بسم الله الرحمـٰن الرحيم

{وَإِذَا مَرِضْتُ فَهُوَ يَشْفِينِ}

PBL Clinical | FINAL 2

Rheumatology

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Introduction to Rheumatology

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Lecture Outline

- Introduction to rheumatology and its clinical relevance
- Overview of key rheumatologic diseases
- Approach to joint pain: inflammatory vs mechanical
- Clinical relevance of the patterns and distributions of joint involvement
- Basic pathology of rheumatic diseases
- Key dermatological manifestations of rheumatologic diseases
- Principles of treatment: NSAIDs, corticosteroids, DMARDs, biologics, urate-lowering therapy

Overview of key rheumatological diseases

Inflammatory Arthritis

RA, psoriatic arthritis, reactive arthritis, ankylosing spondylitis

Connective Tissue Disease

SLE, systemic sclerosis, inflammatory myopathies, Sjögren syndrome

Crystal Arthropathy

Gout and calcium pyrophosphate deposition disease

Degenerative Disease

Osteoarthritis and periarticular mechanical disorders

Vasculitis

ANCA vasculitis, GCA, Behcet's disease

The First Branch Point: Inflammatory vs. Mechanical

Inflammatory		Mechanical
> 30-60 min; improves with movement	**Morning Stiffness**	< 30 min; gelling after rest
Often worse after rest or at night	**Pain Pattern**	Worse with use; relieved by rest
Warmth, swelling, tenderness, effusion	**Exam Findings**	Bony enlargement, crepitus, limited ROM
Fatigue, fever, weight loss, rash	**Systemic Clues**	Usually absent
RA, gout, psoriatic arthritis	**Classic Examples**	OA, tendinopathy, bursitis

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The patient complains of joint pain so we ask, “does the joint that hurts u in the morning feel stif(inability to move the joint). DURATION MATTERS

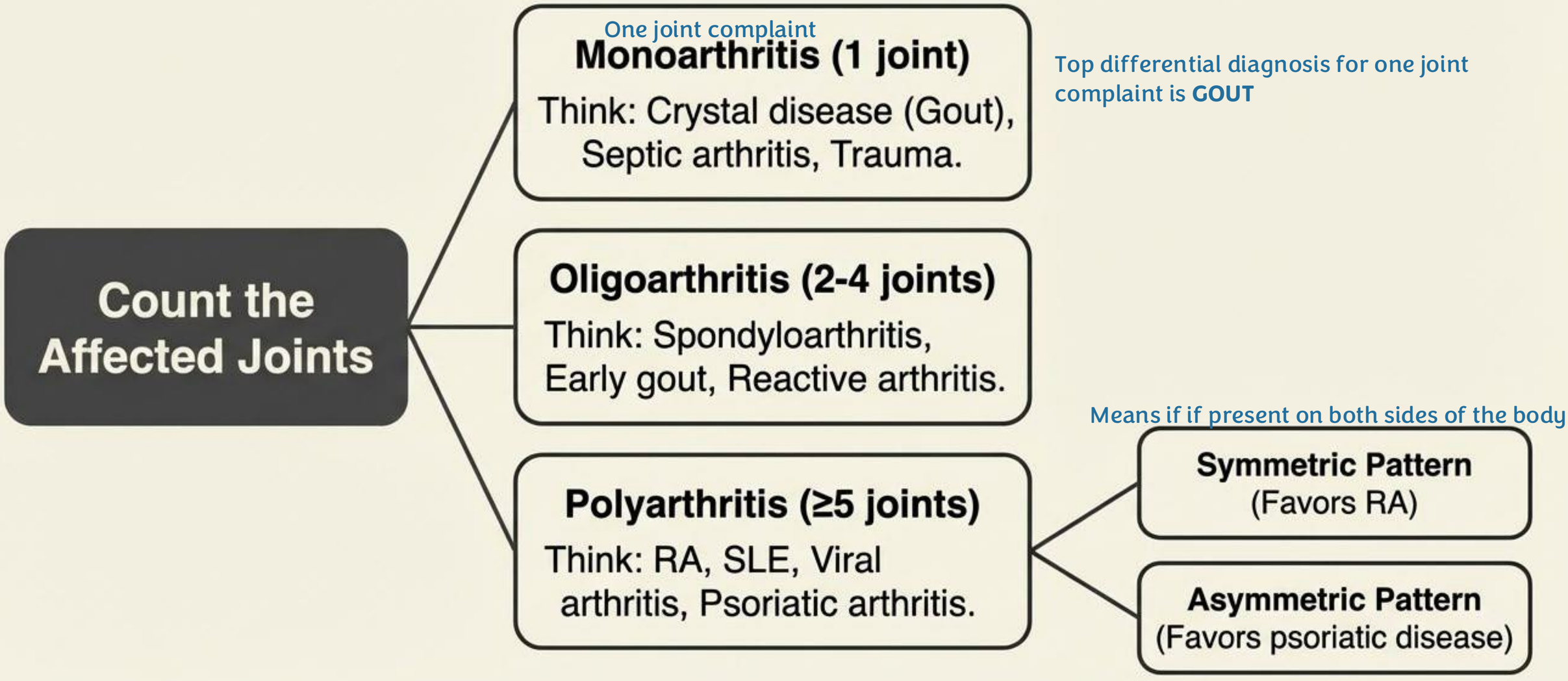
The more they use it, the more mobilization of inflammatory component in the joint, giving them relief (improved symptoms or less pain with exercise)

