Basics of

Rheumatology and Rehabilitation

STAFF OF
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Mansoura Faculty of Medicine

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Preface

This book is written by the staff members of Rheumatology, Physical Medicine and Rehabilitation Department, Faculty of Medicine, Mansoura University. It has been made to provide, in brief, the basic knowledge of this specialty in a systemic, concisely written, well-illustrated and comprehensive manner to be easily memorized by the undergraduate students.

We hope that this book provides our students with adequate basic rheumatological knowledge to make accurate clinical observations, arrive at a diagnosis and be aware of relevant differential diagnosis. We hope that this book can also provide our students with different modalities of physical medicine and role of interdisciplinary rehabilitation program in different medical conditions.

Also we hope that this book will be beneficial to general practitioner helping them to diagnose and manage some medical disease with rheumatological manifestation (how to deal with! And when to consult!).

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1 APPROACH TO A PATIENT WITH RHEUMATIC DISEASE

3 SIMPLE SCREENING QUESTIONS

1. Have you any pain or stiffness in your muscle, joints or back?
2. Can you dress yourself without any difficulty?
3. Can you walk up and down stairs easily?

If any answer is positive to any question, then detailed history must be obtained.

Rheumatism: is any painful disorder affecting the musculoskeletal system including: joints, muscles, connective tissues, soft tissues around the joints and bones.

Rheumatology: science dealing with the diagnosis and management of painful conditions in musculoskeletal system from conservative aspect.

The diagnosis of many rheumatic diseases is mainly clinical.

ARTICULAR SYMPTOMS

Pain

- Articular pain: localized to joint.
- Non-articular pain: originates from peri-articular structures e.g. tendon or bursae.
- Referred pain: e.g. cervical spondylosis presenting as shoulder pain.
- Inflammatory disease: joint pain tends to be worse at night.
- Mechanical disorder: pain is worse at the end of the day and after activity; relieved by rest.

Swelling

- Diffuse: synovial effusion; synovial hyperplasia.
- Localized: swelling of the structures surrounding the joint (e.g. bursa); Heberden's nodes; Bouchard's nodes.

Fatigue

- Important feature of many rheumatic disorders (e.g. rheumatoid arthritis and SLE).
- Prominent feature in fibromyalgia.

Stiffness

- Early morning stiffness: inflammatory arthritis (may last for several hours).
- Joint stiffness after rest may indicate osteoarthritis (gelling).
- Fibromyalgia.

Weakness

Caused by: muscle weakness, pain, mechanical factors (e.g. tendon and joint impairment) and nerve damage.

Limitation of movement

Caused by pain, contracture, arthritis, capsular fibrosis (e.g. frozen shoulder).

Deformities

e.g. genu varus, genu valgum, Boutonnière deformity, Swan-neck deformity, Dupuytren's contracture.

ANALYZING SYMPTOMS

Precipitating Factor

e.g. recent trauma, administration of a new drug, recent infection … etc.

Acute or Chronic

- Acute (<6 weeks duration): infectious arthritis, gout.
- Chronic: OA, RA.

Onset and Course of the Disease

- Slow insidious pattern: degenerative arthritis.
- Rapid onset, severe, self-limiting: Crystal-related inflammation.
- Remission and exacerbation: Inflammatory arthritis.

Inflammatory or mechanical in nature

See Table 1.1

Table 1.1. Pain due to mechanical versus inflammatory causes

Feature	Inflammatory	Mechanical
Morning stiffness	>1 hour	<30 minutes
Fatigue	Significant	Minimal
Activity	Improve symptoms	Worsen symptoms
Rest	Worsen symptoms	Improve symptoms
Systemic involvement	Yes	No
Corticosteroid requirements	Yes	No

Which Joints are Involved

- Peripheral: RA, OA, psoriatic.
- Axial: sero-ve arthropathy, OA.

Number of Joints Affected

- Mono: Septic arthritis, trauma, crystal arthritis
- Oligo (≤ 4 joints): Lower limb oligo-arthritis in reactive arthritis.
- Poly (≥ 5joints): RA, SLE.

Symmetry

- Symmetric: RA, SLE, SSc, PM/DM.
- Asymmetric: sero-ve arthropathy.

Sequence of joint involvement

- Additive: e.g. OA, RA.
- Migratory: e.g. Rheumatic fever, viral arthritis.
- Intermittent: e.g. gout.

EXTRA-ARTICULAR MANIFESTATIONS

- Constitutional symptoms (fever, weight loss, fatigue): connective tissue disease (CTD), vasculitis.
- Nodules: RA; rheumatic fever; connective tissue diseases; sarcoidosis; gout.
- Mucocutaneous: SLE (malar rash; discoid lesion; alopecia; oral ulcers), psoriasis, Behcet's disease (oral ulcer), Reiter's disease (circinate balanitis), Sjogren (dry eye: sicca syndrome; dry mouth: xerostomia).
- Raynaud's syndrome: Systemic sclerosis, SLE, mixed CTD.
- Diarrhea: enteropathic arthritis (ulcerative colitis; Crohn’s disease); coeliac disease; Whipple’s disease; proceed reactive arthritis.
- Urethritis: Reiter's disease.

