CHAPTER

35

Approach to Arthritis

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ABSTRACT

Arthritis is the inflammation of the joints which is a term derived from Greek in which arthro- means joint and –itis means inflammation. Once the source of pain is confirmed as originating from joint then decide whether the disease is inflammatory or non-inflammatory in nature. Patients with an inflammatory arthritis are more likely to have palpable synovitis and morning stiffness; if the condition is severe, they may have fever, weight loss, and fatigue. Then evaluate the temporal pattern of the disorder; especially acute versus chronic duration. Then classify the arthritis according to the spatial pattern: primarily, monoarthritis or poly arthritis and the presence of axial involvement. Then search for the existence of extra-articular and/or systemic manifestations.

Arthritis is the inflammation of the joints which is a term derived from Greek in which arthro- means joint and–itis means inflammation. 12th October has been declared as World Arthritis day. Musculoskeletal diseases are among the most common reasons for which medical help is sought. Anywhere between 25% and 30% individuals will have a musculoskeletal complaint in their life

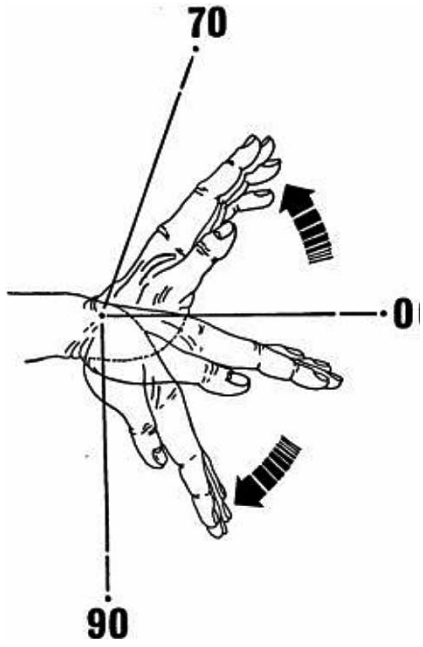


Fig. 1: Stress pain and restriction at the wrist – there is no pain in the neutral 'loose-pack' position, but progressive pain and some restriction as the wrist moves towards full extension or full flexion

Table 1 : Distinctive features of regional syndromes

	Periarticular pain	Articular pain	Neurogenic pain	Referred pain
Enquiry	Only a few selective movements are painful	All joint movements are painful	Dysaesthesic; aggravated by compression of nerve or movement of the spine	Unrelated to movement; 'visceral' timing; poorly localised, may be improved by rubbing
Pain on motion	Active> passive; selected movements	Active~passive; several directions	Normal; if root pain: pain on movement of the affected spine segment	Normal
Range of motion	Active movement may be limited by pain; passive movement: full	May be limited equally for both active and passive movement	Normal	Normal
Resisted active movement	Pain on specific manoeuvres	No effect	No effect	No effect
Local palpation	Tenderness over affected periarticular structure (away from joint line)	Possible tenderness over joint line, crepitus, capsular swelling, effusion, increased heat	Normal	Normal
Neurological examination	Normal	Normal	May be abnormal	Normal

Table 2: Differences between inflamed and damaged joints

	Inflamed joint	Damaged joint
Early morning stiffness	Prolonged	Brief
Inactivity stiffness	Prolonged	Brief
Increased warmth	+	–
Stress pain	Yes	No
Capsular soft-tissue swelling	+	–
Effusion	+++	+/–
Coarse crepitus	–	+++
Erythema	+/–	–
Malalignment/ deformity	–	+/–
Instability	–	+/–

Table 3: Shows a broad classification of the causes of arthritis with a focus on major causes of monoarthritis

Acute arthritis	Chronic arthritis
Inflammatory	
Monoarthritis Crystal induced arthritis (gout and pseudogout) Septic arthritis Gonococcal arthritis Acute onset of inflammatory polyarthritis (like RA, SLE)	**Monoarthritis** Tubercular arthritis Fungal arthritis Other infections (e.g Brucellosis) Immunoinflammatory arthritis Crystal induced arthritis
Polyarthritis (e.g., acute onset of polyarthritis, reactive arthritis)	Polyarthritis (e.g., RA, psoriatic arthritis, spondyloarthritis)
Non-inflammatory	
Monoarthritis Hemarthrosis Trauma	**Monoarthritis** Single joint osteoarthritis Neuropathic arthropathy Osteonecrosis Pigmented villo nodular synovitis
Polyarthritis	Polyarthritis (e.g., osteoarthritis)

time.[1,2] A significant proportion of patients who present with musculoskeletal complaints have in fact systemic illness such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE) etc. which may be potentially life-threatening if not detected, correctly diagnosed and treated. These conditions have to be distinguished from other musculoskeletal conditions, which have no systemic component. The evaluation should proceed to ascertain if the complaint is (1) articular or non-articular in origin, (2)