Patient will tell u that they have joint paint, BUT, the more they use it the more it hurts—> mechanical , because it indicates to us that the joint is degenerating from inside (shoulder tendinitis or inflammation of tendon).

If u see the 5 cardinal signs of inflammation (erythema, warmth, swelling, pain and loss of function).

Systemic complaints: feeling tired, fatigue, fever and night sweats

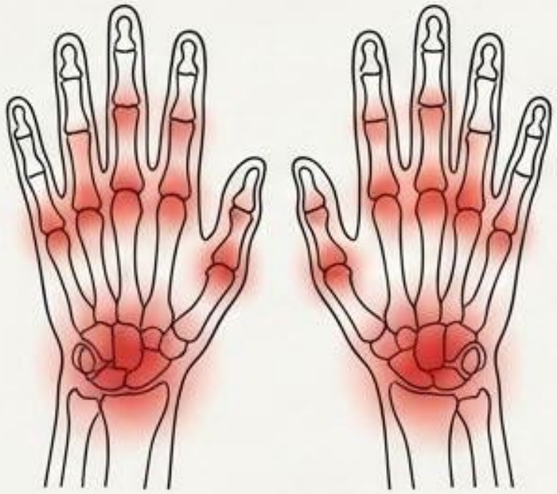
The Diagnostic Funnel: Joint Count & Symmetry



Which joints are effected

The Anatomy of Distribution

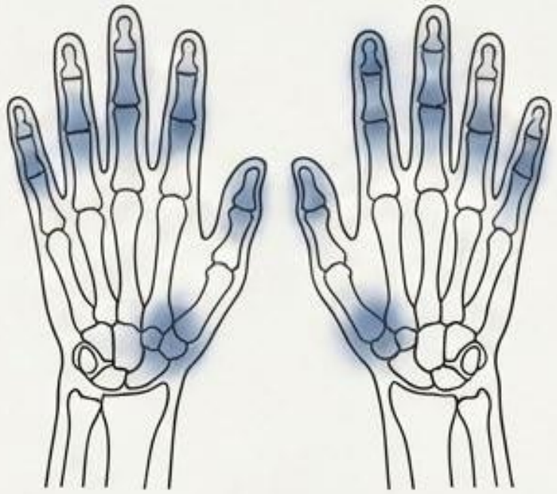
CLASSICALLY Spares DIP joint, must effect the hand joint (wrist, metacarpal, proximal interphalangeal)



RA Signature

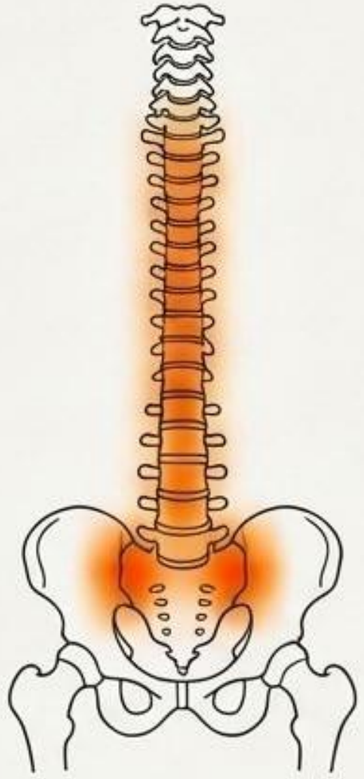
Small joints of hands/wrists (MCP, PIP).

Effects distal interphalangeal joint and base of thumb, NEVER THE WRIST JOINT (MCP)



OA Signature

DIP involvement, 1st CMC.



SpA Signature

Axial spine and sacroiliac joints.

When attack happens it usually presents as acute in one joint (usually big toe).
Classic gout or podagra.