- Eyes: conjunctivitis; iritis (Reiter's syndrome), uveitis (seronegative spondylo-arthropathies), episcleritis (RA), scleritis (RA), kerato-conjunctivitis sicca (RA and Sjogren's syndrome).
- Cardio-respiratory: Episodes of pericardial or pleuritic chest (connective tissue disease). Dyspnea due to pulmonary fibrosis or cardiac affection e.g. aortic regurgitation (spondyloarthropathies).
- Neurological:
  - Peripheral neuropathies, e.g. entrapment neuropathy e.g carpal tunnel syndrome.
  - Migraine; depression; stroke (e.g. SLE, vasculitis; antiphospholipid syndrome).

OTHER RELEVANT HISTORY

- Prodromal symptoms and events: Acute rheumatic disease may follow events e.g. upper respiratory tract infections, diarrhoea, genitourinary infection, insect bites (e.g. Lyme disease) and vaccinations.
- Medication: e.g. hydralazine induces drug induced lupus.
- Past history: previous attacks of the symptoms; psoriasis; diarrhea; risk of sexually transmitted infection.
- Family history: inflammatory arthritis, psoriasis.

EXAMINATION

General

- Patient appears ill: septic arthritis.
- Check for associated features: skin or eye involvement; disorders of the respiratory, cardiovascular, abdominal or neurological systems.

Joint examination

- Check joints for tenderness and swelling; asymmetry of colour; deformity; limitation of movement; muscle wasting.
- Check both passive and active range of joint movements.

A) Upper limbs

- Shoulder examination: test glenohumeral, acromioclavicular and sternoclavicular joints.
- Check for swelling or deformity of the elbow and hand.
- Assess pronation, supination and grip, and dexterity by placing tip of each finger on tip of thumb.
- Pain when 2nd to 5th metacarpals are squeezed suggests synovitis.

B) Lower limbs

- Observe patient standing to check for deformity of upper leg, lower leg or foot.
- Gait: observe the patient walking, turning, and walking back.
- Knee and hip examination:
  - with patient on couch: check hip and knee ROM; knee crepitus.
  - Examine each knee for joint effusion: patellar tap, cross fluctuation tests.

- Check for quadriceps bulk.

- Check feet for synovitis, for callosities, deformities and high or low arch.

C) Spine

- With the patient standing, check from behind to detect lateral spinal curvature, difference in level of the iliac crests and asymmetry of the paraspinal muscles.
- From the side, check for anteroposterior curvature.
- Assess all movements of neck and lower back.
- Check lumbar spine and hip flexion (modified Schober's test).

INVESTIGATIONS

Full blood count

- Anaemia: of chronic disease or blood loss from gastric irritation secondary to NSAIDs.
- White cells: leucopenia (SLE); neutropenia (Felty's syndrome); neutrophilia(septic arthritis), eosinophilia (polyarteritis nodosa),
- Platelets: may be ↑ in RA and may be ↓ in SLE.

Acute phase proteins

↑ ESR and CRP in inflammatory activity.

Serologic

- Rheumatoid factor support diagnosis of RA.
- Anti-CCP: more specific than rheumatoid factor in RA.
- ANA (Table 2.2).

Genetic

HLA B27 – Increased positivity in ankylosing spondylitis and other spondyloarthropathies.

Synovial fluid

- Raised White cell count (infection)

Table 2.2. Selected ANA with High Sensitivity or Specificity for Rheumatic Diseases

Anti-	SLE	Other conditions
dsDNA	60–80%, Highly Specific to SLE (>97%) Serum Level correlate with lupus nephritis and SLE flare	
Sm	10–40%, Highly Specific to SLE	
U1 RNP	30–40%	MCTD: 100%
Ro (SS-A)	50%, associated with photosensitivity, subacute cutaneous lupus, interstitial lung disease. Can cross the placenta causing neonatal cutaneous lupus and congenital complete heart block.	Sjögren syndrome: 75% RA: 10–15%
La (SS-B)	10–15%	Primary Sjögren syndrome: 40–50% congenital complete heart block: 90% neonatal cutaneous lupus: 70%
centromere		CREST: 60% (Highly specific: >98%) Scleroderma: 15%
Scl-70		Scleroderma: 40% (Specificity, 100%).
Histones	SLE: 50–70%	Drug induced lupus: >95%

- Gram stain (tuberculosis)
- Culture and sensitivities.
- Crystal identification – urate, calcium pyrophosphate.

Others

- Urine: proteinuria (SLE).
- Serum uric acid: may be raised in gout.

Imaging:

- X-rays: RA (juxta-articular osteoporosis; erosions); OA (osteophytes; asymmetric narrowing).
- Ultrasound.
- CT scan
- MRI – much greater information of bone, joint and soft tissue.

Arthroscopy

Direct view of joint and intra-articular structures.

2 CONNECTIVE TISSUE DISEASE

- A group of chronic inflammatory disorders predominantly affecting females.
- They involve many different organs ➔ therefore exhibit a wide spectrum of clinical manifestations.
- Their etiology is unknown but generally thought to be multifactorial involving immunological, genetic, environmental and possibly viral factors.

CTD include

- Rheumatoid arthritis.
- Systemic Lupus Erythematosus.
- Systemic sclerosis.
- Polymyositis and dermatopolymyositis.
- Mixed connective tissue disease.
- Vasculitis

Common features of CTD

- Constitutional features.
- Overlapping clinical features.
- Overlapping pathologic features.
- Prominent immunologic abnormalities.

