

Rheumatology

Ankylosing spondylitis	
Inflammatory back pain	• Insidious onset at age <40 • Symptoms >3 months • Relieved with exercise but not rest • Nocturnal pain
Examination findings	• Arthritis (sacroiliitis) • Reduced chest expansion & spinal mobility • Enthesitis (tenderness at tendon insertion sites) • Dactylitis (swelling of fingers & toes) • Uveitis
Complications	• Osteoporosis/vertebral fractures • Aortic regurgitation • Cauda equina
Laboratory	• Elevated ESR & CRP • HLA-B27 association
Imaging	• X-ray of sacroiliac joints • MRI of sacroiliac joints

• ** Anterior uveitis (iritis) is the most common extraarticular manifestation of AS and occurs in 25%-40% of patients. It is characterized by inflammation of the uveal tract (iris, ciliary body, and choroid). Anterior uveitis typically presents with intense pain and photophobia in one eye.

• (Choice C) Episcleritis is characterized by inflammation seen at the white of the eye, without involvement of the uveal tract. It is most strongly associated with rheumatoid arthritis and inflammatory bowel disease but is not associated with AS.

Common causes of low back pain		
	Condition	Clinical clues
Musculoskeletal	Mechanical (muscle strain, spasm, degenerative arthritis)	• Normal neurologic examination • Negative straight leg raise • Possible paraspinal tenderness
	Herniated nucleus pulposus/ disk disease	• Radiculopathy (usually L4-L5) • Possible positive straight leg raise • Possible neurologic deficits
	Spinal stenosis	• Pseudoclaudication • Better with spine flexion • Worse with extension • Older age
	Compression fracture	• Older age • More common in women • Trauma/fall (may be minor)
Inflammatory	Ankylosing spondylitis, reactive arthritis, psoriatic arthritis, inflammatory bowel disease	• Better with activity or exercise • No improvement with rest • Gradual onset • HLA-B27 present
Malignancy	Metastatic cancer to bone	• History of malignancy • Age >50 • Worse at night • Unintentional weight loss • Cauda equina syndrome (weakness, urine retention/incontinence, saddle anesthesia)
Infectious	Osteomyelitis, discitis, abscess	• Recent infection • IV drug abuse • Diabetes • Fever, exquisite point tenderness

Lumbosacral strain is the most common cause of back pain. It is estimated that the lifetime risk of lumbosacral strain is close to 80%. The clinical scenario described is typical. The pain starts acutely after physical exertion, and it is concentrated in the lumbar area, usually without radiation to the thighs. Physical examination reveals local tenderness and contraction of the paraspinal muscles. A straight-leg raising test and neurologic examination are typically normal. The treatment includes NSAIDs and early mobilization.

Management of low back pain	
Acute pain	• Maintain moderate activity • NSAIDs or acetaminophen • Consider: muscle relaxants, spinal manipulation, brief course of opioids
Chronic pain	• Intermittent use of NSAIDs or acetaminophen • Exercise therapy (stretching/strengthening, aerobic) • Consider: tricyclic antidepressants, duloxetine
Secondary prevention	• Exercise therapy • Education

• NSAIDs = nonsteroidal anti-inflammatory drugs.

Indications for imaging in low back pain	
X-ray	• Osteoporosis/compression fracture • Suspected malignancy • Ankylosing spondylitis (eg, insidious onset, nocturnal pain, better with movement)
MRI	• Sensory/motor deficits • Cauda equina syndrome (eg, urine retention, saddle anesthesia) • Suspected epidural abscess/infection (eg, fever, intravenous drug abuse, concurrent infection, hemodialysis)
Radionuclide bone scan or CT scan	• Indications for MRI but patient not able to have MRI

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- Red Flag
- Age >50
 - History of cancer
 - Constitutional symptoms (eg, fever, unexplained weight loss)
 - Nocturnal pain
 - No response to appropriate treatment
 - Significant or progressive neurologic deficits

• **Rupture** of a popliteal cyst (eg, following strenuous exercise) can cause posterior knee and calf pain, with tenderness and swelling of the calf resembling deep venous thrombosis (DVT). An arc of ecchymosis is often visible distal to the medial malleolus ("crescent sign"). Ultrasound can rule out DVT and confirm the popliteal cyst.

Behçet disease	
Epidemiology	• Young adults • Turkish, Middle Eastern, or Asian descent
Clinical findings	• Recurrent, painful oral aphthous ulcers • Genital ulcers • Eye lesions (eg, uveitis) • Skin lesions (eg, erythema nodosum, acneiform lesions) • Thrombosis
Evaluation	• Pathergy - Exaggerated skin ulceration with minor trauma (eg, needlestick) • Biopsy - Nonspecific vasculitis of different-sized vessels

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This patient with medial knee pain and focal tenderness has **pes anserinus pain syndrome (PAPS)**. This condition is often referred to as anserine bursitis, but most patients do not have true inflammation in the bursa, and multiple regional structures can contribute to the pain. The pes anserinus is formed by the conjoined tendons of gracilis, sartorius, and semitendinosus. The anserine bursa is located anteromedially over the tibial plateau, just below the joint line of the knee and deep to the pes anserinus.

