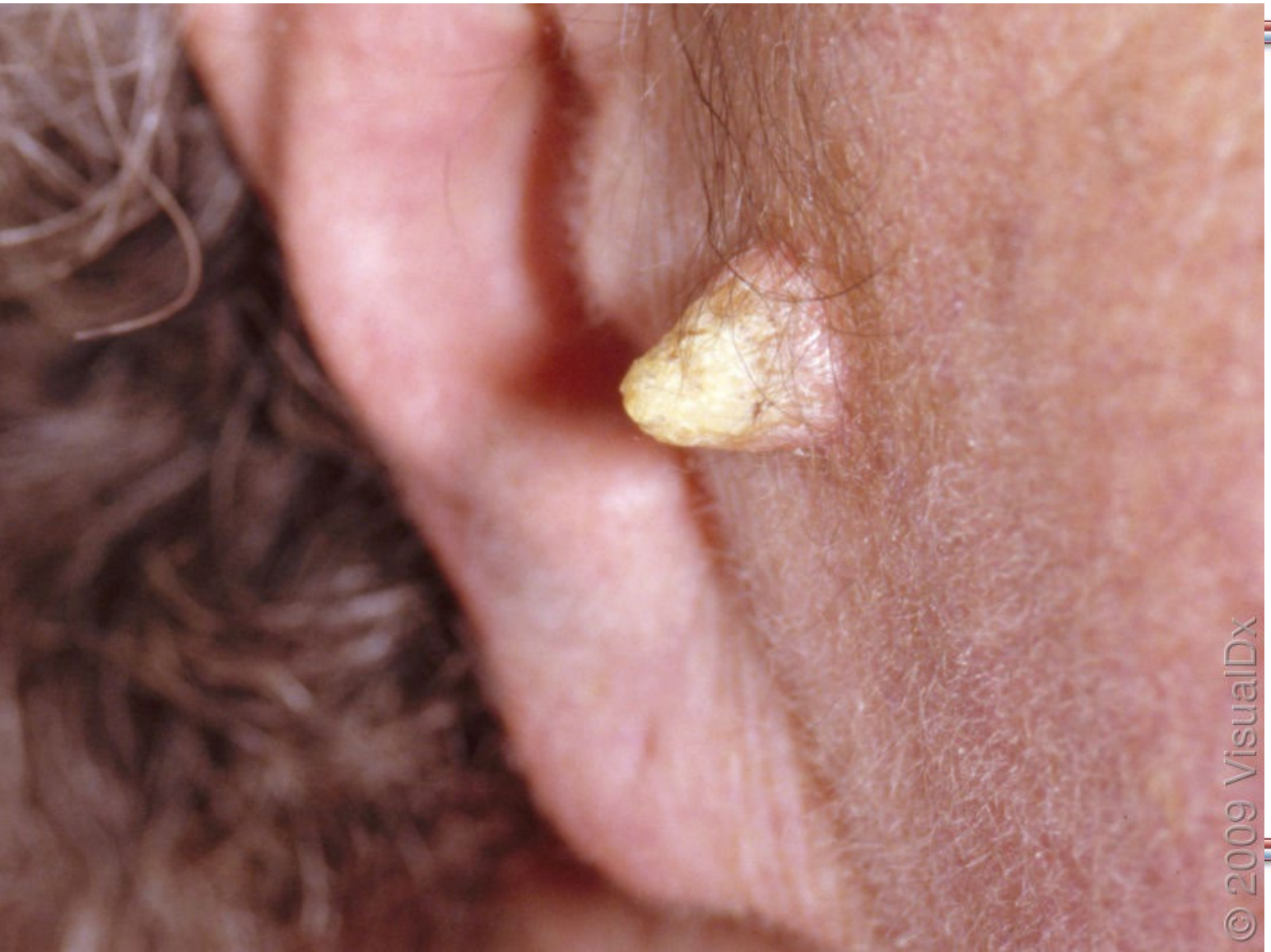
Squamous Cell Carcinoma

