Block N

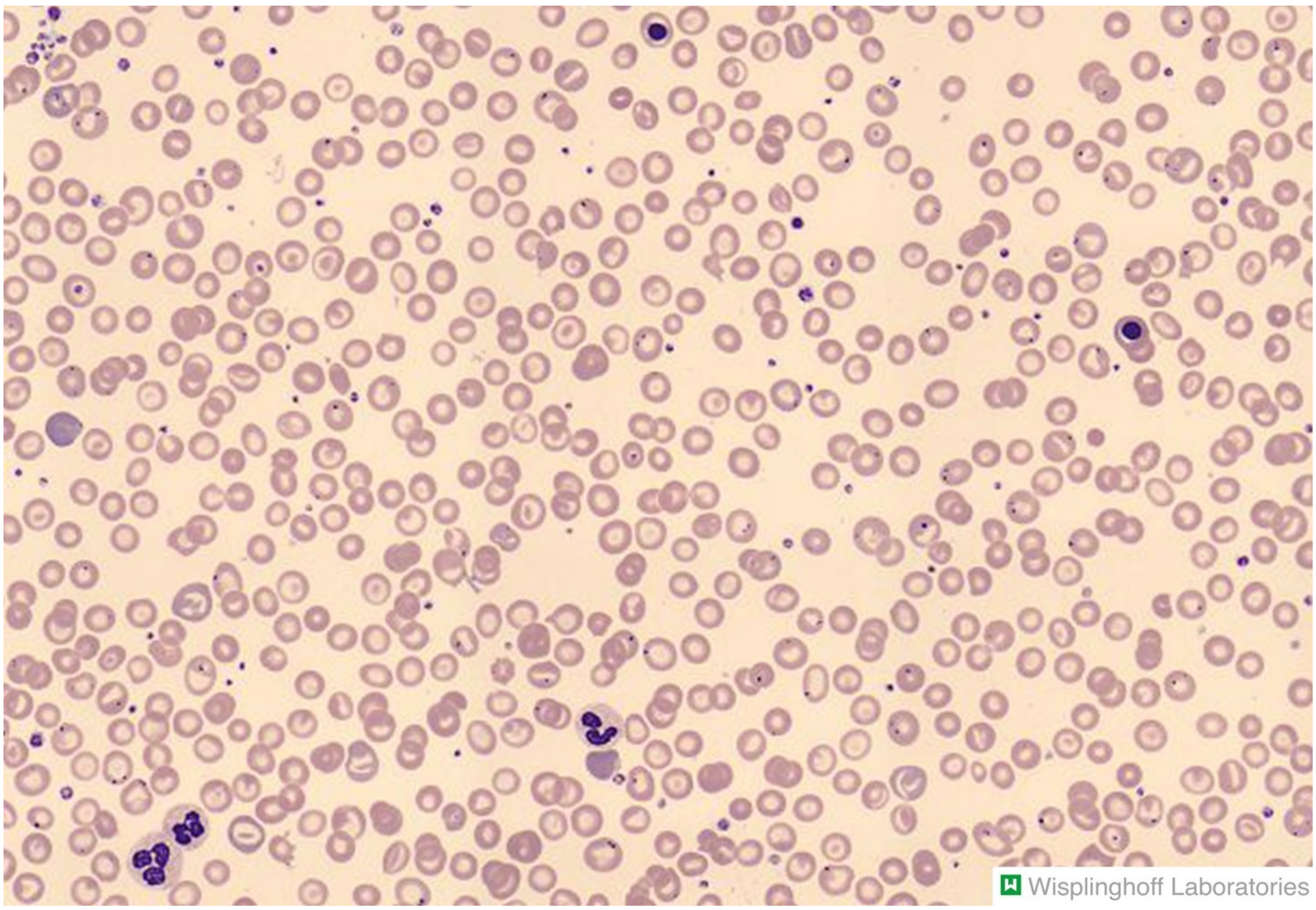
By Fatima Haider

KGMC - 5

Dysmorphic RBCs

* Dacryocytes → Teardrop cells
* Sickle cells
* Schistiocytes → Fragmented RBCs
* Macrocytes / Megalocytes → Large, Spherical
* Spherocytes → Small, spherical, no central pallor
* Elliptocytes → Oval or elliptical
* Echinocytes (Burr cells)
* Target cells → Bullseye appearance
* Acanthocytes (Spur cells)
* Stomatocytes → Slit-like central pallor
* Degmacytes (bite cells)

Target Cells

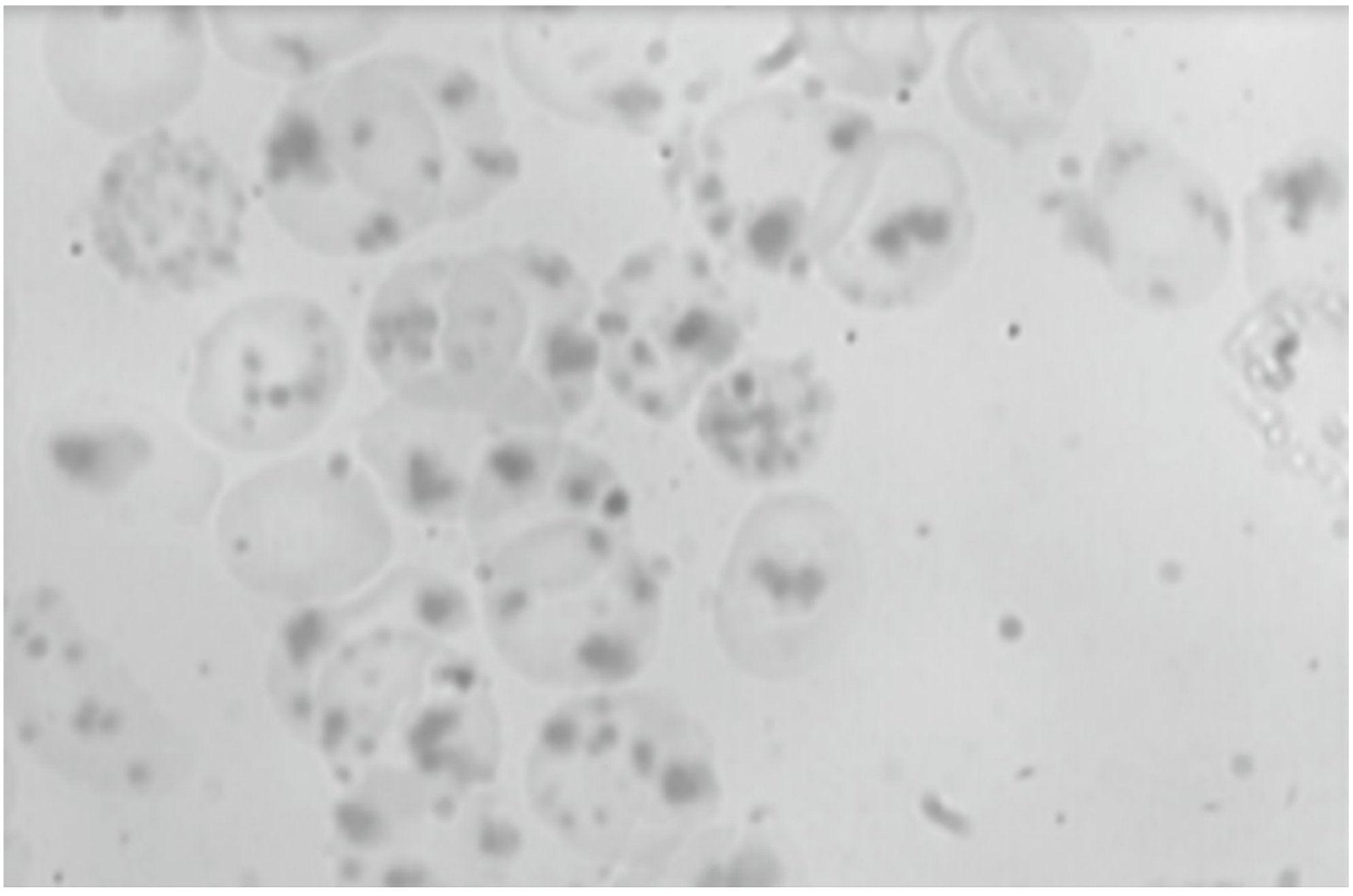


HALT
when u see a
TARGET

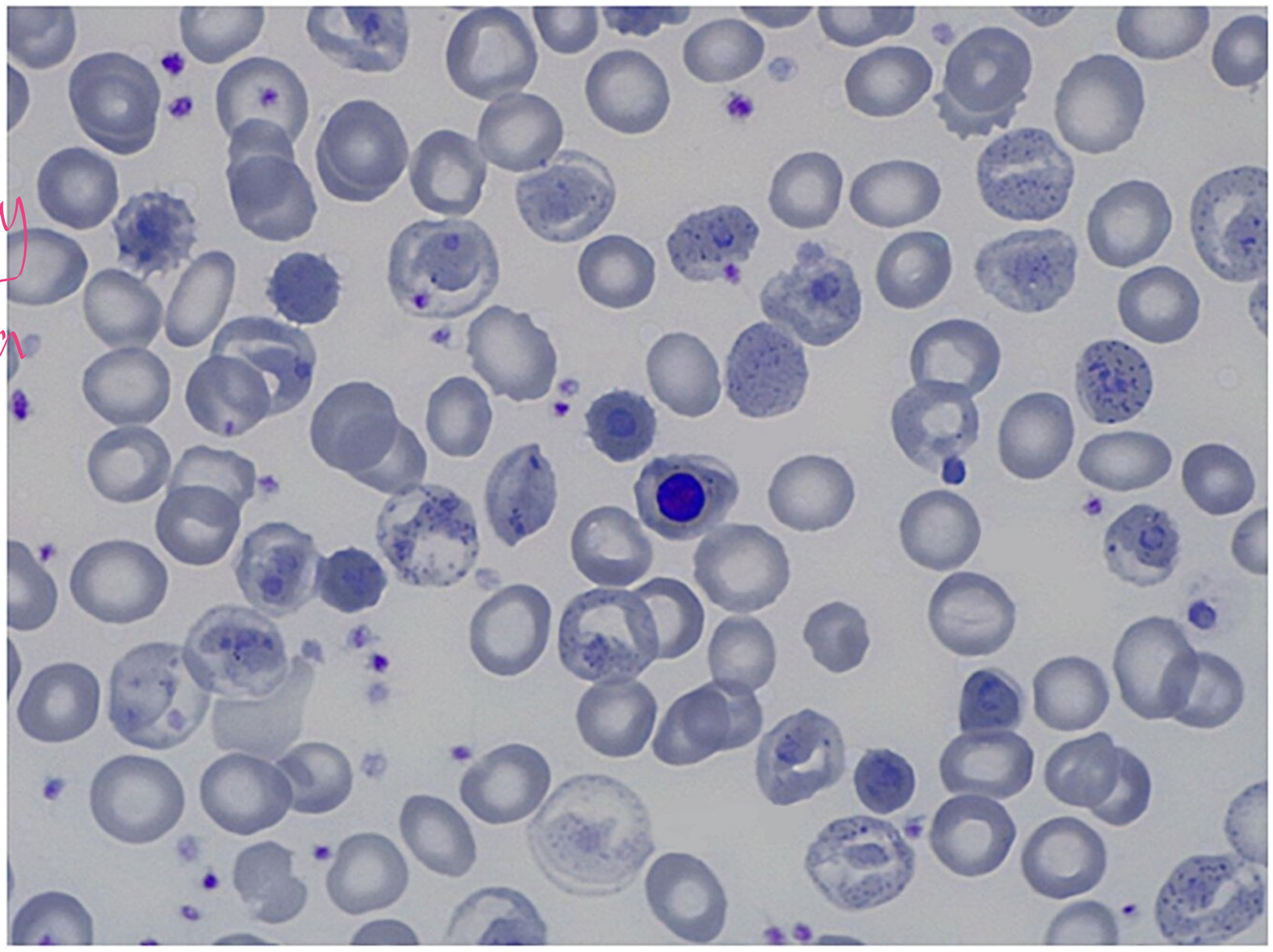
- Hemoglobinopathies
- Asplenia
- Liver disease
- Thalassemia

- Hemoglobinopathies
  - **Thalassemia**
  - Hemoglobin C and S
- Liver disease
- Asplenia
- LCAT deficiency
- Artifacts

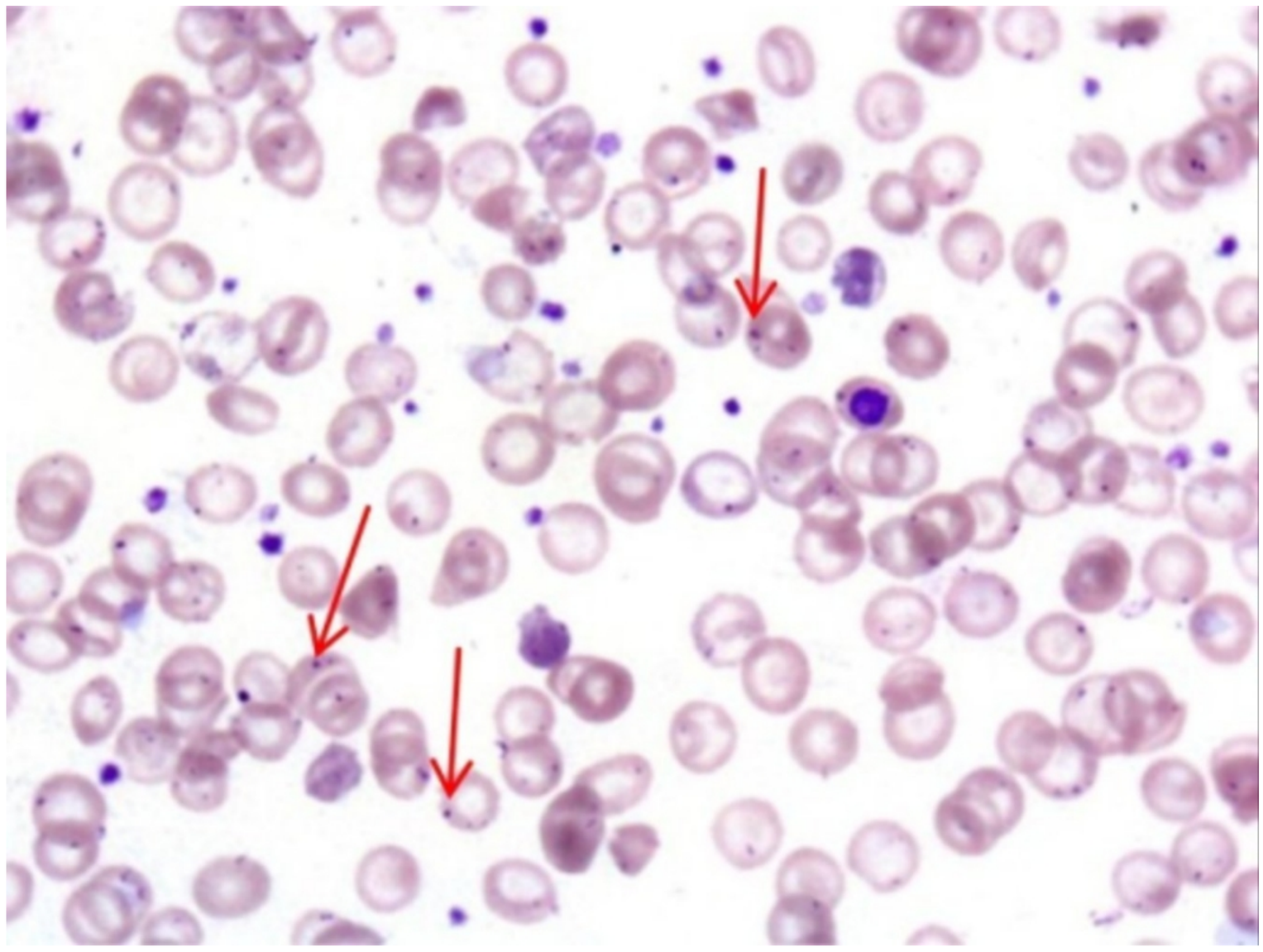
Heinz bodies



- G6PD deficiency
- Thalassemia

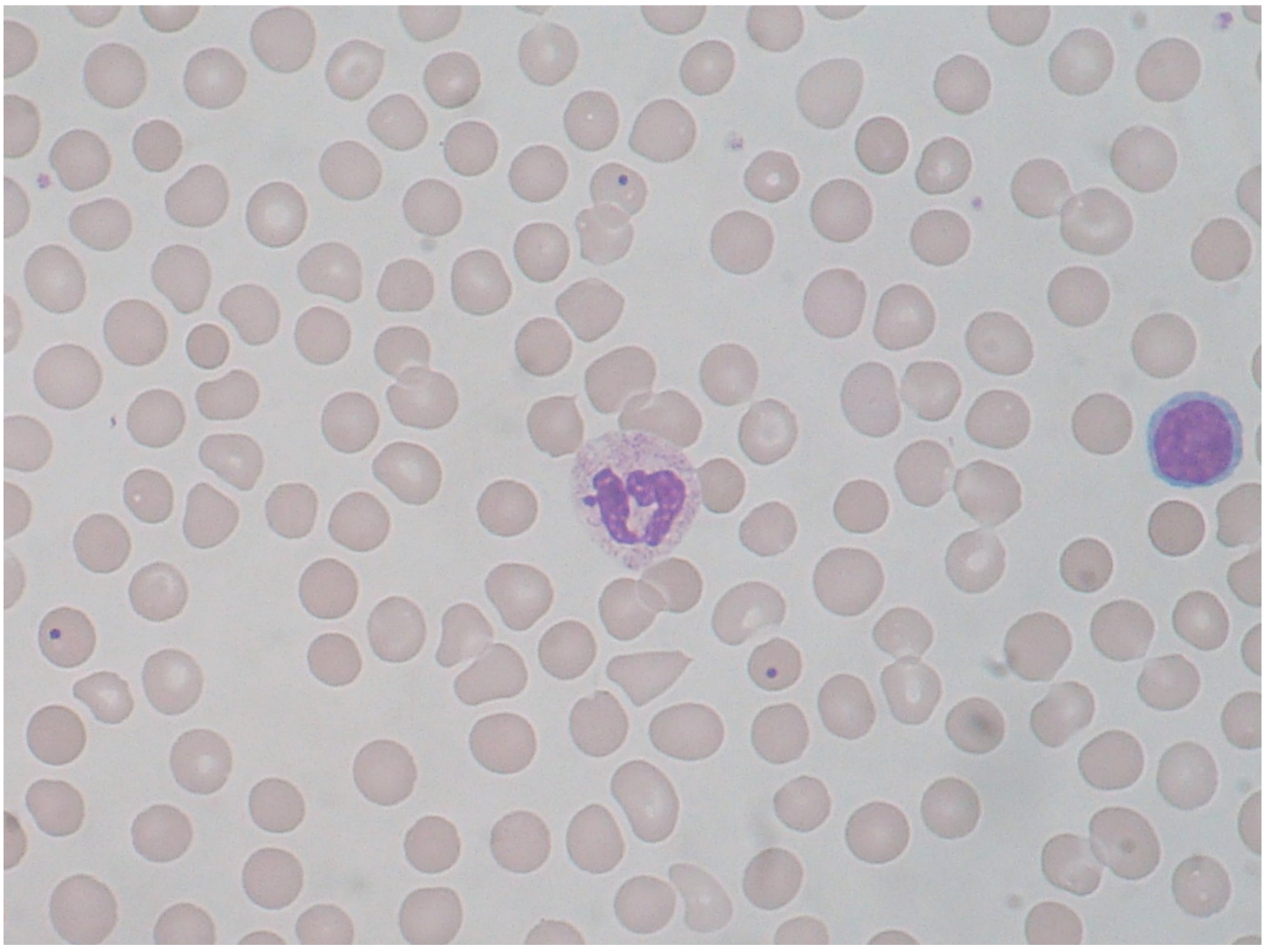


Howel Jolly bodies

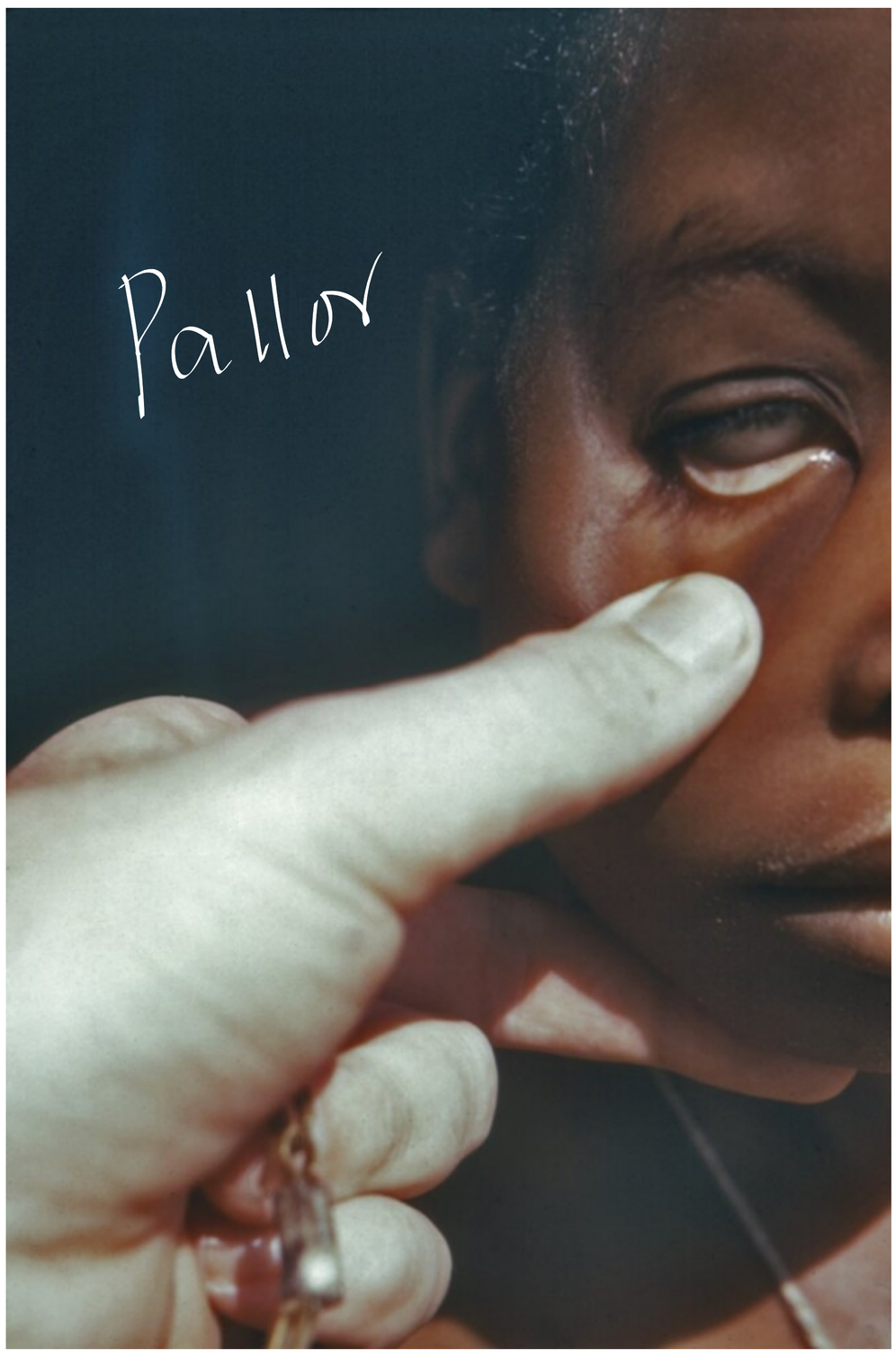


Asplenia

• DNA (nuclear remnants) • Do not contain iron	• A typically small, basophilic spot, located toward the periphery of the cell	• **Asplenia**



Iron Deficiency Anemia



Cyanosis

Glucose-6-phosphate dehydrogenase deficiency

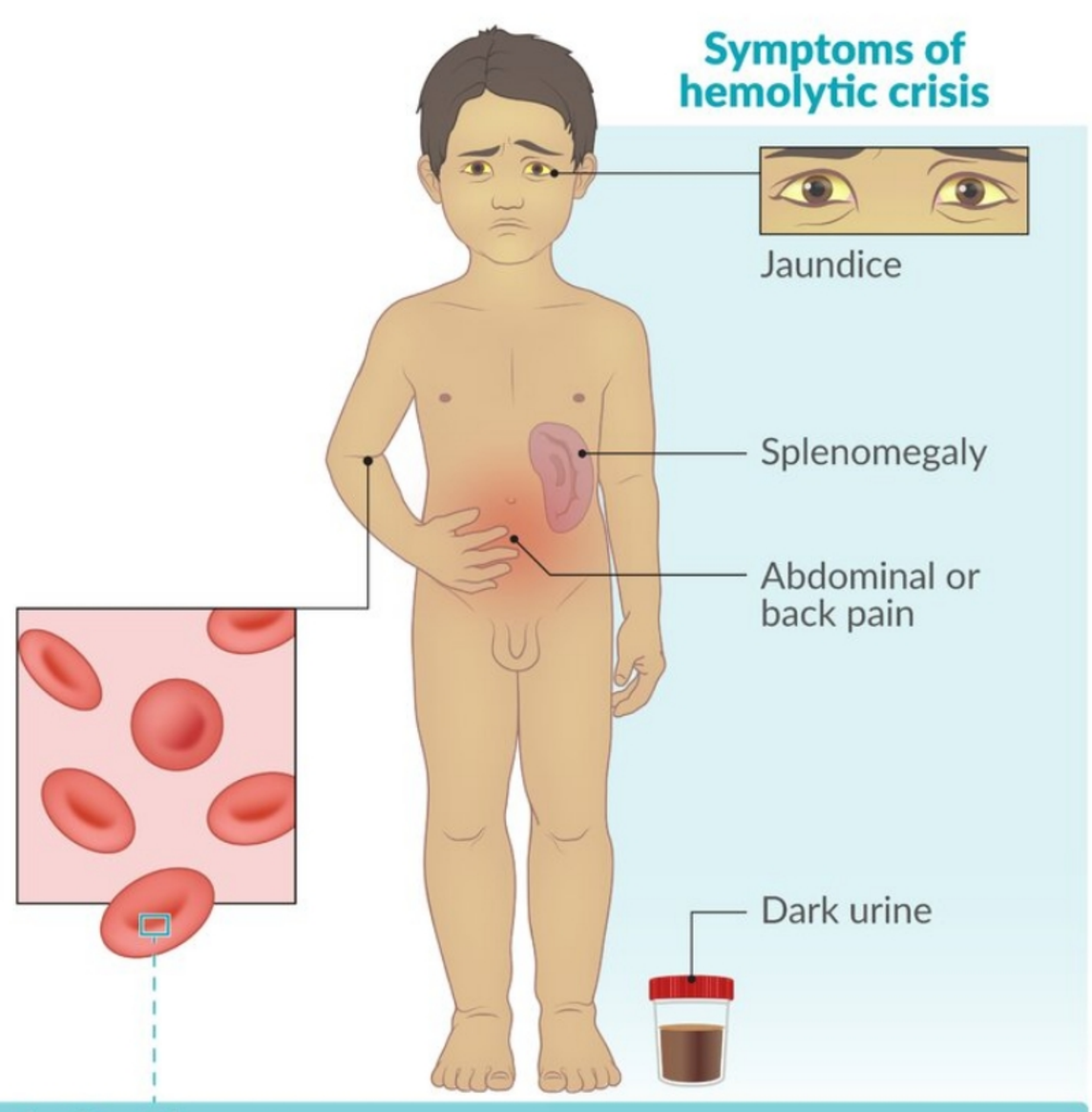
Epidemiology
Most commonly affects male individuals of African, Mediterranean, and Asian descent

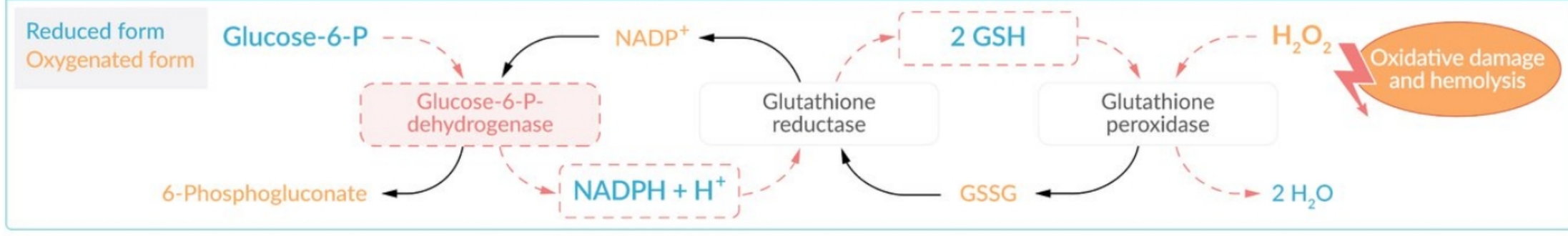
Inheritance
X-linked recessive

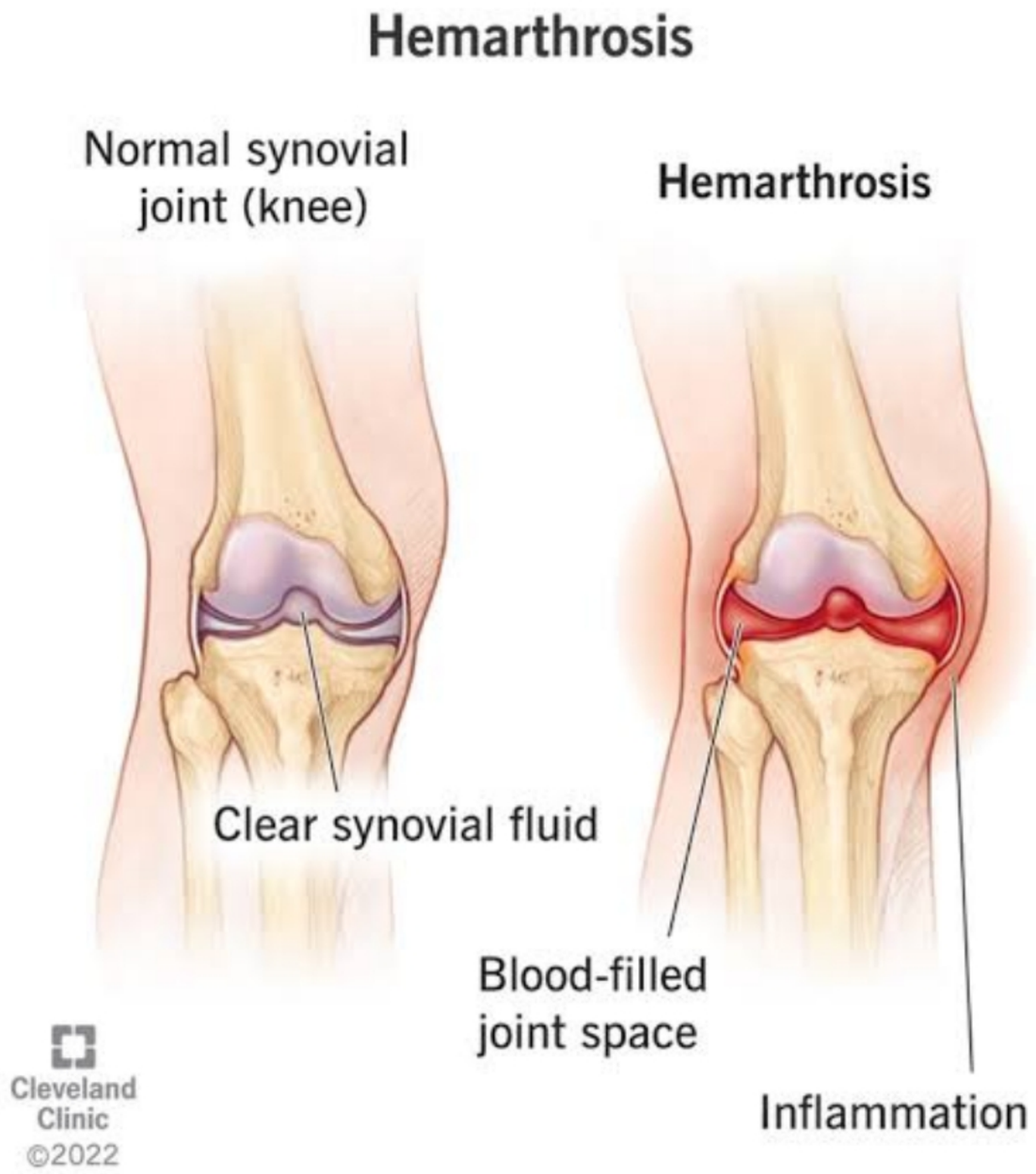
Triggers of hemolytic crisis
- Fava beans
- Infections
- Drugs that cause oxidative stress, e.g., antimalarials, sulfa drugs