PAPS can be caused by an abnormal gait, overuse, or trauma. **Localized pain** is typical over the anteromedial tibia and is often exacerbated by pressure from the opposite knee while lying on the side. Examination shows a well-defined area of tenderness over the medial tibial plateau just below the joint line. A valgus stress test does not aggravate the pain, indicating no medial collateral ligament involvement (**Choice A**). The diagnosis is primarily based on clinical features, although x-ray can exclude concurrent osteoarthritis of the knee.

Carpal tunnel syndrome	
Risk factors	• Obesity • Pregnancy • Diabetes • Hypothyroidism • Rheumatoid arthritis
Clinical presentation	• Pain & paresthesias in median nerve distribution (first 3½ digits) • Positive Phalen & Tinel tests • Severe disease: Weakness of thumb abduction & opposition, atrophy of thenar eminence
Confirmatory test	• Nerve conduction studies
Treatment	• Wrist splinting • Glucocorticoid injection • Surgery for severe or refractory symptoms

Charcot joint (neurogenic arthropathy)	
Associated conditions	• Vitamin B12 deficiency • Diabetes • Peripheral nerve damage • Spinal cord injury • Syringomyelia • Tabes dorsalis (tertiary syphilis)
Clinical manifestations	• Deformed joints • Lacking/decreased sensation (proprioception, pain, temperature) with loss of neurologic input • Arthritis or arthropathy • Mild pain • Fractures (may be unsuspected by patient) • Degenerative joint disease & loose bodies on joint imaging
Management	• Treat underlying condition • Mechanical devices (assist in weight bearing, decrease further trauma) • X-rays (if trauma is present)

Clinical features of dermatomyositis	
Muscle weakness	• Proximal, symmetric • Weakness in UE = LE
Skin findings	• Gottron's papules • Heliotrope rash
Extramuscular findings	• Interstitial lung disease • Dysphagia • Myocarditis
Diagnosis	• ↑ CPK, aldolase, LDH • Anti-RNP, anti-Jo-1, anti-Mi2 • Diagnostic uncertainty ◦ EMG ◦ Biopsy (skin/muscle)
Management	• High-dose glucocorticoids PLUS glucocorticoid-sparing agent • Screening for malignancy

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Paraneoplastic syndrome	Involved site	Clinical features
Myasthenia gravis	Acetylcholine receptor in postsynaptic membrane	Fluctuating muscle weakness • Ocular (ptosis, diplopia) • Bulbar (dysphagia, dysarthria) • Facial, neck & limb muscles
Lambert-Eaton syndrome	Presynaptic membrane voltage-gated calcium channels	• Proximal muscle weakness • Autonomic dysfunction (eg, dry mouth) • Cranial nerve involvement (eg, ptosis) • Diminished or absent deep-tendon reflexes
Dermatomyositis/ polymyositis Windows تنشيط انتقل إلى الإعدادات لتنشيط	Muscle fiber injury	• Symmetrical & more proximal muscle weakness • Interstitial lung disease, esophageal dysmotility, Raynaud phenomenon • Polyarthritis • Esophageal dysmotility • Skin findings (eg, Gottron papules, heliotrope rash) in dermatomyositis

This patient's presentation is consistent with likely fibromyalgia (FM). FM presents most commonly in young to middle-aged women with widespread pain, fatigue, and cognitive/mood disturbances. Patients tend to have a fairly normal physical examination except for point muscle tenderness in areas such as the mid trapezius, lateral epicondyle, costochondral junction in the chest, and greater trochanter. FM has no specific diagnostic laboratory findings. Revised 2010 American College of Rheumatology criteria suggest using the **widespread pain index and symptom severity scale**, rather than trigger points, for diagnosis. The index and scale better emphasize cognitive problems, fatigue, and severity of somatic symptoms.

Initial management of FM involves a multidisciplinary approach including **patient education** about FM, **regular aerobic exercise**, and **good sleep hygiene**. Education requires validating that FM is a benign condition with a favorable prognosis. Although patients may complain of increased short-term pain during or after activity, a regular exercise program (eg, aerobic conditioning, strength training, stretching) improves long-term pain. Water exercises also can greatly reduce the pain. Medications (eg, tricyclic antidepressants) are reserved for patients failing initial measures. There is limited evidence for other approaches, including trigger-point injections, analgesics, and alternative therapy (eg, tai chi, yoga).