Rheumatoid Arthritis

A chronic systemic inflammatory disease involving synovial joints and occasionally extra-articular manifestations are present.

EPIDEMIOLOGY

- Incidence: 1-3% of the population (most common inflammatory arthritis).
- Age: most often starts at age of 40-60 years (any age can be affected).
- Female : male ratio = 3:1.

ETIOLOGY

Unknown, but many theories are suggested

- Autoimmunity: antibodies against self antigen.
- Genetic: being in more than one member in the family, associated with HLA-DR4.
- Endocrinal: more in females, remission with contraceptive pills and during pregnancy, exacerbate after labour.
- Infection: some organisms isolated from the synovial fluid mostly viruses.
- Trauma: physical or psychological.

PATHOGENESIS

- The primary site of inflammation is the synovium of the joint.
- The thin synovium becomes inflamed and proliferates (thickened) forming pannus.
- As the disease progresses, the pannus invades and damages the cartilage and bone ➔ erosion and deformity (Figure 2.1).

ONSET AND COURSE

- Insidious onset (70% of cases).
- Less common onset
  - Acute mono- or poly-arthritis (15%)
  - Palindromic onset: recurrent episodic self-limited arthritis (5%)
  - Extra-articular onset
- Course: remission and exacerbation.

Triggering factor
(?? Infection, trauma, environmental)

↓

Hitting genetically predisposed patient
(HLA-DR4)

↓

Formation of immune complex
(Antigen+Anti-body+complement)

↓

Immune complex precipitate in synovium

↓

Pannus formation
(inflamed, proliferated synonium)

↓

Invade cartilage, bone and surrounding tissues

↓

Damage and deformity

Figure 2.1. Summary of Etio-Pathogenesis of RA

MUSCULOSKELETAL MANIFESTATIONS

- Chronic polyarthritis (bilateral, symmetric): usually affect peripheral small joints of the hands: MCP, PIP, and wrist joints (sparing DIP) and feet: ankle, MTP joints (Figure 2.2). Also other joints of the body may be affected.
- The affected joint is warm, tender, swollen and painful on movement.
- Morning stiffness: lasting for > 1 hour.
- Tenosynovitis, particularly affecting the flexor tendons in the palm of the hand, can cause trigger finger.
- Bursitis e.g. Baker's cyst.
- Muscle wasting, particularly in the hand.
- Osteoporosis, early juxta-articular. Later, generalized.
- Deformities may occur in long-standing RA:
  Common in sero +ve disease, neglected cases, badly managed cases or late diagnosis cases. Examples (Figure 2.3):
  - ulnar deviation of fingers at level of MCPj
  - Swan neck deformity (hyperextension of PIPj + flexion of DIPj)
  - Boutonniere deformity (flexion of PIPj + hyperextension of DIPj)
  - Z-shaped thumb (flexion of MCP + hyperextension of IPj).
  - Hammer toe (hyperextension of MTPj + flexion of IPj).

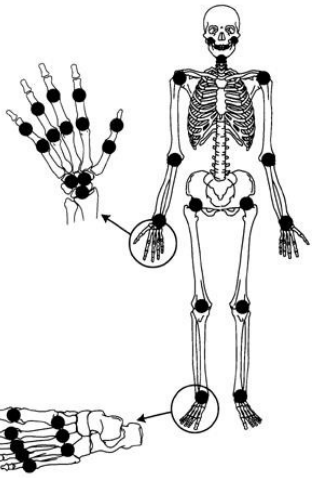


Figure 2.2. Distribution of joint affection in RA.

EXTRA-ARTICULAR MANIFESTATIONS

- Constitutional: low grade fever, anorexia, easy fatigability, weight loss.
- Rheumatoid subcutaneous nodules.

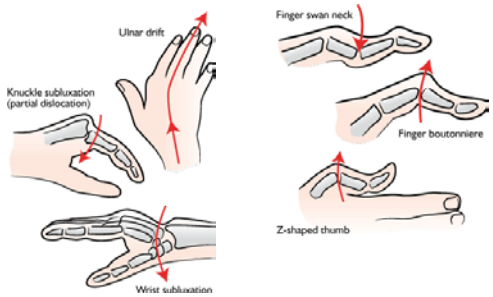


Figure 2.3. common hand deformities in RA.

- 20-30% of sero+ve RA patients.
- Painless.
- Any site, most commonly over joints, extensor surface of forearm and pressure points (Figure 2.4).
- Associated with more severe disease, enlarge when RA is active.

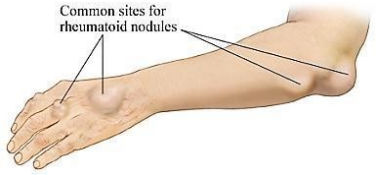


Figure 2.4. Common site for rheumatoid nodules

- Skin: palmar erythema, purpuric eruption, vasculitis, Raynaud's phenomenon.
- Chest: pleural effusion, pleurisy, pulmonary fibrosis, rheumatoid nodules.
- Eye: keratoconjunctivitis sicca (Sjogren syndrome in 10%), episcleritis, scleritis, scleromalacia, scleromalacia perforans.
- Heart: Pericarditis and pericardial effusion (usually asymptomatic).
- Vasculitis:
  - May occur in severe and long-standing RA.
  - Small vessel vasculitis: nailfold infarct, leg ulcers, purpura.
  - Medium vessel vasculitis: large areas of skin necrosis, digital gangrene.
- Nervous system:
  - Compression neuropathy e.g. carpal tunnel syndrome.
  - Peripheral neuropathy: mild glove and stock sensory impairment.
  - Mononeuritis multiplex: occurs as a result of vascultitis.
  - Atlanto-axial subluxation, a common finding in x-ray (25%), usually asymptomatic (cervical cord compression is rare).
- Renal: secondary amyloidosis with proteinuria and nephrotic syndrome.
- Felty syndrome: triad of RA + splenomegaly + neutropenia.