Diagnostics
- Signs of hemolytic anemia (e.g., ↑ reticulocyte count, ↑ unconjugated bilirubin)
- Histology: bite cells, Heinz bodies
- Confirmatory test: quantitative G6PD enzyme analysis

Treatment
Avoidance of triggers

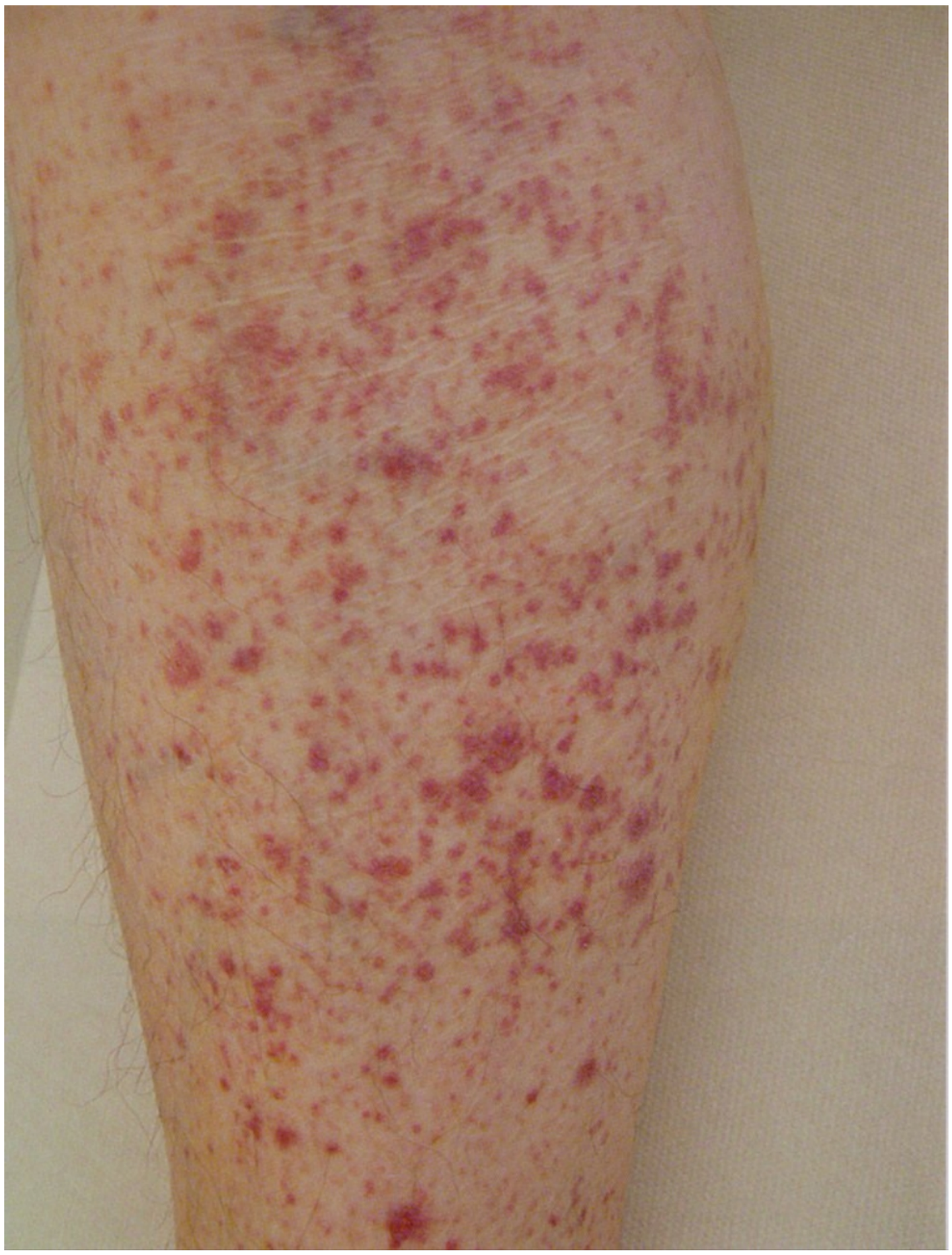






Activated partial thromboplastin time (aPTT): usually prolonged

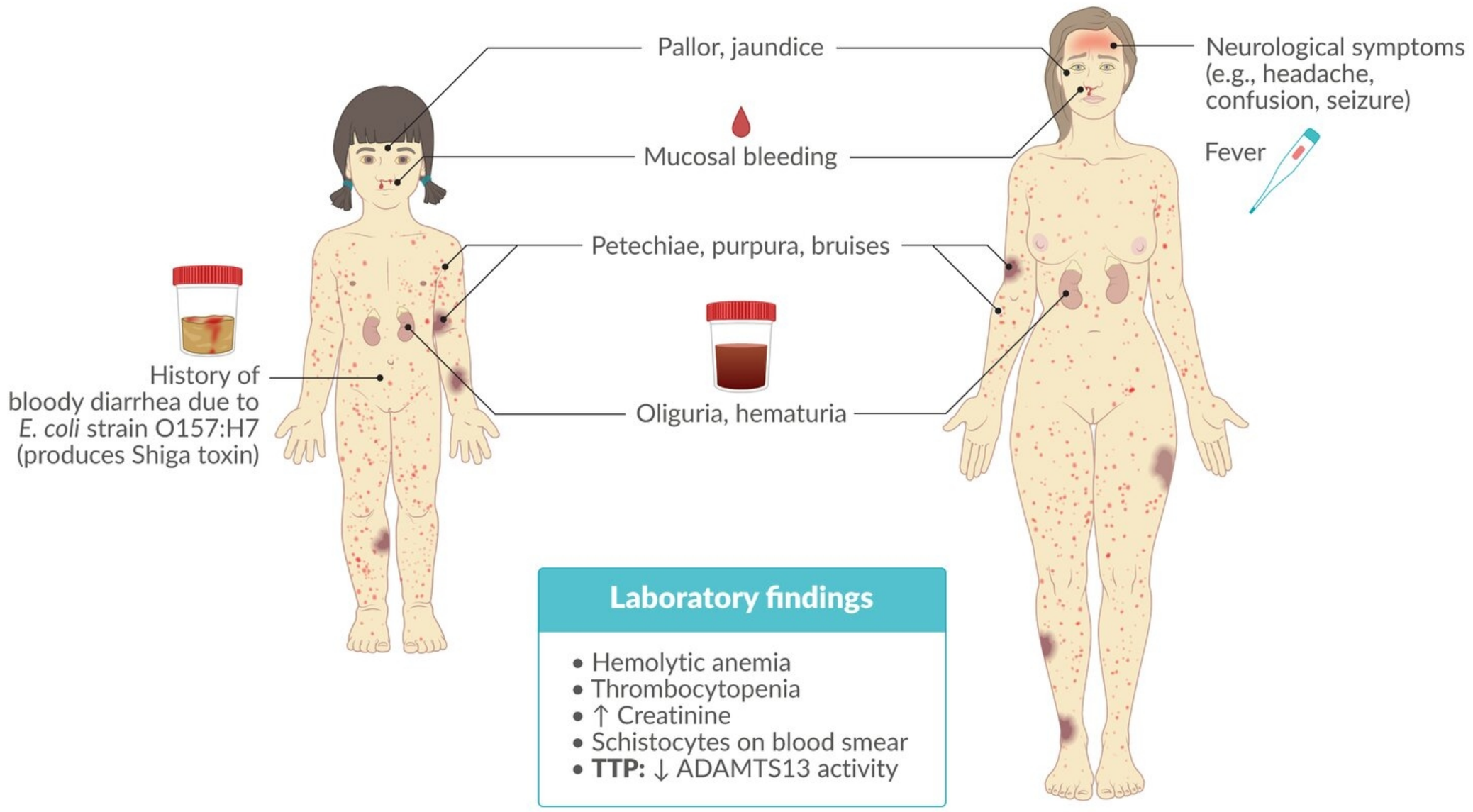
Hemarthrosis in Hemophilia



Petechial bleeding in Thrombocytopenia

Hemolytic uremic syndrome (HUS)

Thrombotic thrombocytopenic purpura (TTP)



Laboratory findings

- Hemolytic anemia
- Thrombocytopenia
- ↑ Creatinine
- Schistocytes on blood smear
- **TTP:** ↓ ADAMTS13 activity

Heparin-induced thrombocytopenia

Definition
An antibody-mediated response to heparin leading to thrombocytopenia and an increased risk of thrombosis

Epidemiology
Occurs in 0.1–7% of patients receiving heparin products

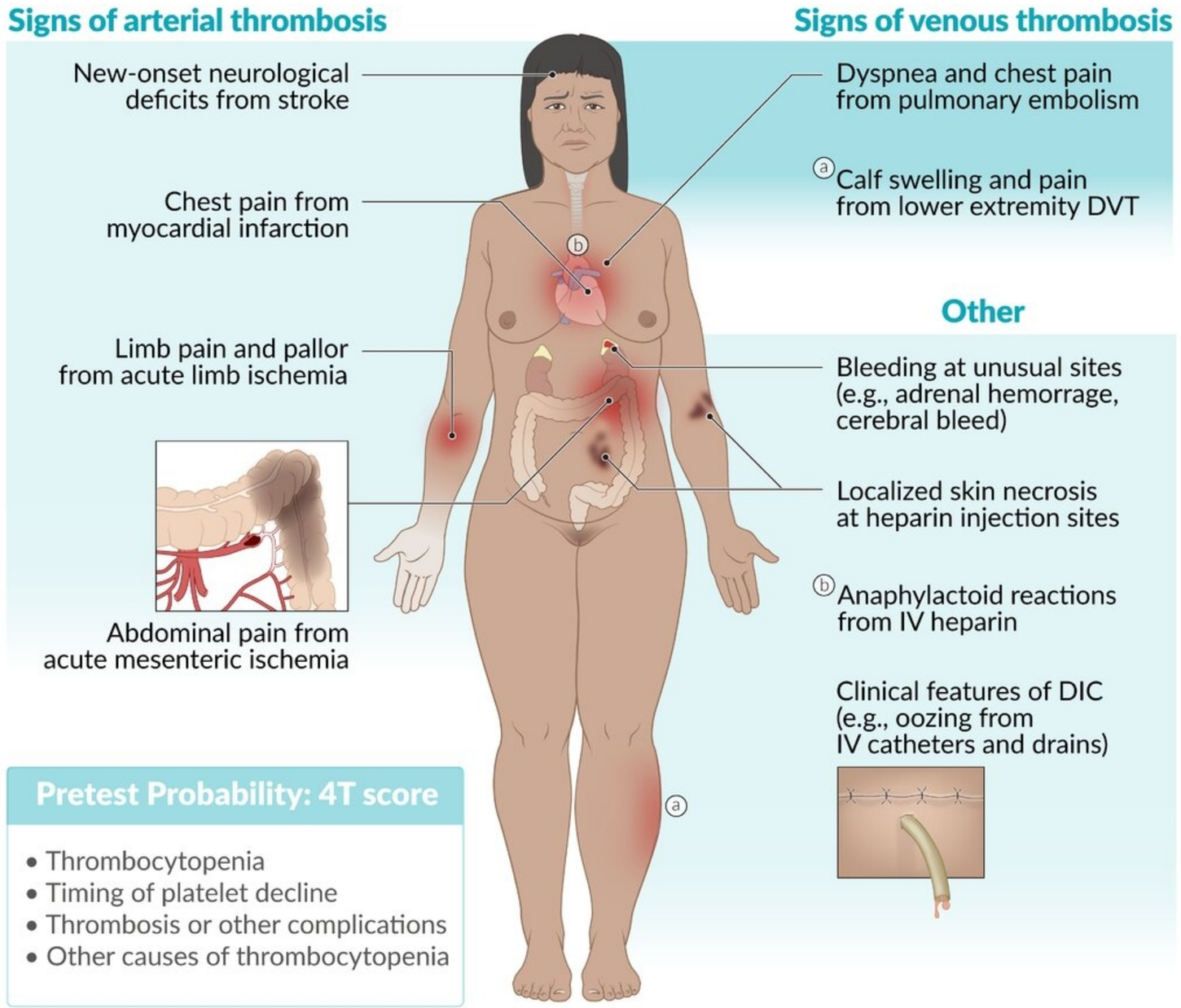
Risk factors
- Female sex
- Surgery
- Major trauma
- Use of unfractionated heparin
- Duration of heparin exposure (> 5 days)

Diagnostics
- CBC, peripheral blood smear
- Coagulation studies (PTT, INR, fibrinogen)
- Confirmatory studies (PF4 heparin immunoassay, functional platelet activation assay)

Treatment
- Stop all heparin exposure
- Initiate nonheparin anticoagulation
- Screen for thrombosis

Pretest Probability: 4T score

- Thrombocytopenia
- Timing of platelet decline
- Thrombosis or other complications
- Other causes of thrombocytopenia



Deep vein thrombosis (DVT) - There is diffuse edema and erythema of the right leg and foot. The diameter of the right calf is visibly greater than that of the left. These features are typical of DVT. Clinical history and investigations (e.g., Doppler ultrasonography, D-dimer levels) help distinguish between DVT and cellulitis, which also manifests with a painful, warm, erythematous, and edematous leg.

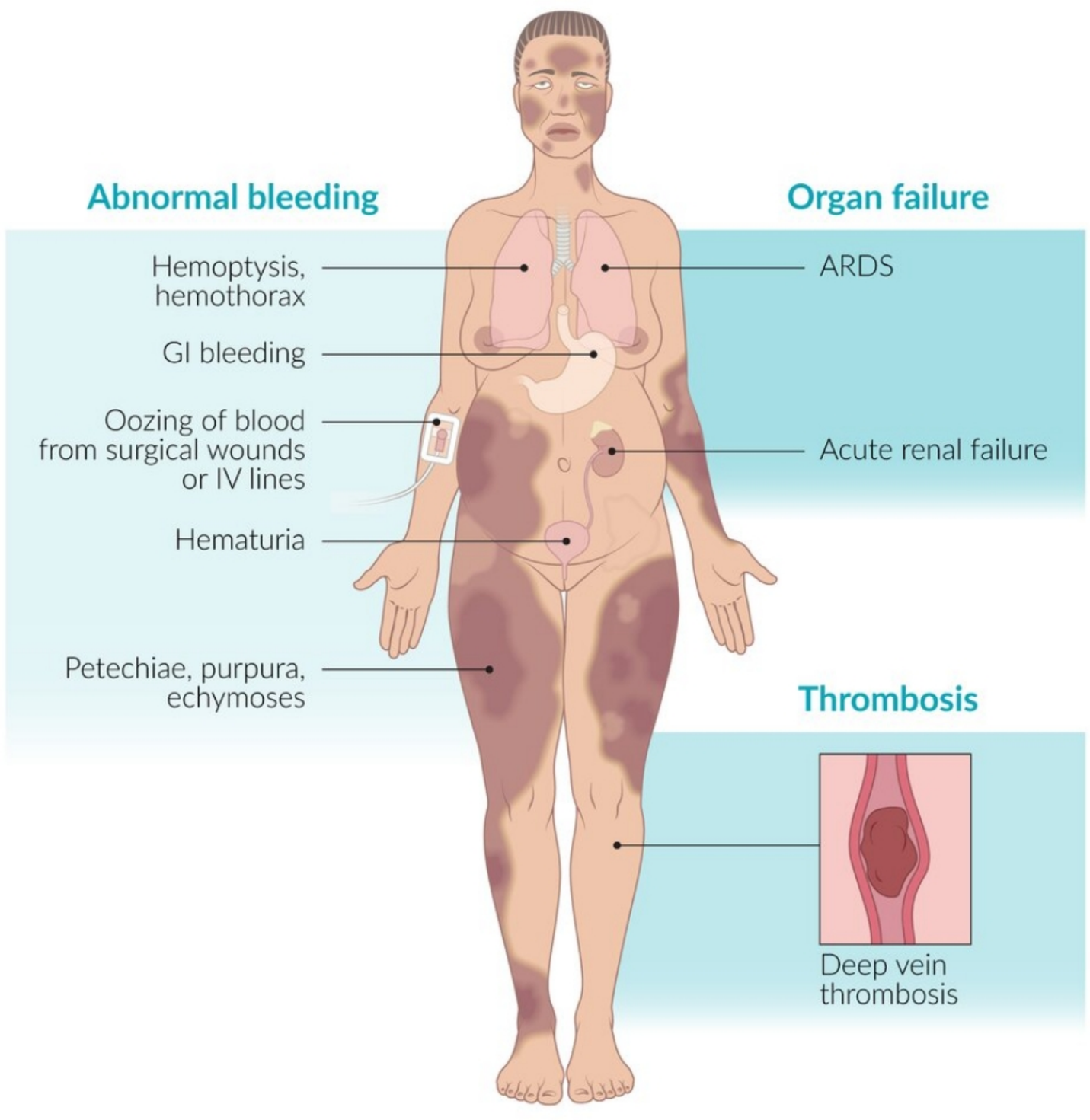
Disseminated intravascular coagulation

Definition
Thrombosis, hemorrhage, and organ dysfunction secondary to platelet consumption and exhaustion of clotting factors

Etiology
Sepsis and other severe infections, trauma, and obstetric complications

Diagnostics
No specific criteria
Clinical findings: abnormal bleeding, organ failure, thrombosis
Laboratory findings: Thrombocytopenia, ↑ D-dimer, ↑ PT and aPTT, ↓ fibrinogen

Management
Stabilize patient as needed.
Start urgent treatment of the underlying cause.
Additional measures may include administration of blood products, anticoagulation, or vitamin K



Warfarin Induced Skin Necrosis

Presentation: painful purpura, hemorrhagic blisters, and large areas of necrosis; mostly affects subcutaneous adipose tissue

Acute Leukemia

Clinical features are either related to bone marrow failure, infiltration of organs by leukemic cells, or a combination of both.

General features of acute leukemia

- Sudden onset of symptoms and rapid progression (days to weeks)
- **Anemia:** fatigue, pallor, weakness
- **Thrombocytopenia:** epistaxis, bleeding gums, petechiae, purpura
- Immature leukocytes: **frequent infections**, fever
- **Hepatosplenomegaly** (caused by leukemic infiltration) [2]
- Oncologic emergencies can be the first sign of leukemia, e.g., an elderly patient presenting with priapism or DIC may have **leukostasis** (more common in AML than ALL): See oncologic emergencies for further details.

Clinical features of ALL	Clinical features of AML
• **Fever**, night sweats, unexplained weight loss • **Painless lymphadenopathy** • **Bone pain** (presenting as limping or refusal to bear weight in children) • Airway obstruction (stridor, difficulty breathing) due to mediastinal or **thymic infiltration** (primarily in T-cell ALL) [15][16] • Features of SVC syndrome • **Meningeal leukemia** (or leukemic meningitis) → headache, neck stiffness, visual field changes, or other CNS symptoms (caused by CNS involvement) [1][16] • Testicular enlargement (rare finding)	• **Leukemia cutis** (or myeloid sarcoma): nodular skin lesions with a purple or gray-blue color • Gingival hyperplasia (AML subtype M4 and M5) • Signs of CNS involvement, e.g., headache, visual field changes (uncommon)

Fever and lymphadenopathy are rare in AML, but can be common first signs in ALL!

Fever in a patient with acute leukemia must always be treated as a sign of infection until proven otherwise!

Remember metastasis for ALL by thinking of the following: ALL meta**S**ta**S**ize**S** to the CN**S** and te**S**te**S**.

Common agents used in chemotherapy regimens for acute leukemia [18][20][25][32][33]	
ALL	• Alkylating agents: e.g., cyclophosphamide • Anthracyclines: e.g., daunorubicin, doxorubicin • Antimetabolites: e.g., cytarabine, methotrexate, 6-mercaptopurine • Enzyme therapy: e.g., L-asparaginase • Alkaloids: e.g., vincristine • Glucocorticoids: e.g., prednisone, dexamethasone
AML	• Anthracyclines (e.g., idarubicin, high-dose daunorubicin) • Antimetabolites (e.g., **cytarabine**, methotrexate) • Hypomethylating agents (e.g., azacitidine)
APL	• Differentiation agents: to induce the maturation of immature malignant WBCs ◦ **ATRA (All-trans retinoic acid)** ◦ **Arsenic trioxide** • Chemotherapy with anthracyclines (e.g., idarubicin)

In APL, the **t(15;17) translocation** and subsequent formation of the *PML-RARA* fusion gene can inhibit myeloblast differentiation under physiological levels of retinoic acid. **High doses of ATRA (a vitamin A derivative)** may induce myeloblast differentiation and **promote remission.**

Splenic rupture due to splenomegaly

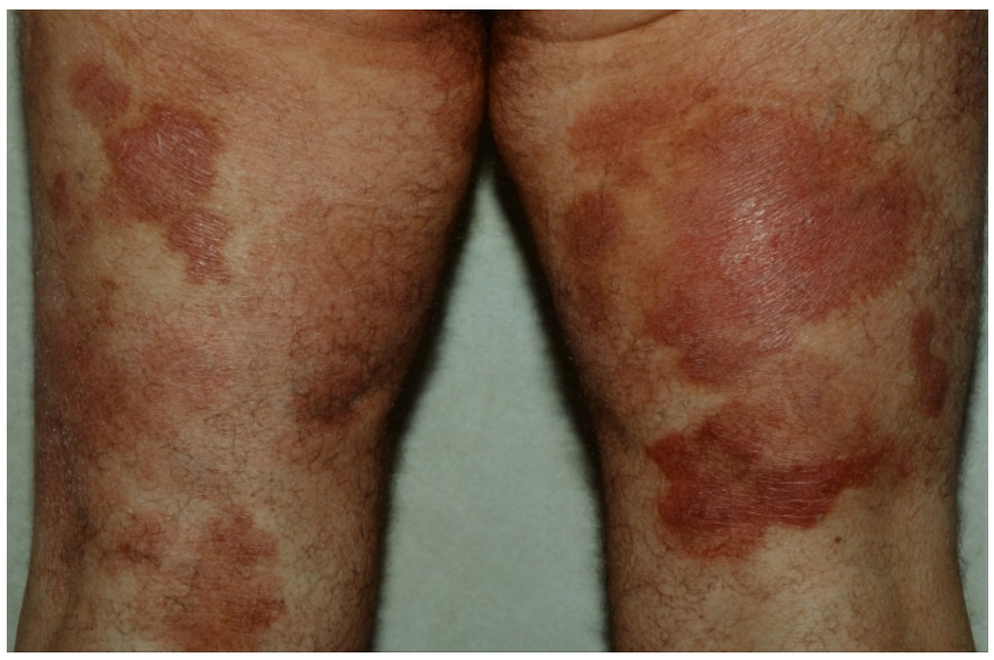
Hodgkin Lymphoma

Clinical features

- **Painless** lymphadenopathy
  - First develops in a single group of lymph nodes, typically **cervical**, supraclavicular and/or mediastinal lymph nodes [5][6][7]
  - Spreads contiguously through the lymphatic system; cervical nodes → supraclavicular lymph nodes → axillary nodes → inguinal nodes [8]
- Splenomegaly **or** hepatomegaly [9]
- **B symptoms** [10]
  - **Night sweats**, **weight loss** > 10% in the past 6 months, **fever** > 38°C (100.4°F)
  - Can occur in a variety of diseases (see "Differential diagnosis of B symptoms" below)
  - In the case of confirmed HL, the presence of a single B symptom suffices for a positive diagnosis of B symptoms.
- **Pel-Ebstein fever**: intermittent fever with periods of high temperature for 1–2 weeks, followed by afebrile periods for 1–2 weeks; relatively rare but very specific for HL
- **Alcohol-induced pain**: pain in involved lymph nodes after ingestion of alcohol; relatively rare but highly specific for HL
- Pruritus: focal or generalized

Cutaneous T cell Lymphoma

Mycosis fungoides



- **Treatment for early stage disease** [7]
  - **Topical corticosteroids** (first-line)
  - Topical nitrogen mustard (second-line)
- **Treatment for moderate to late stage disease** [8]
  - Total skin electron beam therapy (TSEBT)
  - Systemic chemotherapy (e.g., bexarotene)
  - Topical corticosteroids may also be used.

- **Multiple myeloma**: a malignant plasma cell dyscrasia characterized by uncontrolled proliferation and the diffuse infiltration of monoclonal plasma cells in the bone marrow

Classification

- **Based on immunoglobulin type** [2]
  - IgG and IgA: typical multiple myeloma; majority of patients
  - Bence Jones myeloma (free light chains excreted in urine): 15–20% of multiple myelomas
  - IgD, IgE, and IgM: very rare subtypes of multiple myelomas

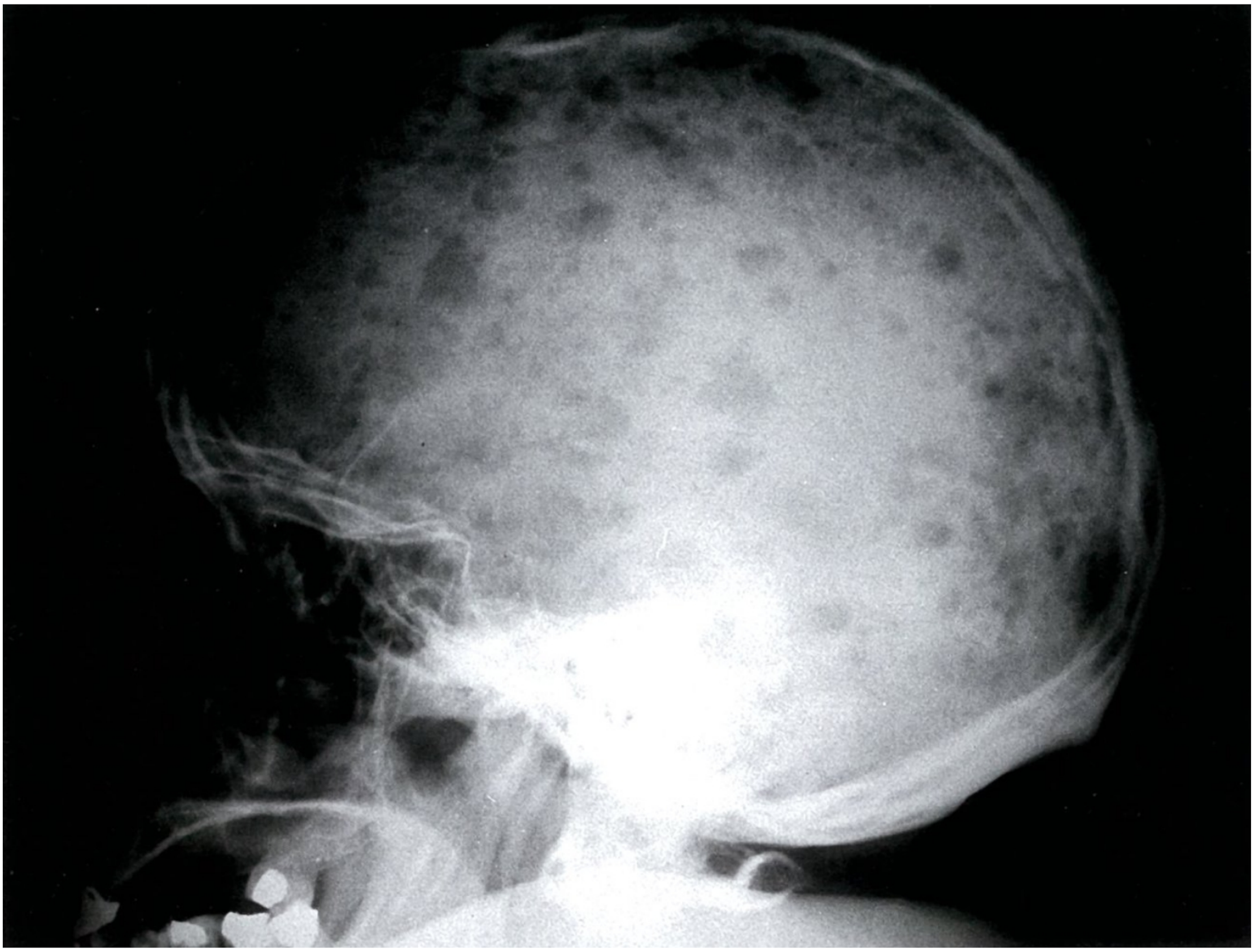
Pathophysiology

- Neoplastic proliferation of **plasma cells**
  - Bone marrow infiltration by malignant plasma cells → suppression of hematopoiesis → leukopenia, thrombocytopenia, anemia
  - Cell proliferation → pro-osteoclastogenic factors (e.g., TNF-α, IL-1, RANK-L) → osteolytic lesions → hypercalcemia
- Overproduction of **monoclonal immunoglobulin** and/or light chains → dysproteinemia (a state of pathologically increased synthesis of immunoglobulins and/or their subunits) → kidney damage (e.g., myeloma cast nephropathy) and/or paraprotein tissue deposition (may cause amyloidosis) [3] [4]
  - Nonfunctioning antibodies → functional antibody deficiency
  - ↑ Serum viscosity → hyperviscosity syndrome

Clinical features

- Often asymptomatic
- Mild fever, night sweats, weakness, and weight loss
- Bone pain, especially **back pain** (most common symptom)
- Symptoms of hypercalcemia
- Spontaneous fractures
- Increased risk of infection
- Increased risk of petechial bleeding
- Foamy urine (caused by Bence Jones proteins in urine)

Multiple Myeloma



Multiple lytic lesions, punched-out holes in the skull seen on X-Ray skull

Osteolytic Lesions
in multiple myeloma

Sickle Cell Disease

Acute manifestations

- **Vaso-occlusive events**
  - Dactylitis in **children < 5 years of age** [6]
    - Typically the earliest manifestation of sickle cell disease
    - Most common in children between 6 months and 2 years of age; uncommon in older children and adults
  - Vasoocclusive crises (sickle cell pain crisis) [1][7]
    - Most common acute complication of sickle cell disease
    - Characterized by recurrent episodes of severe throbbing or sharp pain
    - Typically affects the limbs, chest, and back and lasts for ~ 7 days
    - Often associated with other vasoocclusive events (especially dactylitis in children)
  - Acute chest syndrome
  - Priapism
  - Stroke (common in children)
  - Sickle cell hepatopathy
  - Organ infarctions (any organ; particularly the spleen)
  - Avascular necrosis
- **Infection**
  - Pneumonia
  - Meningitis
  - **Osteomyelitis** (most common cause: ***Salmonella* spp.**, *Staphylococcus aureus*)
  - **Sepsis** (most common cause: ***Streptococcus pneumoniae***)
- **Acute hemolytic crisis**
  - **Splenic sequestration**
  - **Aplastic crisis**

- **Peripheral blood smear** findings may include:
  - Crescent-shaped sickled RBCs (drepanocytes or **sickle cells**)
  - Target cells
  - **Howell-Jolly bodies**: occur with splenic dysfunction
  - Reticulocytosis: indicates the presence of hemolysis

Chronic manifestations

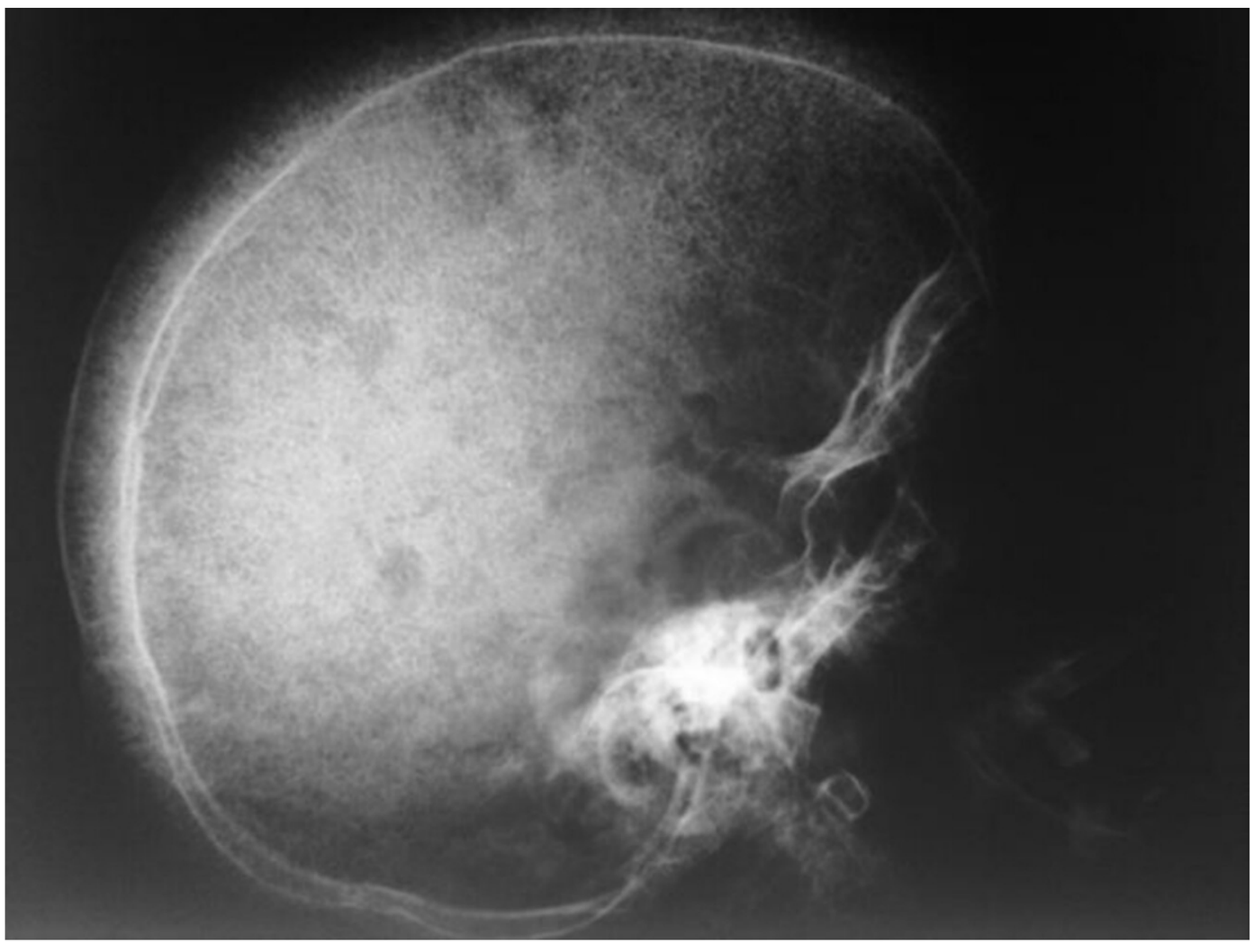
- Chronic hemolytic anemia: fatigue, weakness, pallor; usually well-tolerated
- Chronic pain
- Cholelithiasis (pigmented stones)

Symptoms of other forms of sickle cell syndrome (HbSC disease and HbS/beta-thalassemia) are similar to sickle cell disease but less severe.