Distinguishing features of fibromyalgia, polymyositis & polymyalgia rheumatica		
	Clinical features	Diagnosis
Fibromyalgia	• Young to middle-aged women • Chronic widespread pain • Fatigue, impaired concentration • Tenderness at trigger points (eg, mid trapezius, costochondral junction)	• ≥3 months of symptoms with widespread pain index or symptom severity score • Normal laboratory studies
Polymyositis	• Proximal muscle weakness (eg, increasing difficulty climbing up stairs) • Pain mild/absent	• Elevated muscle enzymes (eg, creatine kinase, aldolase, AST) • Autoantibodies (ANA, anti-Jo-1) • Biopsy: Endomysial infiltrate, patchy necrosis
Polymyalgia rheumatica	• Age >50 • Systemic signs & symptoms • Stiffness > pain in shoulders, hip girdle, neck • Association with giant cell (temporal) arteritis	• Elevated ESR, C-reactive protein • Rapid improvement with glucocorticoids

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GCA

Systemic symptoms	Fever, fatigue, malaise, weight loss
Localized symptoms	Headaches: Located in temporal areas Jaw claudication: Most specific symptom of GCA PMR - Arm claudication: Associated bruits in subclavian or axillary areas - Aortic wall thickening or aneurysms - CNS: TIAs/stroke, vertigo, hearing loss
Visual symptoms	- Amaurosis fugax: Transient vision field defect progressing to monocular blindness AION: Most common ocular manifestation
Laboratory results	- Normochromic anemia - Elevated ESR & CRP - Temporal artery biopsy
Treatment	PMR only: Low-dose oral glucocorticoids (eg, prednisone 10-20 mg daily) GCA: Intermediate- to high-dose oral glucocorticoids (eg, prednisone 40-60 mg daily) GCA with vision loss: Pulse high-dose IV glucocorticoids (eg, methylprednisolone 1000 mg daily) for 3 days followed by intermediate- to high-dose oral glucocorticoids

The fever, knee pain, and white blood cell (WBC) count of 50,000/mm³ on **synovial fluid analysis** strongly suggest septic arthritis. Septic arthritis in a young, sexually active individual is most often caused by *Neisseria gonorrhoeae* (75% of cases).

Gonococcal septic arthritis may present with **asymmetric polyarthralgias** (most often associated with tenosynovitis and skin rash) or an **isolated purulent mono- or oligoarthritis**. In some patients, asymmetric polyarthralgias may precede purulent monoarthritis. Roughly 75% of cases are "silent," meaning that the preceding genitourinary or pharyngeal infection goes unnoticed.

For this patient, migratory asymmetric polyarthralgias of the right wrist and left ankle preceded purulent monoarthritis of the left knee. Although skin rash and tenosynovitis are not present in this patient, **purulent arthritis in a sexually active individual is gonococcal arthritis until proven otherwise**. Synovial fluid white blood cell count is about 50,000/mm³ (slightly lower than other septic arthritides). Gram stain of the synovial fluid (positive in 25% of cases), blood cultures (positive in 20%-50% of cases), and genital/pharyngeal mucosal nucleic acid amplification tests (positive in 90% of cases) are used to confirm the diagnosis. Treatment is ceftriaxone or cefotaxime.

Joint fluid characteristics				
	Normal	Noninflammatory (eg, OA)	Inflammatory (eg, crystals, RA)	Septic joint
Appearance	Clear	Clear	Translucent or opaque	Opaque
WBC count (mm ³)	<200	200-2,000	2,000-100,000	50,000-150,000
PMNs	<25%	25%	Often >50%	>80%-90%

Underlying joint disorders (eg, gout, pseudogout, osteoarthritis) increase the risk for secondary joint infection. In patients with crystal-induced arthritis (eg, gout), the presence of crystals alone does not rule out septic arthritis as these can be present in synovial fluid between attacks. If the Gram stain is positive and joint fluid white cell count is >50,000/mm³, the patient should be started on empiric antibiotics until culture results are known. If crystals are present, the fluid is nonpurulent, and Gram stain is negative, the patient may be managed as for a standard gout flare (**Choice C**).

Risk factors for gout	
Increased risk	• Medications (eg, diuretics, low-dose aspirin) • Surgery, trauma, recent hospitalization • Volume depletion • Diet: High-protein (meat, seafood), high-fat, fructose or sweetened beverages • Heavy alcohol consumption • Underlying medical conditions (eg, hypertension, obesity, chronic kidney disease, organ transplant)
Decreased risk	• Dairy product intake • Vitamin C (≥1500 mg/day) • Coffee intake (≥6 cups/day)

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Uric acid tophi are virtually pathognomonic for gout, and even in the absence of microscopic confirmation of crystals, the diagnosis can be made provisionally in patients with visible tophi and a history of episodic monoarthritis.