Table 4: Diagnostic Clues in Patients Presenting with Joint Pain

Clues from history and physical examination	Diagnoses to consider
Sudden onset of pain in seconds or minutes	Fracture, internal derangement, Trauma, loose body
Onset of pain over several hours or one to two days	Infection, crystal deposition disease, other inflammatory arthritic condition
Insidious onset of pain over days to weeks	Indolent infection, osteoarthritis, infiltrative disease, tumor
Intravenous drug use, immunosuppression	Septic arthritis
Previous acute attacks in any joint, with spontaneous resolution	Crystal deposition disease, other inflammatory arthritic condition
Recent prolonged course of corticosterioid therapy	Infection, avascular necrosis
Coagulopathy, use of anticoagulants	Hemarthrosis
Urethritis, conjunctivitis, diarrhea, and rash	Reactive arthritis
Psoriatic patches or nail changes such as pitting	Psoriatic arthritis
Use of diuretics, presence of tophi, history of renal stones or alcoholic binges	Gout
Eye inflammation, low back pain	Ankylosing spondylitis
Young adulthood, migratory polyarthralgias, inflammation of the tendon sheaths of hands and feet, dermatitis	Gonococcal arthritis
Hilar adenopathy, erythema nodosum	Sarcoidosis

inflammatory or non-inflammatory in nature, (3) acute or chronic in duration, and (4) localized (monoarticular) or widespread (polyarticular) in distribution.

ARTICULAR VERSUS NONARTICULAR

The first step in approach to a patient with arthritis is to confirm that the origin of pain is from the joint. (anatomical basis).[3] Questioning and examination will allow the distinction of four main origins (Table 1):

a. Articular pain

b. Extra articular pain:

- periarticular pain
- neurogenic pain
- referred pain.