LABORATORY INVESTIGATIONS

Blood count

- Anemia of chronic disease, iron deficiency anemia.
- Leukocytosis.
- Neutropenia (Felty syndrome).

Rheumatoid Factor

+ve in 85% of RA patients (70% in early RA).

- Value of RF:

Table 2.1. conditions other than RA with positive RF.

Normal population	Overall Elderly	4% 25%
Other Rheumatic diseases	Sjogren syndrome SLE Scleroderma Dermatomyositis	90% 35% 30% 5%
Other immunologic diseases	Sarcoidosis Cryoglobulinaemia Transplant recipients	
Chronic infections (usually low titer)	HCV TB Bacterial endocarditis Syphilis Leprosy Parasitic: bilharziasis, malaria	60% 15% 25% 10% 10%
Miscellaneous	Neoplasms especially after radiation and chemotherapy.	

- Help in diagnosis (one of the criteria for diagnosis).
- +ve RF does not make diagnosis: as RF can be found in other conditions (Table 2.1).
- -ve RF does not exclude possible RA.
- +ve FR factor indicate bad prognosis.
- Help in follow up of treatment (titer decreases with good control).

Anti-Cyclic Citrullinated peptides (Anti-CCP) antibodies

- Highly specific for RA (98%).
- Found in 33% of RF –ve RA patients.
- Can be detected in early RA.
- Useful in differentiating RA from disorders with articular symptoms and are RF +ve e.g. HCV.

ESR and CRP

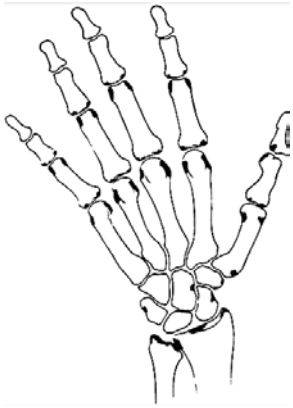
- Increased especially in active disease

Synovial Fluid

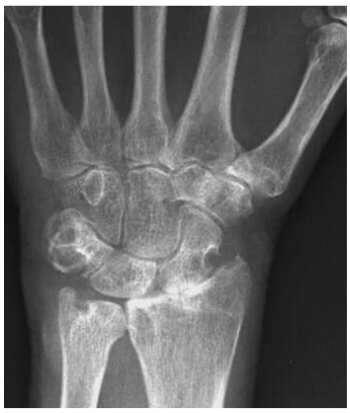
- Yellow, cloudy, low viscosity, cell count 2000-40000/mm^3.

RADIOLOGIC FEATURES (Figure 2.5)

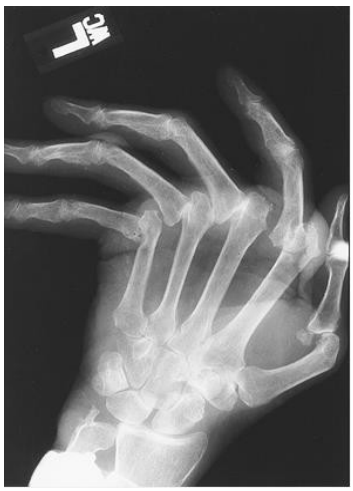
- Early plain x-ray may be normal.
- Juxtra-articular osteoporosis.
- Soft tissue swelling.
- Erosions and joint space narrowing.
- Deformities.



Sites susceptible to direct attack by pannus



Erosion in carpal bones, distal radius and ulnar styloid process.



Hands damage in advanced RA

Figure 2.5. Common radiologic features in RA.

FACTORS INDICATING BAD PROGNOSIS

- Generalized poly-arthritis (>20 total joints).
- Male patient.
- Extra-articular affection.
- Persistent elevation of ESR and CRP.
- Positive RF.

- Functional disability at 1 year after start of disease
- Radiographic erosions within the first 2 years from onset.
- HLA-DR4 genetic marker.

Rheumatoid Arthritis Classification Criteria

1. The 1987 American College of Rheumatology (ACR) Criteria

To be diagnosed as having RA, a patient must meet 4 or more of the following 7 criteria (*Criteria 1,2,3,4 should present for at least 6 weeks*):

1. Morning stiffness in or around joints for at least l hour before maximal improvement.
2. Soft-tissues swelling (arthritis) of 3 or more joint areas.
3. Swelling (arthritis) of PIP, MCP, or wrist joints.
4. Symmetric arthritis
5. Subcutaneous nodules
6. Positive test for RF
7. Radiographic erosions or peri-articular osteopenia in hand or wrist joints.

2. The 2010 ACR / EULAR Criteria

Every patient with a point total of 6 or higher is classified as an RA patient, provided he has synovitis in at least one joint and given that there is no other diagnosis better explaining the synovitis (Table 2.2).