Prevention of future gout attacks

- Weight loss to achieve BMI $<25 \text{ kg/m}^2$
- Low-fat diet
- Decreased seafood & red meat intake
- Protein intake preferably from vegetable & low-fat dairy products
- Avoidance of organ-rich foods (eg, liver & sweetbreads)
- Avoidance of beer & distilled spirits
- Avoidance of diuretics when possible

- Risk is also increased by heavy intake of beverages and foods containing **fructose** and other **refined sugars**

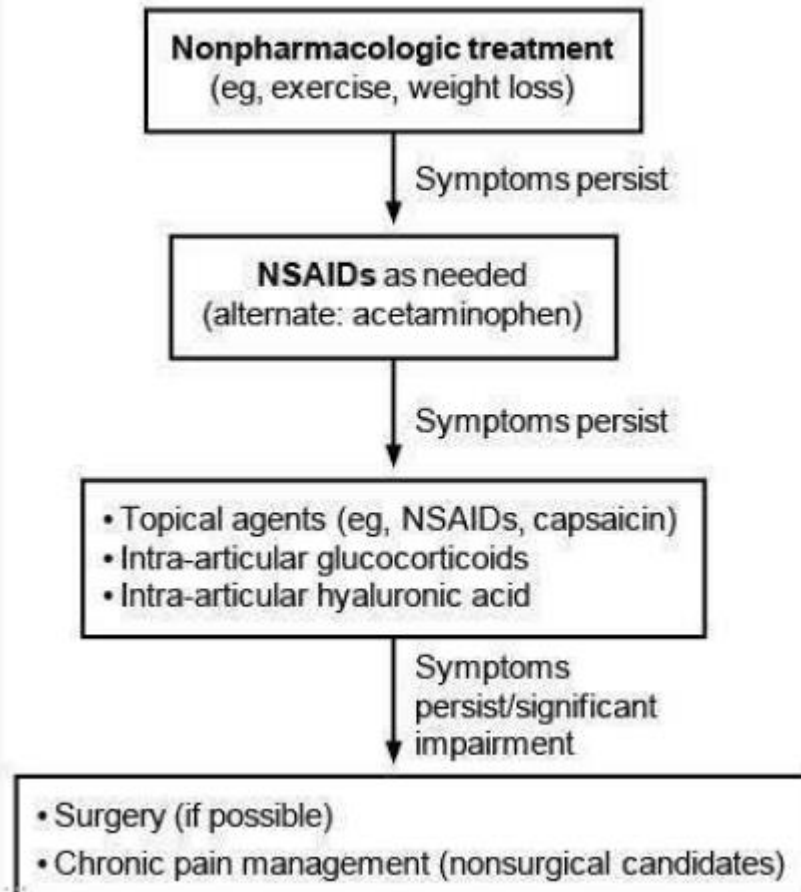
Myeloproliferative disorders are common secondary causes of gout. This patient has several clinical features suggesting **polycythemia vera** (PV), including pruritus triggered by hot baths (aquagenic pruritus), headaches, and hepatosplenomegaly. PV is characterized by increased cell turnover due to clonal hyperproliferation in all 3 primary bone marrow lineages (ie, red cells, white cells, platelets). Up to 40% of patients with PV have gout. Allopurinol inhibits uric acid formation and is used to prevent gout attacks in patients with hyperuricemia due to PV.

- **Negatively birefringent** (appear **yellow** when lying **parallel** to the polarizing axis, and **blue** when lying **perpendicular**)

Lateral epicondylitis

Clinical presentation	• Subacute to chronic lateral elbow pain • History of repetitive or forceful wrist extension • Peak incidence age 45-54
Diagnosis	• Tenderness at epicondyle & proximal extensor muscles • Pain with resisted wrist extension or supination • Pain with passive wrist flexion
Management	• Modified activity & ergonomics • Inelastic counterforce brace • Nonsteroidal anti-inflammatory drugs (topical or oral) • Stretching & progressive resistance exercise • Physical therapy

Management of osteoarthritis



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NSAIDs = nonsteroidal anti-inflammatory drugs

Sjögren syndrome

Exocrine features	• Keratoconjunctivitis sicca • Dry mouth, salivary hypertrophy • Xerosis of skin
Extraglandular features	• Raynaud phenomenon • Cutaneous vasculitis • Arthralgia/arthritis • Interstitial lung disease
Diagnostic findings	• Objective signs of decreased lacrimation (eg, Schirmer test) • Positive anti-Ro (SSA) &/or anti-La (SSB) • Salivary gland biopsy with focal lymphocytic sialoadenitis • Classification: primary if no associated CTD, secondary if comorbid CTD (eg, SLE, RA, scleroderma)

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Systemic sclerosis (scleroderma)	
Pathogenesis	● Progressive tissue fibrosis ● Vascular dysfunction
Clinical features	● Systemic: Fatigue, weakness ● Skin: Telangiectasia, sclerodactyly, digital ulcers, calcinosis cutis ● Extremities: Arthralgias, contractures, myalgias ● Gastrointestinal: Dysphagia, dyspepsia ● Vascular: Raynaud phenomenon
Serology	● Antinuclear antibody ● Anti-topoisomerase I (anti-Scl-70) antibody ● Anticentromere antibody
Complications	● Lung: Interstitial lung disease, pulmonary arterial HTN ● Kidney: HTN, scleroderma renal crisis (oliguria, malignant HTN, thrombocytopenia, MAHA) ● Heart: Myocardial fibrosis, pericarditis, pericardial effusion