Articular structures include the synovium, synovial fluid,

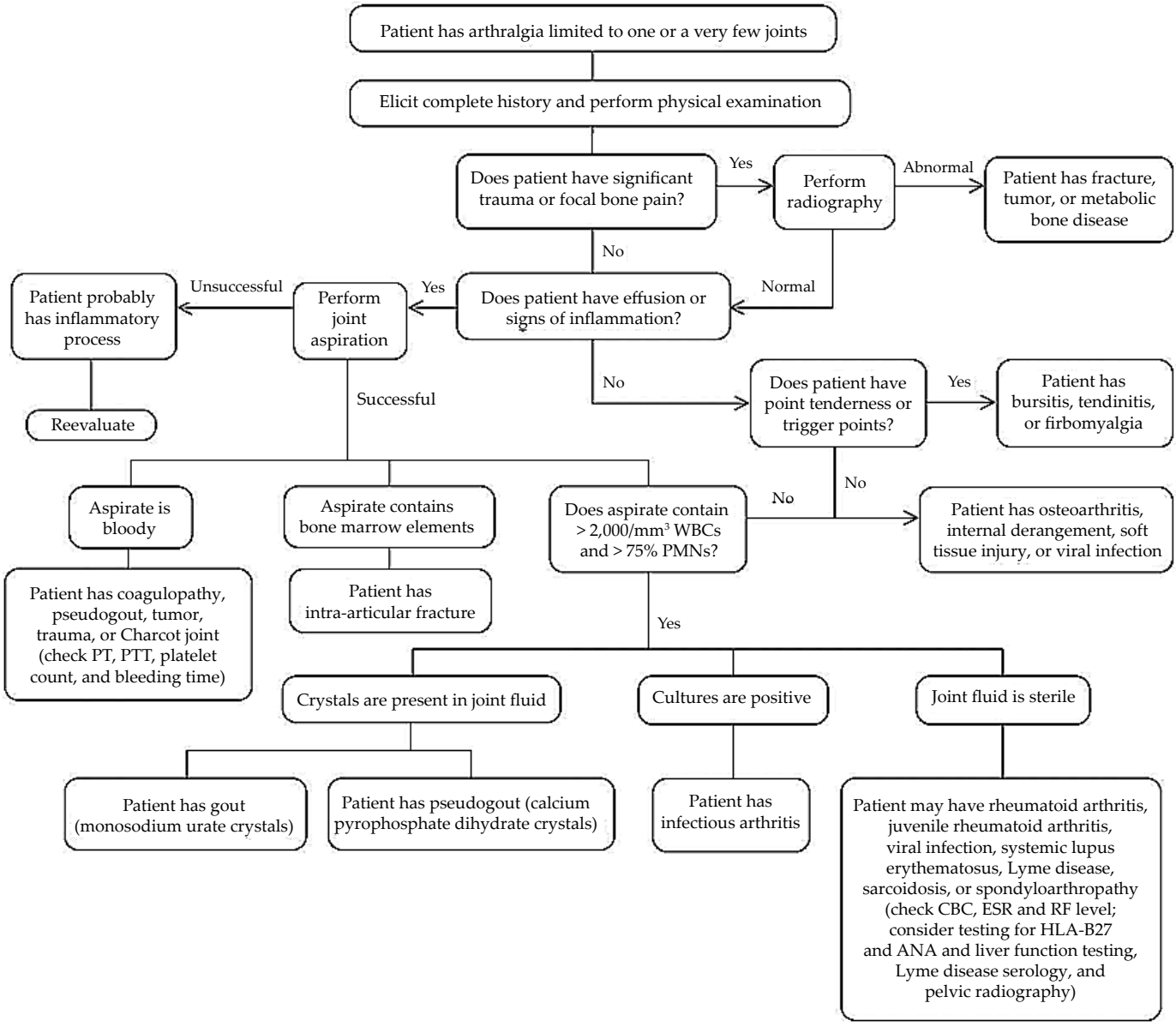


Fig. 2: Diagnosing Acute Monoarthritis

articular cartilage, intraarticular ligaments, joint capsule, and juxta-articular bone. Non articular (or periarticular) structures, such as supportive extra articular ligaments, tendons, bursae, muscle, fascia, bone, nerve, and overlying skin, may be involved in the pathologic process.

Arthropathies – that is, diseases affecting the joints – are at the heart of rheumatology.

As the first step we have to recognise that this is an articular syndrome. Once this is done four fundamental features of the articular pattern should be defined:

1. Whether the disease is inflammatory or non-inflammatory in nature.
2. The temporal pattern of the disorder; especially acute versus chronic duration.
3. The spatial pattern: primarily, monoarthritis or polyarticular arthritis and the presence of axial involvement.
4. The existence of extra-articular and/or systemic manifestations.

INFLAMMATORY VERSUS NON-INFLAMMATORY DISORDERS

Determine the nature of the underlying pathologic process and whether inflammatory or non-inflammatory findings exist. Inflammatory Disorders may be infectious (Neisseria gonorrhoeae or Mycobacterium tuberculosis), crystal-induced (gout, pseudogout), immune-related (rheumatoid arthritis [RA], systemic lupus erythematosus [SLE]), reactive(rheumatic fever, reactive arthritis), or idiopathic. Non-inflammatory disorders may be related to trauma (rotator cuff tear), repetitive use (bursitis, tendinitis), degeneration or ineffective repair (Osteoarthritis), neoplasm (pigmented villonodular synovitis) or pain amplification (fibromyalgia).

The most important goal is to differentiate the features of joint damage, predominantly caused by OA, from those of inflammatory joint disease (Table 2).

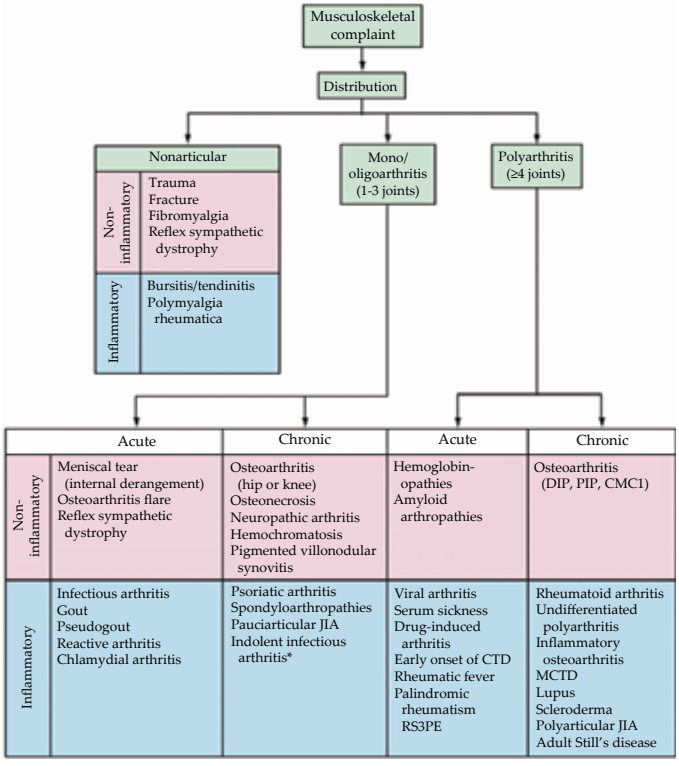
Table 5: Synovial Fluid Characteristics in the Clinical Situations, with Imaging and Investigation Techniques Best Used to Identify the Cause