→ Dactylitis in Sickle cell disease

Dactylitis - The entire left index finger of this patient with psoriatic arthritis is swollen. This appearance is typical of dactylitis, which can be caused by a variety of conditions, including spondyloarthropathies, infections, and sickle cell disease.

Seen in Thalassemia and Sickle cell anemia



Skull x-ray [14]

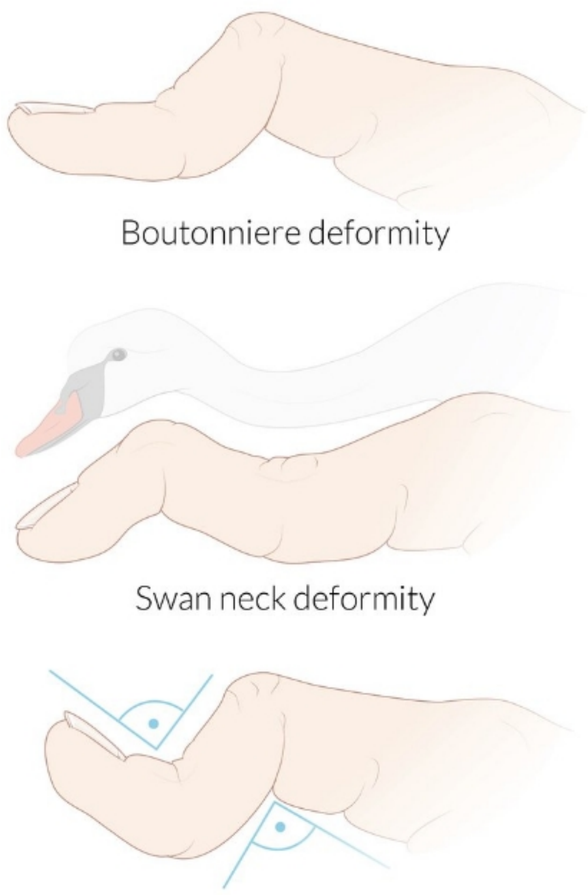
- May show hair-on-end (“crew cut”) sign
- Caused by periosteal reaction to erythropoietic bone marrow hyperplasia

Rheumatoid Arthritis

- Rheumatoid hand is characteristic and typically manifests with one or more of the following deformities:
  - Deepening of the interosseous spaces of the dorsum of hand
  - Swan neck deformity: PIP hyperextension and DIP flexion
  - Boutonniere deformity: PIP flexion and DIP hyperextension.
  - Hitchhiker thumb deformity (Z deformity of the thumb): hyperextension of the interphalangeal joint with fixed flexion of the MCP joint [13]
  - Ulnar deviation of the fingers
  - Piano key sign: dorsal subluxation of the ulna

Rheumatoid Nodules

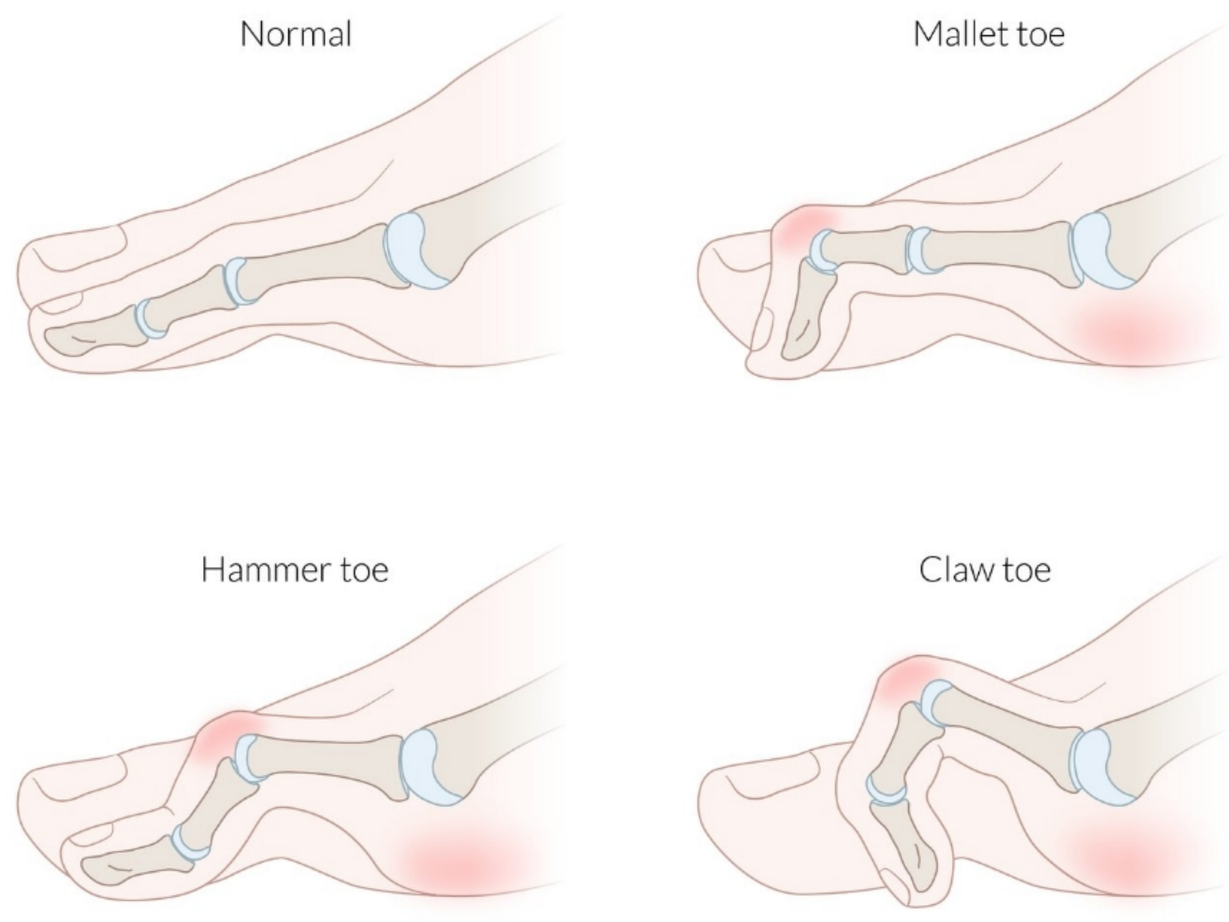
Swan Neck Deformity ↓



Boutonniere deformity ↑

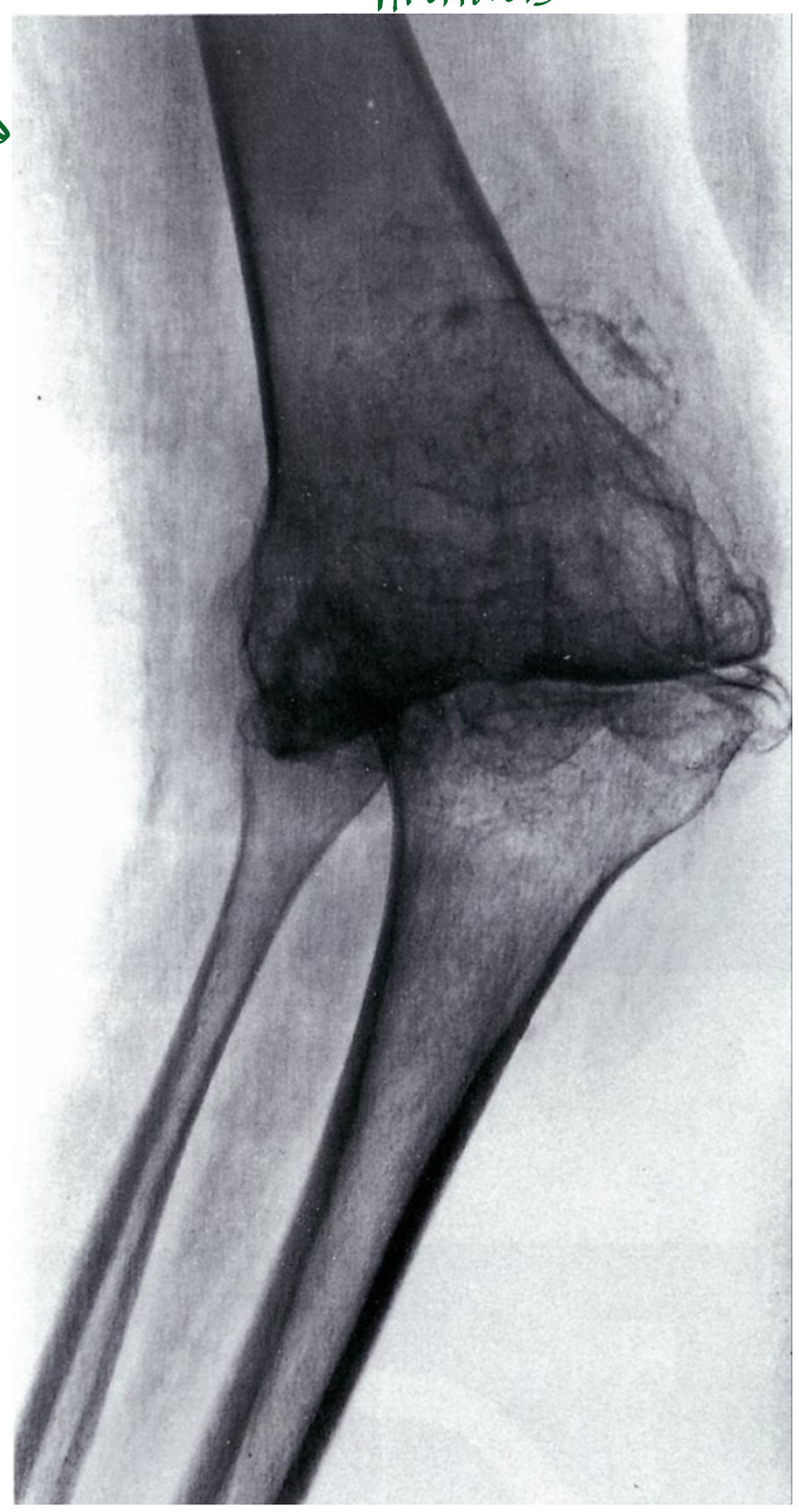
Rheumatoid Arthritis

  - Hammer toe or claw toe
  - **Atlantoaxial subluxation** (see "Rheumatoid arthritis of the cervical spine" below)
- Physical examination: compression test (Gaenslen squeeze test)
  - Painful compression of hands (or feet) at the level of the MCP joint (metatarsophalangeal joint)
  - Painful handshake is an early sign of arthritis



MRI cervical Spine
Showing atlanto axial subluxation
in Rheumatoid Arthritis

X-Ray knee showing knee
joint destruction in Rheumatoid
Arthritis



Popliteal (Baker) Cyst →


Podagra


Podagra


Soft tissue tophi

GOUT


Gouty tophi


Gouty tophi of hands


Gouty tophi

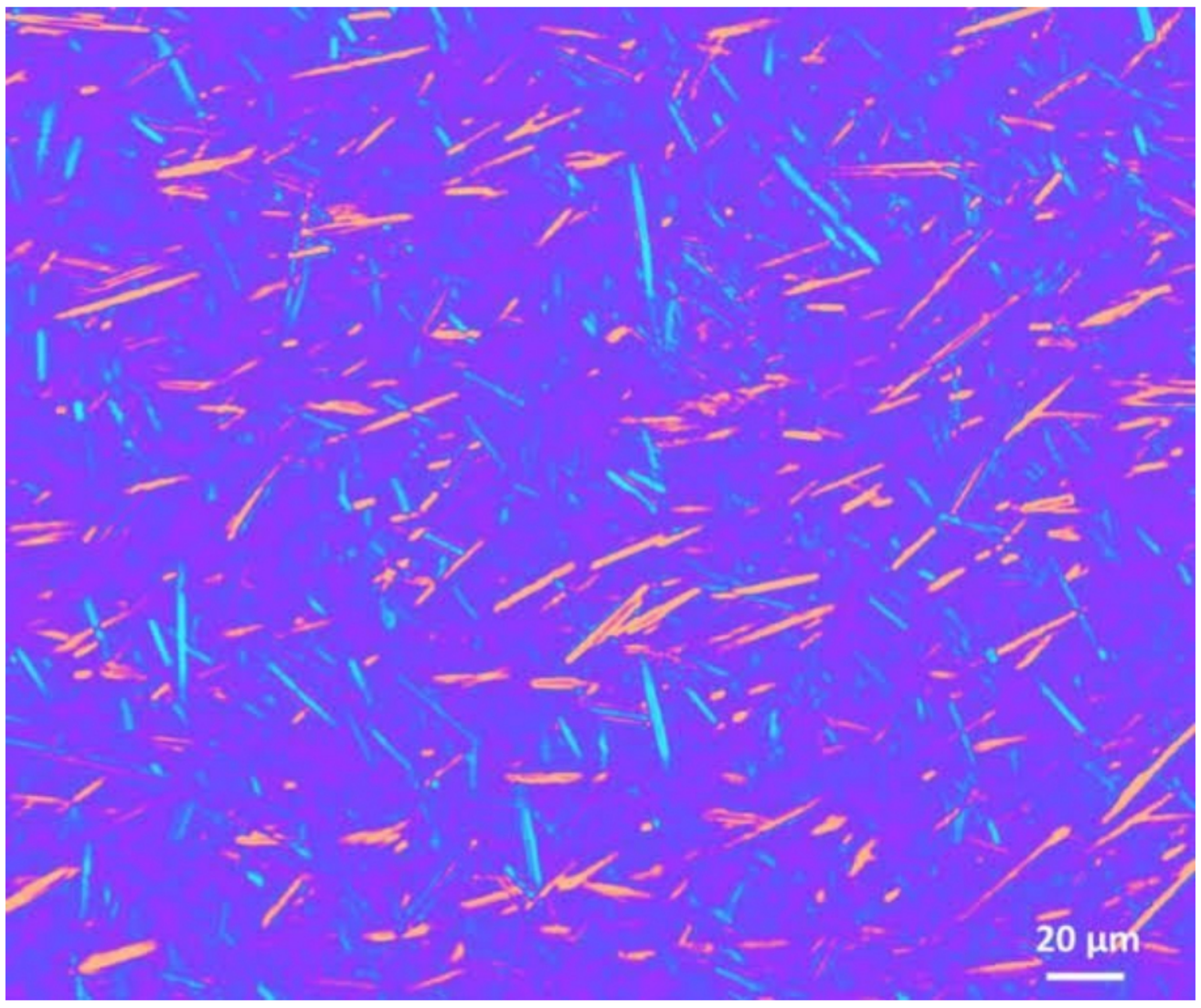

Mono arthritis in acute gouty arthritis


Gouty Tophi

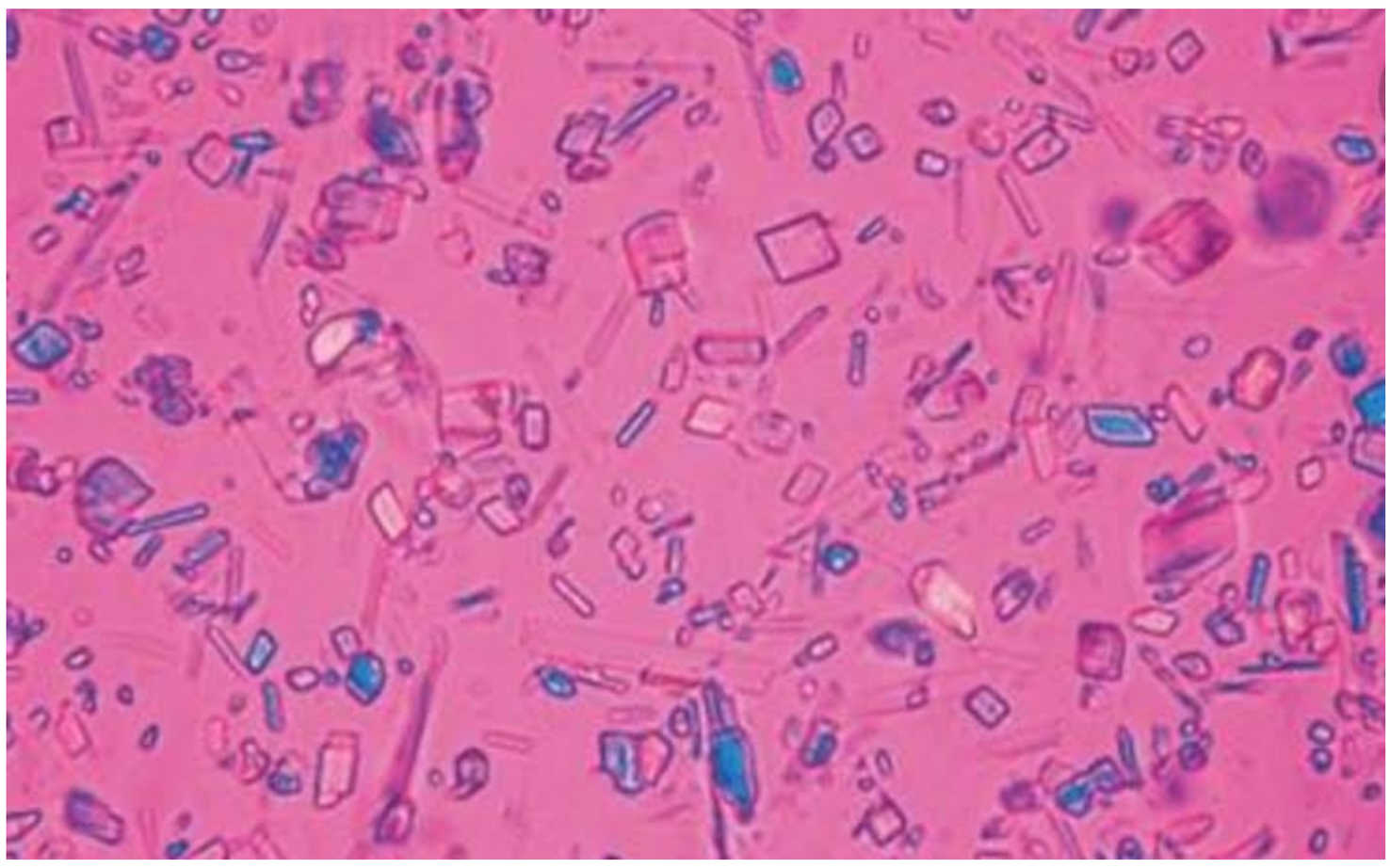
- Deposition of mono Sodium urate (MSU) crystals
- Joint Aspiration is the most accurate test

Gouty tophi

Podagra X-Ray foot

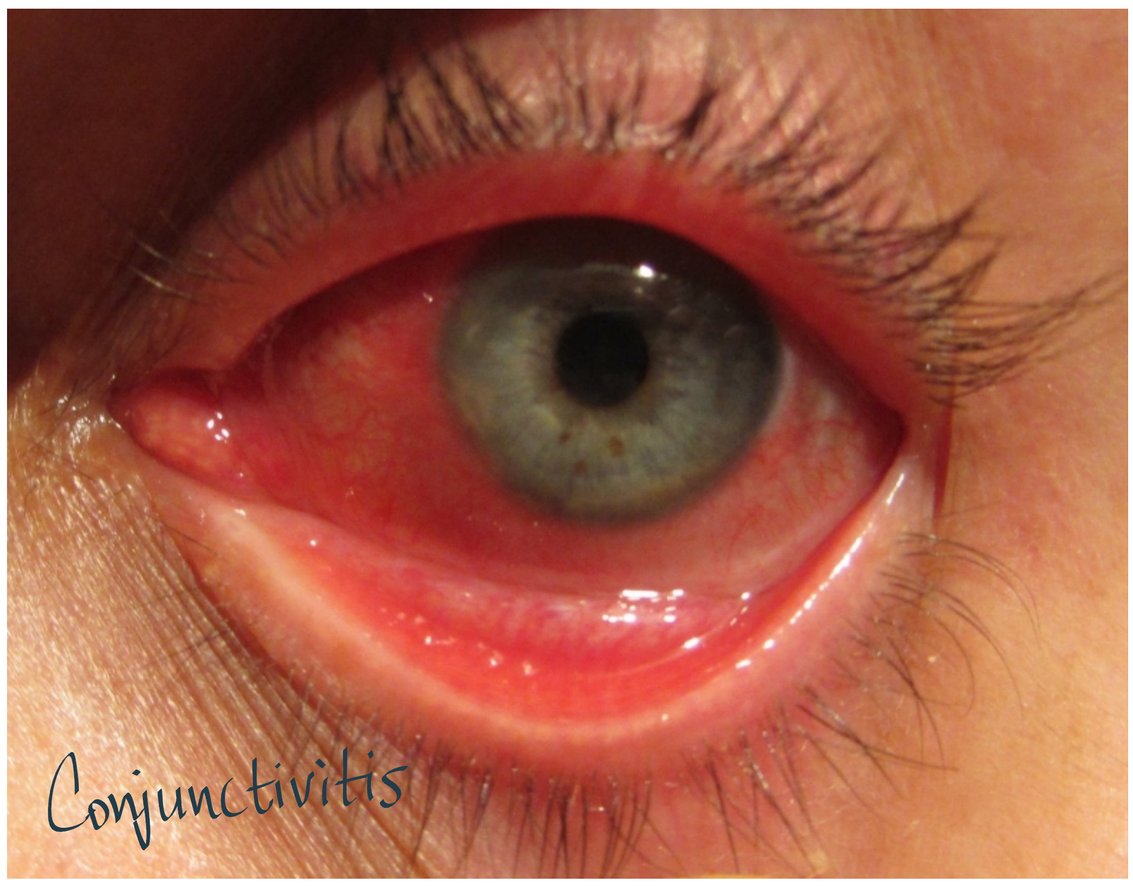


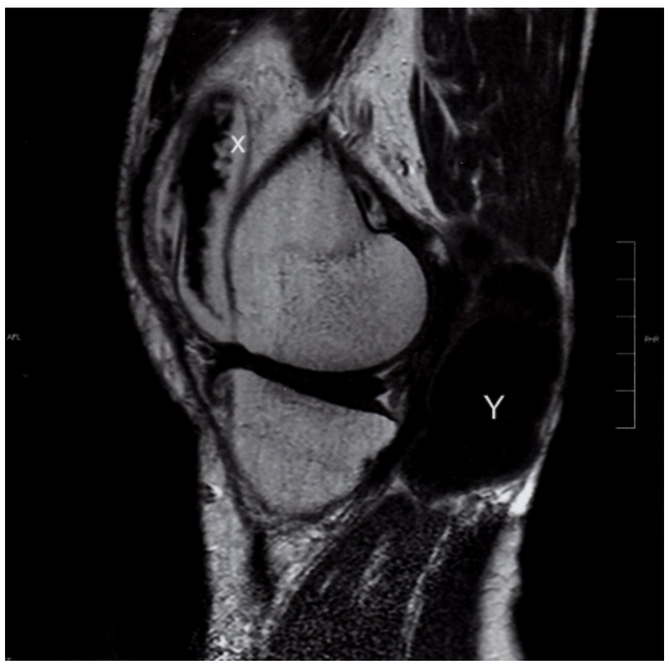
Gout → Negatively birefringment needle-like crystals under polarized light



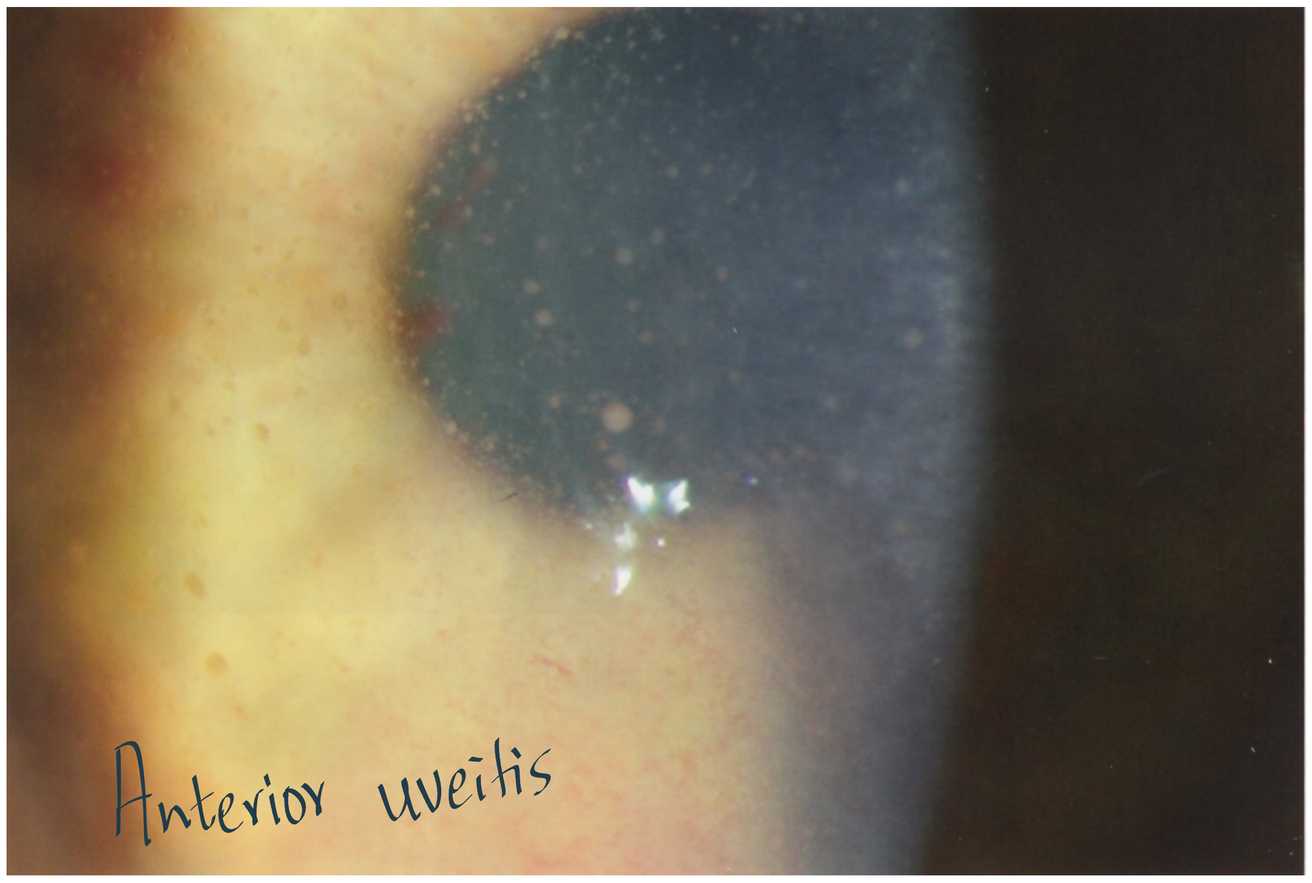
Pseudogout → Positively birefringent rhomboid shaped crystals under polarized light