The diagnosis of SSc is based primarily on clinical manifestations and serologic markers. **Antinuclear antibodies** are present in almost all patients but are nonspecific. **Anti-topoisomerase I (anti-Scl-70)** and **anti-RNA polymerase III** are less sensitive but more specific and are associated with extensive disease. **Anticentromere antibodies** may also be seen, primarily in patients with limited disease.

Clinical features of systemic sclerosis (scleroderma)	
Systemic	• Fatigue • Joint stiffness & pain
Skin	• Telangiectasia • Sclerodactyly • Digital ulcers • Calcinosis cutis
Vascular	• Raynaud phenomenon
GI	• Dysphagia, dyspepsia • Angiodysplasia of stomach (watermelon stomach) with GI bleeding • Malabsorption due to bacterial overgrowth
Pulmonary	• Pulmonary fibrosis • Pulmonary arterial hypertension
Renal	• Scleroderma renal crisis ◦ Acute onset oliguric renal failure with malignant hypertension ◦ Thrombocytopenia ◦ Microangiopathic hemolytic anemia
Cardiac	• Myocarditis, pericarditis, pericardial effusion

A wide range of **pulmonary complications** can occur in patients with SSc. They affect up to 80% of patients, and are the leading cause of death in SSc. The most common pulmonary complication in patients with diffuse SSc is **interstitial fibrosis**, which develops in about 40% of patients.

(Choice D) Pulmonary arterial hypertension, clinically similar to idiopathic pulmonary hypertension, is a possible but less common complication of SSc. It occurs in up to 15% of patients, **primarily in those with limited SSc (eg, CREST syndrome).**

Antiphospholipid antibody syndrome	
Clinical features	Venous or arterial thromboembolic disease • Deep venous thrombosis • Pulmonary embolism • Ischemic stroke/transient ischemic attack Adverse pregnancy outcomes • Unexplained embryonic or fetal loss • Premature birth due to placental insufficiency or preeclampsia
Laboratory findings	• Lupus anticoagulant effect: Paradoxical aPTT prolongation not reversed on plasma mixing studies • Presence of specific antiphospholipid antibodies ○ Anticardiolipin antibody ○ Anti-beta2-glycoprotein-I antibody

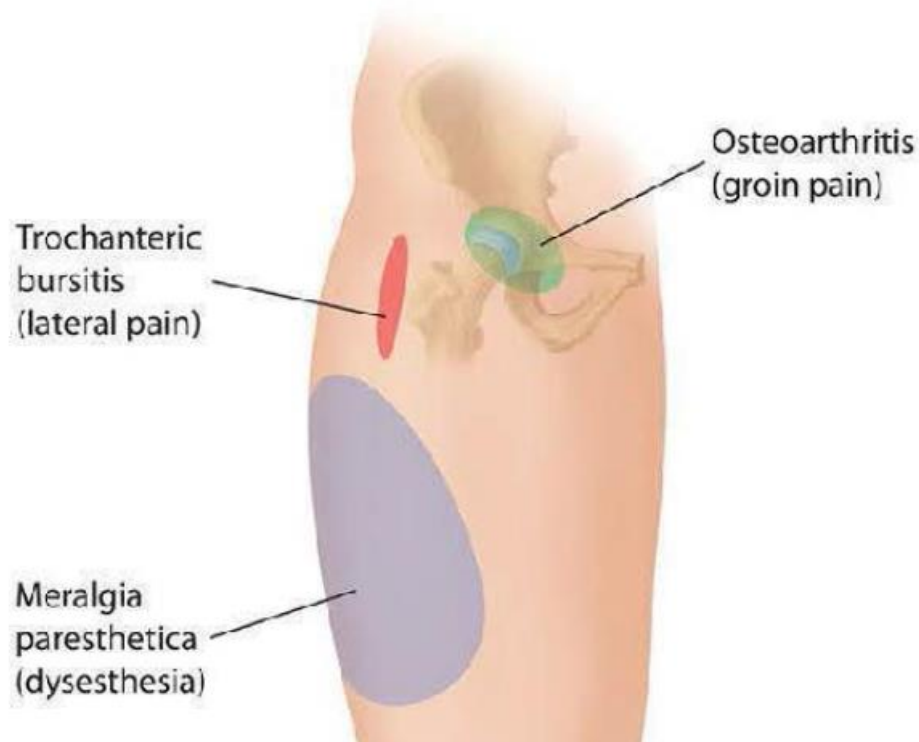
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- The biggest risk factor for APS is **SLE**; APS occurs in up to 40% of these patients.
- A minority of healthy individuals may develop transient antiphospholipid antibodies, so all positive serology for APS should be repeated at 12 weeks to confirm diagnosis.
- Arthritis and arthralgias affect 95% of patients with SLE and tend to be migratory, symmetric, polyarticular, and accompanied by brief morning stiffness (much shorter in duration than rheumatoid arthritis [RA]). The knees, carpal joints, and joints of the fingers are most commonly affected. Joint pain often exceeds the objective findings on examination, and K-rays usually show no evidence of joint destruction or erosion (unlike other inflammatory arthritides such as RA).