Diagnosis	Cells	Microorganisms	Appearance	Imaging Modality	Comments
Bacterial arthritis	Neutrophils 10,000-100,000	Gram stain usually positive	Turbid/pus	Aspiration to dryness: may need ultrasound	Systemic symptoms. Gram stain Blood and synovial fluid culture
Gonococcal arthritis	Neutrophils 10,000-100,000	Gram stain usually positive	Turbid/pus	Aspiration to dryness; may need ultrasound	Systemic symptoms, Gram stain Blood and synovial fluid culture
Crystal arthritis	Neutrophils 10,000-100,000		Turbid/pus	XR,CPPD	Presence of appropriate crystals Acute serum urate unreliable
Tuberculous arthritis	Mononuclear 5000-50,000	Acid-fast stain often negative	Turbid/pus		At-risk population Ziehl-Neelsen stain biopsy may be necessary
Inflammatory moioarthropathies	Neutrophils 5000-50,000		Slightly turbid	Ultrasound/ MRIfor early synovitis and erosions	Serum autoantibodies such as RF, AC PA, ANA
Osteoarthritis	Mononuclear 0-2000	—	Clear	XR changes	Usually noninflammatory CPPD may be present
Internal derangement	Red blood cells	—	Clear/turbid	MRI	Arthroscopy may be necessary
Trauma	Red blood cells		Clear/turbid	XR	Tc bone scan may aid diagnosis if radiograph normal
Ischemic necrosis		—		MRI in early disease	XR abnormal only in advanced cases
Rarer Causes					
Sarcoidosis	Mononuclear 5000-20,000	—		CXR	
PVNS	Red blood cells	—	Turbid	Ultrasound and MRI	Synovial biopsy essential
Charcot's	Mononuclear 0-2000	—		XR	CPPD may be present
Lyme disease	Neutrophils 0-5000		Clear/turbid		SF eosinophilia may be found Serology for *Barmlia*
Amyloid	Mononuclear 2000-10,000		Turbid		Synovial biopsy for Congo red stain

ACPA, anticitrullinated protein antibody; ANA, antinuclear antibody; CPPD, calcium pyrophosphate dehydrate deposition; CXR, chest radiograph; MRI, magnetic resonance imaging; PVNS, pigmented villonodular synovitis; RF, rheumatoid factor; SF, synovial fluid; XR, radiograph.

Inflammatory disorders may be identified by any of the four cardinal signs of inflammation (erythema, warmth, pain, or swelling). In active inflammatory disease pain is worst in the morning (often waking the patient up a little early) and is relieved as they get up and start to move their joints. Morning stiffness is often prolonged, lasting for more than 30 min and sometimes for several hours. Stiffness after rest may persist for more than 5 min. With inflammatory disease sufficient to trigger the acute phase response, the patient may additionally complain of non-specific features such as fatigability, weight loss, night sweats (the commonest symptom of pyrexia

Table 6: Algorithm for assessing initial history and examination. CMC, carpometacarpal; CTD, connective tissue disease; DIP, distal interphalangeal; JIA, juvenile idiopathic arthritis; MCTD, mixed connective tissue disease; PIP, proximal interphalangeal



Joint damage/OA is typically associated with pain that increases with repeated use of the joint, which is relieved by rest, and which is often worst towards the end of the day. Patients may describe pain and stiffness(gelling) that increases again after resting that subsides after just a few minutes. Early morning stiffness in OA is 'worn off' in well under 30 minutes. Although OA signs may be detected in many joints on examination (many being asymptomatic), OA usually causes pain in just one or a few joints at any one time. Extra-articular manifestations (eg, anterior uveitis, skin lesions, lung or bowel problems) are not associated with OA, which is purely a condition of the joints, although age-related co-morbidities (eg, obesity, hypertension, depression) may commonly occur in older patients with OA and contribute to their participation restriction.