Reactive Arthritis

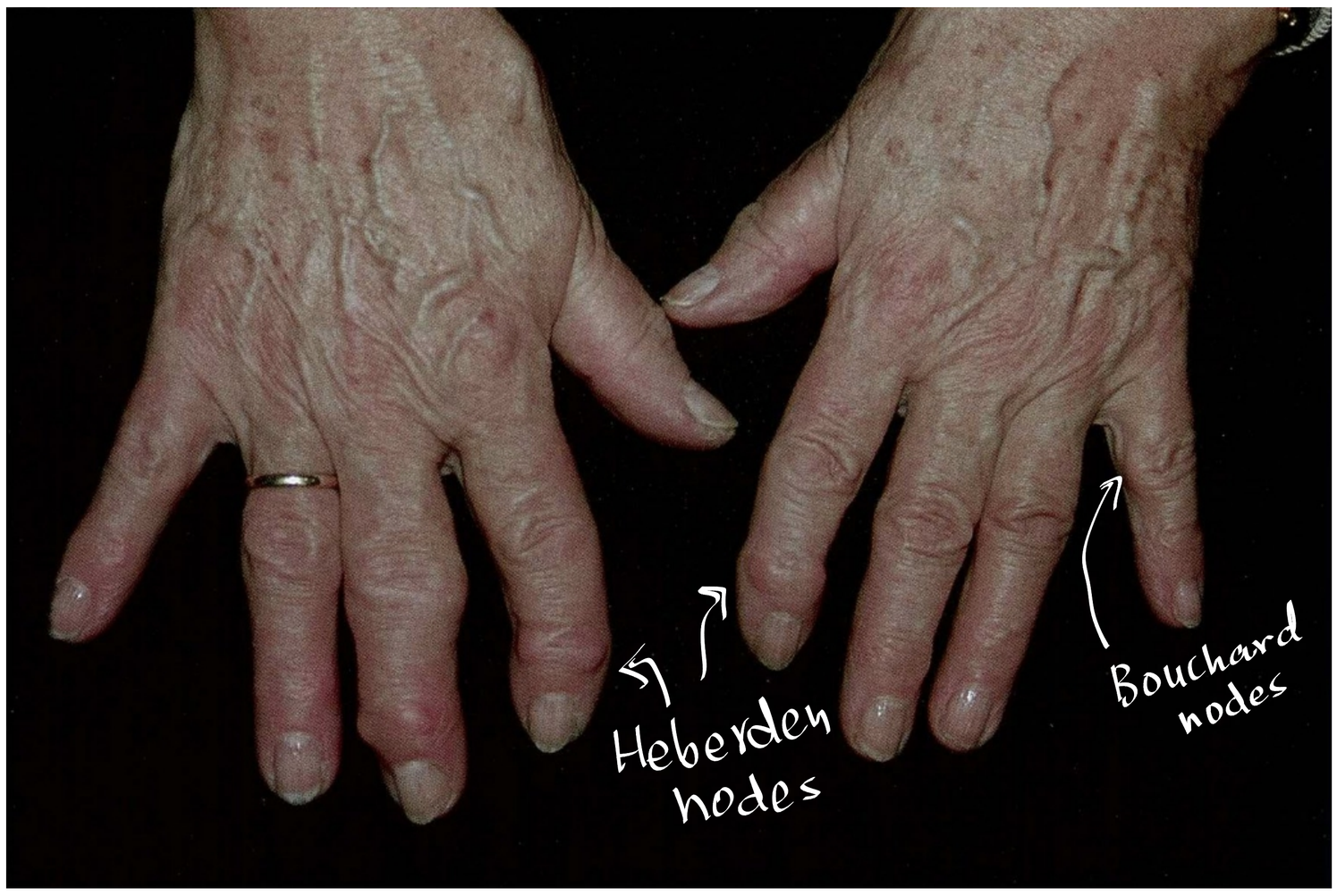


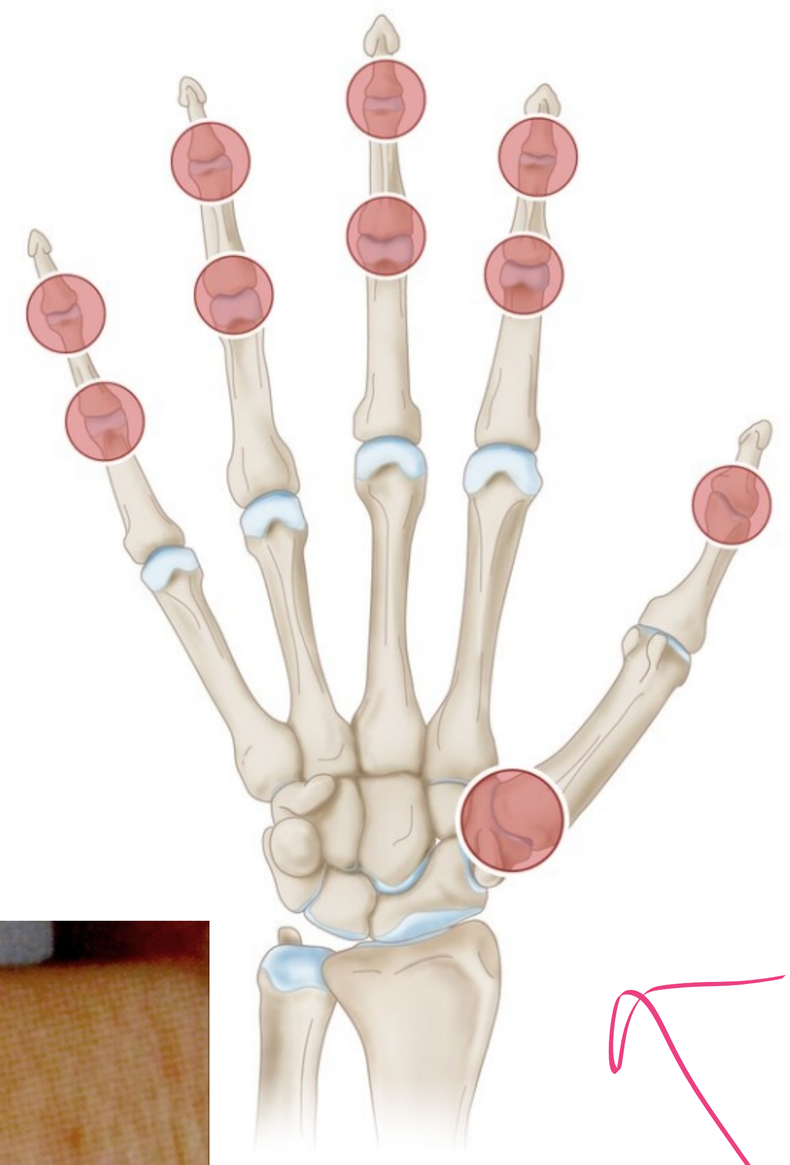


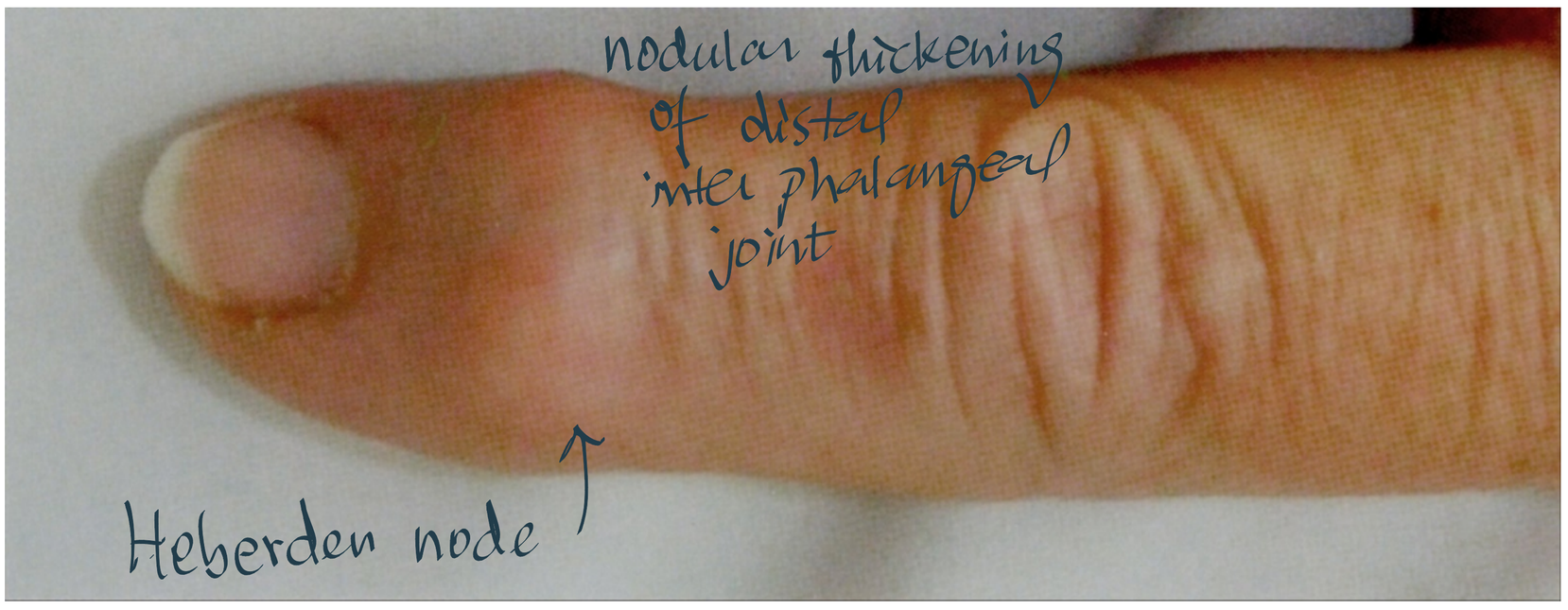
MRI knee
Showing
Synovitis of
knee joint



OSTEO ARTHRITIS







Distribution pattern of osteoarthritis of the hand - Osteoarthritis of the hand affects especially the distal interphalangeal joints (Heberden nodes), proximal interphalangeal joints (Bouchard nodes), and the first carpometacarpal joint, between the trapezoid and the first metacarpal (Rhizarthrosis).

Sarcoidosis

Etiology
Multisystem disorder with T-cell dysfunction and noncaseating granuloma formation

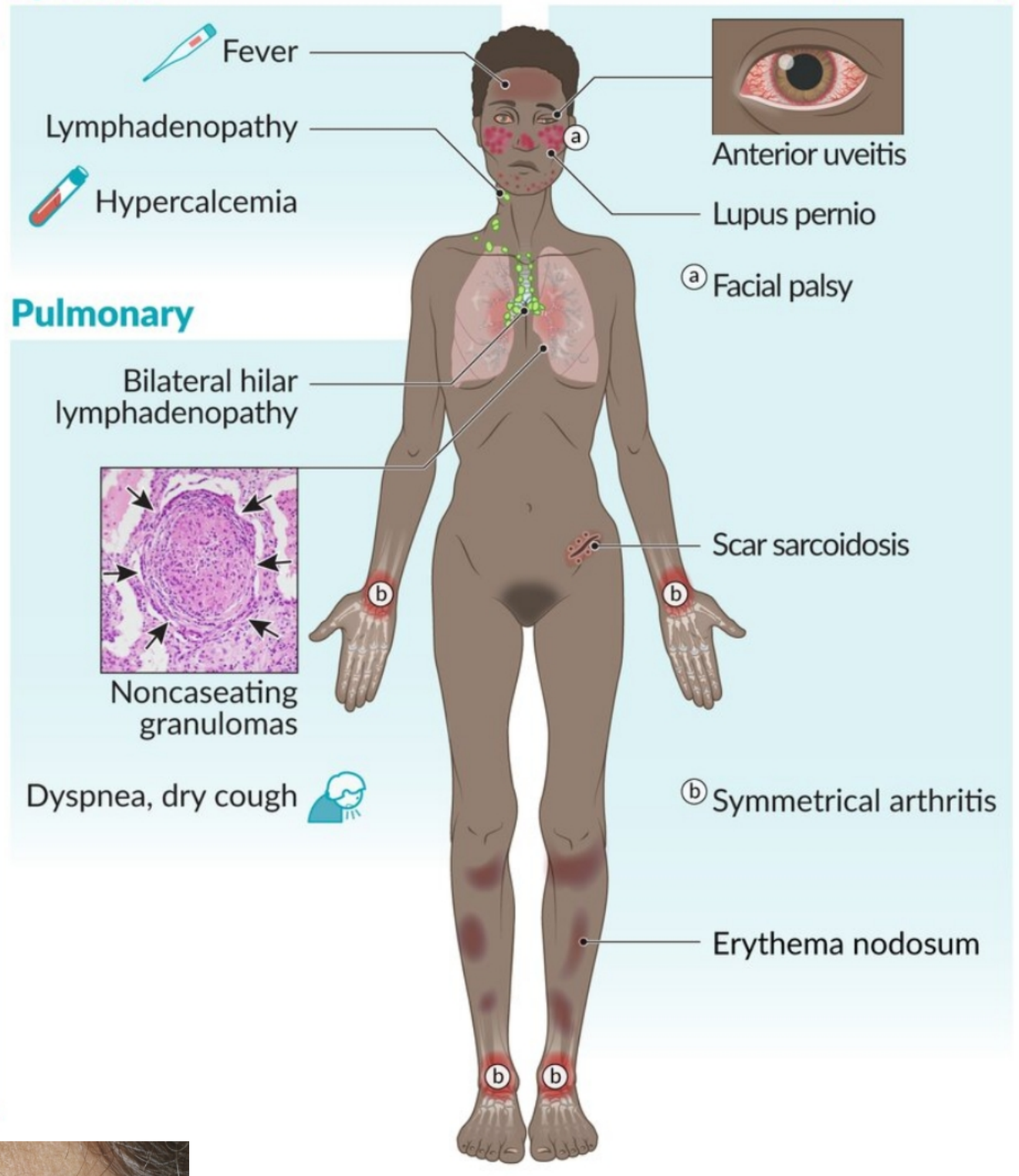
Epidemiology
♀ > ♂; peak age: 30–55 years
Incidence highest in the US among African American women

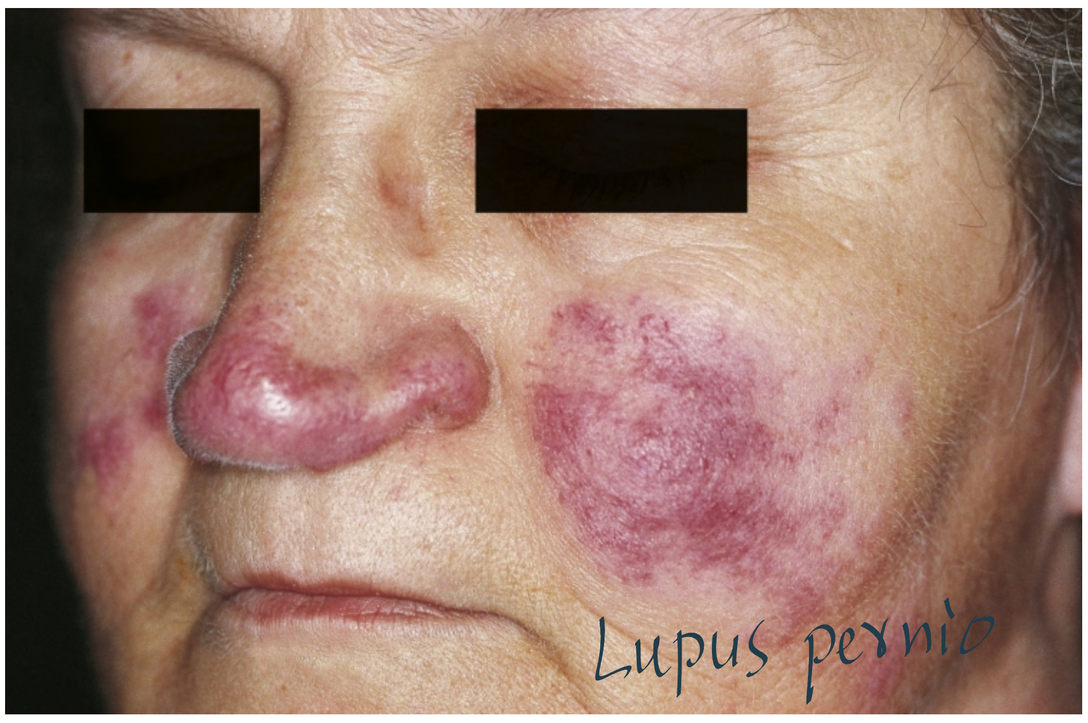
Histopathyoogy (gold standard)
Noncaseating granulomas on biopsy; exclude more common granulomatous diseases

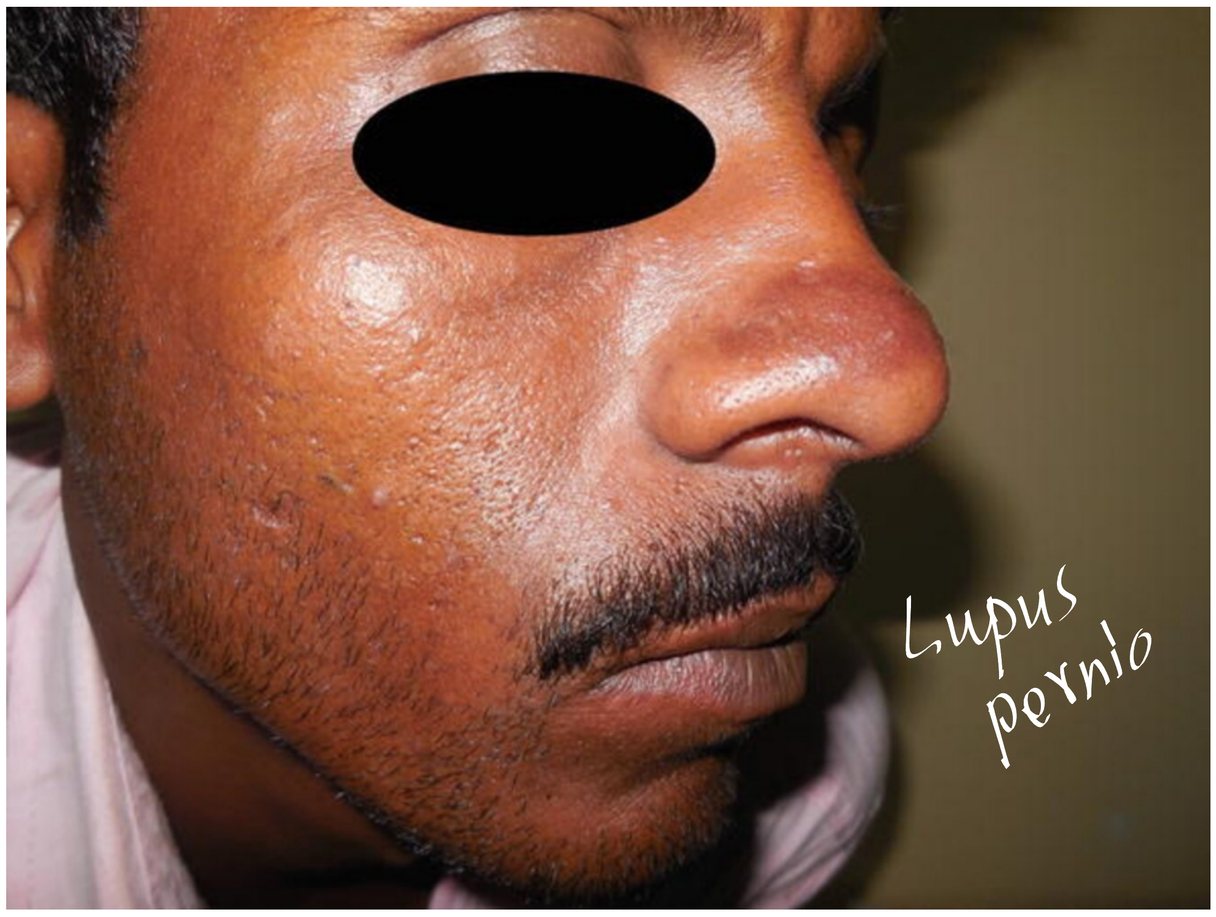
Diagnostic studies
- Chest x-ray: bilateral hilar lymphadenopathy
- Bronchoalveolar lavage: CD4:CD8 ratio > 3.5 supports the diagnosis

Complications
Bronchiectasis, pulmonary fibrosis, chronic renal failure

Prognosis
Spontaneous remission in 20%–70% of patients







Lupus pernio ← (pathognomonic of sarcoidosis)


Annular Sarcoidosis


Cutaneous manifestation of Sarcoidosis

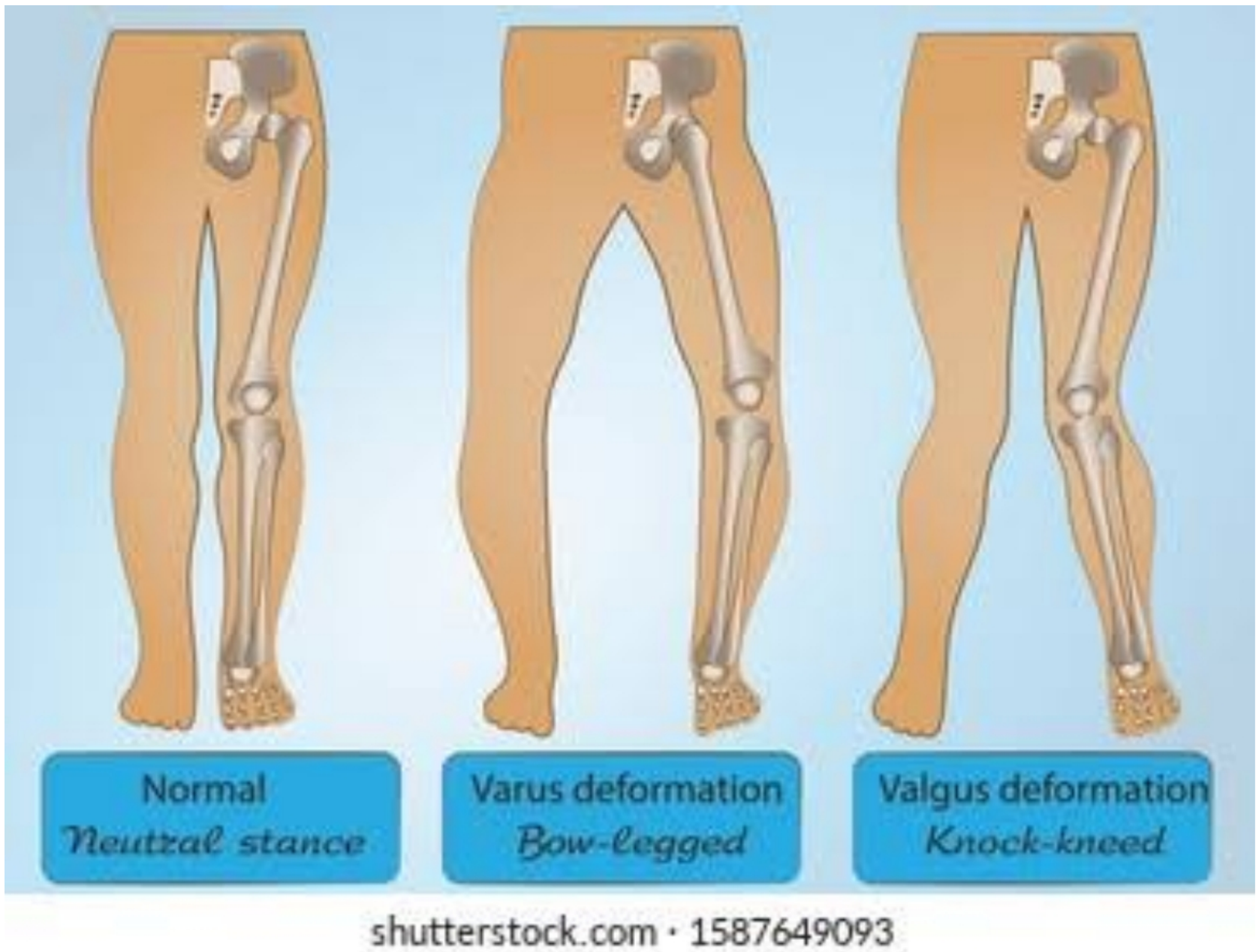

Maculopapular Sarcoidosis


Scar Sarcoidosis


Morpheaform Sarcoidosis


Plaque Sarcoidosis

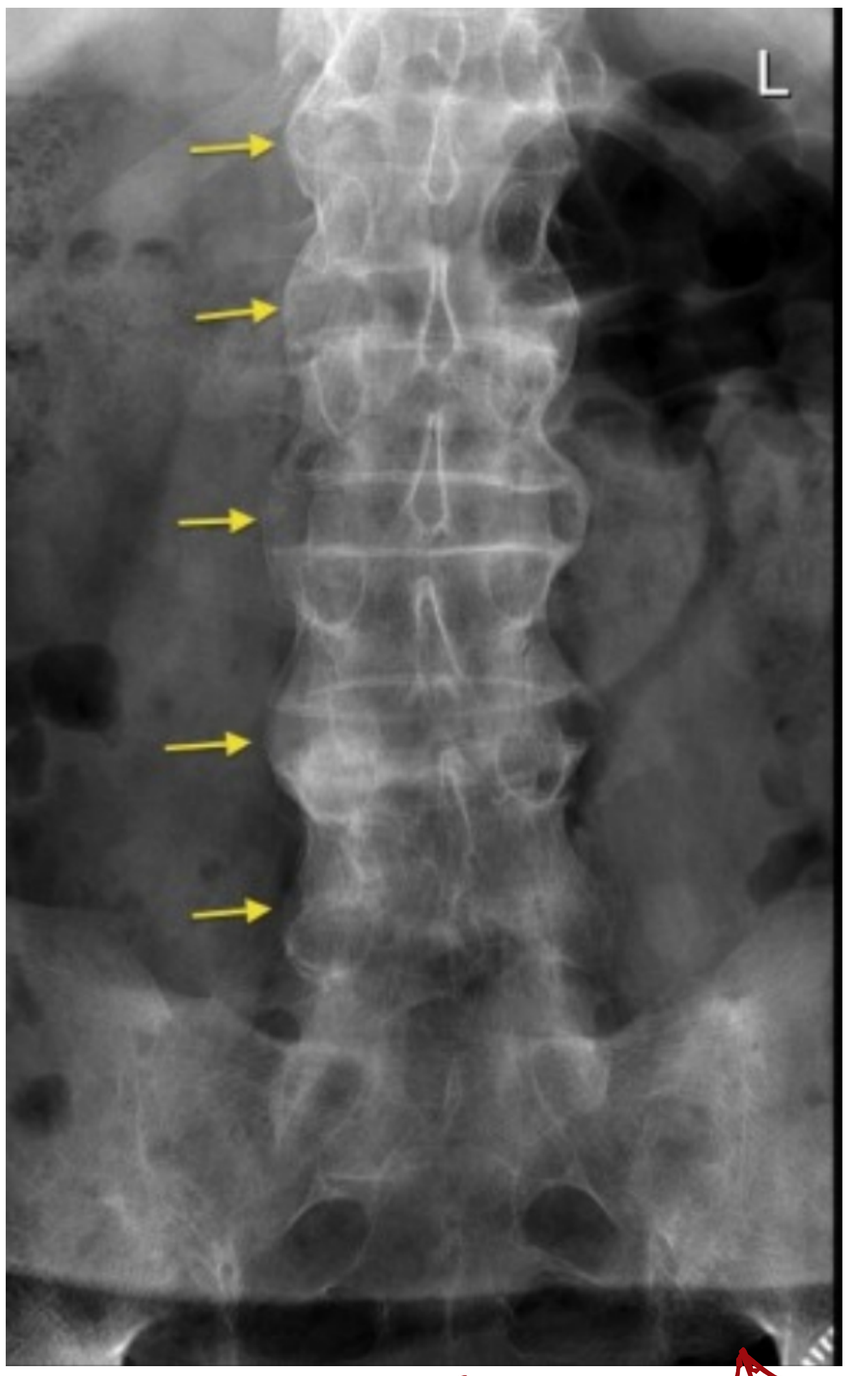
Psoriasiform Sarcoidosis



Varus Deformity seen in knee osteoarthritis

Valgus Deformity in Rheumatoid arthritis

Ankylosing Spondylitis



Bamboo Spine

x-Ray

Ankylosing spondylitis.
There is ossification along the margins of the disk spaces in this patient's lumbar spine (arrows), producing an appearance similar to a stick of bamboo. Note how difficult it is to see the sacro-iliac joints, as they have fused.

* X-Ray → best initial test
* MRI Spine → most accurate test
* HLA-B27 positive
* ↑ ESR, CRP

Psoriatic Arthritis

Pencil-in-cup deformity at DIP seen on X-Ray

Onycholysis ↑

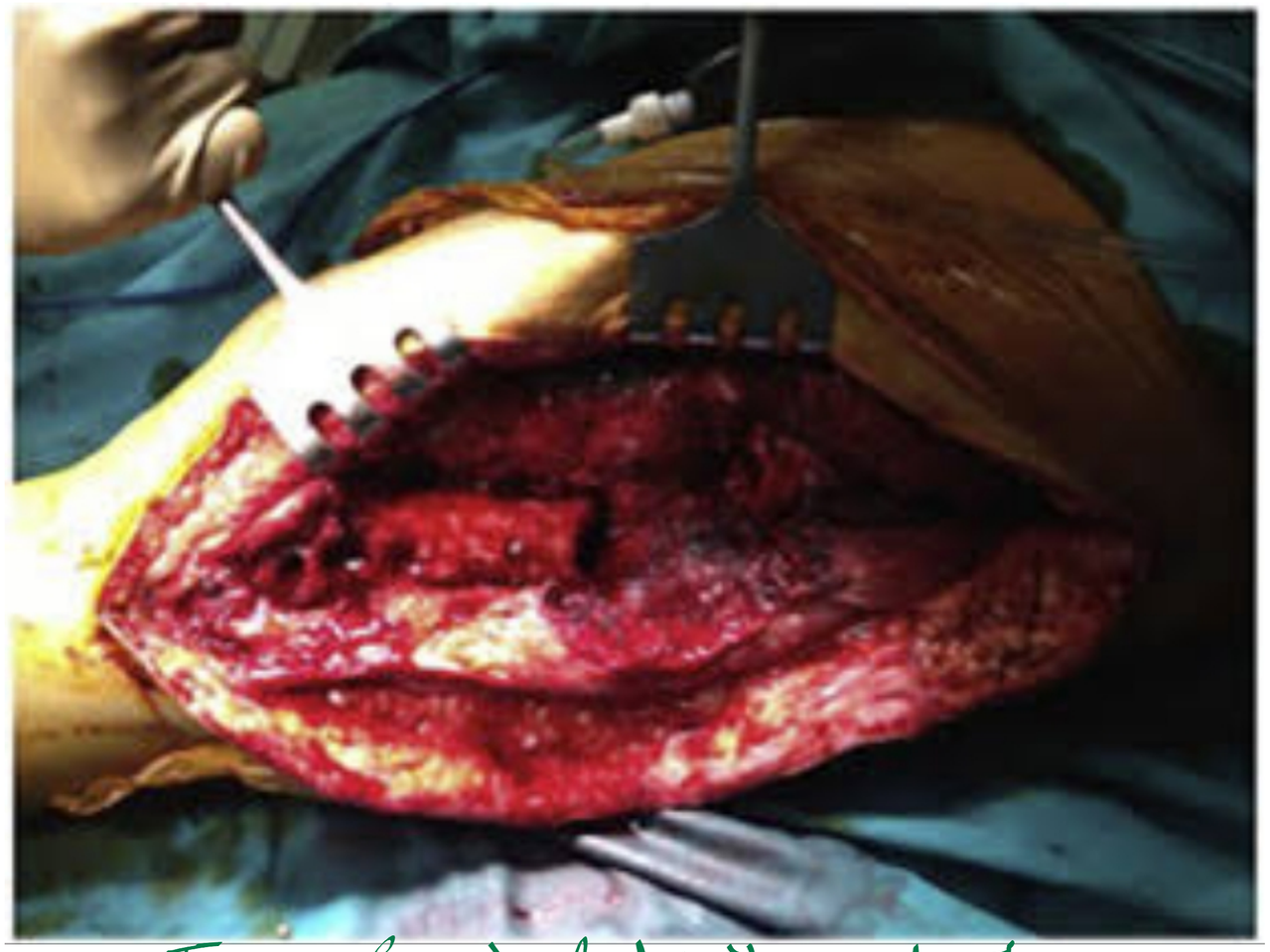
* Methotrexate (DMARDs) is the first treatment of choice

Telescopic fingers seen in Arthritis mutilans

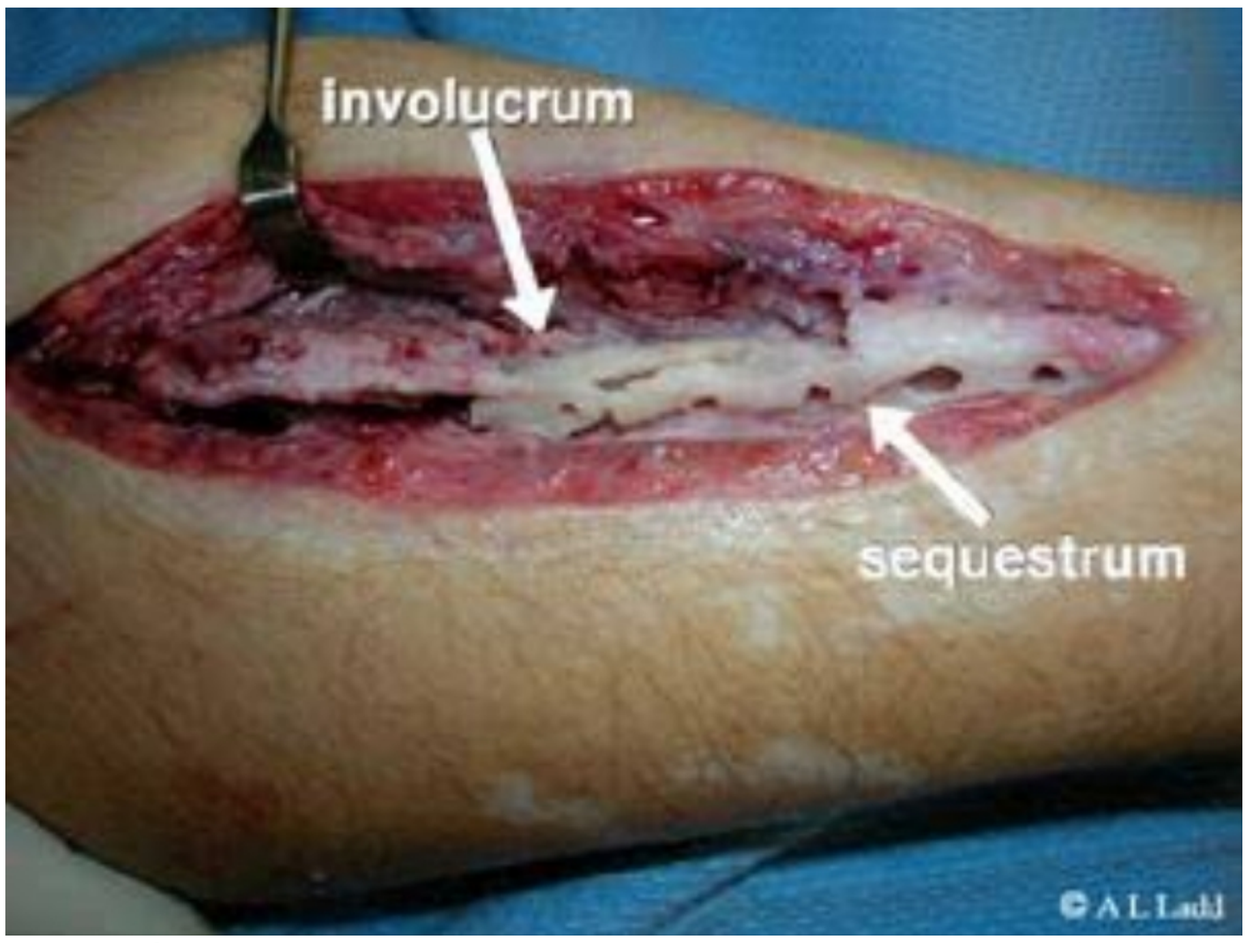
Osteomyelitis

X-Ray → Lytic lesions with surrounding sclerosis

- Biopsy is the most accurate test.