Disease-modifying antirheumatic drugs		
Agent	Mechanism	Adverse effects
Methotrexate	Purine antimetabolite	• Hepatotoxicity • Stomatitis • Cytopenias
Leflunomide	Pyrimidine synthesis inhibitor	• Hepatotoxicity • Cytopenias
Hydroxychloroquine	TNF & IL-1 suppressor	• Retinopathy
Sulfasalazine	TNF & IL-1 suppressor	• Hepatotoxicity • Stomatitis • Hemolytic anemia
TNF inhibitors • Adalimumab • Certolizumab • Etanercept • Golimumab • Infliximab		• Infection • Demyelination • Congestive heart failure • Malignancy

Osteoarthritis	
Age of onset	>40; increases with age
Joint involvement	• Knees • Hips • Distal interphalangeal joints • 1st carpometacarpal joint
Morning stiffness	None/brief (<30 min)
Systemic symptoms	Absent
Examination findings	• Hard, bony enlargement of joints • Reduced range of motion

Symptomatic differentiation of common hip disorders



Examination findings suggesting OA in the knee include:

- Periarticular **bony hypertrophy** and tenderness
- Limited range of motion
- **Crepitus** and pain with motion
- Small joint effusion without erythema or warmth
- Varus or valgus angulation of the tibia
- Popliteal (Baker) cyst behind the joint

Avascular necrosis	
Etiology	• Steroid use • Alcohol abuse • Systemic lupus erythematosus • Antiphospholipid syndrome • Hemoglobinopathies (eg, sickle cell) • Infections (eg, osteomyelitis, HIV) • Renal transplantation • Decompression sickness
Clinical manifestations	• Groin pain on weight bearing • Pain on hip abduction & internal rotation • No erythema, swelling, or point tenderness
Laboratory findings	• Normal white blood cell count • Normal ESR & CRP
Radiologic imaging	• Crescent sign seen in advanced stage • MRI is most sensitive modality

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CRP – C-reactive protein; ESR – erythrocyte sedimentation rate

Osteoporosis risk factors
• Advanced age • Medical history of fracture with minimal trauma (fragility fracture) • Low body weight (<58 kg [127 lb]) • Family history of hip fracture • Current smoking • Excessive alcohol intake • Medications (eg, steroids, anticonvulsants) ◦ Secondary causes of osteoporosis ◦ Premature menopause ◦ Hypogonadism ◦ Malabsorption (celiac disease) ◦ Inflammatory disorders (eg, inflammatory bowel disease, rheumatoid arthritis) ◦ Hyperthyroidism & hyperparathyroidism ◦ Cushing syndrome ◦ Vitamin D deficiency ◦ Chronic liver or renal disease

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Paget disease of bone	
Clinical features	• Most patients are asymptomatic • Bone pain & deformity ◦ Skull: Headache, hearing loss ◦ Spine: Spinal stenosis, radiculopathy ◦ Long bones: Bowing, fracture, arthritis of adjacent joints • Giant cell tumor, osteosarcoma
Pathogenesis	• Osteoclast dysfunction • Increased bone turnover
Laboratory testing	• Elevated alkaline phosphatase • Elevated bone turnover markers (eg, PINP, urine hydroxyproline) • Calcium & phosphorus are usually normal
Imaging	• X-ray: Osteolytic or mixed lytic/sclerotic lesions • Bone scan: Focal increase in uptake
Treatment	• Bisphosphonates

PINP = procollagen type I N propeptide.

Polymyalgia rheumatica	
Clinical features	Findings
Symptoms	• Age >50 • Bilateral pain & morning stiffness >1 month • Involvement of 2 of following: ○ Neck or torso ○ Shoulders or proximal arms ○ Proximal thigh or hip ○ Constitutional (fever, malaise, weight loss)
Physical examination	• Decreased active ROM in shoulders, neck & hips
Laboratory studies	• ESR >40 mm/h, sometimes >100 mm/h • Elevated CRP • Normocytic anemia possible • ~20% can have normal studies
Treatment	Response to glucocorticoids

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- Muscle enzymes (eg, creatine kinase) are invariably elevated, and autoantibodies (eg, antinuclear antibodies, anti-Jo-1 antibodies) are present in most cases. A muscle biopsy is the most definitive diagnostic test and shows a mononuclear infiltrate surrounding necrotic and regenerating muscle fibers