Table 2.2. The 2010 ACR/EULAR classification criteria for RA

Finding	Points
Joint involvement*	
1 large joint	0
2-10 large joints	1
1-3 small joints (with or without involvement of large joints)	2
4-10 small joints (with or without involvement of large joints)	3
Involvement of more than 10 joints (with involvement of at least 1 small joint)	5
Serological parameters	
-ve RF and -ve ACPA	0
Low +ve RF or low +ve ACPA	2
High +ve RF or +ve ACPA	3
Acute phase reactants	
Elevated ESR or CRP	1
Duration of arthritis	
Symptoms lasting six weeks or longer	1

Joints examined are: MCPj, PIPj, IPj of the thumb, 2nd through 5th MTPj a nd wrist as small joints, and shoulders, elbows, hip, knee and ankles as large joints.

TREATMENT

Current strategy for treatment of RA include early aggressive treatment with one or more disease-modifying antirheumatic drugs (DMARDs) and/or biologic agents in addition to symptomatic therapy with NSAIDs, low-dose prednisone, physical therapy occupational therapy, rest, and patient education.

Patient education

Explain the chronic nature of the disease and the value of follow-up and the drug side effects.

Measures to decrease pain and stiffness

- Physical: heat therapy, paraffin wax bath, ultra-sound therapy, interferential current and TENS for muscles and tender points.
- Medical:
  - NSAIDs: relief pain and stiffness but have no disease modifying effect.
  - Systemic steroids: 5-10 mg daily to achieve symptomatic control as a "bridge therapy" before the onset of action of DMARDs.
  - Local steroid injections: of inflamed tendons, bursae or intra-articular.

Measures to prevent disease progression

- Conventional (synthetic) DMARDs:
  - Early treatment with DMARDs significantly results in better outcome (as articular damage in RA occurs in the early stages of the disease).
  - They decrease the levels of inflammatory indices and retard radiographic progression of articular affection.
  - Used either single or in combination of more than one drug.
  - Methotrexate (MTX):
    a. MTX is the most effective anti-rheumatic drug used and can induce low disease activity as monotherapy in about 30% of patients.
    b. In patients who fail to respond to an adequate dose (15 to 25 mg/wk) of MTX advance by the addition of synthetic DMARDs to MTX referred to as triple therapy (methotrexate, sulfasalazine, hydroxychloroquine).
- Leflunamide: a valuable alternative for patients intolerant to MTX.
- Hydroxychloroquine and sulfasalazine: used in milder diseases or if the previous two drugs are contraindicated.
- Biologic agents
  - Newly developed targeted therapies with rapid onset of action and highly effective for control of disease activity and prevention of structural joint damage.
  - Patients who fail to respond to DMARDs therapy within 6 months should receive a biologic agent (e.g. tumor necrosis factor [TNF] inhibitors such as etanercept or adalimumab) usually in combination with MTX or as monotherapy.
  - RA patients who fail to respond to an initial biologic agent should be switched to another biologic agent with a different mode of action.
  - Rituximab (monoclonal antibody against the protein CD20, which is primarily found on the surface of immune system B cells) is typically reserved for seropositive RA patients who have failed one or more biologic agents including at least one TNF inhibitor.
  - Limitations: very high cost and unknown long term consequences.

Measures to prevent or correct deformity

- Splints

- Static exercise during pain and inflammation.
- Active graduated exercises when pain subsides.
- Passive stretching
- Hydrotherapy
- Ultrasound
- Electric muscle stimulation: faradic stimulation help strengthening weak muscles.

Surgical for fixed uncorrectable deformities.

Systemic Lupus Erythematosus

A chronic inflammatory multisystem connective tissue disease predominantly affect females in the child bearing period, characterized by a wide range of clinical manifestations accompanied by striking immunologic abnormalities.

EPIDEMIOLOGY

- Female:male ratio is 9:1
- Age: mainly in age of 18-45 years (females in the child bearing period).

CLINICAL FEATURES

Constitutional manifestations

- Low grade fever.
- Anorexia.
- Malaise.
- Chronic fatigue.

Musculoskeletal manifestations

- Arthralgia (commonest > 90%).
- Arthritis (non-erosive).
- Joint deformities due to tendon or ligament laxity (joint erosion is uncommon).
- Inflammatory myopathy may cause muscle wasting.

Table 2.3. WHO classification of glomerulonephritis in SLE

WHO classification	Clinical features
I. Normal glomeruli	Asymptomatic
II. Mesangial disease	Low grade hematuria or proteinuria
III. Focal proliferative GN	Nephritic urinary sediment (hematuria, casts), proteinuria
IV. Diffuse proliferative GN	Hypertension, variable renal insufficiency.
V. Membranous nephropathy	Nephrotic syndrome
VI. Sclerosing nephropathy	Inactive urinary sediment, azotemia

Dermatologic and mucosal manifestations

- Butterfly rash: erythema over cheeks and nose (malar rash) sparing nasolabial fold.
- Discoid lesions: coin shapes.
- Non-specific rash in exposed areas.
- Mucous membrane ulcerations (painless).
- Alopecia: focal or generalized.

- Vasculitis: lesions at finger tips and around nail fold.
- Photosensitivity: skin rash as a result of unusual reaction to sun light.
- Raynaud's phenomenon.