It is noteworthy that people with OA may suffer 'flares' of pain which may relate to minor inflammation or be biomechanically initiated. During such pain exacerbations patients may have more prolonged morning and activity stiffness. However, inflammation is not a prominent clinical feature and OA does not trigger the acute phase response. Conversely, longstanding but 'inactive' inflammatory arthritis will be associated with 'mechanical' pain reflecting joint damage caused by their inflammatory disease.

Physical examination will support joint damage/OA if there is coarse crepitus, joint-line tenderness (often localised rather than universal as in inflammatory arthritis) and/or bony swelling (osteophyte) along the joint margin. Deformity may also be present in later stages of joint damage and OA.

In inflammatory diseases the synovium becomes inflamed, engorged and eventually hypertrophied and the volume of synovial fluid increases. Causing intra-articular hypertension leading to pain, stiffness and restriction of movement. A joint with intra-articular hypertension is most comfortable in the position that minimises the pressure increase. This position, generally mild to mid flexion, and is termed the 'loose-pack' position, in which the capsule is normally at its loosest and therefore can accommodate an increase in fluid and soft tissue. Conversely, the positions in which the capsule is naturally tight – the 'tight-pack' positions at the extremes of range of movement – are the positions that are the first to be painful when synovitis is developing, and the first movements to become restricted.

This uneven distribution of pain, maximal in all tight pack positions, is called 'universal stress pain' – the most sensitive sign of synovitis, occurring even before there is visible swelling or restricted movement (Figure 1). Joint damage is associated with a more even spread of pain throughout the range of movement.

Joint inflammation may also cause increased warmth palpable over the capsular contour. The summated features that allow distinction between joint damage and joint inflammation are shown in table. Age is also an important factor. Joint conditions before the age of 40 are likely to be inflammatory if not traumatic. Inflammatory arthritis will usually establish itself in a matter of days to weeks or months, whereas patients with OA tend to present to doctors only after years of variable but very slowly increasing pain.

THE SPATIAL PATTERN

Monoarthritis

Monoarthritis which is arthritis of a single joint can either be acute(of < 6 weeks duration) or chronic (of > 6 weeks duration) or be either inflammatory or non-inflammatory as given in the Table 3. Acute monoarthritis in adults can have many but crystals, trauma, and infection are the most common. Prompt diagnosis of joint infection, which often is acquired hematogenously, is crucial because of its destructive course (Figure 2). A prospective, three-year study[4] found that the most important risk factors for septic arthritis are a prosthetic hip or knee joint, skin infection, joint surgery, rheumatoid arthritis, age greater than 80 years, and diabetes mellitus. Intravenous drug use and large-vein catheterization are predisposing factors for sepsis in unusual joints (e.g., sternoclavicular joint).[4] Gonococcal arthritis is the most common type of non-traumatic acute monoarthritisin young, sexually active persons in the United States. It is three to four times more common in women than in men.[4,5] Nongonococcal septic arthritis, the most destructive type, generally is monoarticular (80 percent of cases) and most often affects the knee (50 percent of cases).[4,7] Staphylococcus aureus is the most common pathogen in non-gonococcal