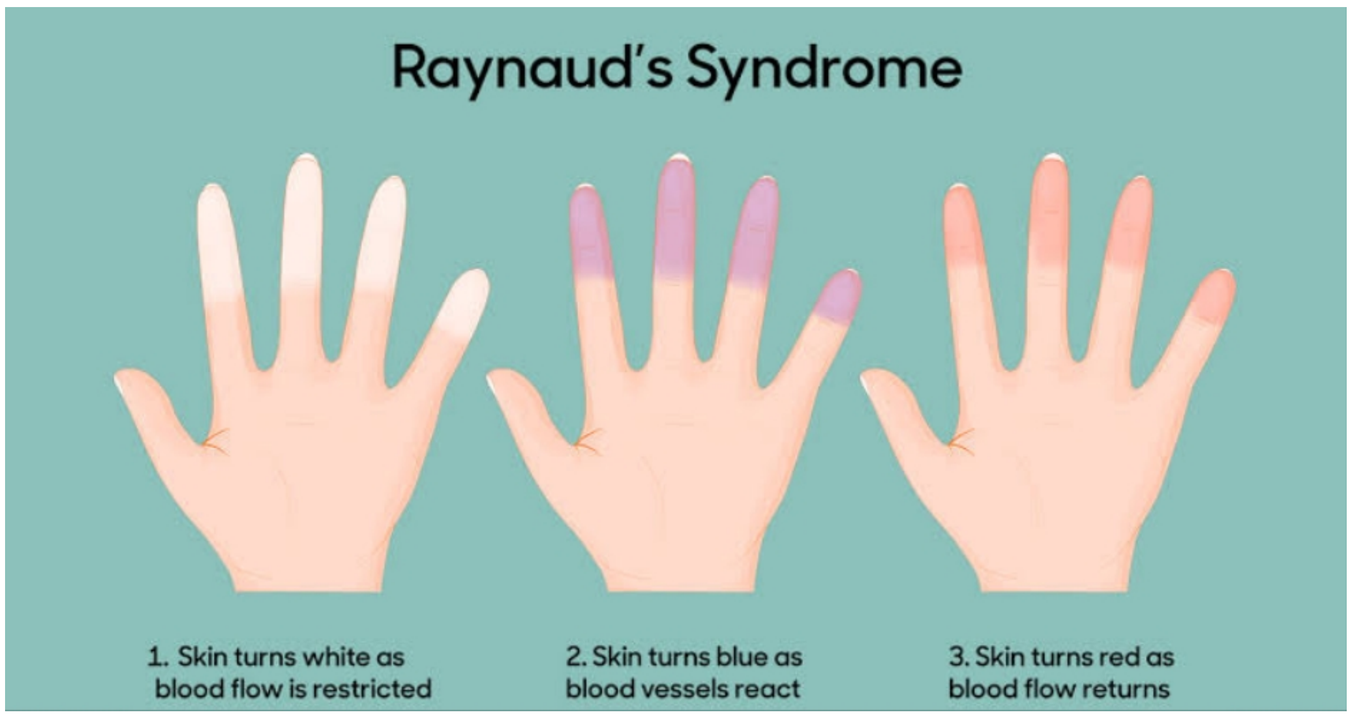


Tx → Surgical debribement of necrotic infected bone



* Sequestrum → dead piece of bone
* Involucrum → new bone formation

SLE



↓ Discoid Rash ↑

Malar Rash

* ANA positive
* Anti dsDNA antibody
* Anti-Smith Antibodies

Systemic Sclerosis

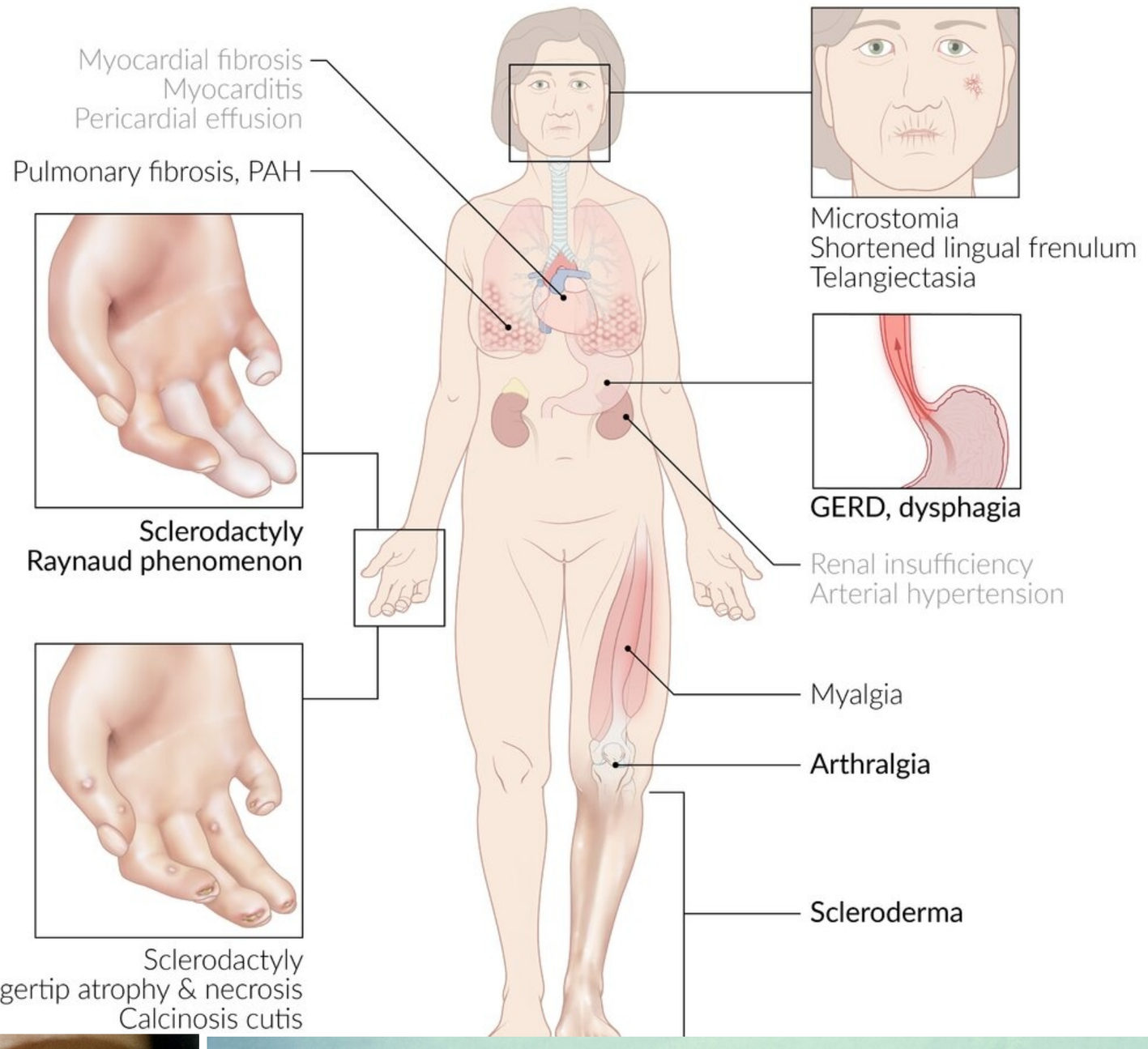
Systemic sclerosis (SSc)

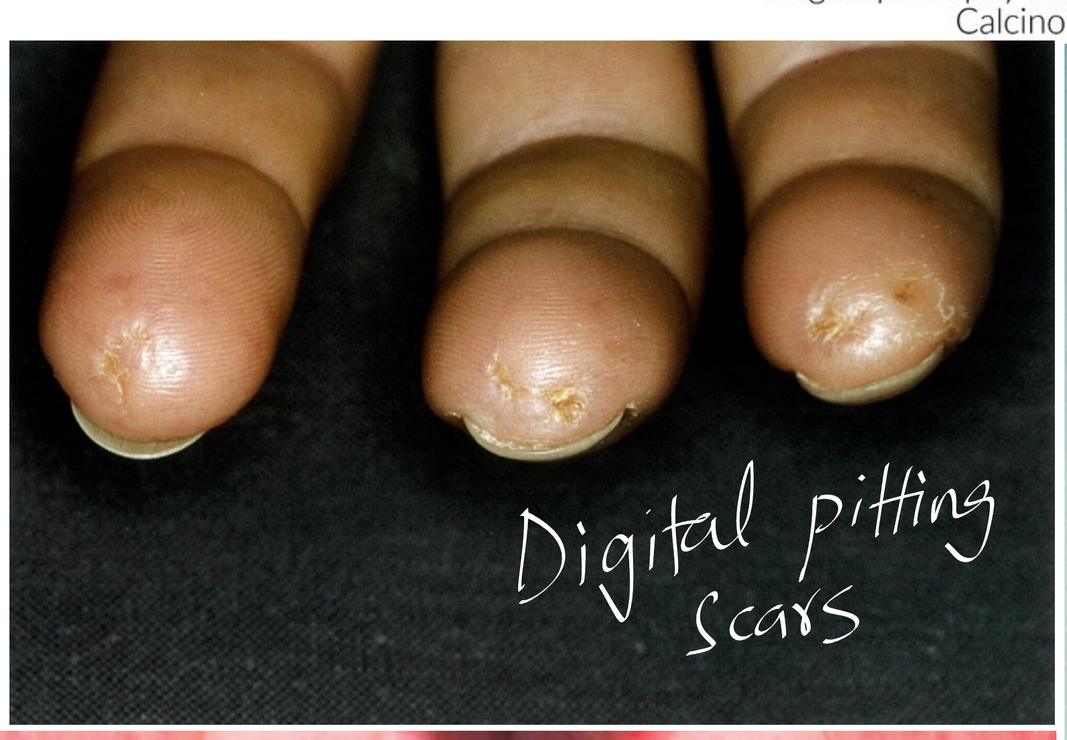
Epidemiology
♀ > ♂
Peak age: 30–50 years

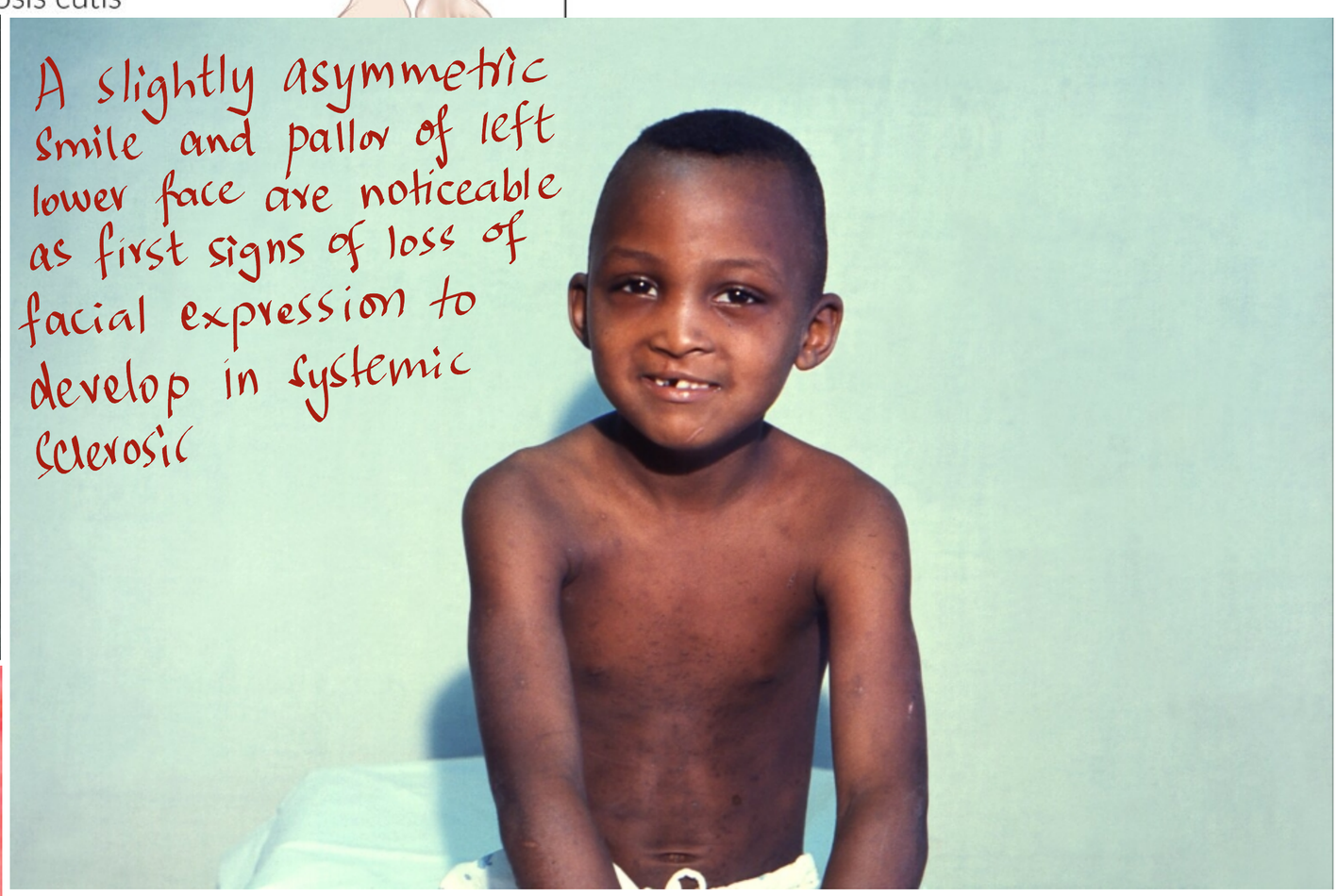
Antibody serology
(usually mutually exclusive)
Antinuclear antibodies (ANA)
Anti-Scl-70 antibodies
Anticentromere antibodies (ACA)

Patterns of cutaneous involvement
Limited SSc (neck, face, and distal limbs, sparing the trunk)
Diffuse SSc (trunk to the elbow)

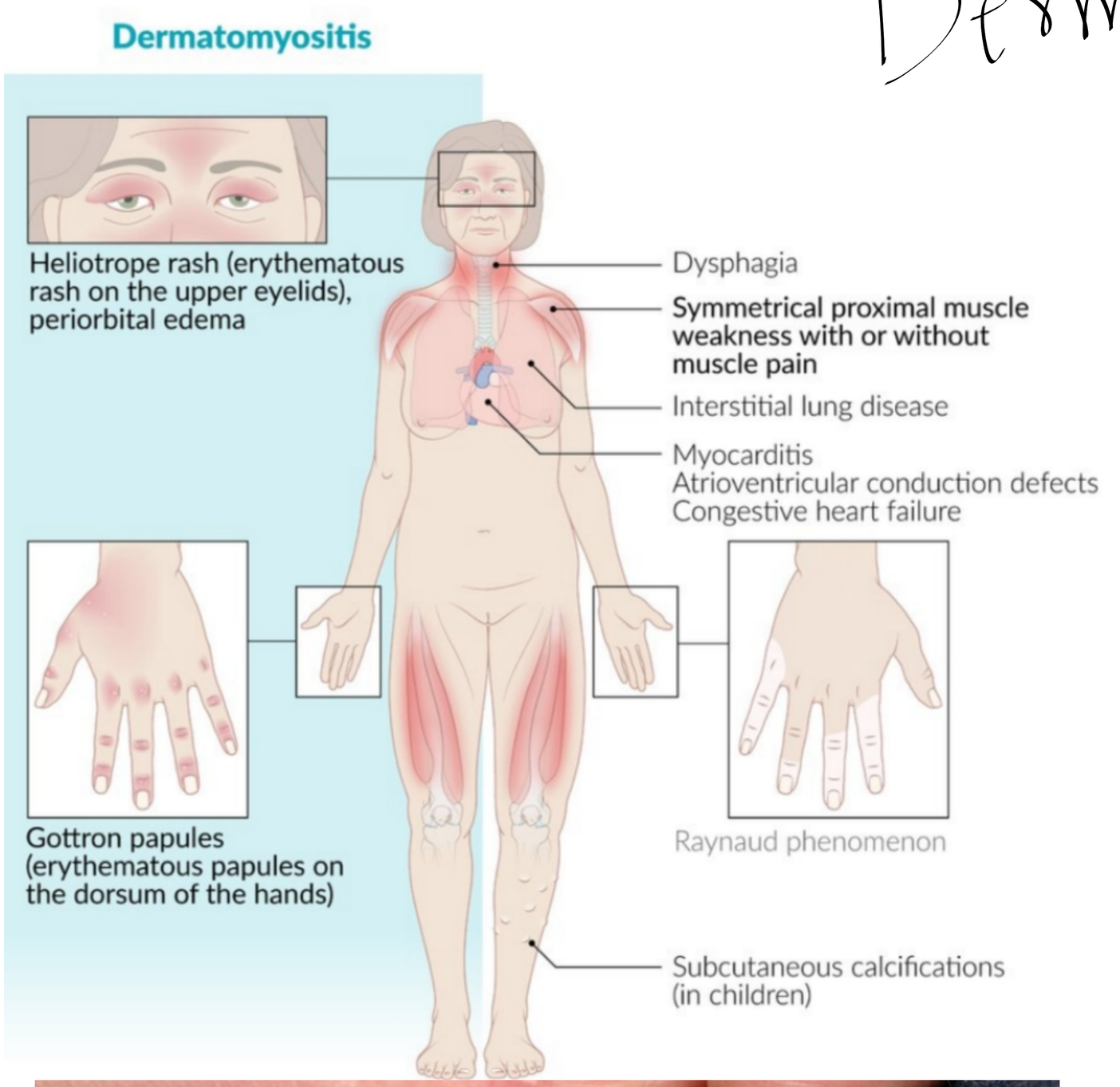
CREST syndrome
(more common in limited SSc)
C: Calcinosis cutis
R: Raynaud phenomenon
E: Esophageal hypomotility
S: Sclerodactyly
T: Telangiectasias

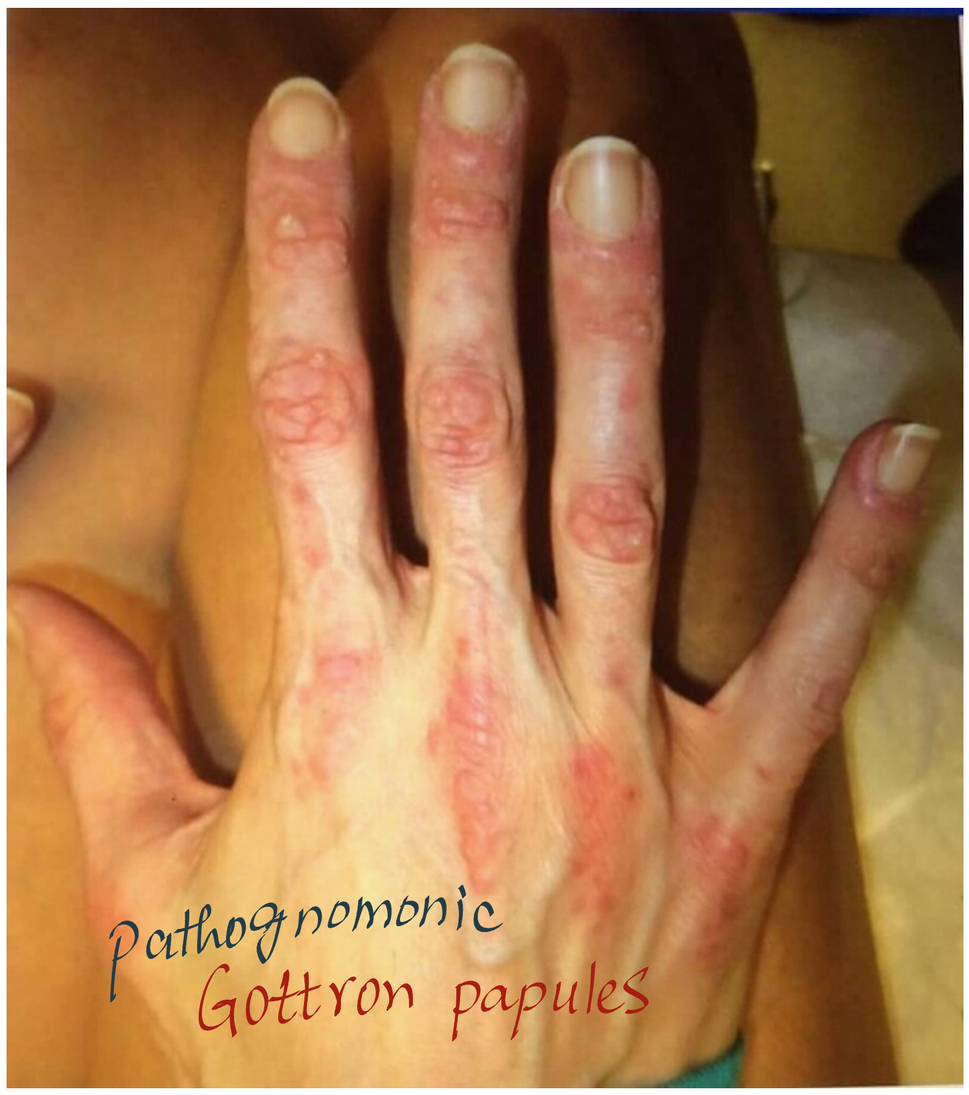






Dermatomyositis





Serology
ANA (nonspecific)
Anti-Mi-2 antibodies (specific for DM)

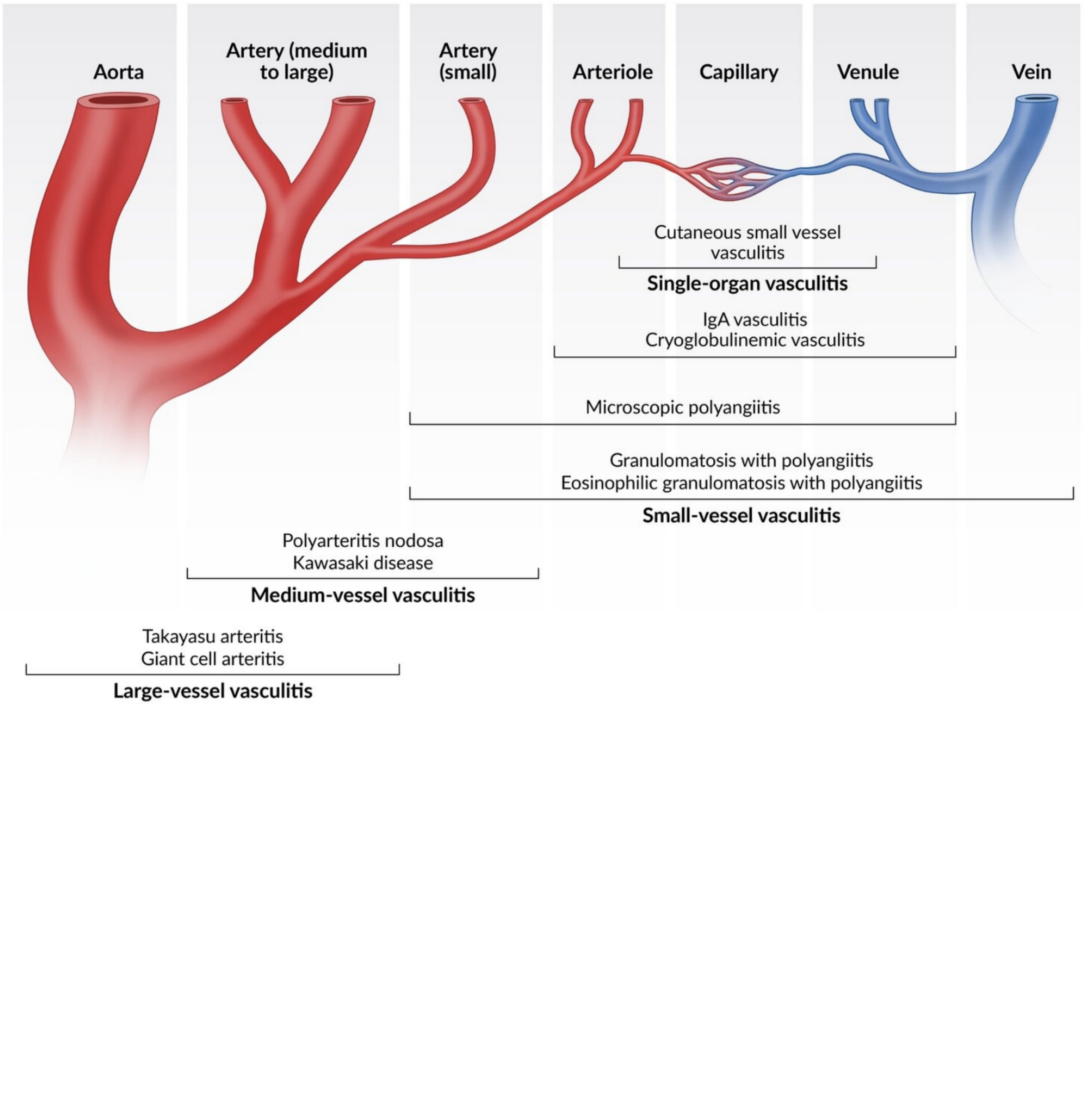
Laboratory findings
↑↑ Serum creatine kinase,
↑ Aldolase, ↑ myoglobin, ↑ LDH,
↑ AST, ↑ ALT

Note
Associated with malignancy (esp. in patients with dermatomyositis)
Polymyositis (DM without skin involvement) is a diagnosis of exclusion in the absence of the characteristic findings of the other inflammatory myopathies

Heliotrope Rash ↑

Gottron papules →

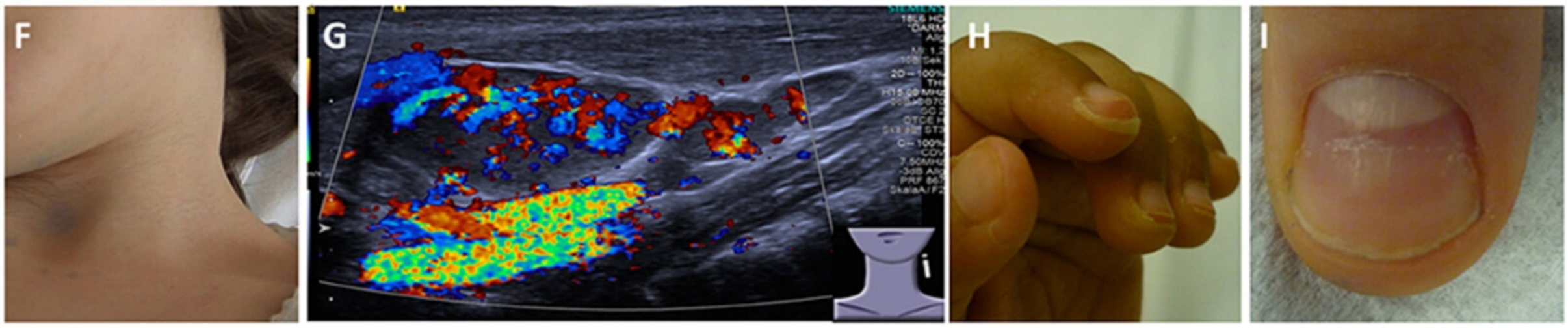
Vasculitides



Giant Cell Arteritis

Kawasaki Disease

↗ Also known as "mucocutaneous lymph node syndrome"



A- Bilateral, non purulent Conjunctivitis

B- Stomatitis with bright red lips

C - Erythema and edema of hands

D- Erythema and edema of feet

E- Truncal Rash

F- Cervical Lymphadenopathy

G - U/s of enlarged cervical Lymph node

H- Desquamation of fingertips

I - Beau lines (Deep grooves in the nails)

IgA Vasculitis

Buerger Disease (Thrombophlebitis obliterans)

- **Migratory superficial thrombophlebitis** (recurrent): often seen prior to the onset of limb ischemia [3][6]
  - Tender nodules along the course of the affected vein
  - Induration
  - Erythema
- **Raynaud phenomenon**
- **Chronic or acute limb ischemia**: may progress from distal to proximal vessels
  - Intermittent claudication
  - Pain at rest, cool extremities, and/or diminished or absent pulses
  - Ulceration and/or gangrene of fingertips and/or toes (digits may autoamputate)

- Associated with smoking

- **Ankle-brachial index (ABI)**: may be decreased [3]

- **Angiography**
  - Findings
    - Segmental occlusions in the distal vessels of the extremities
    - Corkscrew-shaped collateral vessels around the site of occlusion
    - Normal proximal arteries without evidence of atherosclerosis