Gout Signature

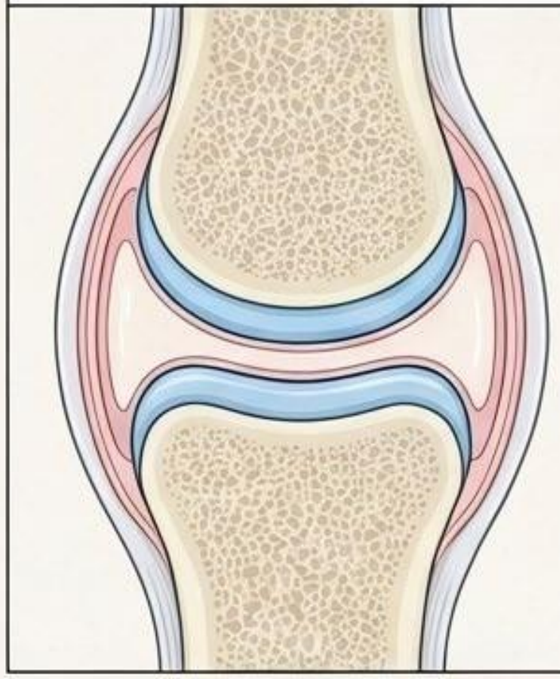
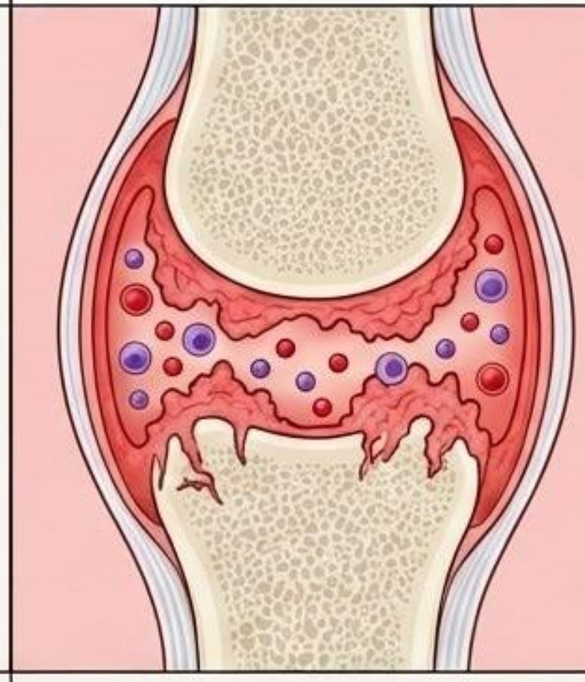
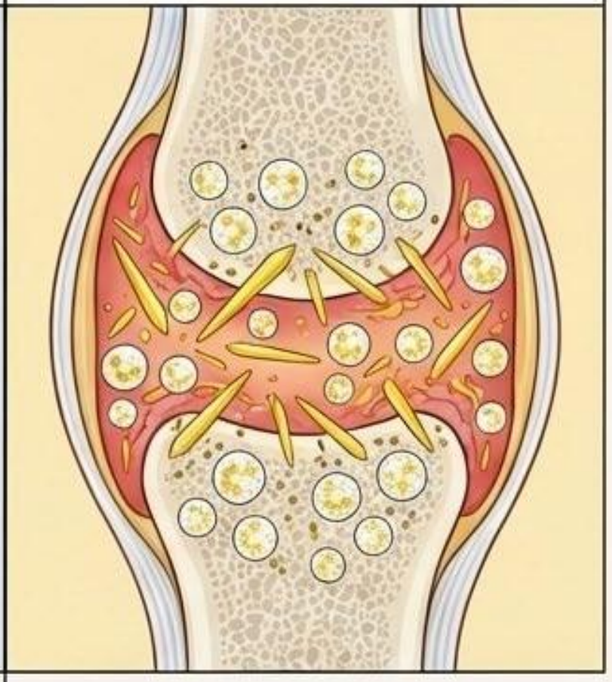
First MTP joint.

Pathology Dictates Presentation

Autoimmune, inflammation, cytokines activated, results in pannus formation

Not autoimmune

Crystal deposition from high uric acid lvl (hyperuricimia) cause attacks .

Normal	Rheumatoid Arthritis	Osteoarthritis	Gout
			
Normal • Intact cartilage and smooth synovial lining. • Clear, well-defined joint space.	**Rheumatoid Arthritis** • Chronic, systemic inflammatory synovitis. • Pannus formation erodes cartilage and bone.	**Osteoarthritis** • Progressive cartilage degradation and loss. • Bony remodeling and osteophyte formation.	**Gout** • Acute inflammation from urate crystal deposition. • Intense neutrophilic response in the joint.

RA Rheumatoid Arthritis (RA) – Part 1

Definition

Chronic autoimmune synovitis causing symmetric polyarthritis. Without treatment, persistent synovitis leads to erosion and deformity.