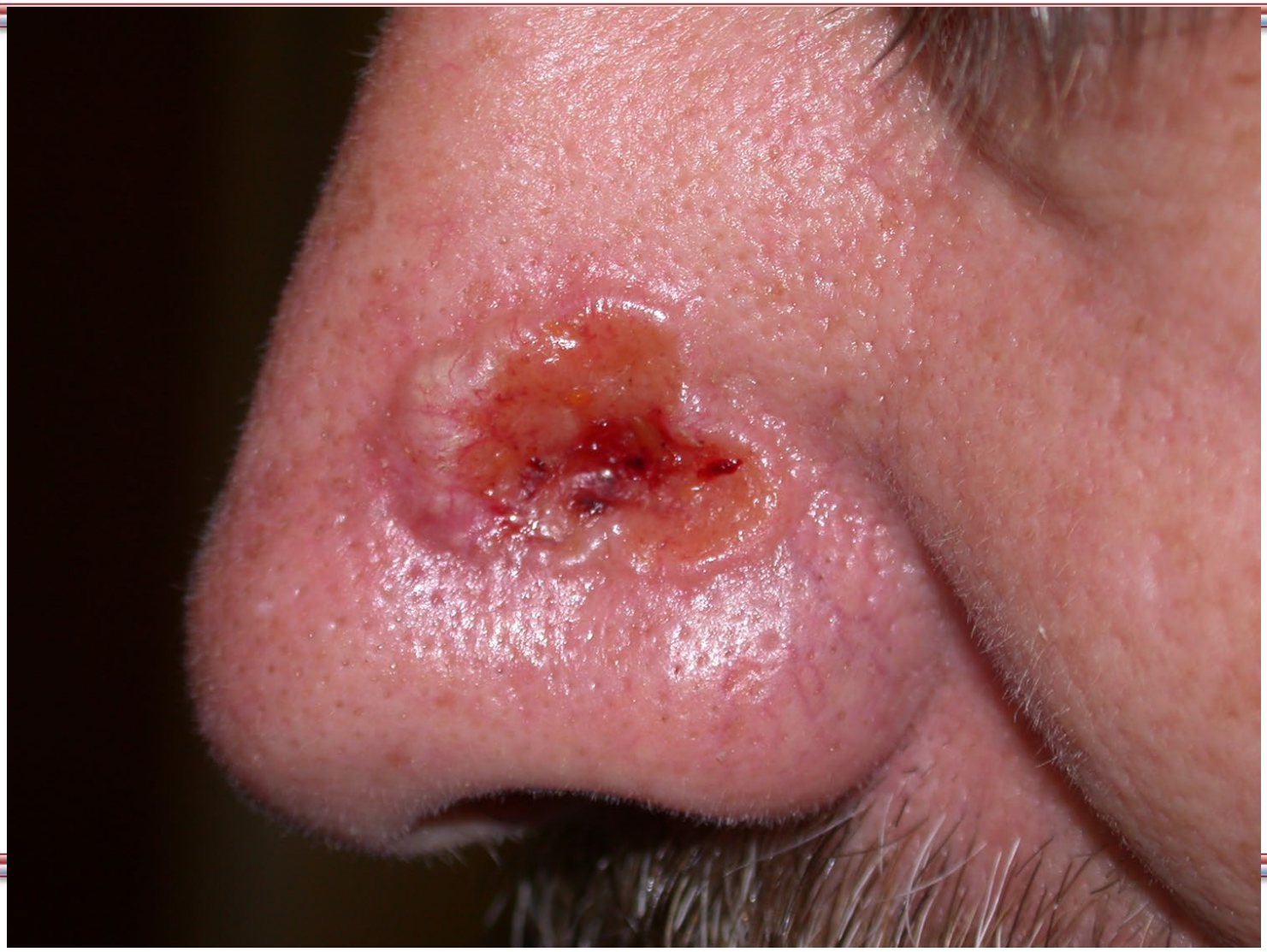


(VisualDx, 2009)