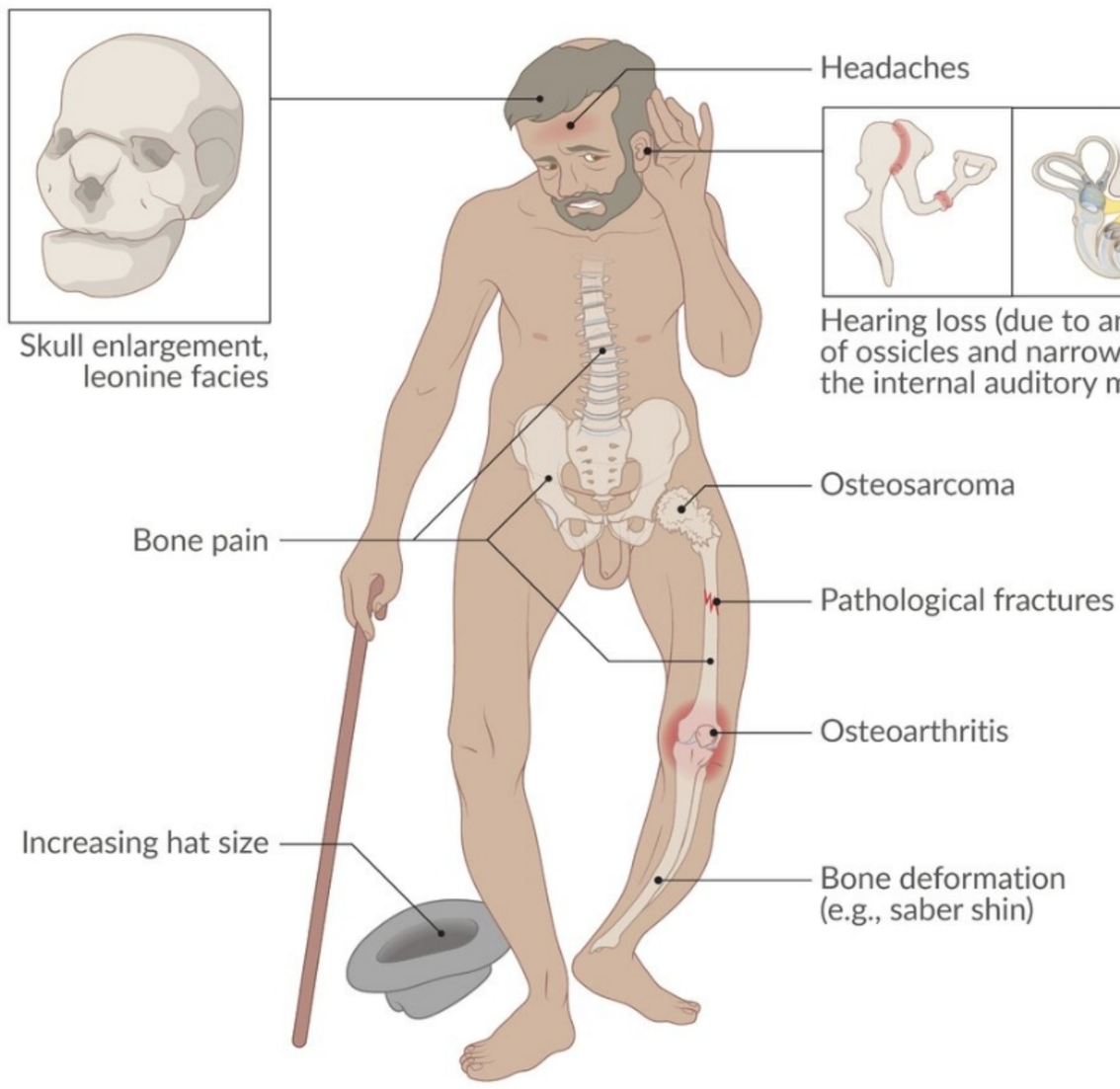
Paget Disease

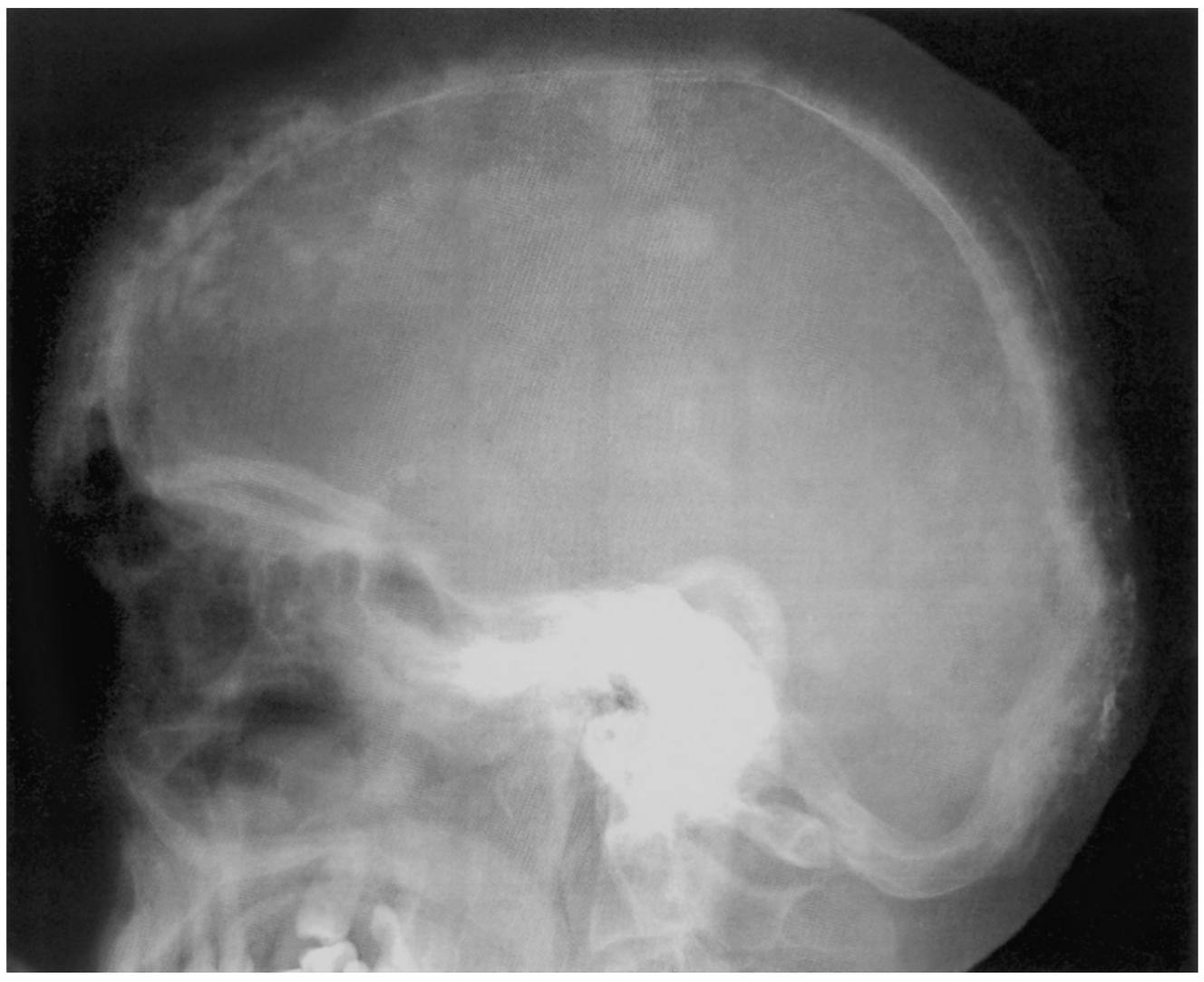
Paget disease of the bone (osteitis deformans)

Pathophysiology
Increased bone remodeling (↑ osteoclastic and osteoblastic activity) → replacement of lamellar bone with weak woven bone

Diagnosis
- Isolated elevation of ALP with normal calcium, phosphate, and PTH
- Urinalysis (markers of collagen degradation)
- X-ray (bone deformation with sclerotic and osteolytic lesions)
- Skeletal scintigraphy (bony lesions)

Treatment
- Indicated in active disease (↑ ALP or active foci on skeletal scintigraphy)
- First-line: bisphosphonates





* Bisphosphonates are the mainstay of treatment

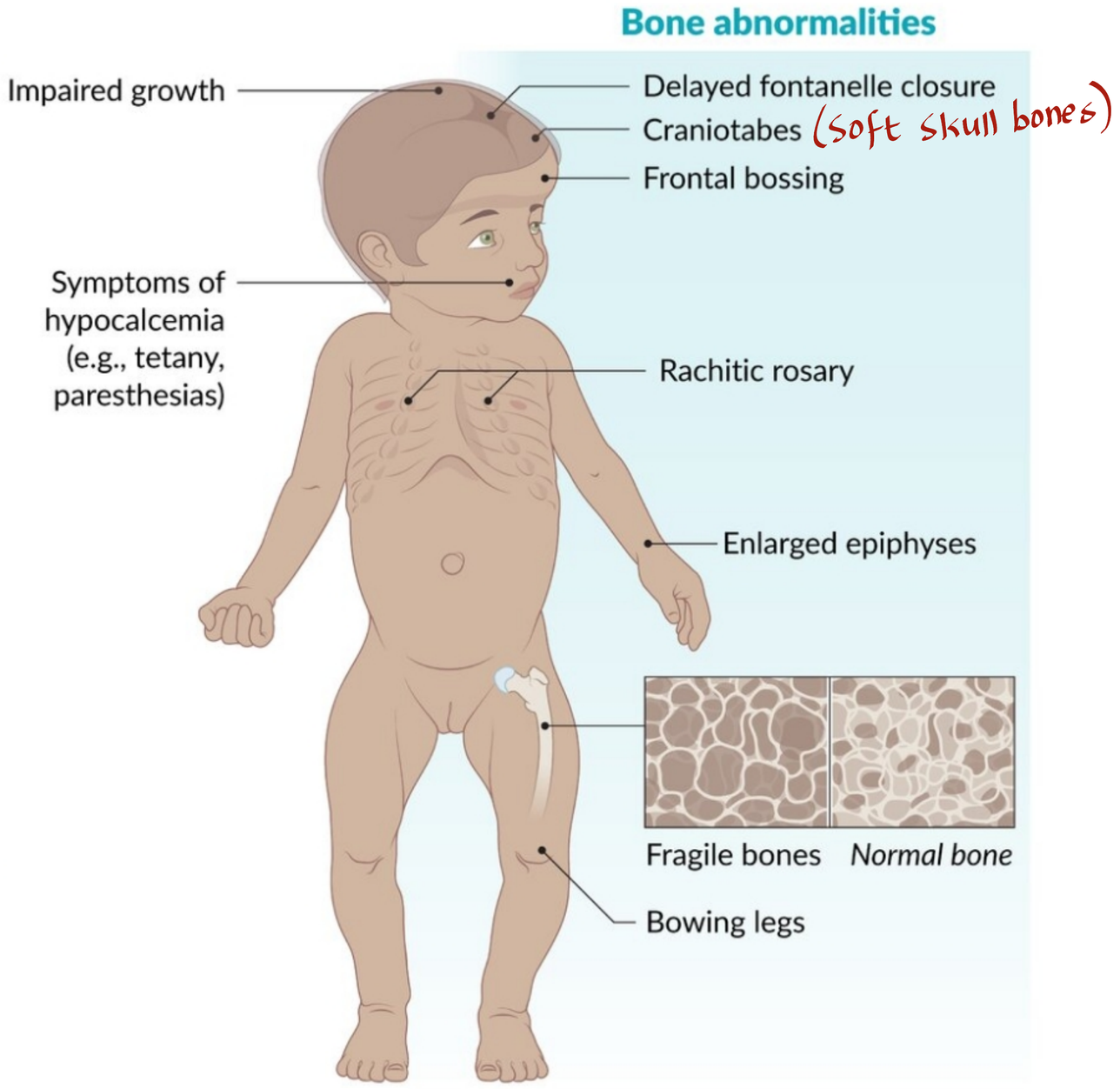
Rickets secondary to vitamin D deficiency

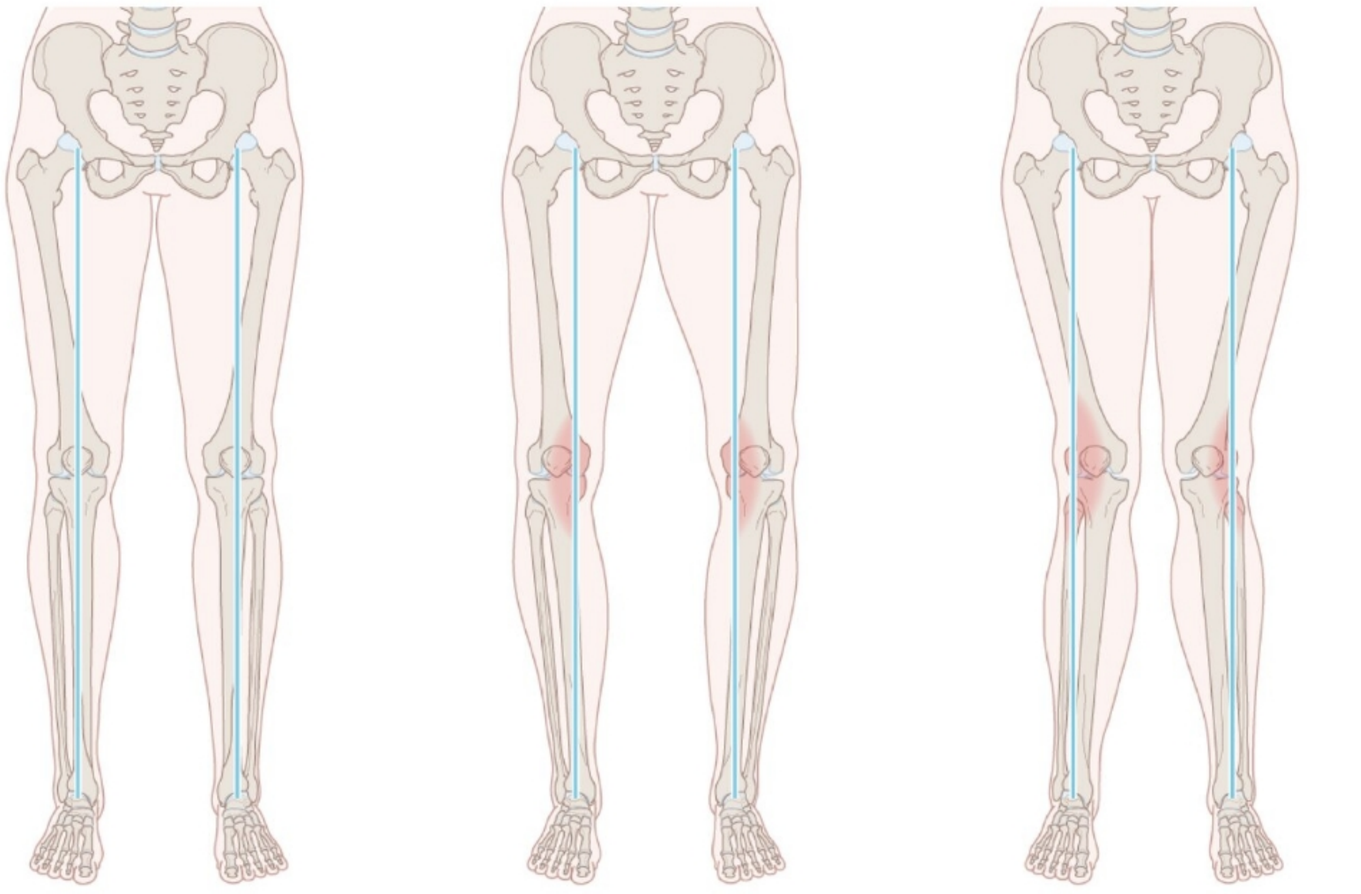
Etiology
- Vitamin D deficiency due to:
  - Insufficient synthesis due to low UV radiation exposure (e.g., northern climates) and/or dark skin
  - Insufficient oral intake (e.g., in infants who are exclusively breastfed)
  - Malabsorption
  - Defective vitamin D metabolism

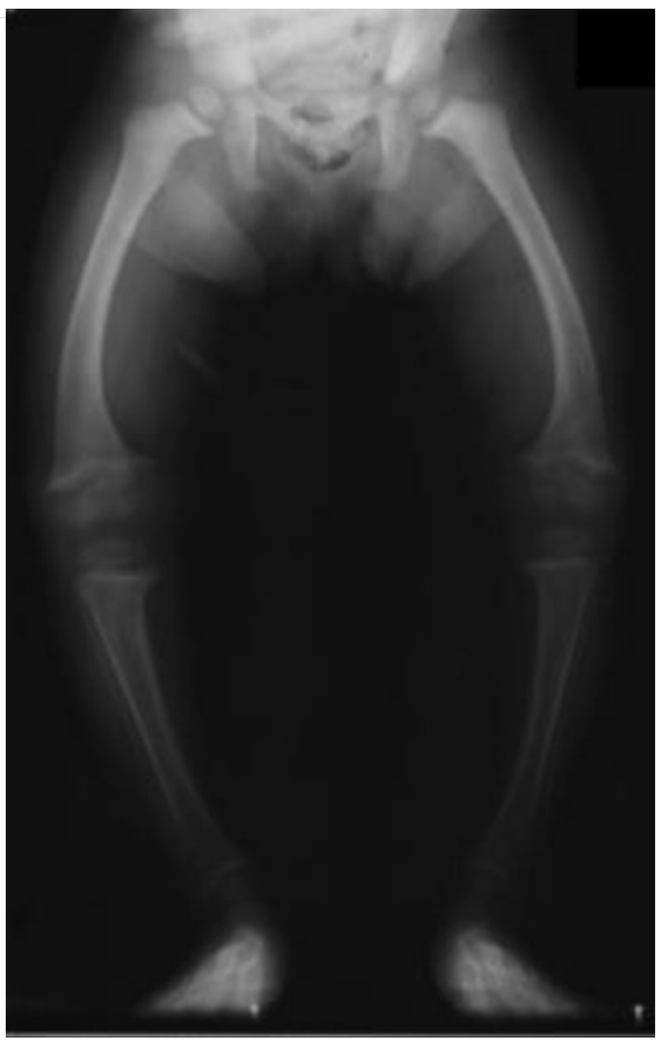
Pathophysiology
Vitamin D deficiency → defective mineralization of osteoid and growth plates

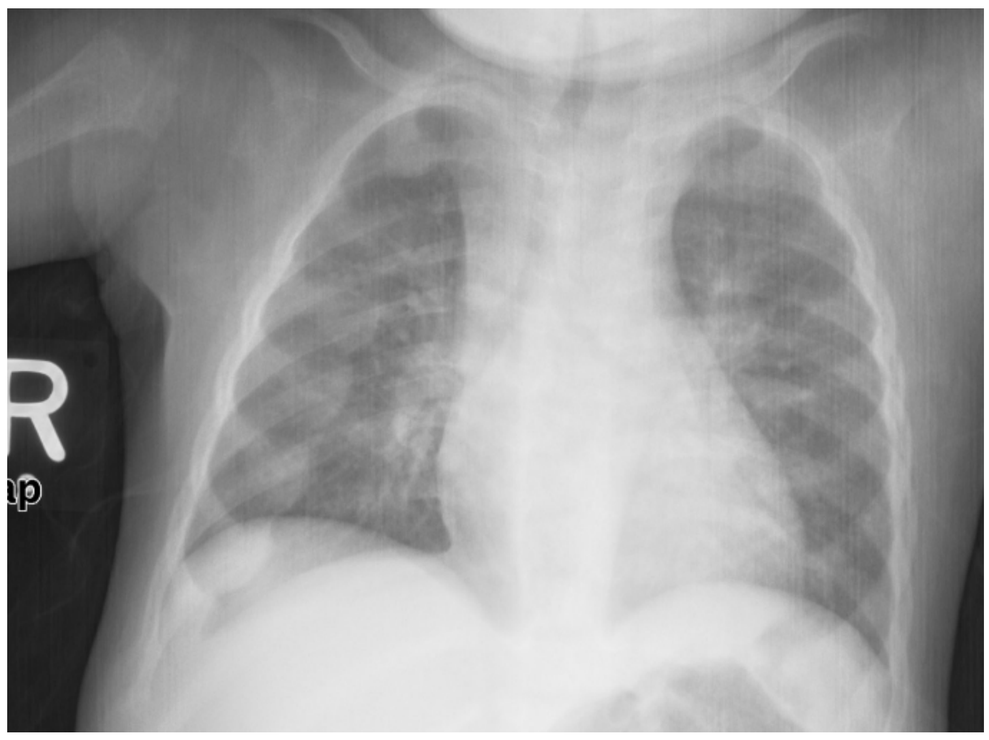
Diagnostics
↓ Vitamin D, ↓ serum Ca^{2+}, ↑ PTH, ↓ PO_4^{3-}, ↑ ALP

Treatment
- Dietary supplementation (vitamin D, calcium)
- Treatment of underlying cause









Chicken pox
Varicella

Pathogen
Varicella-zoster virus

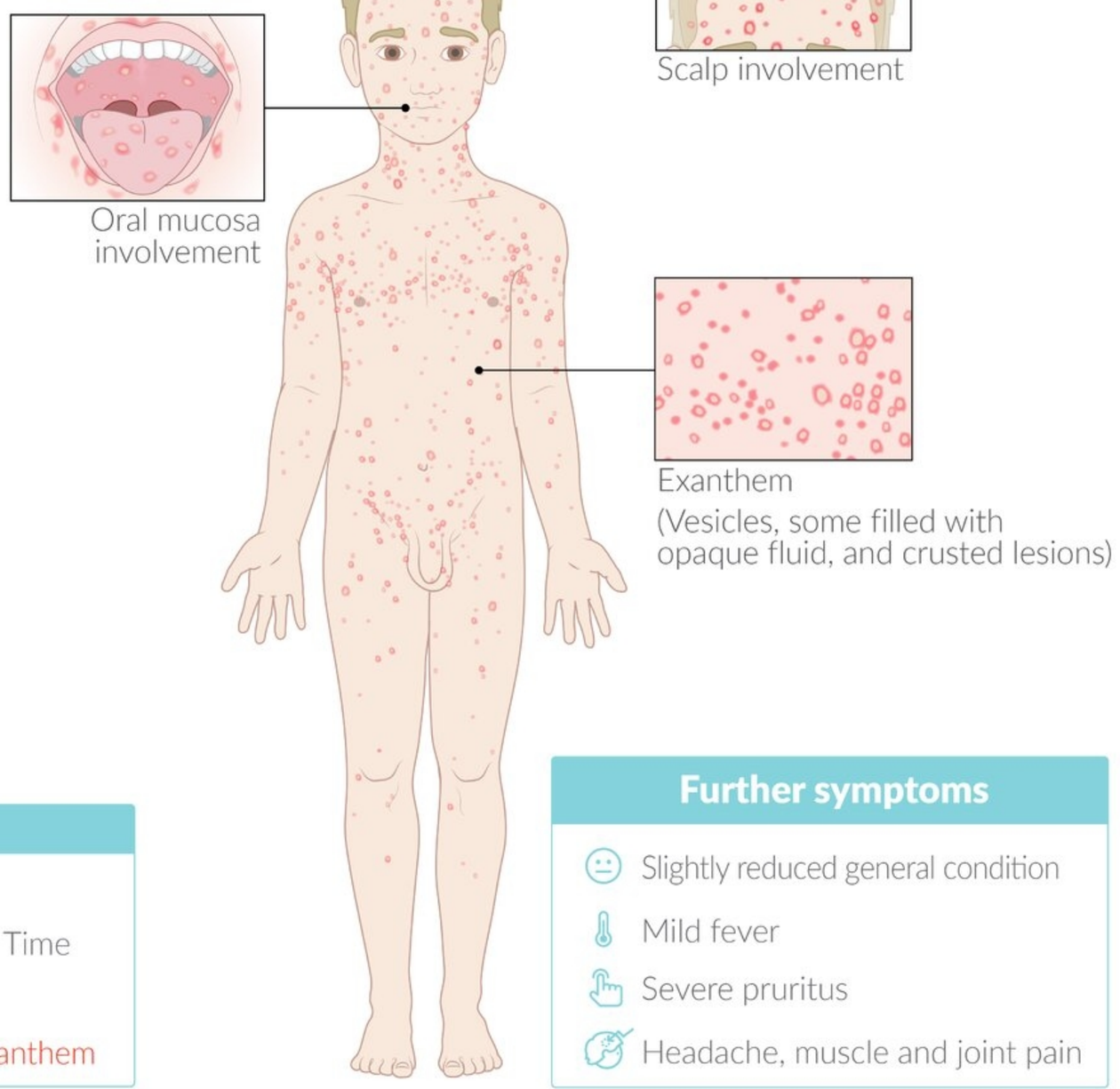
Course
Widespread vesicular rash with lesions beginning as macules and rapidly becoming papules
Simultaneous occurrence of various rash stages
Remission of exanthem after 8 days

Complications
Bacterial superinfection
Meningitis, acute cerebellar ataxia, encephalitis
Herpes zoster (Shingles) from virus reactivation
Congenital varicella syndrome

Treatment
Symptomatic

Vaccine
Yes

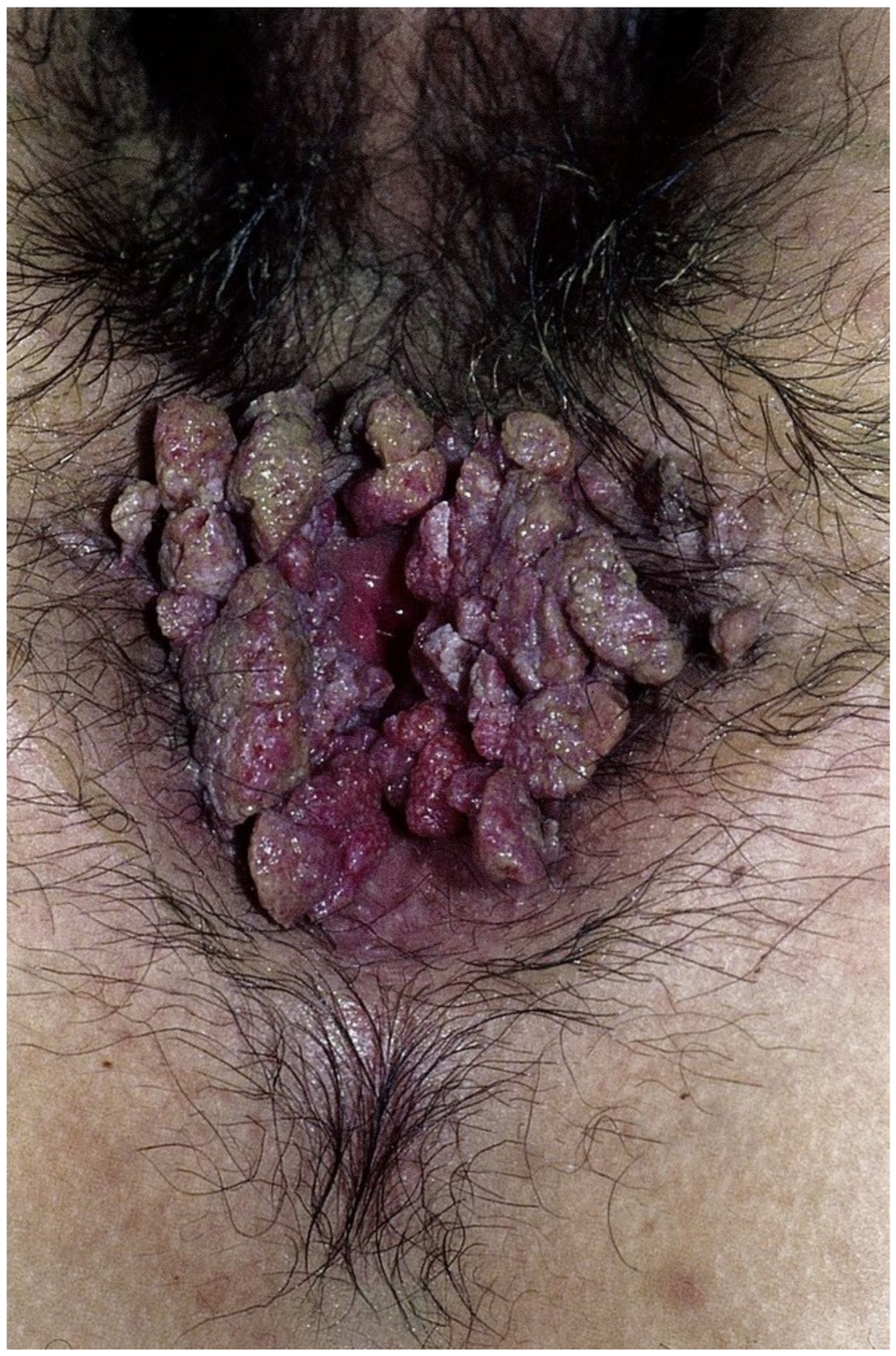
Course of disease



Further symptoms

- Slightly reduced general condition
- Mild fever
- Severe pruritus
- Headache, muscle and joint pain

Warts (HPV)

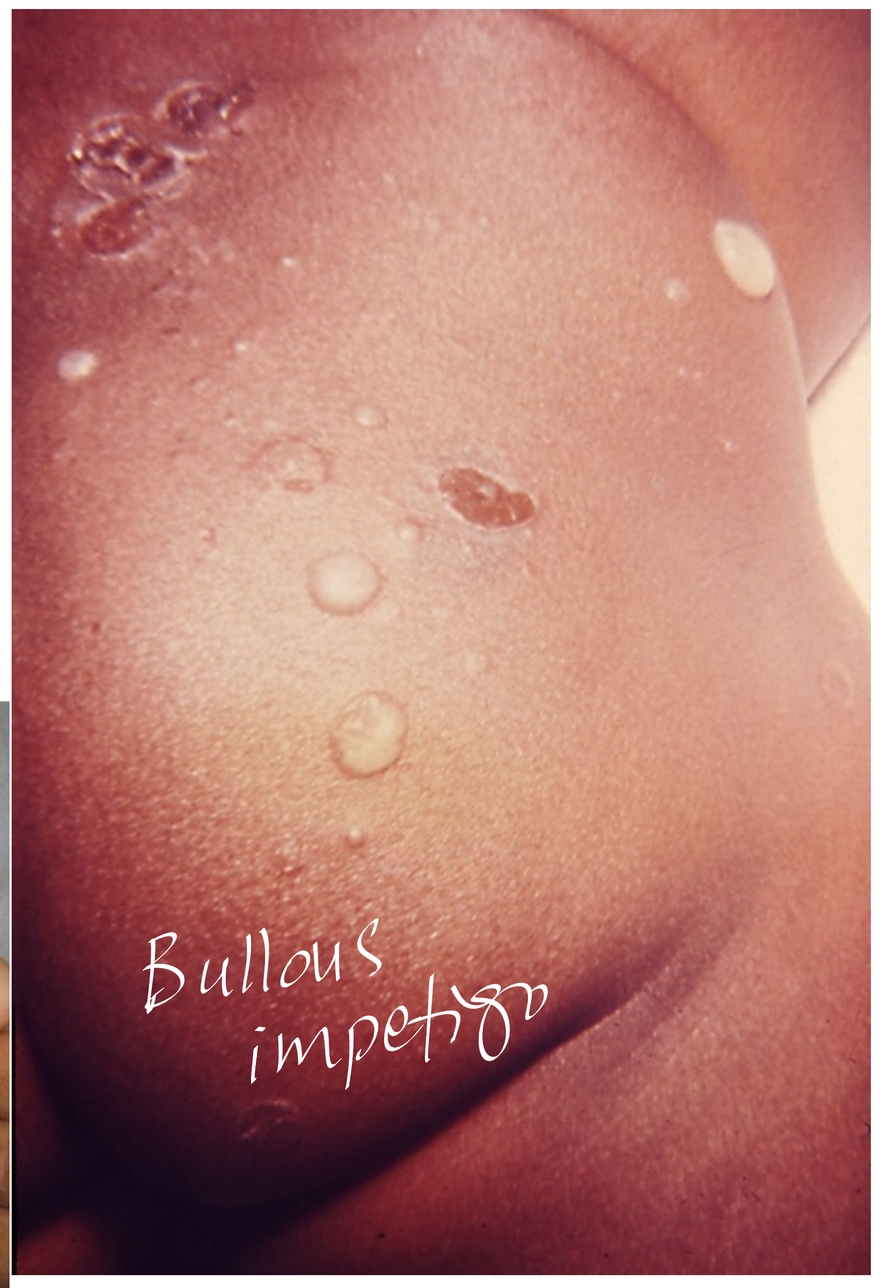


Molluscum Contagiosum

Etiology

- **Pathogen**: a DNA poxvirus (molluscum contagiosum virus)
- **Transmission**:
  - **Direct skin contact** (contact sports, sexually transmitted)
  - **Autoinoculation** (scratching or touching lesion, e.g., while shaving)
  - Fomites (e.g., on bath sponges/towels)
- **Risk factors**: immunosuppression , active atopic dermatitis (in children), hot and humid climates, crowded living conditions [2]

IMPETIGO



Impetigo - Confluent scabbed erosions and honey-colored crusts are visible in the perioral region. This appearance is typical of impetigo, which, in this patient, developed secondary to preexisting eczema and immunosuppression caused by HIV infection.