Clinical Manifestations

Morning stiffness > 1 hour; swelling of MCP, PIP, and wrists; notable sparing of the DIP joints.

Female-to-male 3:2, typically fourth and fifth decades.

- Small joints of hand.
- Patient present with inflammatory complaints, morning stiffness, swelling in MCP and PIP, sparing DIP.
- Disease of women, usually in middle age groups in 4-5th decade

Any complaint in the joint that exceeds 6 weeks → chronic
Less than 2 weeks → acute
Between 2-6 → subacute

Rheumatoid arthritis

- MCP are swollen, this Hand is in advanced stage , they weren't treated causing hand deformity (Ulnar deviation of fingers and extension of PIP)
- If not treated:

1) Deformity
2) Dislocation or subluxation of joint

Figure 1

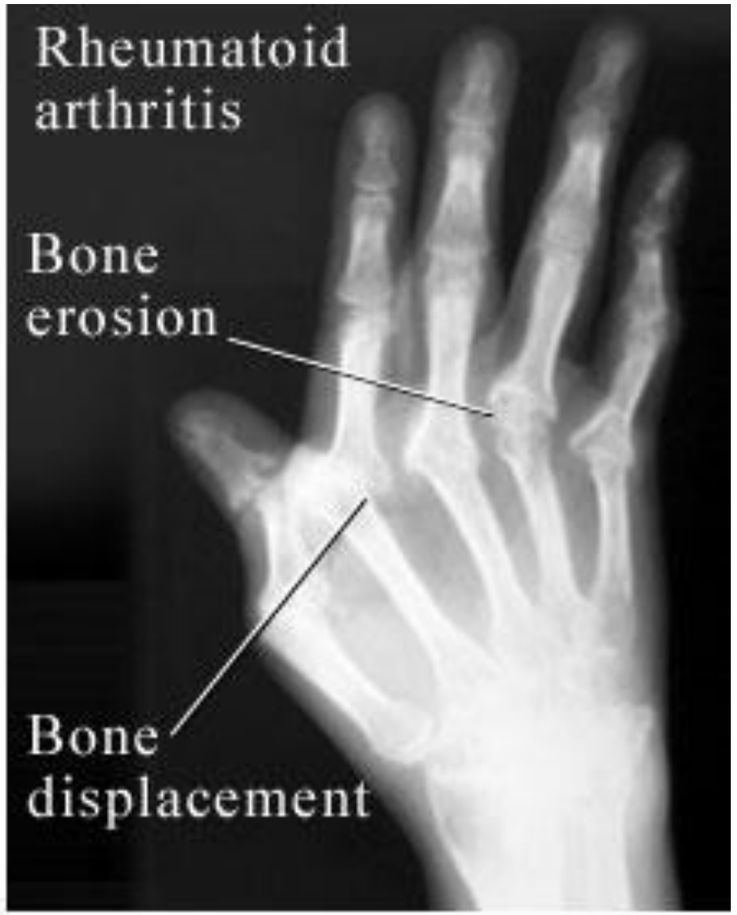


Figure 2

- Damage and erosion of joint from chronic inflammation and pannus formation in joint resulting in subluxation

OA Osteoarthritis (OA)

Definition

Degenerative joint disease characterized by focal cartilage loss and compensatory osteophyte formation.

Chronic, results from degeneration of joint. Patient complains of pain that worsens with use or movement indicating joint from inside is degenerating

Clinical Manifestations

Pain is gradual, activity-related, and worse after weight-bearing. Brief stiffness after rest. Exam reveals crepitus and bony enlargement (Heberden nodes at DIP, Bouchard nodes at PIP).
Typically females after the age of 50.

MCP are normal cause OA doesn’t effect them
PIP are enlarged but if u feel it, it will feel bony not soft tissue (in RA u will feel synovitis as spongy feeling)
DIP enlarged (RA never effects DIP).

OA patient

Gout: Crystal Arthritis

attacks triggered by stress, medication or dehydration resulting in deposition of crystals which go to a joint and causes locals inflammation.
Most effected is the 1st MTP of big toe.
Usually effects Males in 4th–5th decade, 20 yrs after puberty.