Table 7 : Distinguishing Different Causes of Polyarthritis

Arthritis	Patient Profile	History/Onset	Joints Involved	Type of Arthritis	Supportive Tests
GC	F > M, young, active sexually	Fever, acute oligoarthritis or polyarthritis	Wrist, knee, tenosynovitis	Inflammatory	↑ESR/CRP, ↑WBC
Gout	Men, postmenopausal women	Intermittent oligoarticular early, polyarticular later	MTP, toes, ankle, knee [hands late]	Acute sudden onset severe pain with attacks	↑CRP, ↑WBC Normal uric acid in 40% acutely
HHC	M > F. mean age, 50	Intermittent oligoarticular or polyarticular	MCP, hip, knee, feet	Intermittent or chronic inflammatory	↑ESR/CRP, ↑LFTs, HFE gene, x-rays—chondrocalcinosis and osteophytosis
OA	F > M. ↑Age men w/ knee or hip	Additive oligoartictlar or polyarticular	DIP, PIP, first CMC1, knee, hip, MTP, spine	Noninflammatory asymmetric or symmetric, bony swelling	Normal laboratory results
PMR	M= F, older white	Prolonged AM stiffness or soreness, weight loss	Girdle (hip, shoulder) muscles; seldom synovitis	Inflammatory, chronic	Anemia, ↑ESR/CRP, ↑LFTs
PsA	Long history of psoriasis	Insidious, additive	DIP, PIP, knees, feet, spine	Inflammatory, asymmetric oligoarticjular	↑CRP/ESR, negative RF, HLA-B27, ↑Uric acid
Pseudogout	M = F, older patients	Intermittent oligoarticular or polyarticular	Knee, wrist finger, MTP	Intermittent or chronic inflammatory	↑CRP, ↑WBC
RA	F>M. 35-50 yr	Insidious, additive	PIP, MCP, wrist MTP, knee, ankle	Symmetric inflammatory	↑CRP/ESR, +RF, +CCP
UPA	F>M	Insidious, one to four joints	Same as RA	Inflammatory	↑CRP/ESR
Viral (HBV. HCV)	Hepatitis risk factors	Acute, additive polyarthritis	PIP, MCP, wrist, knee, ankle	Inflammatory	↑ESR/CRP, ↑LFTs, +HCWHBV serologies

CCP, cyclic citrullinated protein; CMC, carpometacarpal; CRP, C-reactive protein; DIP, distal interphalangeal; ESR, erythrocyte sedimentation rate; GC, gonococcal arthritis; HBV, hepatitis B virus; HCV, hepatitis C virus; HHC, hereditary hemochromatosis; LFT, liver function test; MCP, metacarpophalangeal; MTP, metatarsophalangeal; OA, osteoarthritis; PIP, proximal interphalangeal; PMR, polymyalgia rheumatica; PsA, psoriatic arthritis; RA, rheumatoid arthritis; RF, rheumatoid factor; UPA, undifferentiated polyarthritis; WBC, white blood cell; SLE = systemic lupus erythematosus; IBD = inflammatory bowel disease; RA = rheumatoid arthritis; PAN = polyarteritis nodosa; DIP = distal interphalangeal; PIP = proximal interphalangeal. *—The clues listed in this table are not, in themselves, diagnostic or complete; they are presented for illustrative purposes only.

septic arthritis (60 percent in some series), but non–group-A beta-hemolytic streptococci, gram-negative bacteria, and Streptococcus pneumoniae can be present.[3] Anaerobic and gram-negative infections are common in immunocompromised persons. Inflammation of a single large joint, especially the knee, may be present in Lyme disease. Mycobacterial, fungal, and viral infections are rare. Monoarticular inflammation can be the initial manifestation of human immunodeficiency virus (HIV) infection.[8] Many types of crystals can trigger acute monoarthritis, but monosodium urate (which causes gout) and calcium pyrophosphate dihydrate (CPPD, which causes pseudogout) are the most common. Calcium oxalate (especially in patients who are receiving renal dialysis), apatite, and lipid crystals[9] also elicit acute monoarthritis (Table 4). Transient arthritis sometimes results from intra-articular injection of corticosteroids. Osteoarthritis may worsen suddenly and manifest as pain and effusion. Spontaneous osteonecrosis may occur in patients with risk factors such as alcoholism or chronic corticosteroid use. Aseptic loosening is often the source of pain in a prosthetic joint. Infection, commonly from a skin source, is also possible and requires urgent attention.

Table 8: Selected Extra-Articular Manifestations Associated with Conditions that Result in Polyarticular Joint Pain*

Physical finding	Diagnoses to consider
Skin and mucous membranes	
Rash	
Erythema infectiosum	
Reticulated (lacy) rash	Human parvovirus B19 infection
Facial exanthem (slapped cheek)	Human parvovirus B19 infection
Malar rash	SLE, human parvovirus B19 infection, Lyme disease, rosacea, seborrhea, dermatomyositis
Plaques (scalp, navel, gluteal cleft)	Psoriasis
Heliotrope	Dermatomyositis
Erythema chronicum migrans	Lyme disease
Erythema marginatum rheumaticum	Rheumatic fever
Erythema nodosum	Sarcoidosis, Crohn's disease
Pyoderma gangrenosum	IBD, RA, SLE, anklyosing spondylitis, sarcoidosis, Wegener's granulomatosis
Palpable purpura	Hypersensitivity vasculitis, Schonlein-Henoch purpura, PAN
Livedo reticularis	Antiphospholipid-antibody syndrome, vasculitis, cholesterol emboli
Lesions	
Keratoderma blennorrhagicum	Reactive arthritis, psoriatic arthritis
Discoid skin lesions	Discoid lupus erythematosus, SLE, sarcoidosis
Gottron's papules or plaques	Dermatomyositis
Vesicopustule on erythematous base	Gonococcal arthritis
Eyes	
Iritis or uveitis	Spondyloarthropathies, sarcoidosis, Wegener's granulomatosis
Conjunctivitis	Spondyloarthropathies, SLE, Wegener's granulomatosis
Cytoid bodies (retinal exudates)	SLE