Folliculitis

Leprosy

Syphilis

Syphilis

Epidemiology
Sex: Approx. 6:1 (♂:♀)
Incidence of primary and secondary syphilis (US): Approx. 10.8/100,000 (rising)
Peak incidence: 20–29 years

Etiology
Pathogen: Treponema pallidum
Transmission: Sexual; vertical; bloodborne

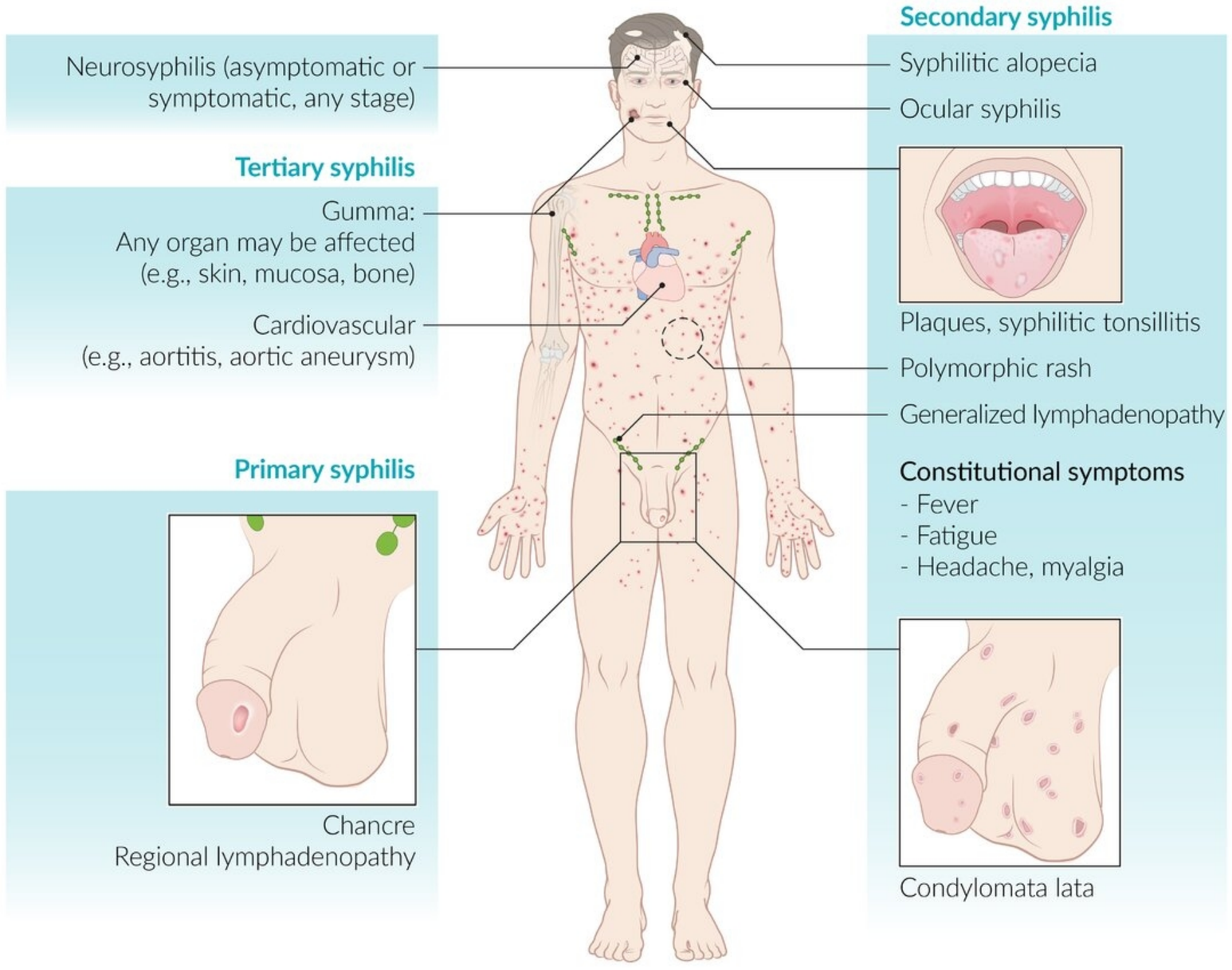
Classification
Early stages: primary and secondary syphilis
Latent syphilis
Late stage: Tertiary syphilis

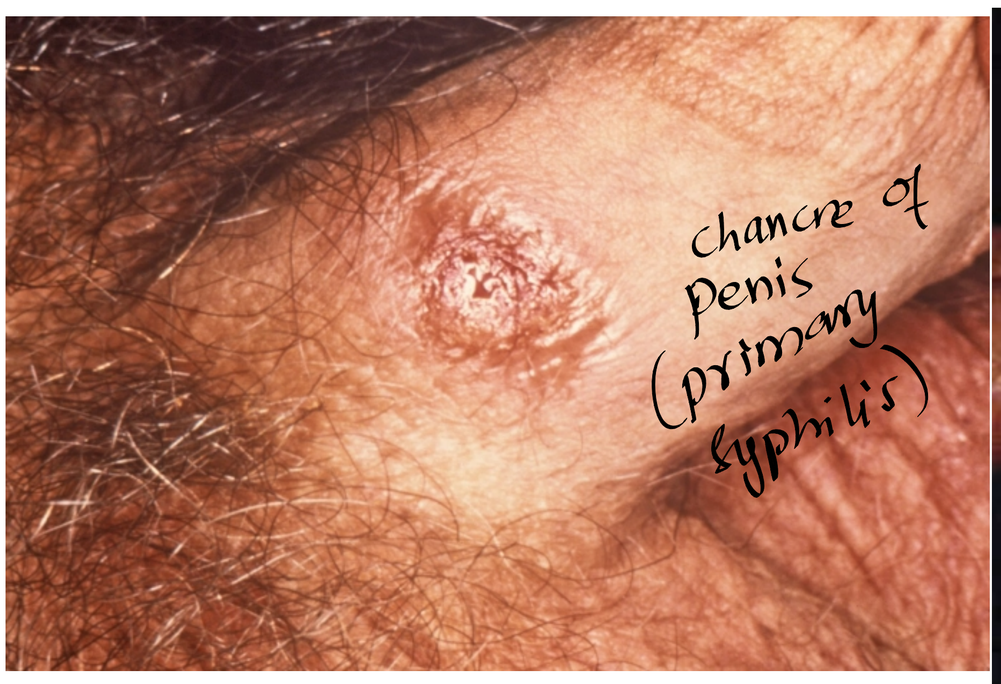
Diagnosis
Direct detection of the pathogen (e.g., PCR, darkfield microscopy)
Serology (e.g., RPR, TPPA, FTA-ABS, VDRL)

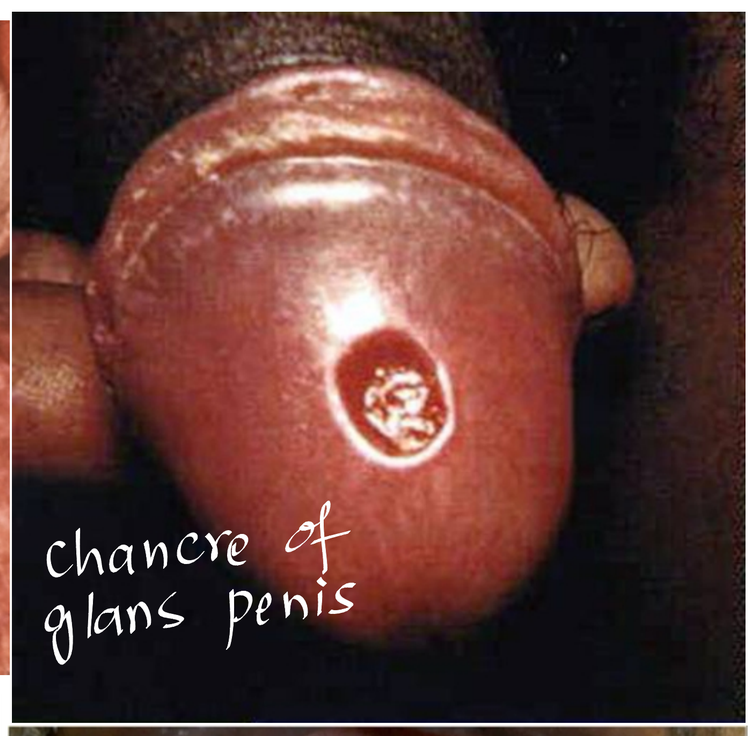
Therapy
First-line therapy: penicillin G

Complications
Vertical transmission (congenital syphilis)
Malignant syphilis (rare; may be seen in immunocompromised individuals)
Treatment complication: Jarisch-Herxheimer reaction

Note
Primary and secondary stages are highly infectious
Coinfection with other sexually transmitted infections is possible (e.g., HIV)
National notifiable condition

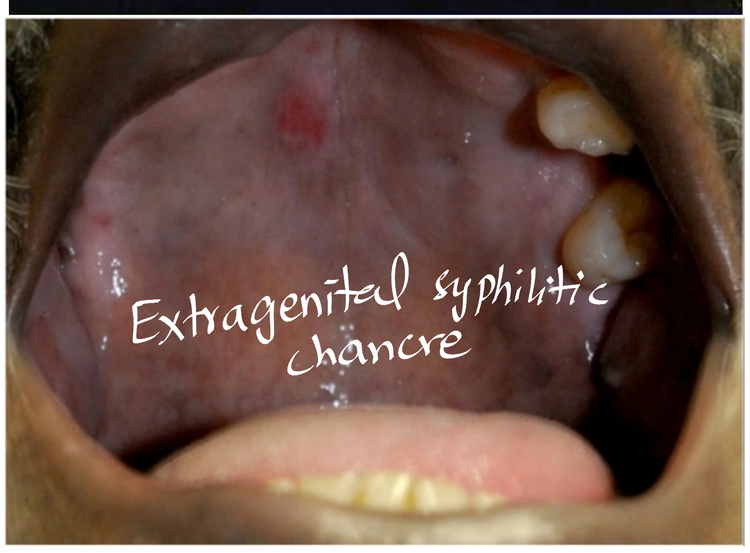



Chancre of Penis (primary syphilis)


Chancre of glans penis


Facial gummas in tertiary syphilis

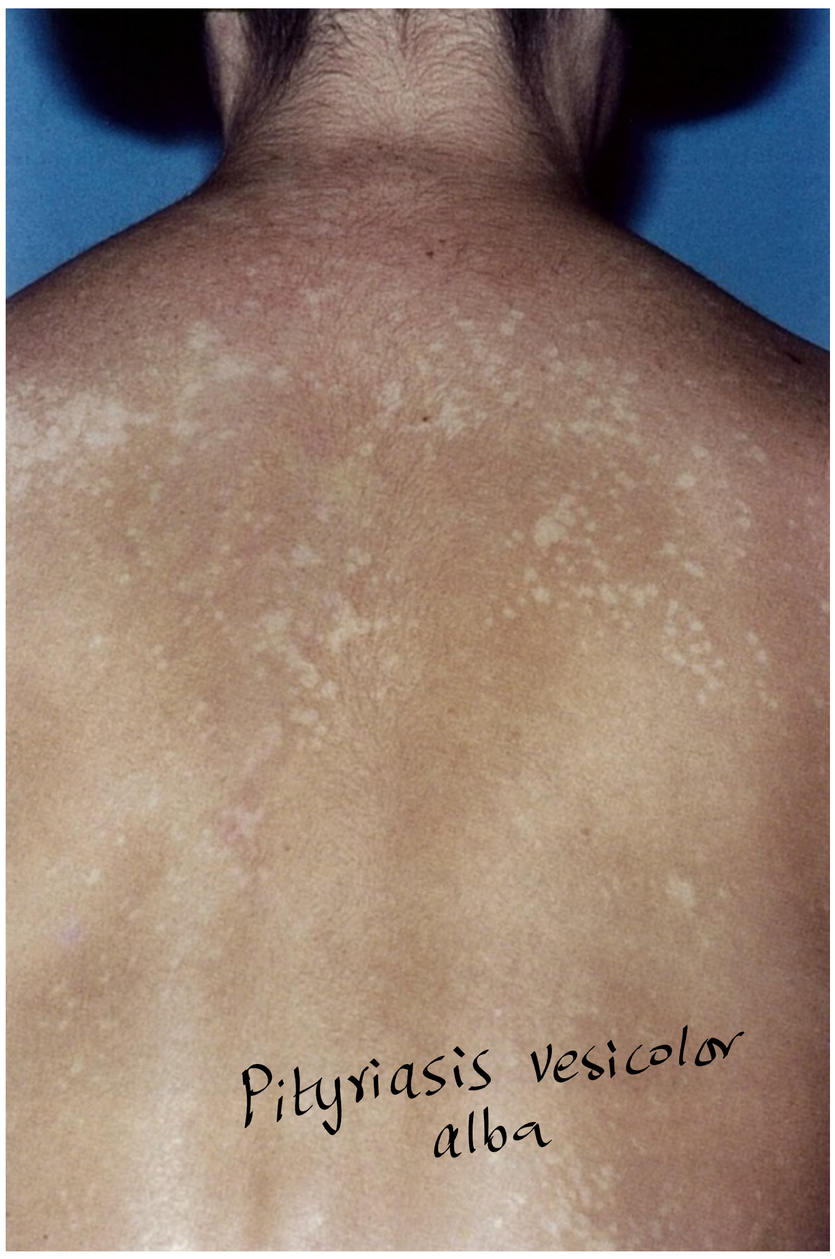

Chancre


Extragenital syphilitic chancre


↑ Maculopapular rash in secondary syphilis

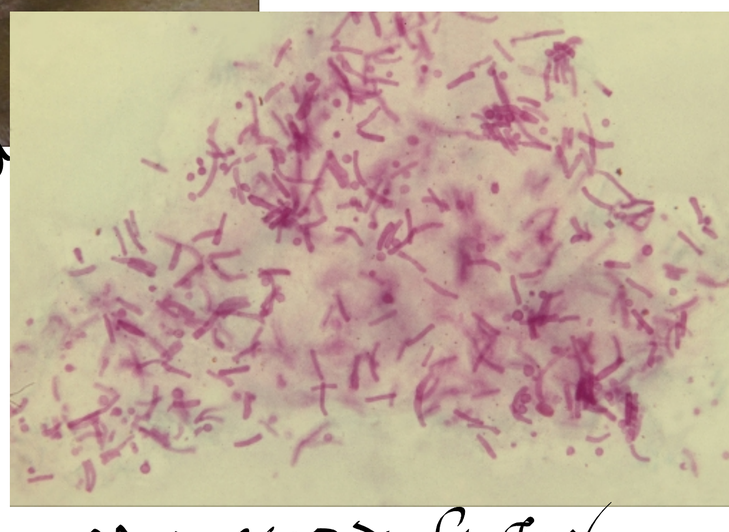

↖ Rash in secondary syphilis

Pityriasis Versicolor (Tinea versicolor)



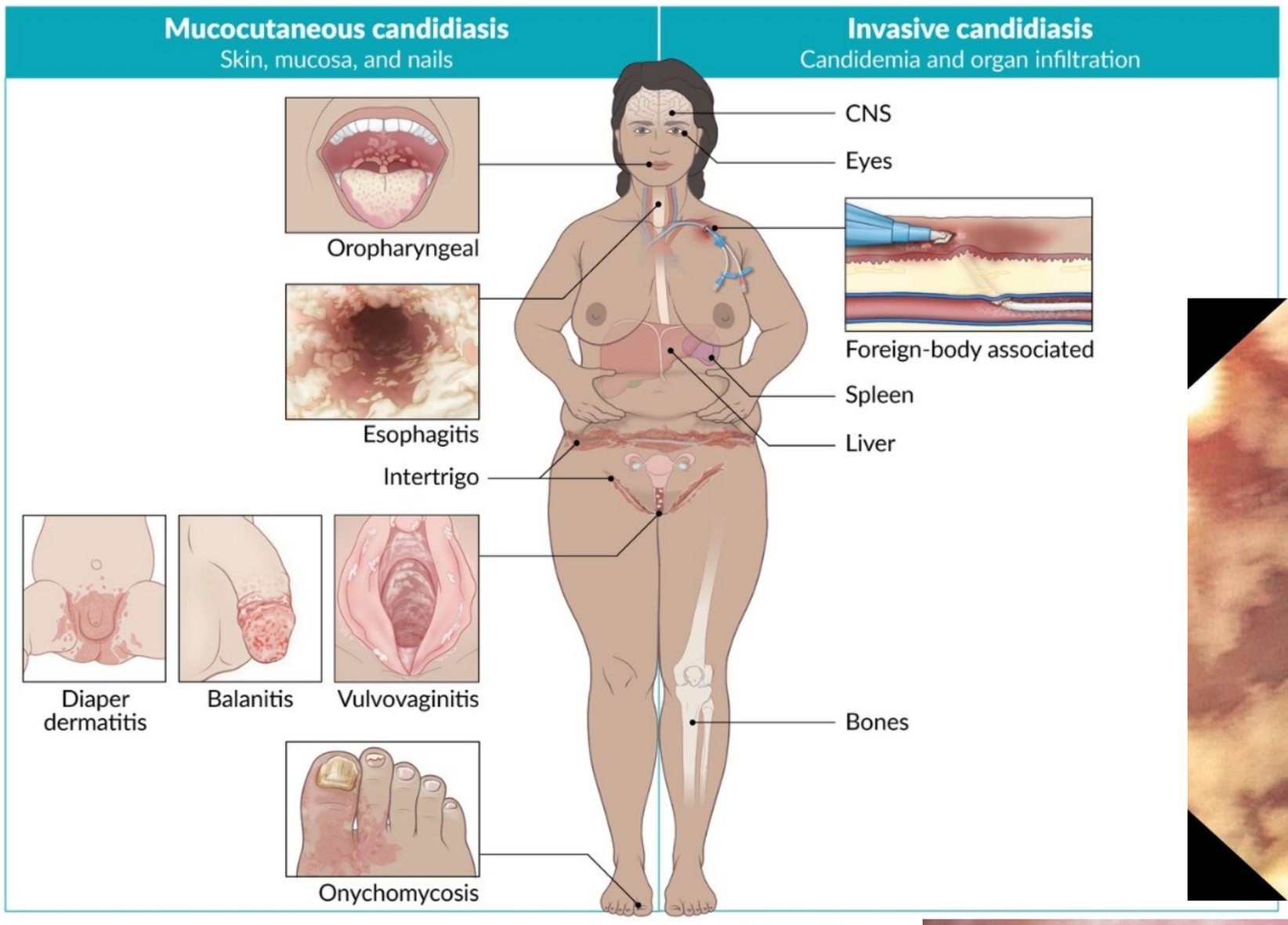
Tinea versicolor alba

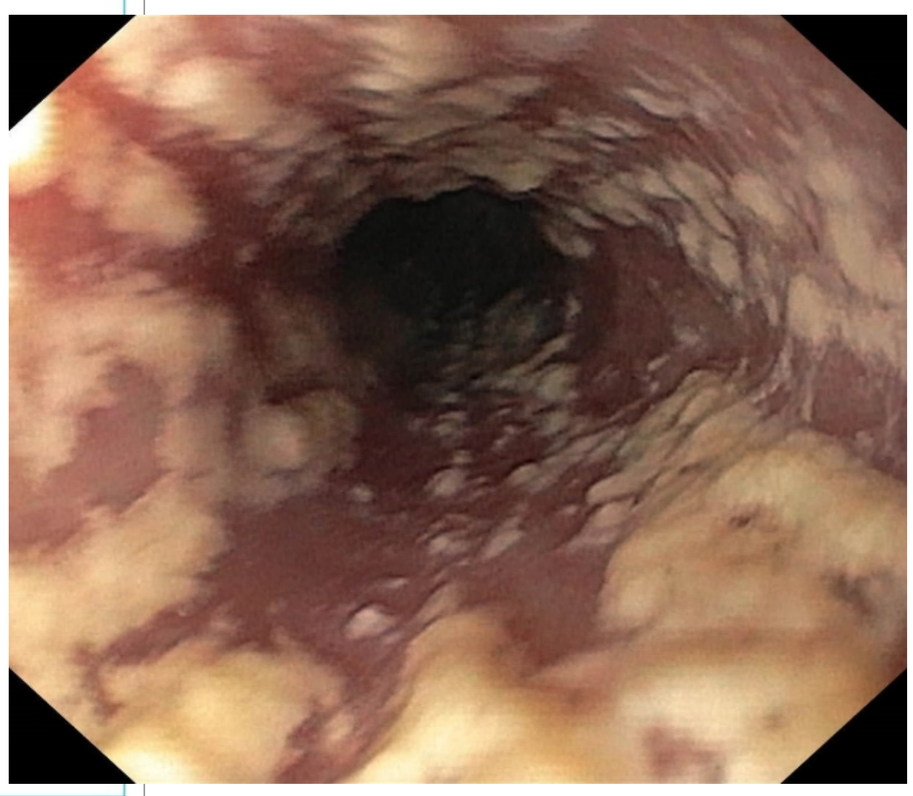
↑ Pityriasis versicolor alba

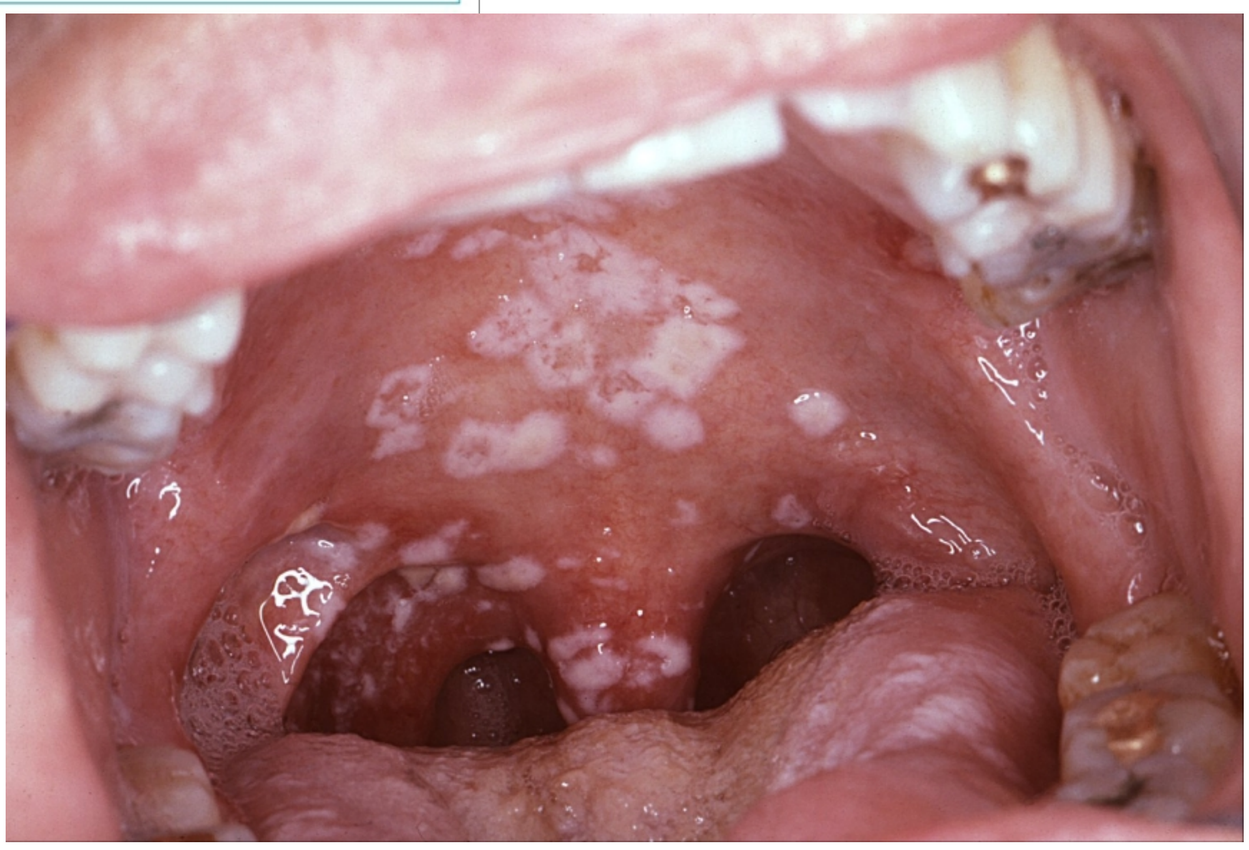


Malassezia furfur

Candidiasis



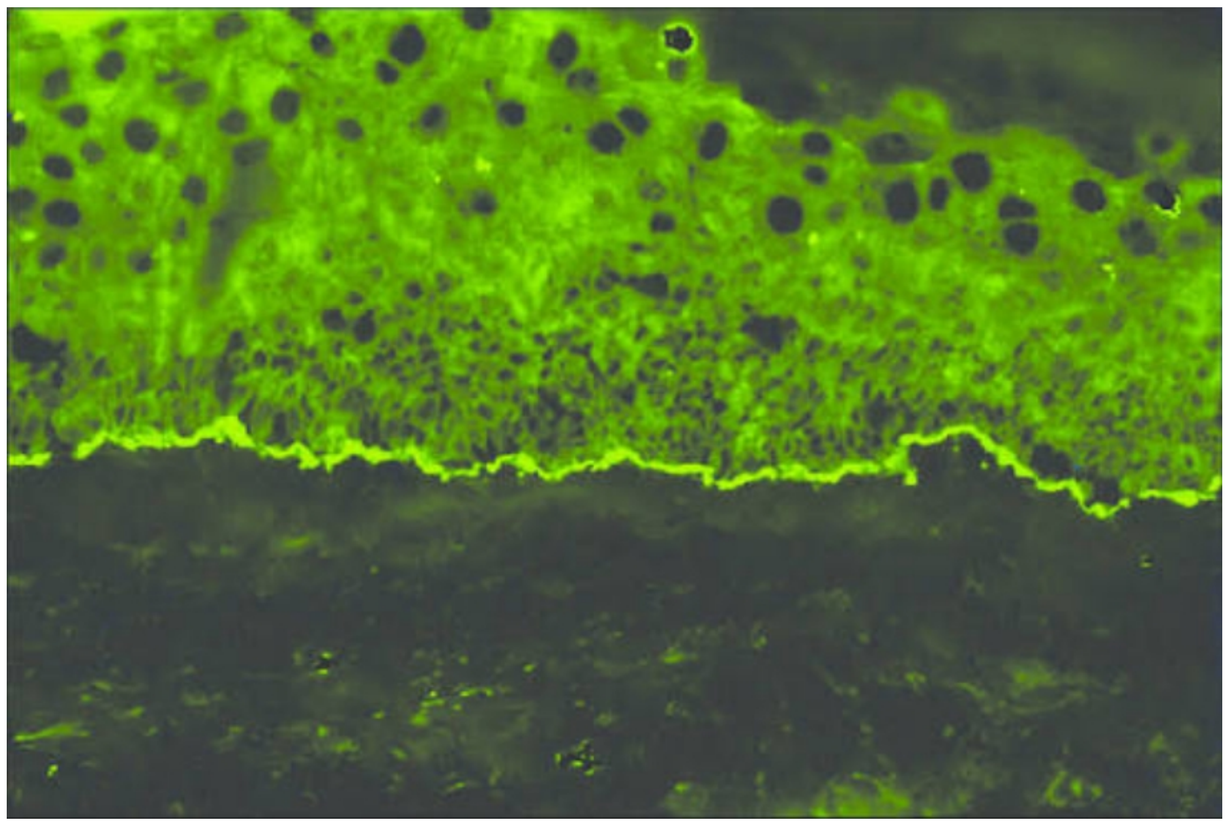




Bullous pemphigoid

* Autoimmune blistering disorder
* Large tense bullae
* Blisters are very itchy
* Blisters are tense and don't rupture easily

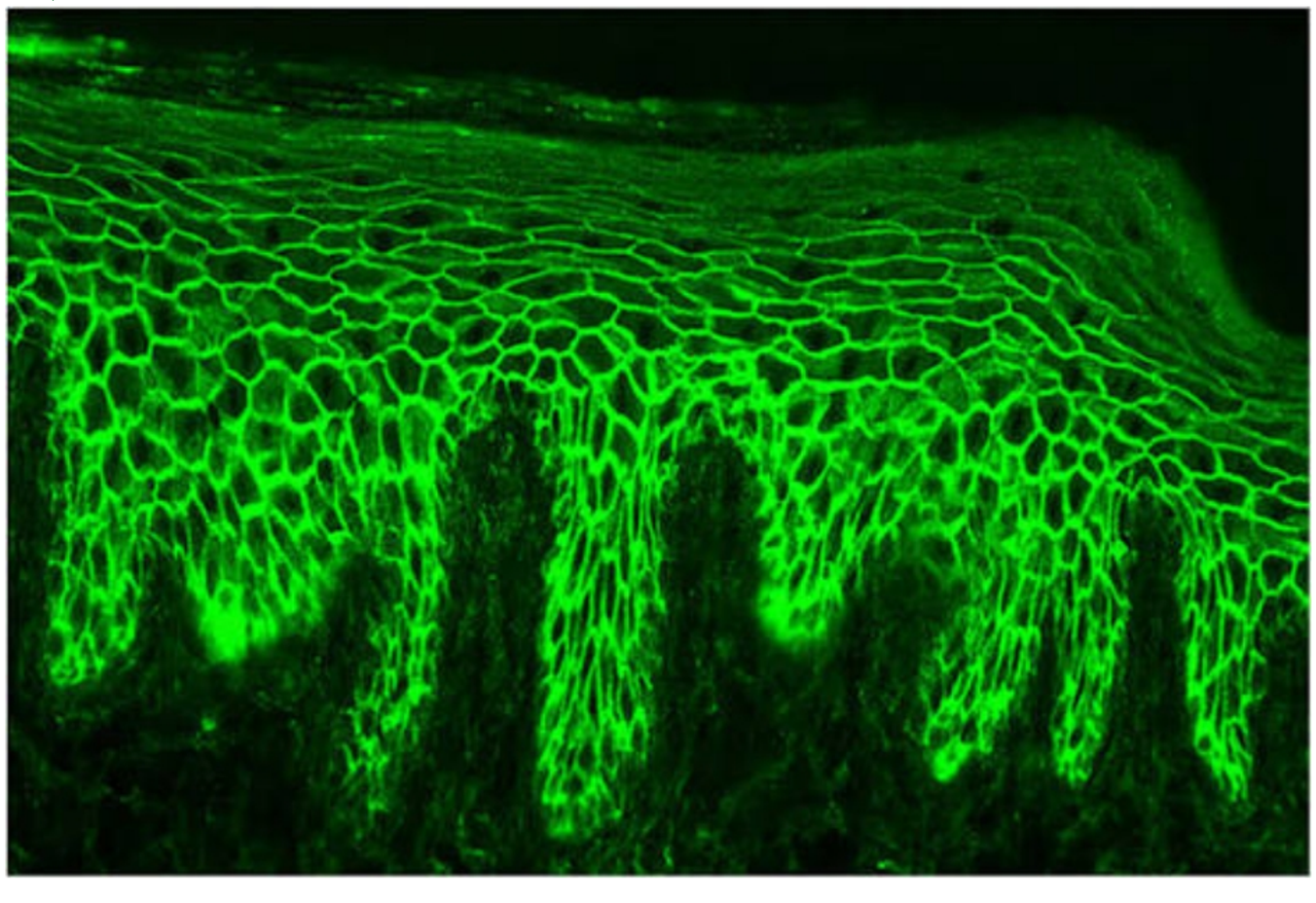
Direct Immunofluorescence → Linear deposition of immunoglobulins along the basement membrane with increased eosinophils in dermis