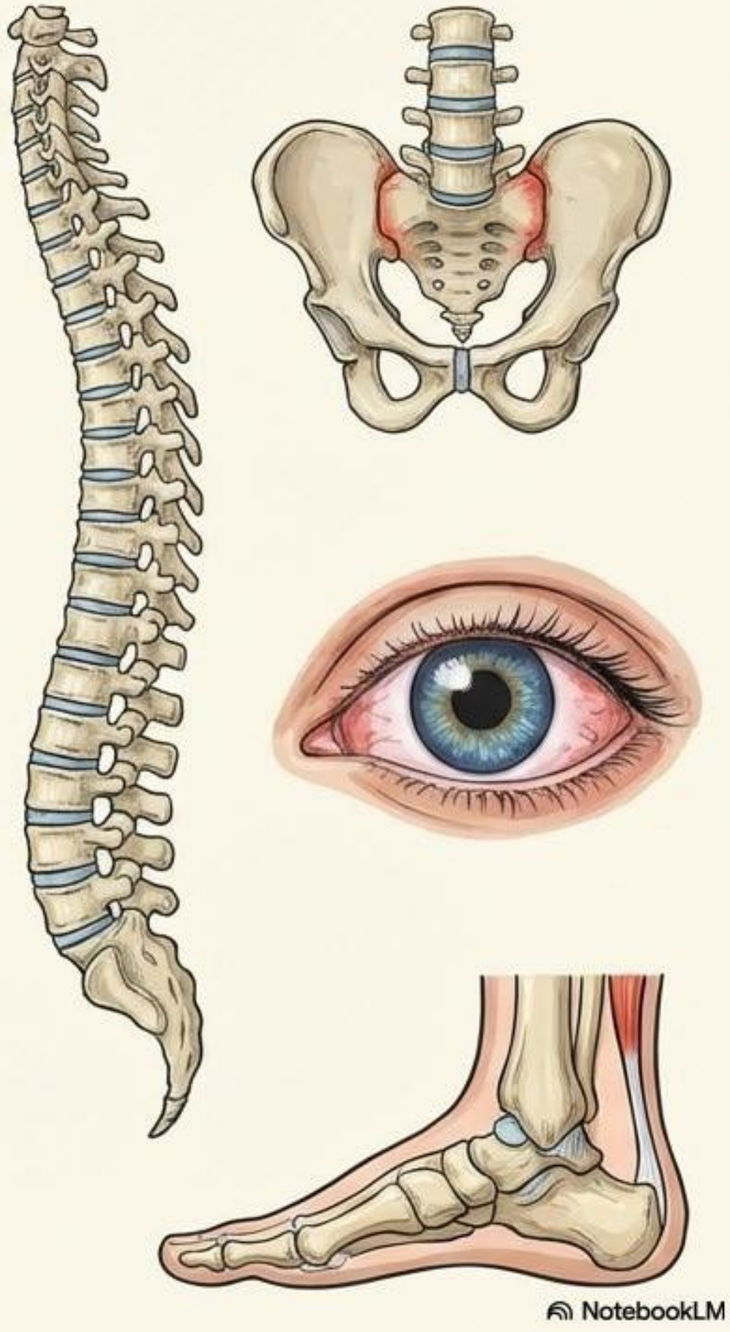
Definition	Clinical Manifestations
Deposition of monosodium urate triggering intense, abrupt neutrophilic flares.	Abrupt, exceptionally painful monoarthritis. Initial attack often strikes the 1st MTP (podagra). Presence of tophi in chronic cases; typically males 20 years after puberty.

Local sign of inflammation, swelling erythema, very painful to move and very tender if touched.

SpA Spondyloarthritis (SpA)

Definition (Core Pathology)	Clinical Manifestations (Key Bedside Clues)
Inflammation of the axial skeleton, sacroiliac joints, and entheses (tendon insertions), often starting in young adults.	Typically males 16-40 years of age. Inflammatory back pain (nocturnal, morning stiffness) ± asymmetric lower-limb arthritis. Manifestations outside spine Look for heel pain (Achilles enthesitis), dactylitis (sausage digits), a red painful eye (uveitis), or a history of psoriasis/IBD.

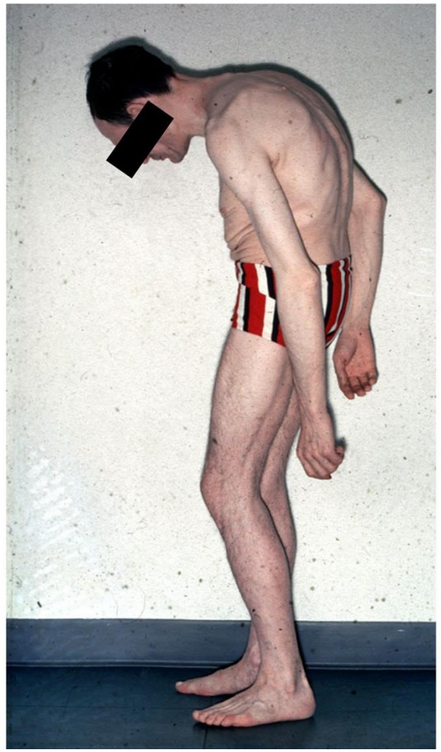
Effect spine mostly plus sacroiliac. Axial spondyloarthritis (ankylosing spondylitis) effects male between 16-40. classic presentation includes back pain that is inflammatory (morning stiffness and improves with use)