Renal manifestations

- Common in SLE (> 50%).
- Patients with active lupus nephritis have proteinuria >0.5 gm/day (commonest, may be asymptomatic), hematuria (microscopic).
- ↑ serum creatinine and BUN.
- Renal biopsy in patients with active urinary sediment to determine type and activity of renal pathology (Table 2.3).

Pulmonary manifestations

- Recurrent pleurisy and pleural effusion (common),
- Pulmonary hypertension (secondary to pulmonary vasculitis).

Cardiac

- Pericarditis with small pericardial effusion (common).
- Myocarditis.
- Coronary artery vasculitis (in severe cases).
- Premature atherosclerosis (especially in patients treated with steroids).
- Non-bacterial endocarditis (Libman sack's endocartitis).

Nervous system manifestations

- Central: depression, psychosis, cognitive abnormalities, seizures.
- Peripheral: sensory or sensorimotor neuropathies, vasculitis may cause mononeuritis multiplex.

Gastrointestinal manifestations

- Non-specific symptoms: nausea, vomiting, abdominal pain are frequent.
- Vasculitis of mesenteric vessels ➔ bowel ischemia, infarction, perforation.
- Pancreatitis secondary to disease or steroid use.
- Gastritis secondary to NSAIDs or steroids.

Hematological manifestations

- Anemia (of chronic disease, hemolytic)
- Lymphopenia.
- Thrombocytopenia.
- Elevated ESR, normal CRP but raised if secondary infection occurs.
- Coagulation abnormalities:
    - Phospholipid antibody (lupus anti co-agulant). Interference with coagulation profile causing prolongation of the PTT.

- However, patients are not prone to bleeding but rather have higher incidence of thrombosis.
- Recurrent 2nd trimester abortion.

Immunological abnormalities

- Low C3 and C4 level reflect activation of immune complex cascade.
- Hyper gammaglobulinaemia due to hyperactivity of B cells.
- Autoantibodies are common:
  - ANA are present almost in all SLE patients (patients with –ve ANA are unlikely to have SLE).
  - +ve ANA are found in many other conditions (high sensitivity but low specificity).
    - Anti-dsDNA: specific to SLE but present in 60% of patients, levels of these antibodies rise with active disease.
    - Anti-Sm: specific for SLE.
    - Anti-histone antibodies specific for drug induced lupus.
    - Anti-Ro and anti-La antibodies: seen in SLE and Sogren's Syndrome.
    - Antiphospholipid antibodies (40%), but only minority has thrombotic events.

DIAGNOSTIC CRITERIA (SLICC "Systemic Lupus International Collaborating Clinics" 2012 CRITERIA)

Patient must have ≥ 4 criteria (at least 1 clinical and 1 laboratory criteria) or biopsy-proven lupus nephritis with positive ANA or Anti-DNA:

Clinical Criteria	**Immunologic Criteria**
1- Acute cutaneous lupus	1- ANA
2- Chronic cutaneous lupus	2- Anti-DNA
3- Oral or nasal ulcers	3- Anti-Sm
4-Non-scarring alopecia	4- Antiphospholipid Ab
5- Arhritis	5- Low complement (C3, C4, CH50)
6- Serositis	6- Direct Coombs' test (do not count in the presence of hemolytic anemia)
7- Renal	
8- Neurologic	
9- Hemolytic anemia	
10- Leukopenia	
11- Thrombocytopenia (<100,000/mm$^{3)}$	

TREATMENT

Treatment should be tailored to the patients findings.

Preventive measures

- Patient education.
- Regular evaluation.
  - Assess lupus activity
  - Routine investigations
  - Control of blood pressure

    - Control of hyper-lipidemia
- Photoprotection
    - Avoid exposure to sun.
    - Sun screens
- Pregnancy
    - Birth control with active lupus (especially nephritis) and with cytotoxic drugs.
- Infection control.
    - Suspect infection whenever there is fever.
    - Antibiotic prophylaxis for dental, gynecologic procedures.
    - Influenza and pneumococcal immunizations

Mild disease

- NSIADs: for joint pain, fever and mild systemic features e.g. serositis.
- Hydroxyl-chloroquine: for arthritis not controlled by NSAIDS, skin lesions and fatigue.
- Steroids:
    - Topical preparations: for skin lesions.
    - Low dose steroids (5-10 mg/day).

Moderate and severe disease

- Systemic steroids
    - The mainstay for treatment.
    - Indicated for: arthritis, serositis, severe hemolysis, thrombocytopenia, pneuminitis, vasculitis, cardiac involvement, central or peripheral nervous involvement, renal disease.
    - The starting dose is determined according to the disease activity and severity.
    - In acute and life-threatening manifestations, start with doses of 40-80 mg prednisolone orally daily.
    - With remission reduce dose gradually to maintain at 5-10 mg/day.
- Pulse steroid: methylprednisolone 1 gm IV for 3 successive days for lefethreatening lupus (severe renal, CNS, cardio-pulmonary or hemolytic abnormalities).
- Cytotoxic therapy:

  For patients with more serious manifestations e.g. severe nephritis or active cerebral disease.

  -Azathioprine – 2 mg/kg orally daily. Important as a steroid-sparing agent in patients with moderate-to- severe lupus.