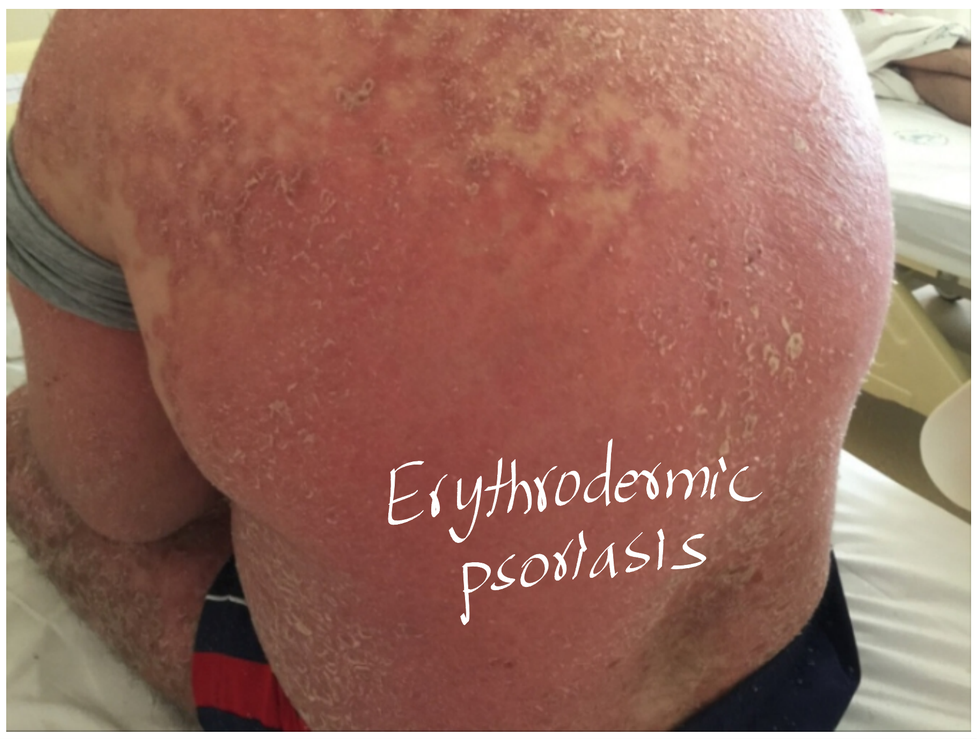
Pemphigus vulgaris

* Auto immune blistering disorder
* Mucosal involvement is common
* Blisters are NOT itchy
* Blisters rapidly denude
* Nikolsky's Sign → separation of superficial skin layers with slight rubbing

Direct IF → Fishnet-like (tombstone) pattern of immunoglobulins surrounding epidermal cells



Psoriasis



* It is a T-cell mediated disease that involves increased keratinocyte proliferation along with inflammation and angiogenesis

↑ Sterile pustules on erythematous base

PSORIASIS

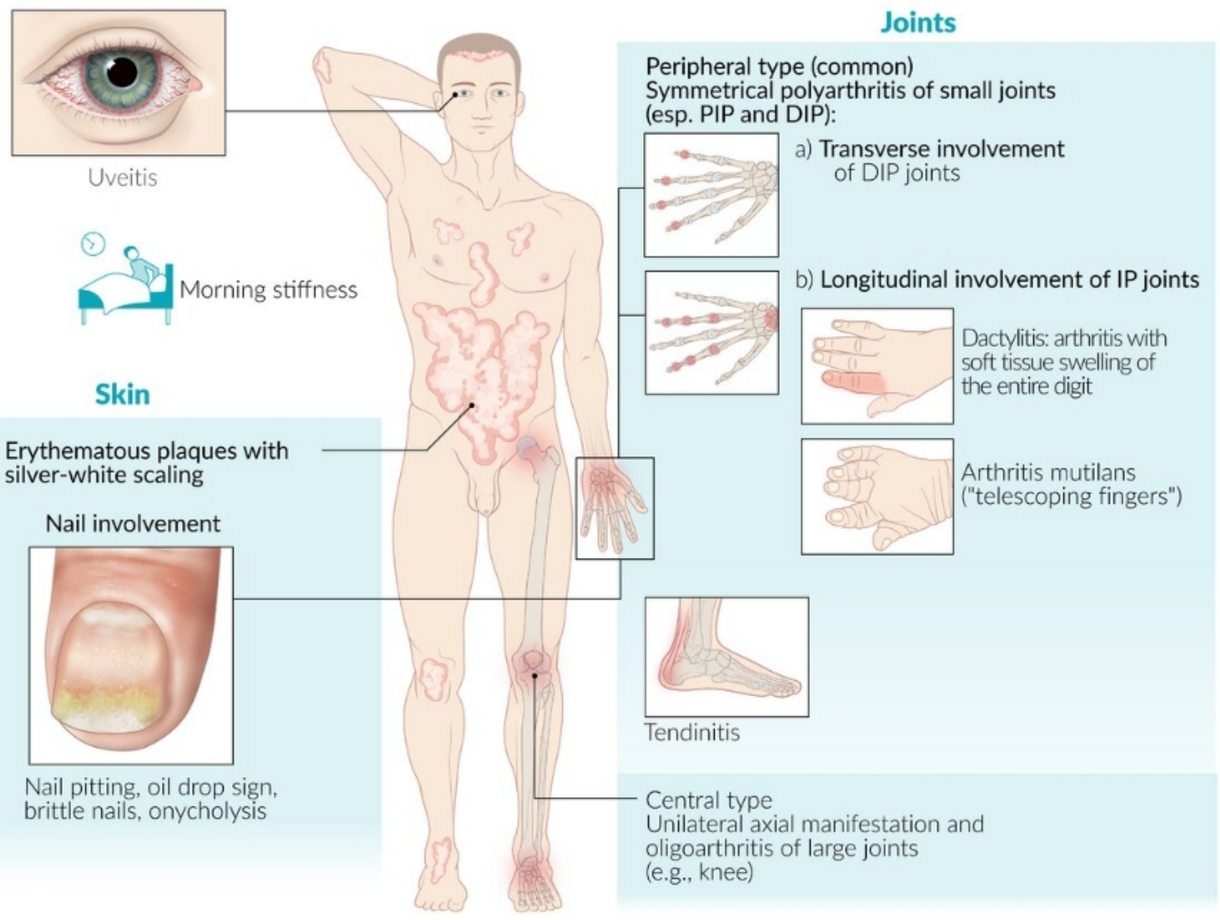
Psoriatic arthritis

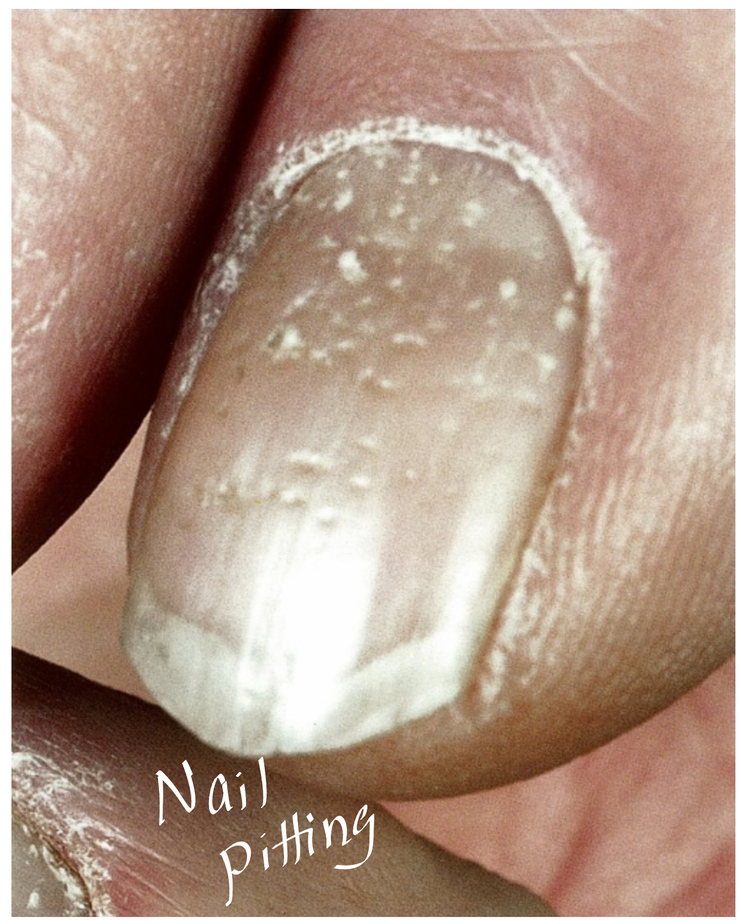
Etiology
Genetic predisposition: associated with HLA alleles (e.g., HLA-Cw6, HLA-B27)
Environmental triggers: infection, trauma, drugs (e.g., beta blockers, aspirin)

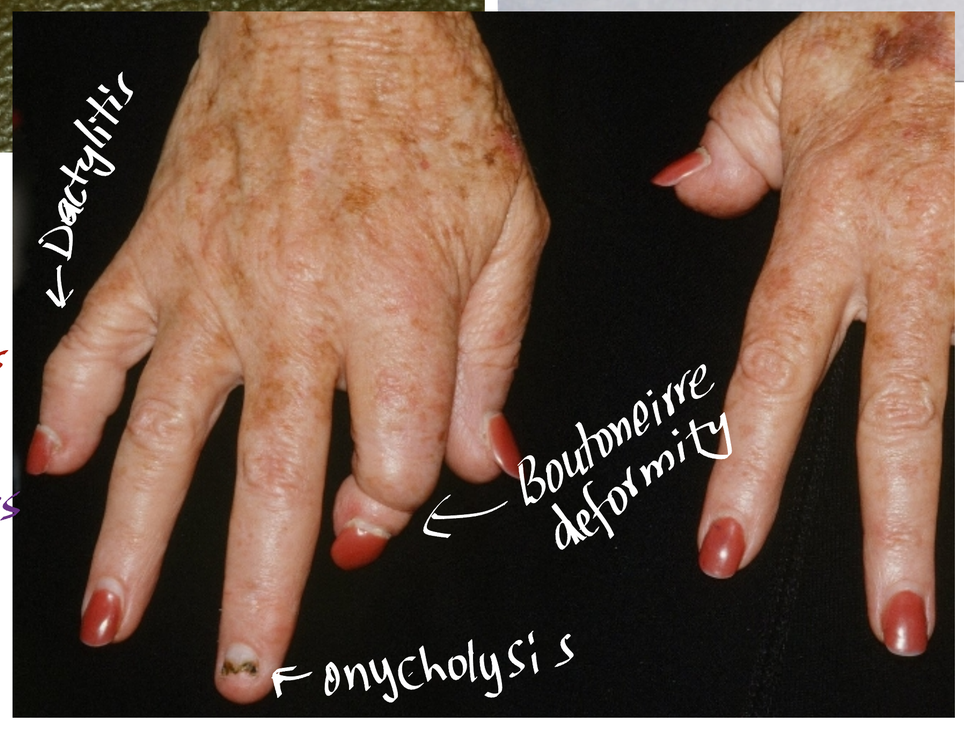
Epidemiology
♂ = ♀
Peak age of onset: bimodal pattern; between 20–30 years and 50–60 years

Serology
Rheumatoid factor negative
Anti-CCP antibodies positive in 10–15% of patients

Note
Psoriasis typically precedes arthritic symptoms by several years.
Nail changes are typically associated with arthritis of the small joints.







* Auspitz Sign i.e visible pinpoint bleeding when scale is removed from plaque due to thin epidermis above dermal papilla
* Koebnerization i.e new plaques occur at sites of repeated trauma

Lichen planus

Etiology

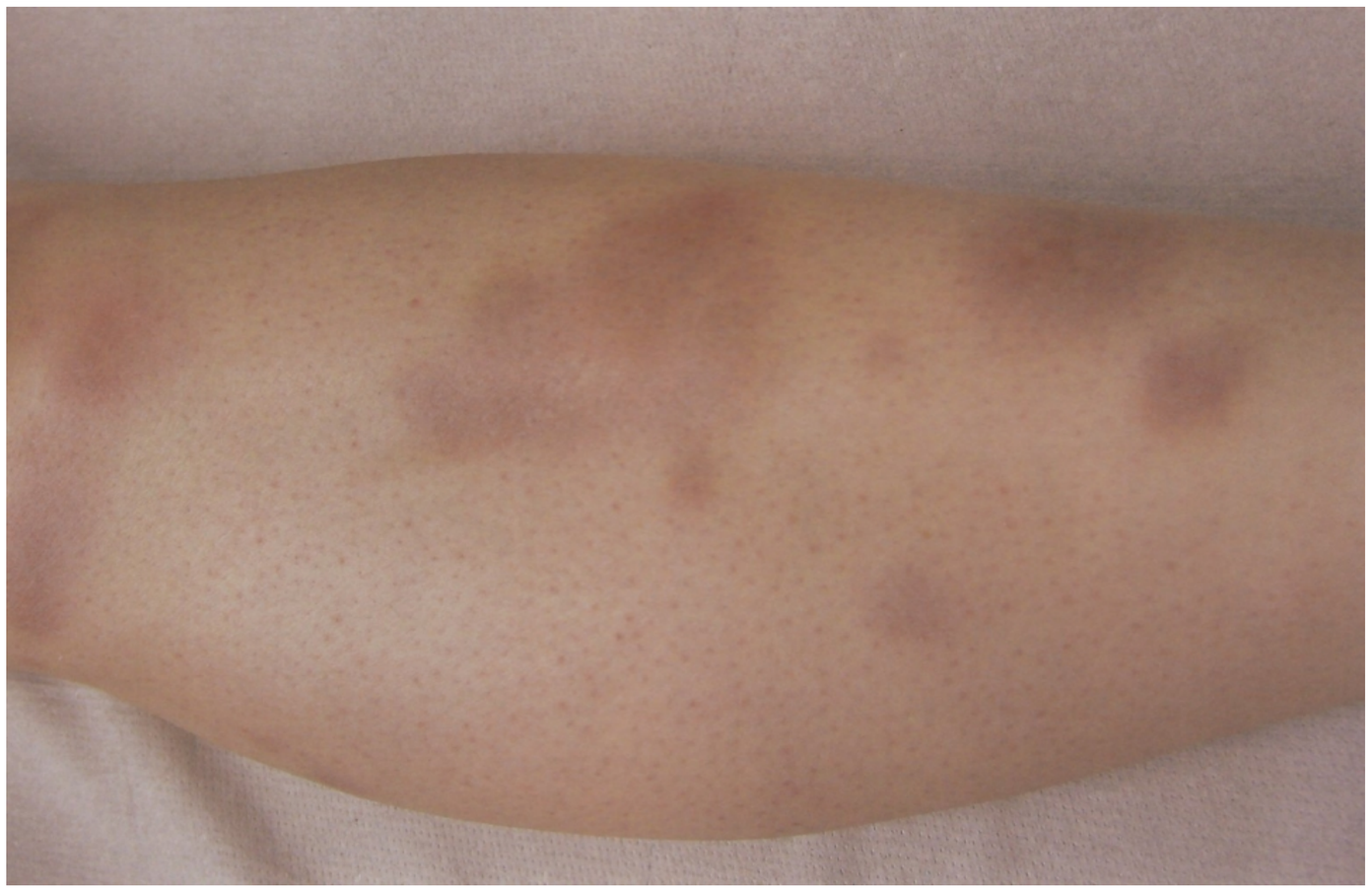
Although the exact etiology of lichen planus is unknown, possible etiologic factors include:

- Altered immune response with T cell activation [5][6]
- Associated with **hepatitis C infections** [7]

Clinical features

Lichen planus can affect the skin, mucosa, scalp, genitalia, and nails, and manifests with varying symptoms depending on the subtype of the disease.

Erythema Nodosum



* painful, erythematous nodules on lower legs and sometimes forearms

Erythema Multiforme

Severe forms of erythema multiforme

Steven's Johnson Syndrome

(epidermal separation of <10% of body surface area)

Toxic epidermal necrolysis

(epidermal separation of >30% of body surface area)

Both show Nikolsky's sign i.e separation of superficial skin layer with slight rubbing

Urticaria (Hives)

* Characterized by superficial, intense erythema and edema in a localized area

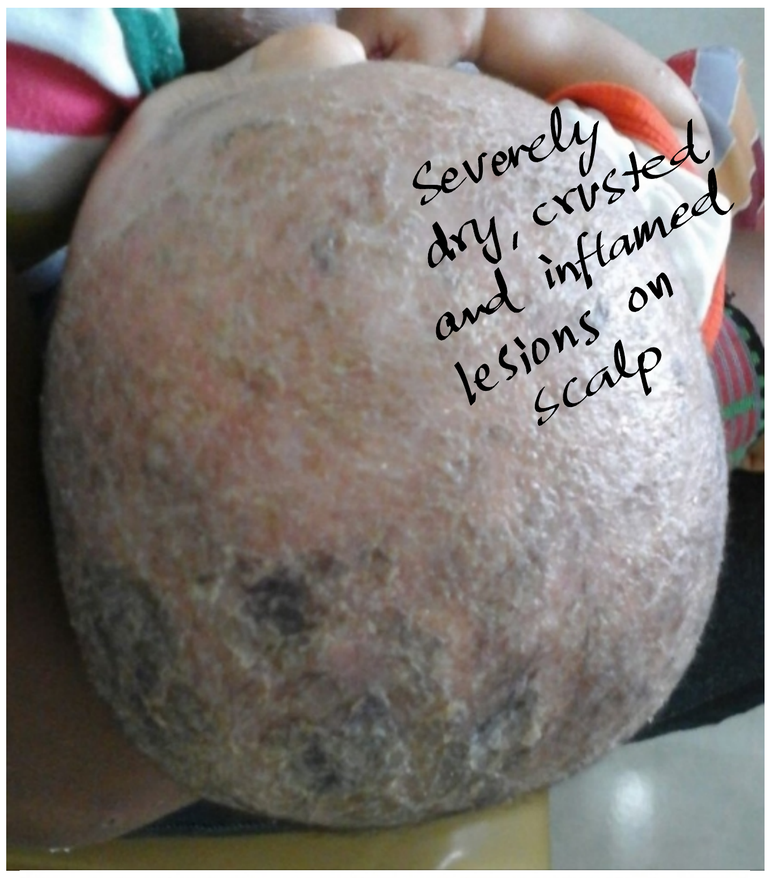
* Tx → Anti histamines

Common Triggers

- **Allergens**: Foods, medications, or insect stings often cause hives.
- **Environmental Factors**: Pollen, sunlight, or water exposure can act as triggers.
- **Stress**: Emotional or physical stress may worsen symptoms.

ECZEMA (DERMATITIS)

Atopic Dermatitis



✱ genotypically complex familial disease with strong maternal influence

Seborrheic Dermatitis

- overgrowth of fungus, Malassezia furfur
- predilection for areas with oily skin

Allergic Contact Dermatitis

List of Important Autoantibodies

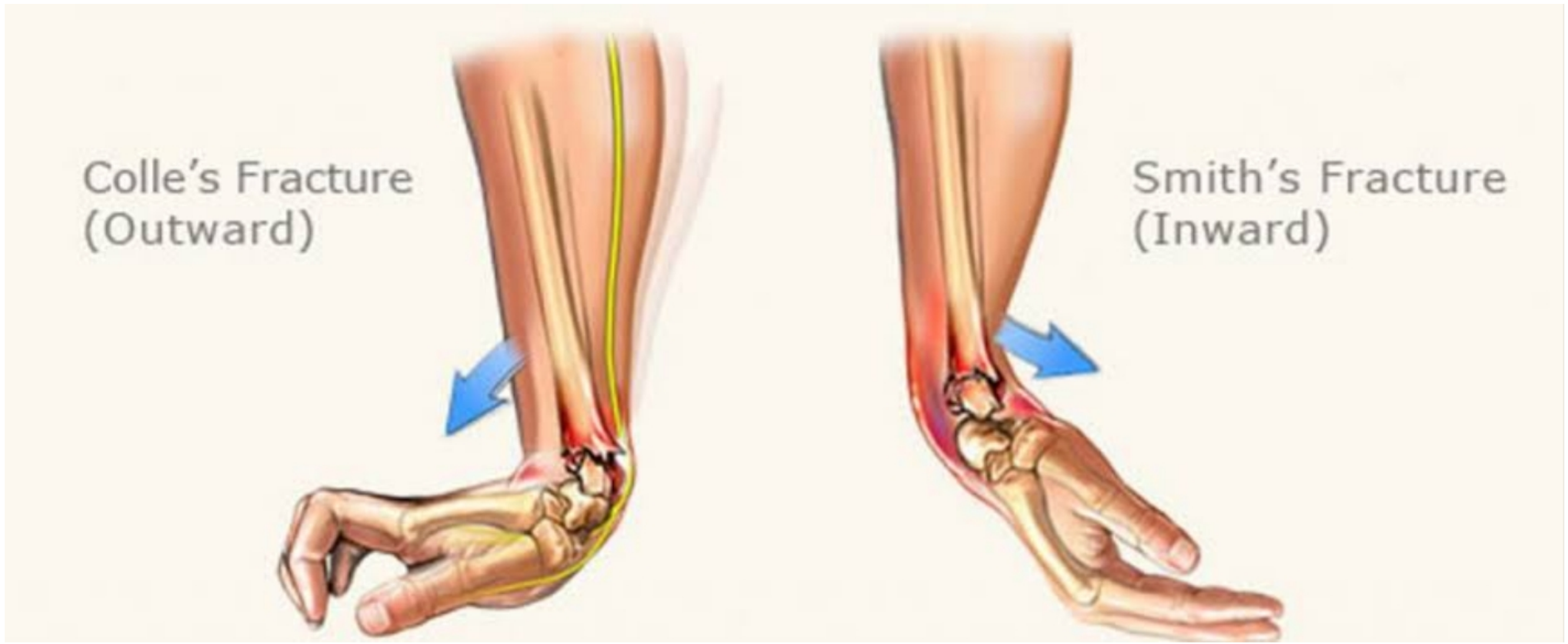
Anti-dsDNA and Anti-smith	SLE (but the initial test is ANA – very sensitive)
Anti-histone	Drug-induced lupus (e.g. Hydralazine)
Anti-scl70	Systemic Sclerosis
Anti-centromere	Limited sclerosis/CREST syndrome
Anti-Jo1	Polymyositis
Anti-Ro, Anti-La	Sjogren's disease
Anti-mitochondrial	Primary biliary cirrhosis
Anti-smooth muscle	Autoimmune hepatitis
pANCA	Churg Strauss (Eosinophilic Granulomatosis with Polyangiitis) / UC / 1ry sclerosing cholangitis.
cANCA	Wegener's Granulomatosis (Granulomatosis with Polyangiitis)
Anti-tissue transglutaminase, Anti-gliadin, Anti-endomysial	Celiac Disease
ANA	RA, SLE (initial), and many other auto-immune diseases.

Scalpel blades for cutting

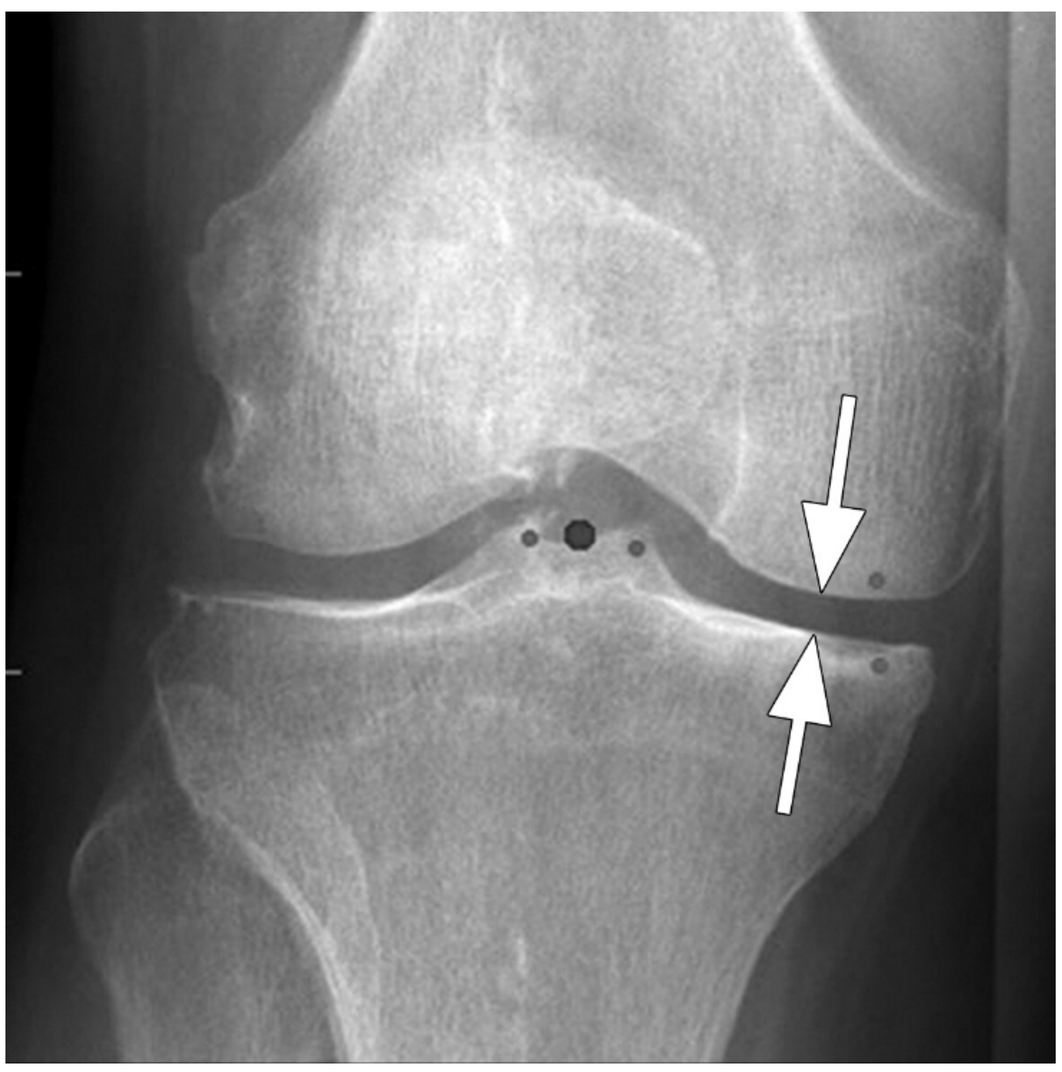
Grasping Instruments

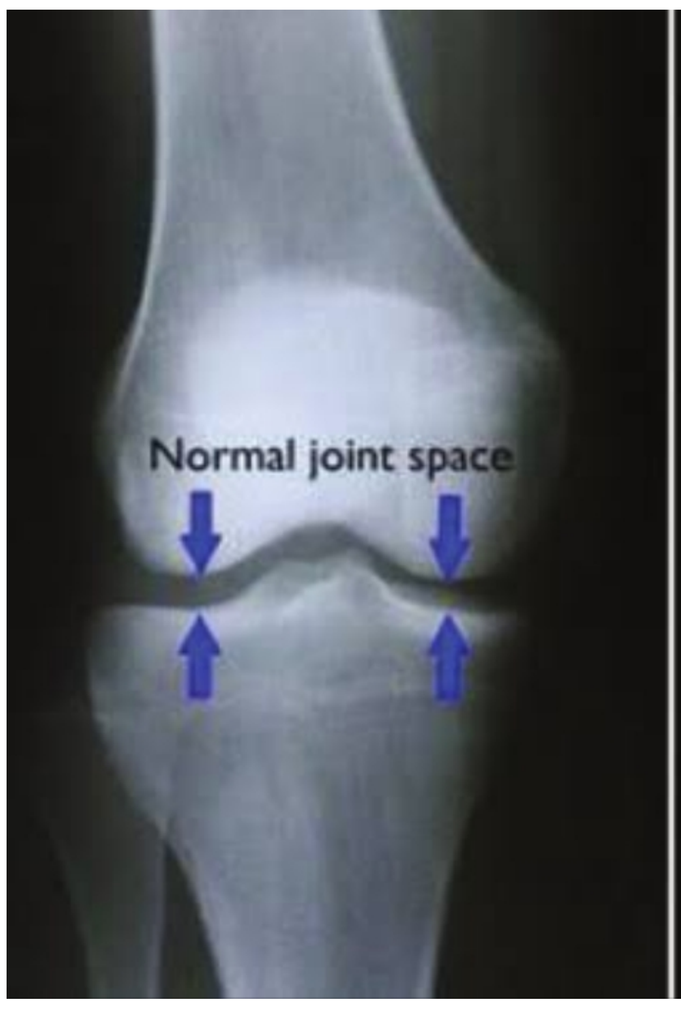
Toothed forceps: Skin (prevents slippage)

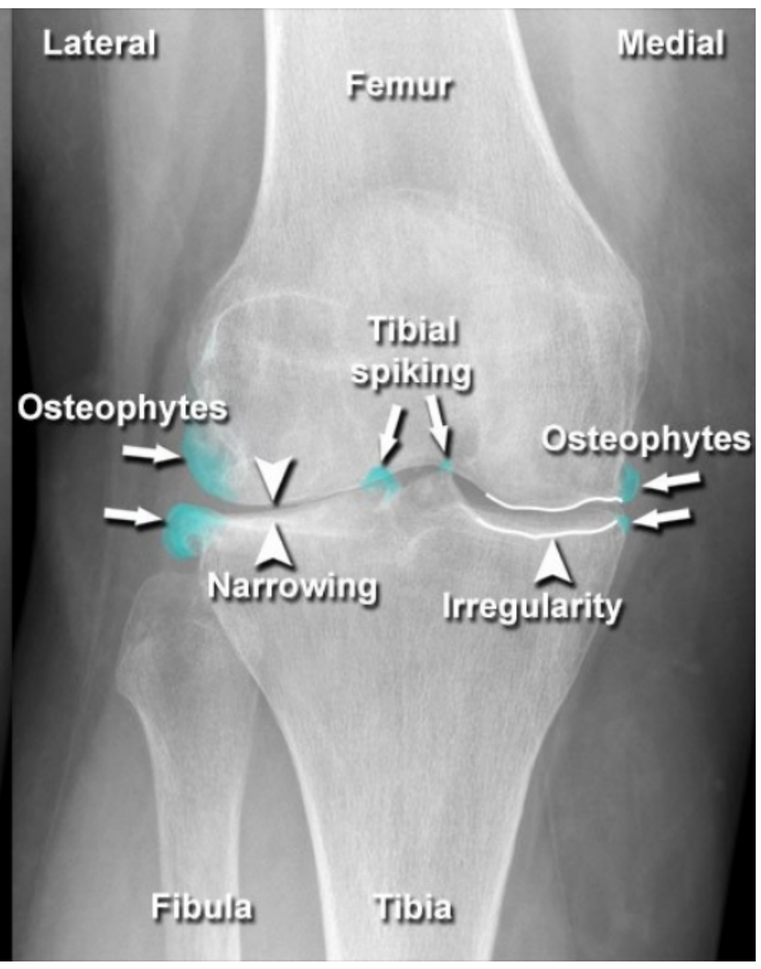
Non-toothed forceps: Viscera, bowel


Colle’s Fracture
(Outward)
Smith’s Fracture
(Inward)

Osteoarthritis







Rheumatoid Arthritis

Figure 1

Figure 2

Cutaneous manifestations of systemic diseases

Lupus
- photosensitivity
- Malar rush - butterfly rash
- Discoid Lupus - round scarring lesions on light exposed areas 5:57 AM

Dermatomyositis
- Heliotrope rash - violaceous color over upper eyelids
- Gottron's papules - flat topped violaceous papules over knuckles of hands 5:58 AM

Scleroderma (Sclerosis)
- Tight and thickened skin due to excessive collagen deposition
- Sclerodactyly
- Telengiectasia and calcification
- Raynaud's phenomenon 5:59 AM

Diabetes
- Shiny atrophic red or yellowish plaques with Telengiectasia over their surface + ulceration 6:00 AM

Cutaneous manifestations of systemic diseases

Cushing Syndrome
- Moon face
- buffalo hump - fat deposit over upper back
- Central obesity
- Atrophy of skin and striae
- Purpura
- Hirsutism
- acne

*

Edited

Addison Disease
- Hyperpigmentation

Chronic Liver disease
- Jaundice
- spider Telengiectasia
- acne
- gynecomastia
- purpura
- palmar erythema

Hypothyroidism
- dry and cold skin
- Myxedema - edematous skin
- hair loss of lateral third of eyebrows
- brittle hair or nails
- yellowish hue to skin due to carotenemia

Hyperthyroidism
- Tibial myxedema
- Thin and fine hair
- Onycholysis - brittle nails
- hyperpigmentation or vitiligo