- Inflammation of spine that wasn’t treated.
- They will start to lose height and their spine starts to fuse.
- patients cannot touch toes or bend a little bit forward.
- Cervical and thoracic can fuse limiting their mobility

Final Stage of AS with Severe Kyphosis of Thoracic and Cervical Spine

Unable to look ahead while walking
(‚patient cannot see the sun‘)

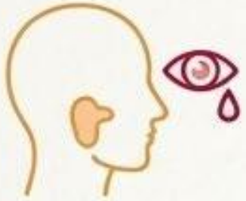


Fused spine that looks like bamboo (bamboo spine)

Connective Tissue Diseases (CTDs)

Systemic, involve diff organs
Skin rash, auto sensitivity, eye dryness, lung, cardiac and neurological complaints

The Systemic Radar



Sjögren Syndrome

Look for extreme dry eyes/mouth and parotid enlargement (can coexist with RA or SLE)

Prototype and MOST important

Systemic Lupus Erythematosus (SLE)

Look for photosensitivity, malar rash, oral ulcers, serositis, nephritis, and hematologic cytopenias

Inflammatory Myopathies

Look for proximal muscle weakness, Heliotrope/Gottron rashes, and elevated CK.

Systemic Sclerosis

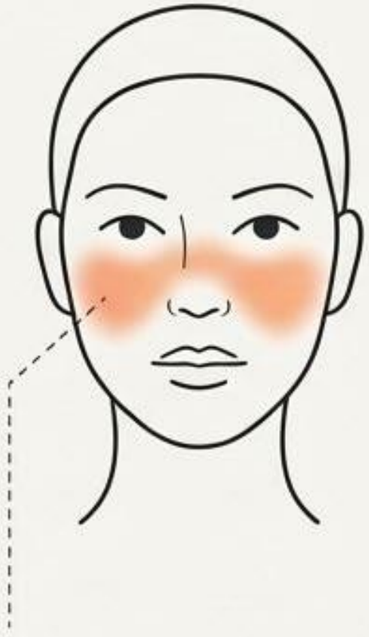
Look for Raynaud phenomenon, skin tightening (sclerodactyly), GI dysmotility, and pulmonary involvement.

Rule: Think systemically beyond the joint.

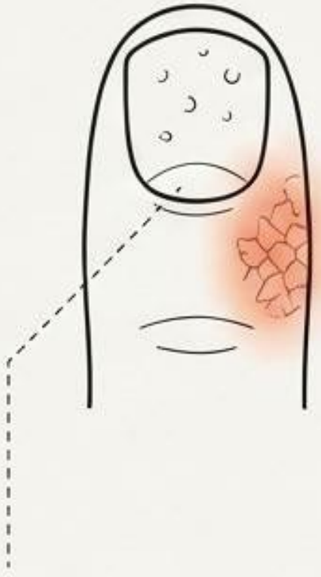
The Rheumatology Field Guide

Key Dermatologic Manifestations

Skin is often the quickest clue to a systemic rheumatic diagnosis



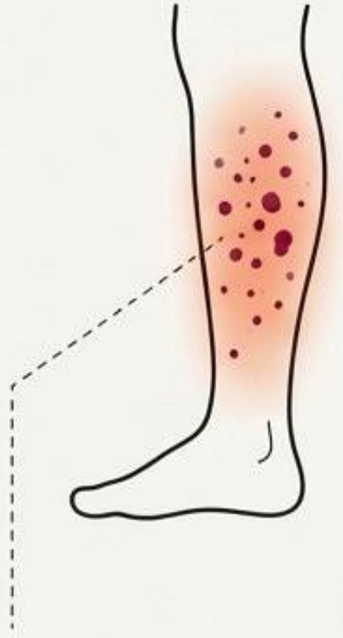
Malar rash / extreme photosensitivity → SLE



Silvery plaques / nail pitting → Psoriatic Arthritis

Nodular Tophi → Chronic Gout

Sclerodactyly and digital ischemia → Systemic Sclerosis



Palpable purpura or livedo → Systemic Vasculitis

Note: The following slides are reserved for clinical photographs of these manifestations.

Chronic disfiguring rash, heals with scarring

Discoid lupus

Photosensitive reaction called acute cutaneous lupus, present in cheek and nose (classic lupus presentation) NO SCARRING

Malar rash(butterfly rash)

Lupus effects young ppl usually

SLE

Skin examination is very essential for MSS evaluation cause if patient presents with joint pain that is an isolated MSS complaint → GOUT
So we must look for other clues that are more systemic to rule out gout and such.