Basal Cell Carcinoma

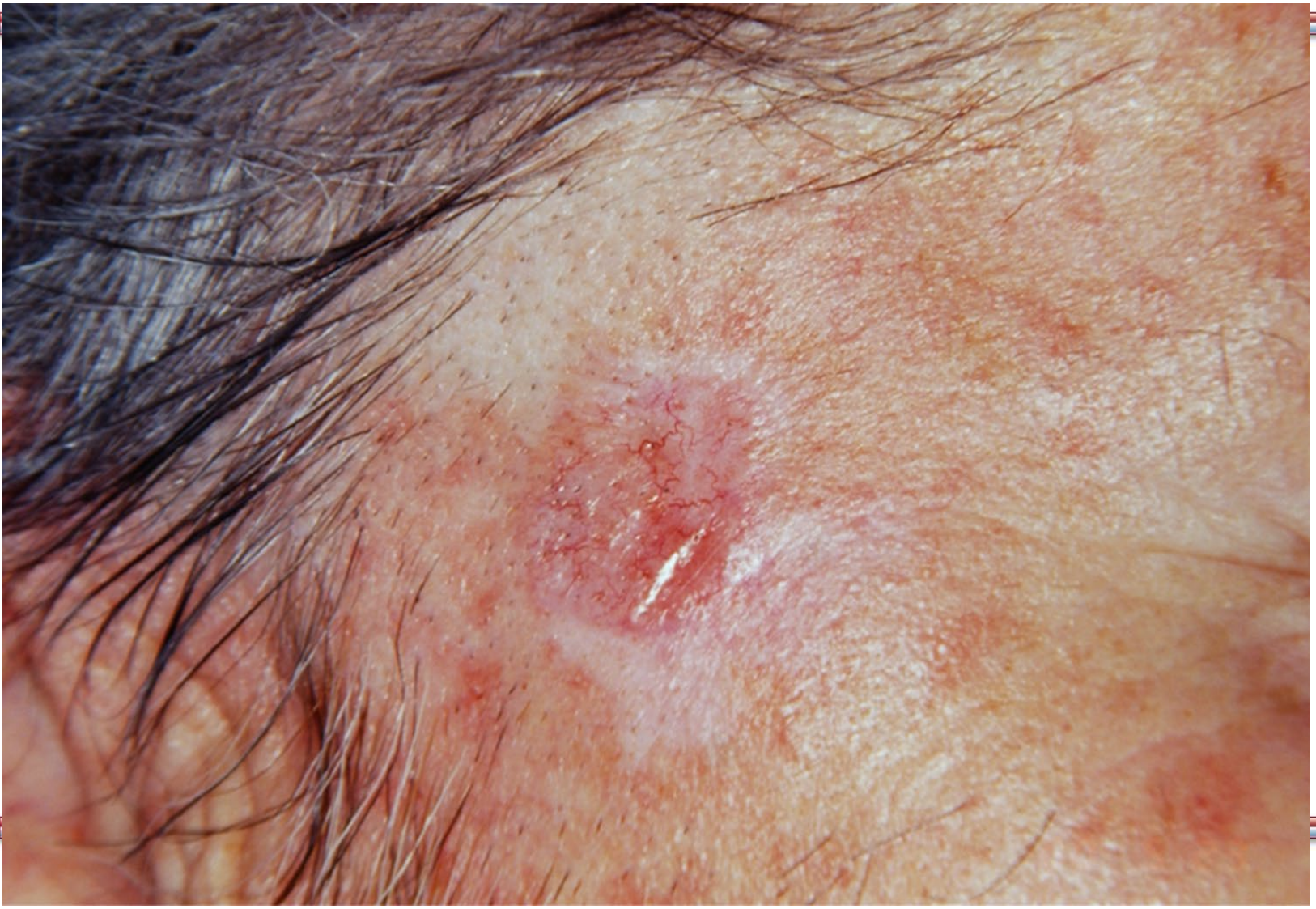
- Slow growing, locally destructive skin cancer
- Risk Factors:
  - Older age, male, skin type
  - Intermittent sun exposure, XRT, blistering sunburns
- Clinical features:
  - Pink pearly papule
  - Ulcerations
  - Pigmented
  - Telangiectasias
- Treatment
  - Excision/Mohs **
  - ED&C **
  - Efudex, Imiquimod
  - Cryotherapy, Radiation, PDT