Physical finding	Diagnoses to consider
Skin and mucous membranes continued.	
Telangiectasia	Scleroderma
Thickened skin	Scleroderma, amyloidosis, eosinophilic fasciitis
Hair thinning	Hypothyroidism, SLE
Musculoskeletal system	
Tender points	Fibromyalgia
Heberden's nodes (DIP joints), Bouchard's nodes (PIP joints)	Osteoarthritis
Boutonniere and swan-neck deformities	RA, SLE, Ehlers-Danlos syndrome
Dactylitis ("sausage digits")	Spondyloarthropathies
Bursitis and enthesitis	Spondyloarthropathies
Constitutional conditions	
Fever	Bacterial or viral infection, Still's disease, subacute bacteria endocarditis, neoplasm
Bradycardia	Hypothyroidism
Cardiovascular system	
Mitral regurgitation and stenosis	Rheumatic fever
Aortic regurgitation	Ankylosing spondylitis, rheumatic fever, relapsing polychondritis, reactive arthritis, Marfan syndrome, Takayasu's arteritis
Cardiomyopathies	Viral infection, amyloidosis, sarcoidosis, SLE, polymyositis
New murmur, fever	Bacterial endocarditis, rheumatic fever
Diminished peripheral pulses	Giant cell arteritis, Takayasu's arteritis
Gastrointestinal system	
Splenomegaly	Felty's syndrome, tumor-associated arthritis
Hepatomegaly	Whipple's disease, hemochromatosis, amyloidosis, Wilson's disease

Contd..

Table 8: Selected Extra-Articular Manifestations Associated with Conditions That Result in Polyarticular Joint Pain*

Physical finding	Diagnoses to consider
Scleritis	RA, relapsing polychondritis
Ischemic optic neuritis	Giant cell arteritis, Wegener's granulomatosis
Ears, nose and throat	
Oral ulcers	SLE, Behcet's syndrome, reactive arthritis, Wegener's granulomatosis
Parotid enlargement	Sjogren's syndrome, sarcoidosis
Macroglossia	Amyloidosis
Scalp tenderness	Giant cell arteritis
Bloody or severe sinusitis	Wegener's granulomatosis
Inflammation of ear lobe	Relapsing polychondritis
Nails	
Onycholysis	Psoriatic arthritis, hyperthyroidism
Pitting	Psoriatic arthritis
Clubbing	IBD, Whipple's disease, hyperthyroidism
Nodules	RA, gout, Whipple's disease, rheumatic fever, amyloidosis, sarcoidosis
Tophi	Gout
Jaundice	Hepatitis, hemochromatosis
Hyperpigmentation	Whipple's disease, hemochromatosis
Positive fecal occult blood test	IBD
Genitourinary system	
Prostatis	Reactive arthritis, ankylosing spondylitis
Urethritis or cervicitis	Reactive arthritis, gonococcal arthritis
Scrotal or vulvar ulcers	Behcet's syndrome
Hypogonadism	Hemochromatosis
Balanitis circinata	Reactive arthritis
Neurologic system	
Entrapment neuropathies	RA, hypothyroidism, hyperparathyroidism
Facial palsy	Lyme disease
Peripheral neuropathy	SLE, amyloidosis
Chorea	Antiphospholipid-antibody syndrome, SLE, rheumatic fever
Mononeuritis multiplex	RA, SLE, Lyme disease, vasculitis (e.g., PAN)
Seizures	SLE
Lymphadenopathy	Tumor-associated arthritis, SLE

SLE = systemic lupus erythematosus; IBD = inflammatory bowel disease; RA = rheumatoid arthritis; PAN = polyarteritis nodosa; DIP = distal interphalangeal; PIP = proximal interphalangeal. *—The clues listed in this table are not, in themselves, diagnostic or complete; they are presented for illustrative purposes only.

Joint aspiration is necessary in monoarthritis. Synovial fluid characteristics are given in Table 5.