Pseudogout (acute calcium pyrophosphate crystal arthritis)	
Symptoms	• Acute mono- or oligoarticular arthritis • Peripheral joints (knee most common)
Diagnosis	• Inflammatory effusion (15,000-30,000 cells/mm³) • CPPD crystals (rhomboid shape, positive birefringence) • Chondrocalcinosis on imaging
Treatment	• Intra-articular glucocorticoids • Nonsteroidal anti-inflammatory drugs • Colchicine

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Clinical features of psoriatic arthritis	
Arthritis	• DIP joints • Asymmetric oligoarthritis • Symmetric polyarthritis, similar to RA • Arthritis mutilans (deforming & destructive arthritis) • Spondylarthritides (sacroiliitis & spondylitis)
Soft tissue & nail involvement	• Enthesitis (inflammation at tendon insertion site to bone) • Dactylitis ("sausage digits") of toe or finger • Nail pitting & onycholysis • Swelling of the hands or feet with pitting edema
Skin lesions	• Arthritis precedes skin disease in 15% of patients • Skin lesions are present but not yet diagnosed in 15% of patients

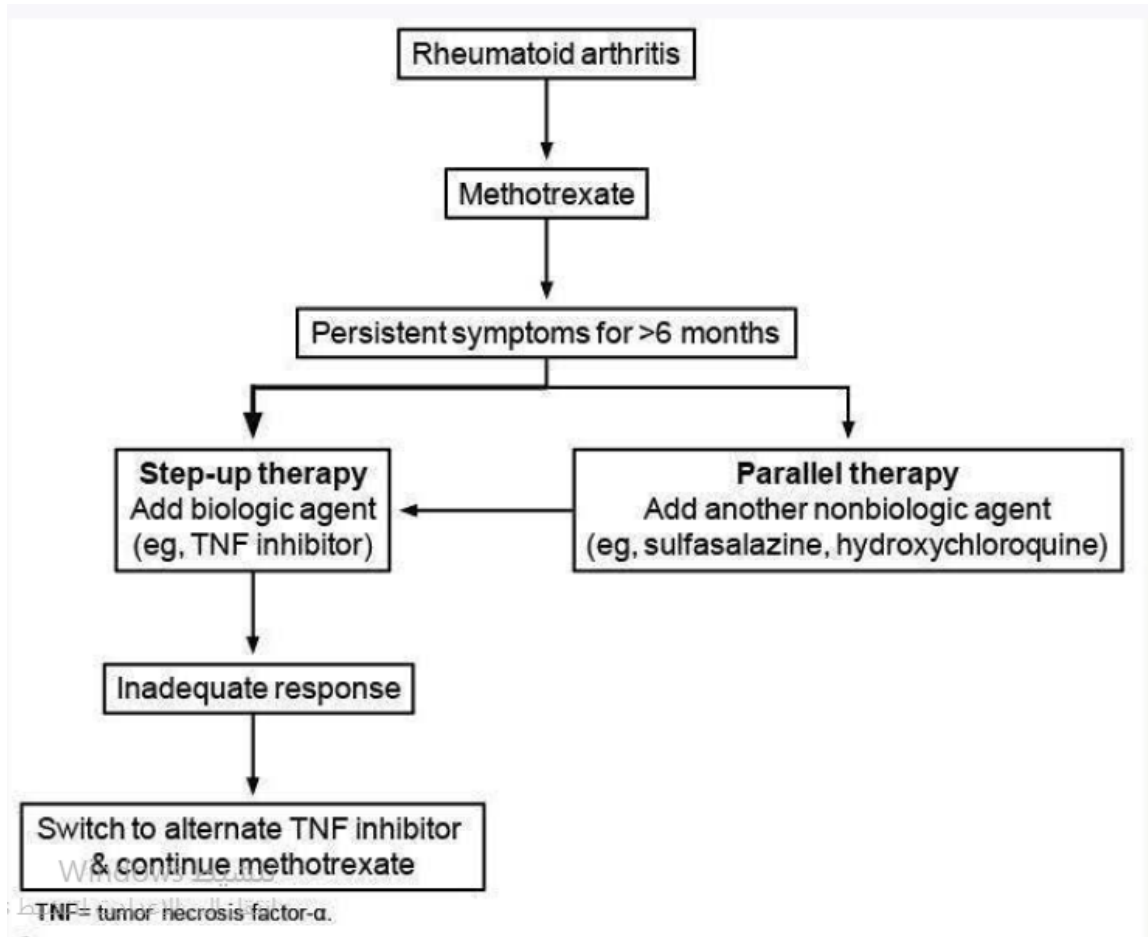
- well-demarcated red plaques with silvery scaling-the classic lesions of psoriasis-are seen on the dorsum of each hand.