Basal Cell Carcinoma



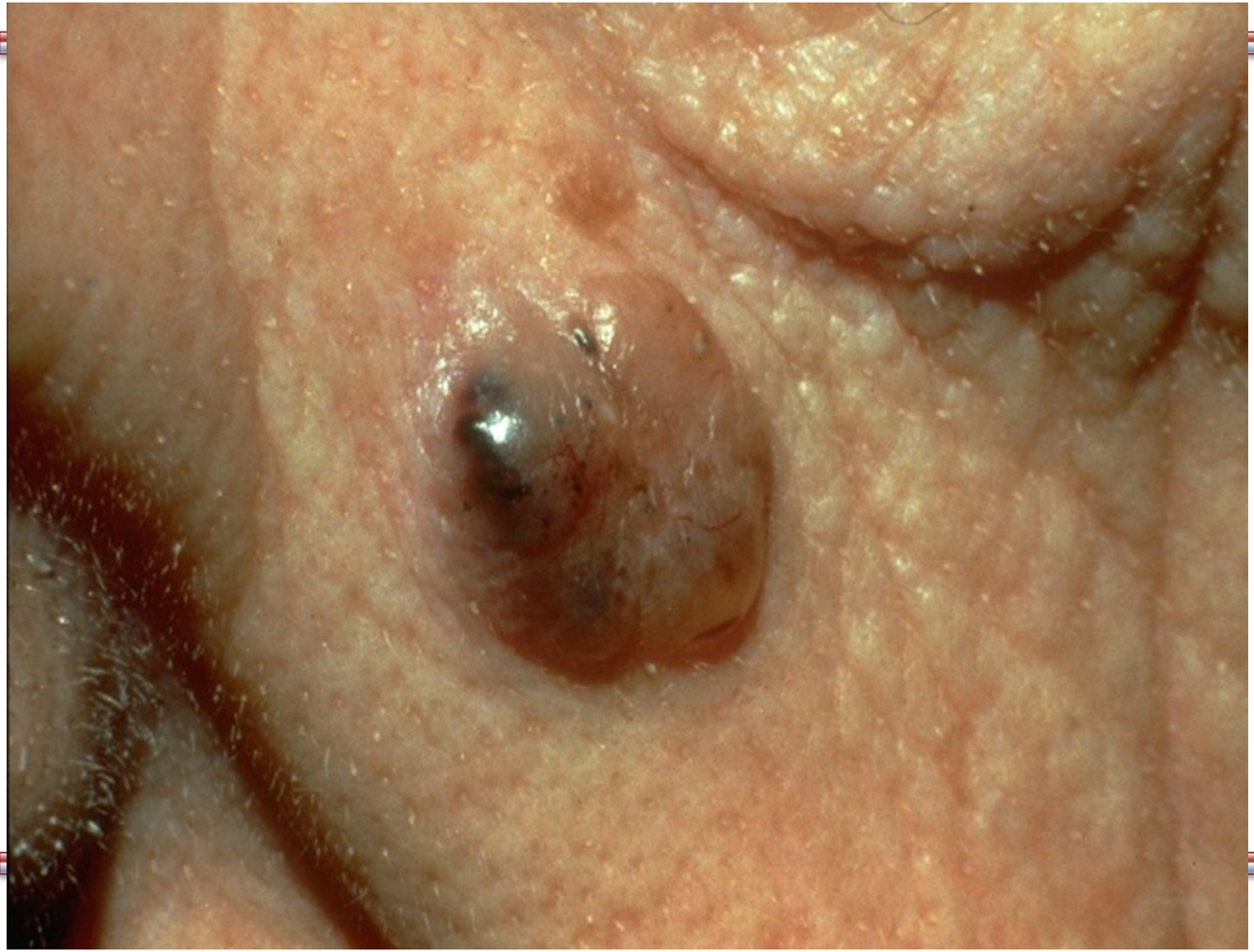
(VisualDx, n.d.)

Basal Cell Carcinoma



(VisualDx, n.d.)

Basal Cell Carcinoma



(VisualDx, n.d.)