  -Cyclophosphamide – 1-3 mg/kg orally daily or 0.5-1 gm/m^2 IV monthly. Full blood count and unrinalysis monthly to monitor side effects (bone marrow suppression, infection, hemorrhagic cystitis, infertility).

  -Cyclosporine and mycophenolate mofetil – used in severe cases e.g. severe nephritis.

  -IVIG and plasmapheresis – may be useful in patients with serious steroid-resistant exacerbations.

3 OSTEOARTHRITIS

Joint symptoms and signs of articular cartilage degeneration, in addition to the related changes in the underlying bone and at the joint margin.

Classification of OA

- Primary: unknown cause, affect certain joints in old age.
- Secondary (can affect any joint at any age): to
  - Local mechanical factors (trauma, menisectomy).
  - Joint diseases (RA, septic arthritis).
  - Systemic diseases (hyperparathyroidism).
  - Congenital anatomical abnormalities (leg discrepancy, scoliosis)

Primary Osteoarthritis

RISK FACTORS

- Age: advancing age ➔ loss of glycosamino-glycan ➔ leaving unsupported cartilage collagen fibers.
- Genetic: may be present especially in generalized OA.
- Sex: both sexes are affected but generalized OA is more common in females especially after menopause.
- Obesity: predisposes to knee OA.
- Repeated overload

PATHOLOGY

- Manifested first by fibrillation of the cartilage articular surface.
- Clefts in the cartilage surface then develop and eventually loss of the cartilage can be seen.
- Synovial membrane hypertrophy, fibrosis and contracture of the capsule.

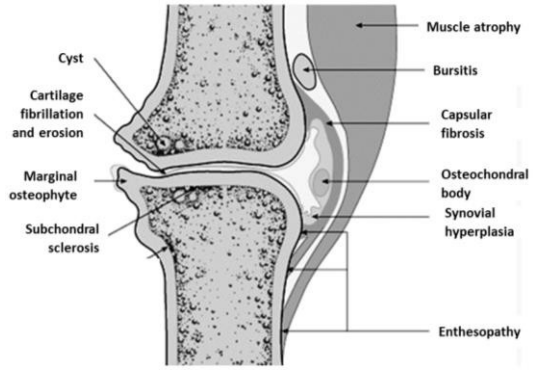


Figure 3.1. Pathologic changes in OA.

- Bone changes include: subchondral sclerosis, marginal osteophytes (Figure 3.1)

DISTRIBUTION OF JOINT INVOLVEMENT

Commonest joints (Figure 3.2)

Knee joint, lumbar and cervical vertebrae, hand PIP joints (Bouchard's nodes), DIP joints (Heberden's nodes), 1st CMC joint and feet (1st MTP joint).

Rarely affected joints

Ankle, shoulders, lateral MTP joints of the feet.

SYMPTOMS

- Pain: arising from several structures (bone, synovium, ligaments, capsules and muscle). Pain worsened by exercise and weight bearing. Pain is aching and poorly localized. As disease progresses, pain during rest.
- Inactivity stiffness: present for few minutes.
- Stiffness: morning stiffness is usually not a prominent feature in OA, and when present lasting no more than ¼ hour.
- Limitation of movement and activity.

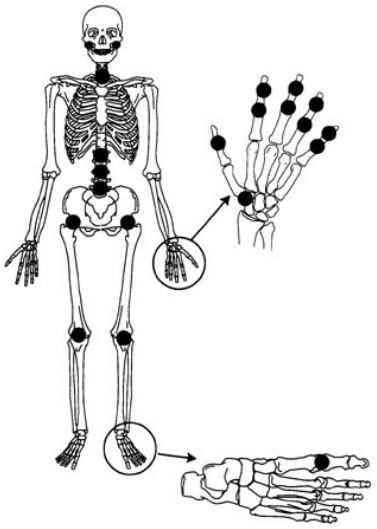


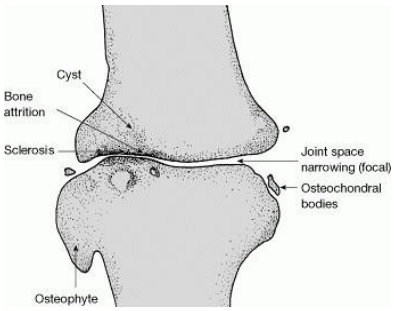
Figure 3.2 joints commonly affected by OA.

SIGNS

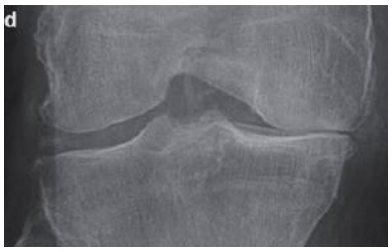
- Swelling due to synovial thickening, effusion or bony swelling.
- Wasting of muscles acting on the affected joints.
- Joint tenderness.
- Joint crepitus (coarse).
- Deformity e.g. flexion deformity of the knee, genu varum, genu valgum.