This patient's skin lesion is consistent with **pyoderma gangrenosum (PG)**, a neutrophilic ulcerative skin disease. PG starts as an inflammatory papule, pustule, vesicle, or nodule, and progresses to form an expanding ulcer with a purulent base and ragged violaceous borders. PG can present as single or multiple lesions, usually on the trunk or lower extremities. Nearly **30%** of cases are triggered by local trauma (pathergy).

More than **50%** of patients with PG have an associated underlying **systemic disorder** such as **inflammatory bowel disease**, arthropathies (eg, **rheumatoid arthritis**), or hematologic conditions (eg, **acute myeloid leukemia**). PG can present before or after diagnosis of these underlying conditions. PG is diagnosed clinically after excluding other diagnoses (eg, venous ulcers, panniculitis, cutaneous cancers), usually with **skin biopsy**. Treatment typically requires local or systemic **corticosteroids**.

	Primary Raynaud's phenomenon	Secondary Raynaud's phenomenon
Etiology	• No underlying cause	• Connective tissue diseases • Occlusive vascular conditions • Sympathomimetic drugs • Vibrating tools • Hyperviscosity syndromes • Nicotine
Clinical presentation	• Usually women age <30 • No tissue injury • Negative ANA & ESR	• Usually men age >40 • Symptoms of underlying disease • Tissue injury or digital ulcers • Abnormal nail fold capillary examination
Management	• Avoid aggravating factors • CCB for persistent symptoms	• Evaluate & treat underlying disorder • CCB for persistent symptoms, aspirin for patients at risk for digital ulceration

- In addition to the classic triad, mucocutaneous lesions and enthesitis (Achilles tendon pain) are common findings in reactive arthritis.



- Most experts suggest a low threshold for starting bisphosphonate therapy in RA patients
- Folic acid supplementation is recommended for all patients taking MTX on a chronic basis as it has been shown to reduce the incidence of adverse effects without the loss of efficacy of MTX therapy

Felty syndrome	
Clinical features	• Rheumatoid arthritis ◦ Severe erosive joint disease & deformity ◦ Rheumatoid nodules ◦ Vasculitis (mononeuritis multiplex, necrotizing skin lesions) • Neutropenia (ANC <1500/μL) • Splenomegaly
Diagnosis	• Anti-CCP & RF are positive in >90% of patients • Markedly elevated ESR, often >85 mm/hr • Peripheral smear & bone marrow biopsy to rule out other causes of neutropenia

- associated with EN include streptococcal infection, sarcoidosis, tuberculosis (TB), coccidioidomycosis, inflammatory bowel disease (IBD), and Behcet disease.
- EN can occur due to inflammatory bowel disease (IBD) (more commonly Crohn disease than ulcerative colitis), typically during periods of active intestinal disease.

Common features of sarcoidosis	
Epidemiology	• Young adults • African Americans
Clinical	• Constitutional symptoms • Cough, dyspnea & chest pain • Extrapulmonary findings ◦ Skin lesions ◦ Anterior/posterior uveitis ◦ Lofgren syndrome
Imaging	• Bilateral hilar adenopathy • Pulmonary reticular infiltrates
Laboratory	• Hypercalcemia/hypercalciuria • Elevated serum ACE level
Pathology	• Biopsy shows noncaseating granulomas that stain negative for fungi & acid-fast bacilli

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Septic arthritis	
Risk factors	• Abnormal joint: OA, RA, prosthetic joint, gout • Age >80 • Diabetes • IV drug abuse, alcoholism • Intra-articular glucocorticoid injections
Clinical features	• Acute monoarthritis: hot, swollen, decreased ROM • Fever • Elevated ESR & CRP
Diagnosis	• Blood cultures • Synovial fluid analysis: leukocytosis ($>50,000/\text{mm}^3$), Gram stain, culture
Initial treatment	• Gram-positive cocci: vancomycin • Gram-negative rod: third-generation cephalosporin • Negative microscopy: vancomycin (+ third-generation cephalosporin if immunocompromised)

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Sjögren syndrome	
Exocrine features	• Keratoconjunctivitis sicca • Dry mouth, salivary hypertrophy • Xerosis of skin
Extraglandular features	• Raynaud phenomenon • Cutaneous vasculitis • Arthralgia/arthritis • Interstitial lung disease
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Takayasu arteritis	
Risk factors	• Female • Asian • Age 10-40
Symptoms	• Constitutional (eg, fever, weight loss) • Arterio-occlusive (eg, claudication, ulcers) in upper extremities • Arthralgias/myalgias
Examination findings	• Blood pressure discrepancies • Pulse deficits • Arterial bruits
Diagnosis	• Elevated inflammatory markers (eg, ESR, CRP) • Chest x-ray: Aortic dilation, widened mediastinum • CT/MRI: Wall thickening, narrowing of lumen
Treatment	• Systemic glucocorticoids