Melanoma

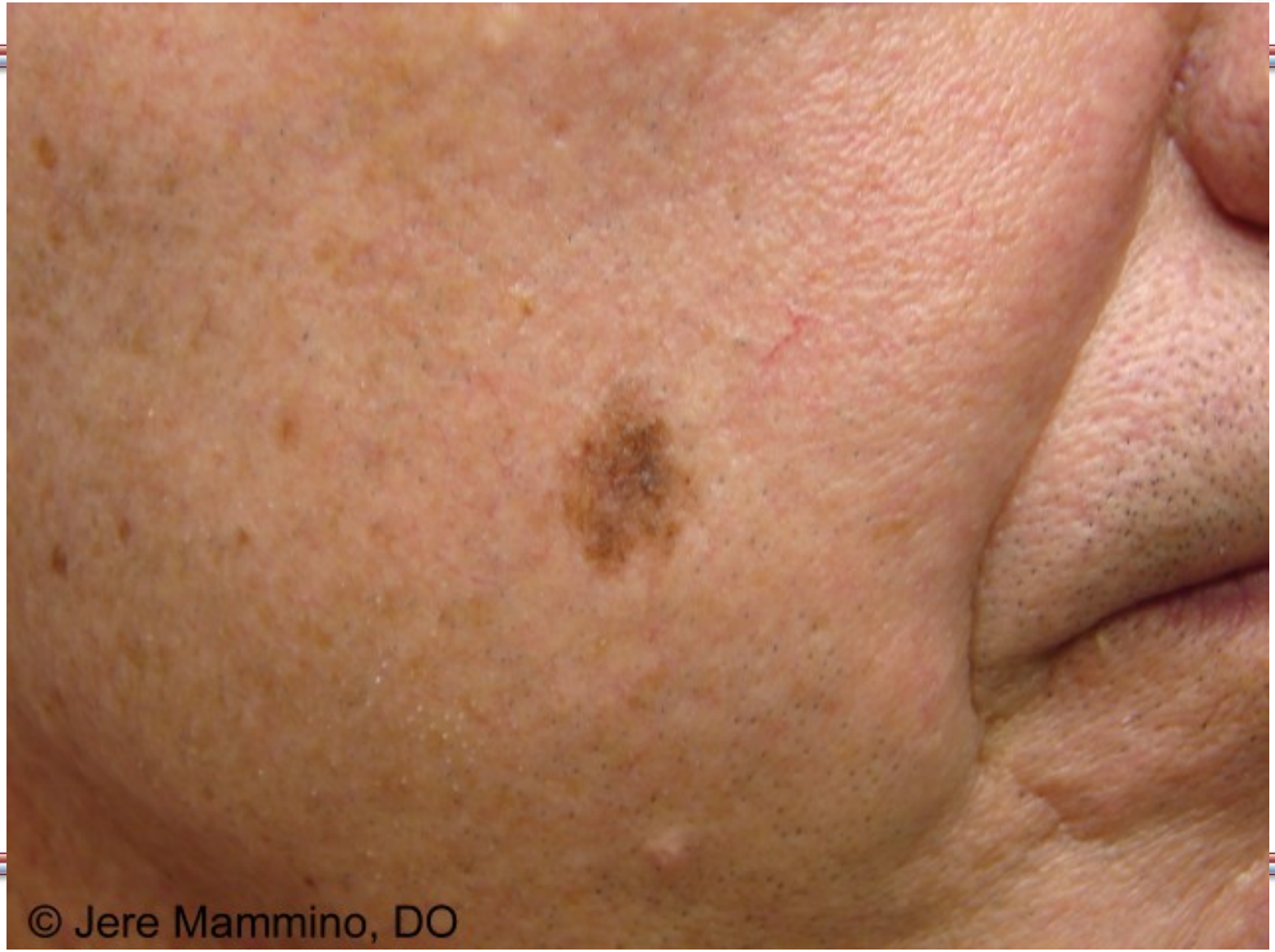
- Comprises <5% of skin cancers
  - 76,100 new cases/year in US
    - 1 in 50 in US will develop melanoma in lifetime
    - MC cancer in women 25-29
  - 9,710 deaths/year in US
    - Majority of skin cancer-related deaths
- De novo or from pre-existing nevus
- Risk Factors
  - Personal history of atypical moles
  - Family history of melanoma
  - >75-100 moles
  - Previous NMSC
  - Giant Congenital Nevus
  - History of melanoma
  - Immunosuppression
  - Other: tanning bed use, childhood sunburns, fair skin/red hair, freckling, intermittent sun exposure

Melanoma

NORMAL		CANCEROUS
	**"A" IS FOR ASYMMETRY** • If you draw a line through the middle of the mole, the halves of a melanoma won't match in size.	
	**"B" IS FOR BORDER** • The edges of an early melanoma tend to be uneven, crusty or notched.	
	**"C" IS FOR COLOR** • Healthy moles are uniform in color. A variety of colors, especially white and/or blue, is bad.	
	**"D" IS FOR DIAMETER** • Melanomas are usually larger in diameter than a pencil eraser, although they can be smaller.	
	**"E" IS FOR EVOLVING** • When a mole changes in size, shape or color, or begins to bleed or scab, this points to danger.	