POLYARTICULAR ARTHRITIS

Polyarticular joint pain (i.e., pain in more than 4 joints) poses a diagnostic challenge because of the extensive differential diagnosis (Tables 6 & 7). Because many rheumatologic laboratory tests lack the desired specificity, results should be interpreted in the clinical context and with caution. The differential diagnosis can be narrowed through investigation of six clinical factors: disease chronology, inflammation, distribution, extra-articular manifestations (Table 8), disease course, and patient demographics. Algorithm 1 gives the summary of approach to arthritis.

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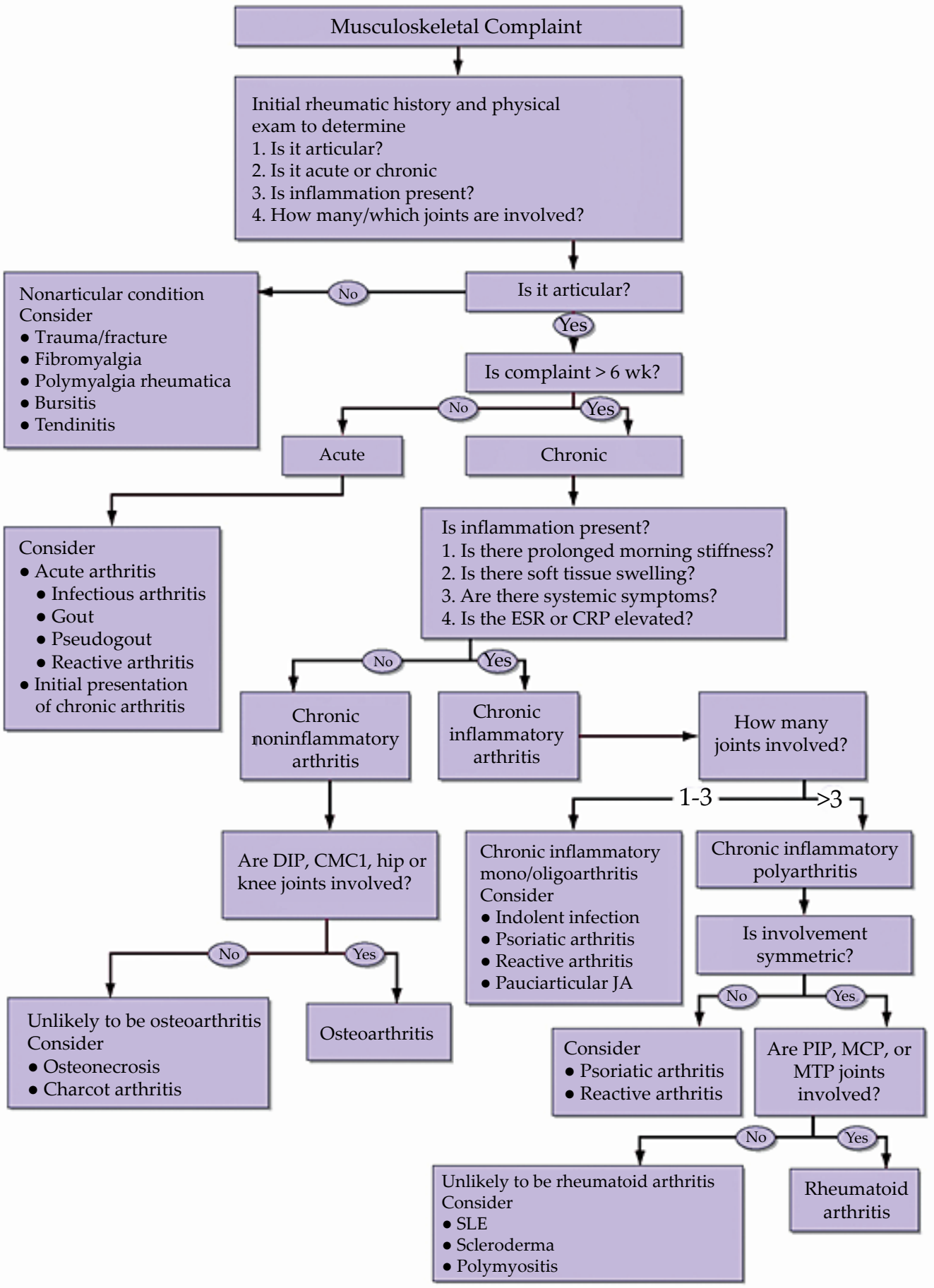

Algorithm 1: Approach to arthritis