Inter-articular dermatitis

VASCULITIS

Livedo Reticularis

Reticular pattern of discoloration

Purpura

Inflammation of small capillaries and venules in skin where they extravasate the RBC outside so u see bleeding under skin.

Raynaud’s phenomenon

- VERY important manifestation for people with rheumatological complaints.
- Results from vaso-spasm in small vessels in hands and and toes and sometimes ears and nose.
- What happens with exposure to cold or stress is that the vessels will occlude (Vaso spams) resulting in blanching (white or yellow fingers).
- Then gradually as u warm up the fingers, the blood flow will return but it will be deoxygenated blood (purple or yellow)
- once u warm up completely the fingers will turn red in color
- If u see such presentation in patient with joint complaints u move more toward C.T disease

Systemic sclerosis (scleroderma)

Facial appearance will be tight cause of deposition of collagen in tissues, absence of wrinkles and change in shape of nose(more beaked) and mouth opening will be very tight, fingers will also be very tight (sclerodactyl) with time they loose range of motion and cannot extend fingers (fixed position) + blanching

Can be associated with arthritis

plaque psoriasis will look on extensor surface as scaly, erythematous and itchy (**scalp** and back of elbow)

can effect nail

Rheumatoid arthritis

Figure 1: Rheumatoid nodules are superficial lesions in the deep subcutaneous tissues, commonly found on the olecranon.

- Cutaneous manifestation, we call them rheumatic nodules.
- Inflammation of subcutaneous tissue resulting from small vessel vasculitis resulting in nodular formation.
- Will be firm but not soft or hard when touched.
- Back of fingers or elbows usually.

Gout

- Monosodium urate crystals deposition in joints but sometime if u have high hyperuricima for long time the tissue will start to get depositions cause of over saturation in serum
- Deposition is called tophus (collection of monosodium urate crystals in tissue).
- Likes cold places thats why big toe is effected the most cause its far from core body so more cold.
- Also ear lobe

The Arthritis Blueprint

SUMMARYYYYYYYYYYYYYY

The Arthritis Blueprint Matrix

Pathology	RA	OA	Gout	SpA	SLE / CTD
Key Clues	Symmetric small joints (MCP, PIP, wrists, MTP)	DIP, PIP, 1st CMC, knees, hips, spine	Often monoarticular (1st MTP, midfoot, knee)	Axial spine/SI joints ± asymmetric lower-limb	Nonerosive polyarthritis/ arthralgia
Pathology	Autoimmune synovitis, pannus formation, cartilage/bone erosion.	Degenerative cartilage loss, osteophytes, subchondral sclerosis.	Monosodium urate crystal deposition (tophi), acute inflammation.	Chronic inflammation, enthesitis, new bone formation (syndesmophytes).	Autoimmune, immune complex deposition, widespread tissue inflammation, vasculitis.

Management Strategies for Rheumatological Diseases

Symptom Control

(Bridging Therapy)

Or SHORT TERM THERAPY

NSAIDs

Rapid symptom relief (inflammatory and mechanical). Useful for acute gout, OA flares, SpA.

- Risks: GI bleeding, renal dysfunction, CV risk.

Corticosteroids

FOR SEVERE BUT DON'T CONTINUE FOR LONG PERIOD CAUSE SIDE EFFECT

Highly effective bridge or flare treatment (oral, IM, IV, intra-articular).

- Risks: Minimize duration due to infection, osteoporosis, and hyperglycemia risks.

Disease Modification

(Long-Term Structural Preservation)

csDMARDs

DISEASE MODIFICATION, U WORK ON IMMUNE SYSTEM LIKE IN RA

(Methotrexate, sulfasalazine, hydroxychloroquine). Used early in RA to prevent damage. Requires lab monitoring.

Biologics/Targeted Therapies

WORK ON CYTOKINES

(TNF inhibitors, IL-6 blockade, JAK inhibitors). Used when csDMARDs fail.

- Risks: Strict screening for infection (TB/hepatitis) before starting.

The Inflammatory Step-Up Approach

Step 1: Symptom Control

Deploy NSAIDs or short corticosteroid courses immediately to relieve suffering while awaiting definitive therapies to work. Do not rely on these long-term.

Step 2: Structural Preservation

Initiate Conventional Synthetic DMARDs (csDMARDs). Methotrexate is the classic first-line anchor drug for RA.

Step 3: Disease Modification

Escalate to Biologic or Targeted Synthetic DMARDs if the disease remains active despite csDMARD optimization. Always screen for latent infections first.

Urate-Lowering Therapy (ULT) Strategies

Treat the flare today; lower urate to prevent the next one.