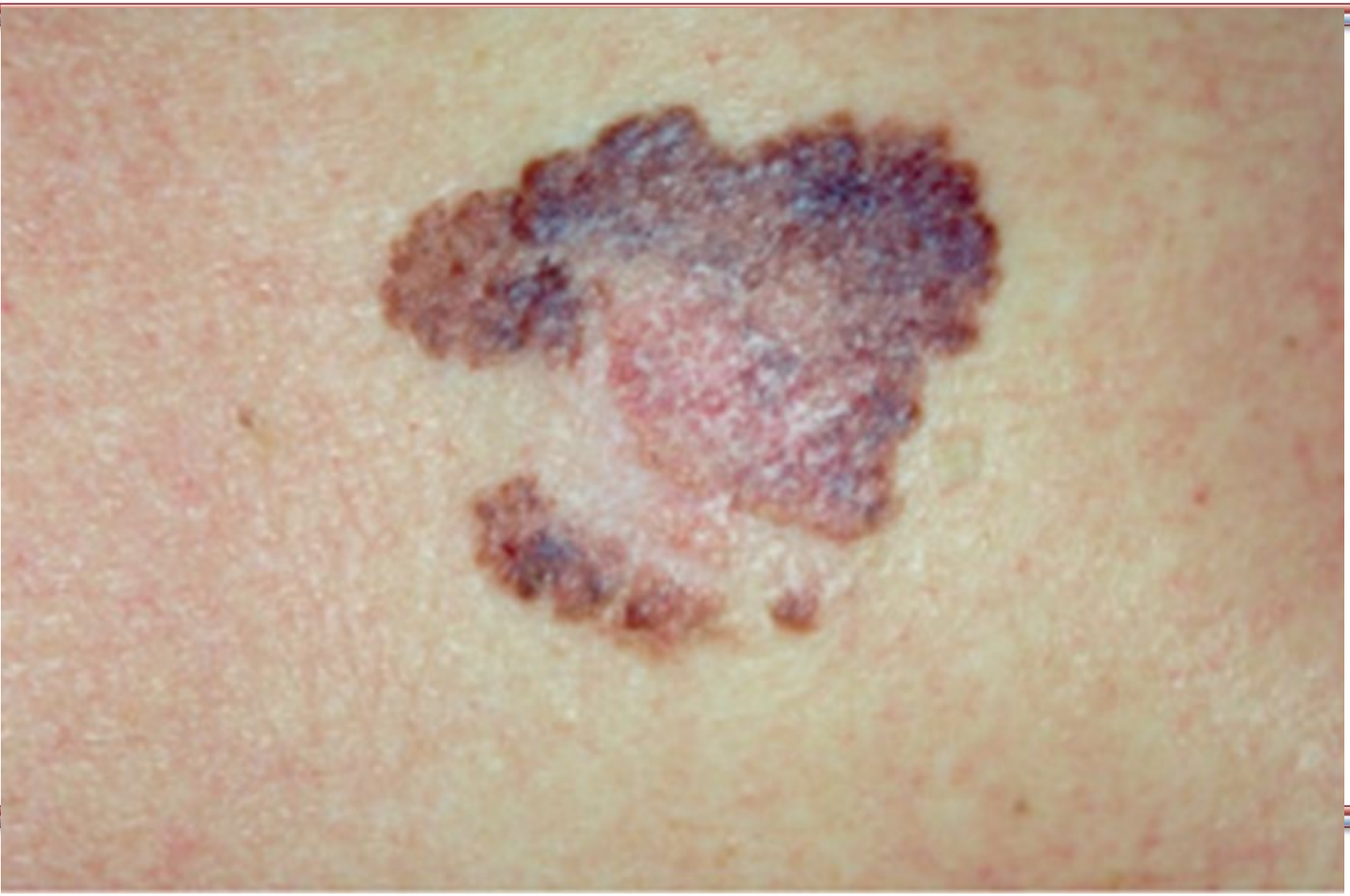
(skincancer.org, n.d.)

Melanoma



(Mammino, n.d.)

Melanoma



(VisualDx, n.d.)

Melanoma

- Treatment:
  - ❑ Excision/Mohs +/- SLNB
  - ❑ Radiation
  - ❑ Chemotherapy/checkpoint inhibitors

SURGICAL TREATMENT OF PRIMARY CUTANEOUS MELANOMA		
Tumor thickness	**Excision margins (cm)**	**Comments**
In situ	0.5	Lentigo maligna of the face may be excised with 1 cm margins (especially when lesions are >1.5–2 cm in diameter) or treated by Mohs micrographic surgery or radiotherapy; postoperative topical imiquimod is often used
≤1 mm	1.0	Mohs micrographic surgery may be considered for facial melanomas
1.01–2 mm	1.0–2.0	
>2 mm	2.0	

(Bolognia et al., 2018)

Key Takeaways

- The skin is an excellent indicator of the overall health of the body.
- Skin cancer is one of the most common forms of cancer in the U.S.
- While it is our “packaging” and its appearance is important, the skin is an active barrier against the outside, an important part of the immune system, and can be a significant site of disease.

References

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Questions?

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   b. Complete the Evaluation
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