Patient presents with acute symptoms and very painful episodes
And high uric acid which is source of problem

Today (Acute Flare)

Only for acute inflammation

Silence the inflammation with NSAIDs, Colchicine, or Corticosteroids.

Clinical Pearl

Prophylaxis with low-dose colchicine or NSAIDs is required when starting ULT to prevent paradoxical early flares.

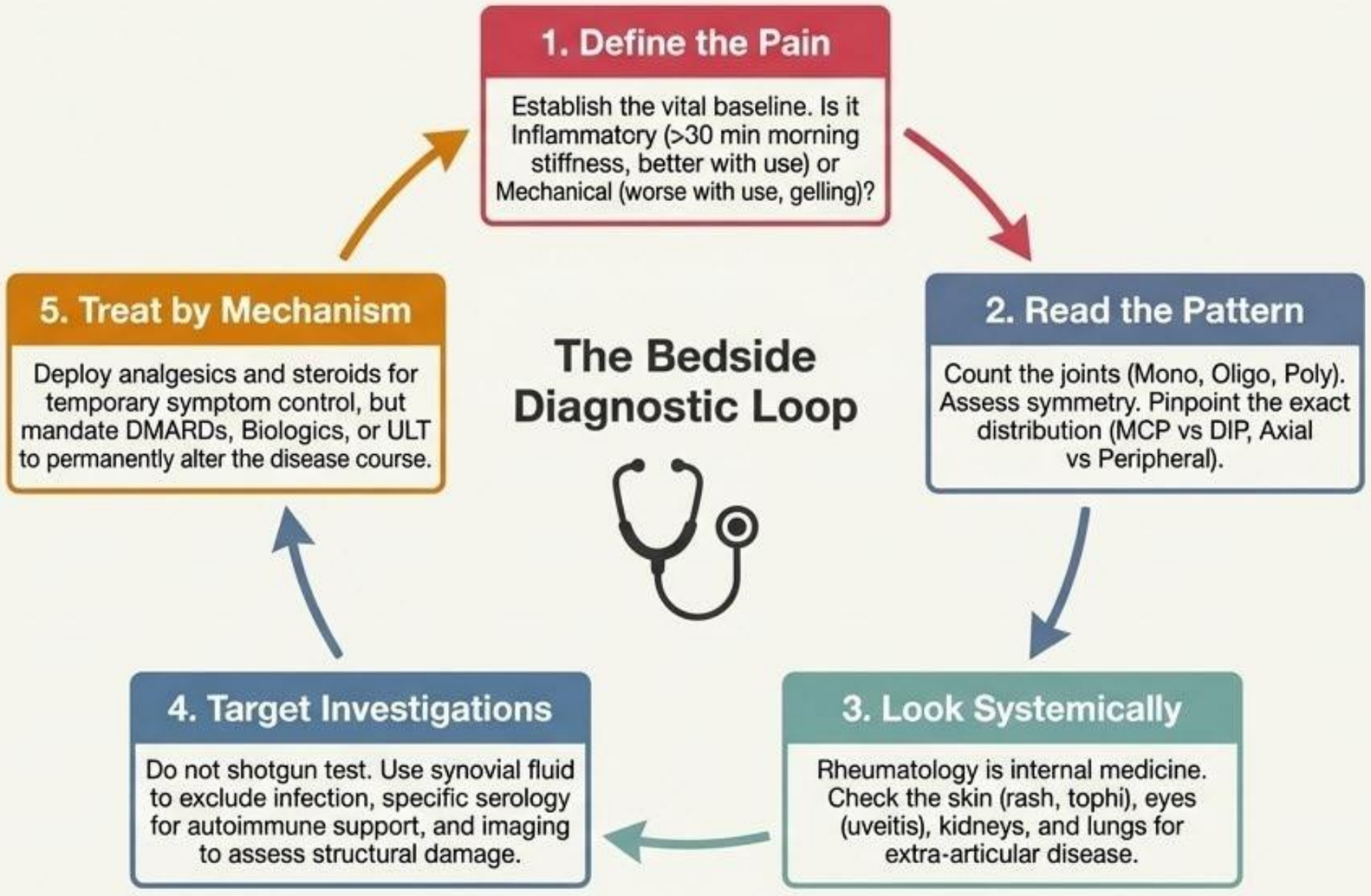
Tomorrow (Long-Term ULT)

Long term, life long, working on disease itself (high uric acid)

Goal is a sustained serum urate below the saturation threshold.

- First-line: Xanthine oxidase inhibitors (Allopurinol; Febuxostat as alternative).
- When to initiate: Recurrent flares, presence of tophi, radiographic damage, or chronic gout burden.

Approach to the patient with joint pain



Thank you

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