INVESTIGATIONS

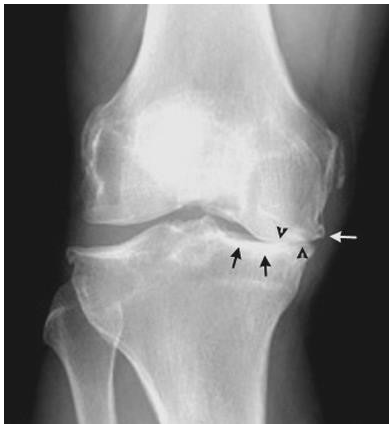
- Laboratory features are normal.
- Synovial fluid: good viscosity, normal mucin clot, slight increase in cell count.
- Plain x-ray: the most useful form of imaging to evaluate OA (Figure 3.3):
  - Joint space narrowing.
  - Subchondral bone sclerosis.
  - Subchondral bone cysts
  - Osteophytes (bone spurs)



Major radiographic features of OA



Severe joint space narrowing in the medial knee compartment. Note the lateral marginal osteophytes



Black arrows point to subchondral sclerosis. White arrow points to osteophytes. Black arrowheads point to joint narrowing in medial compartment

Figure 3.3. common radiologic features in OA

TREATMENT

- Assurance.
- Instructions for joint protection (to avoid overstress the affected joints.):
  - Don't lie or sit too long in one position.
  - Don't use low chairs.
  - Don't stand in same position or walk for long periods.
  - Don't over exercise the affected joints.
  - Don't use faulty postures that place stress on affected joints.
  - Don't load the joint when it is very painful.

- Reduction of body weight in obese patients.

- Physiotherapy (heat, cold, electric stimulation, laser, massage and exercise). Benefits of physiotherapy include
  - Decrease pain, stiffness, muscle spasm.
  - Improve joint range of motion.
  - Strengthen peri-articular structures ➔ improve joint support.
  - Improve blood supply and metabolism.

- Use simple analgesic for pain.
- Short courses of NSAIDS to control symptoms.
- Assistive devices (knee brace; stick) ➔ partially unload the joint.
- Chondroprotective drugs and viscosupplements (debatable).
- Surgical treatment in advanced cases:
  - Osteotomy to correct deformity.
  - Arthroplasty (partial or total joint replacement).

4 GOUT

Disorders of purine metabolism, which are characterized by serum uric acid elevation (hyperuricaemia) and urate deposition in the articular or extra-articluar tissue (Figure 4.1).

CLASSIFICATION

1. Primary gout (90%): hyperuricemia result from disorders of purine metabolism or abnormal excretion of uric acid.
2. Secondary gout (10%): due to either:
   a) Impaired excretion: caused by:
      - Chronic renal diseases.
      - Drugs (thiazide diuretics, low dose aspirin, cyclosporine and INH).
      - Hypertension.
      - Lead toxicity.
      - Hyperparathyroidism.
      - Hypothyroidism.
      - Increased lactic acid production (e.g. alcohol, starvation).
      - Glucose 6 phosphatase deficiency.

   b) Increased uric acid production
      - Myeloproliferative disorders (e.g. polycythemia vera, hemolytic anemia).
      - lymphoproliferative disorders (e.g. leukemia)
      - Others e.g. severe psoriasis.

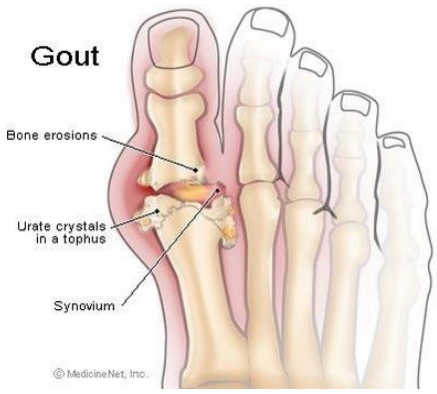


Figure 4.1. Gouty arthritis.

CLINICAL PICTURE

Acute gouty arthritis

Typical attack (95%): Acute gouty arthritis with:

- severe pain develops overnight, reaches a peak within hours
- The patient can't bear weight or even touch of the bed clothes
- The skin is red and may peel.
- Slight fever and chills may present.
- The most commonly affected joints are 1st MTPj, dorsum of the foot, knee (joints of upper limb are rarely affected).

Intercritical gout

- Asymptomatic intervals between acute attacks of gout.
- Some patients never experience 2nd attack.
- With repeated attacks of acute arthritis, the interval between attacks progressively shortens, and finally, joints become permanently mildly swollen and deformed with mild to moderate persistent pain.

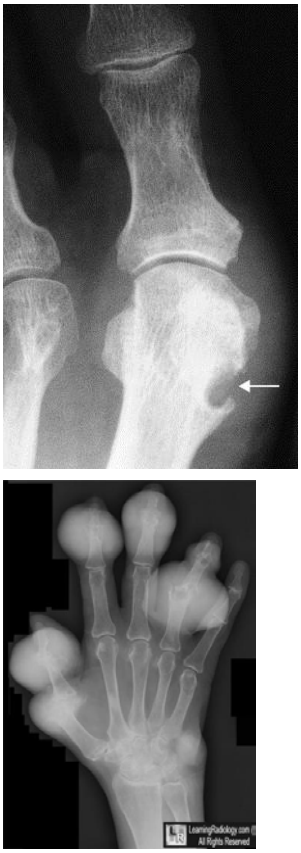
Chronic gout

- Recurrent acute attacks may lead to progressive joint damage, deformity and pain.

- Chronic tophaceous gout: large mono-sodium urate crystals deposits produce firm nodules (tophi), usual sites around extensor surfaces of fingers, hands, elbows, Achilles tendon and the ear.

INVESTIGATIONS

- Evaluation of pateint for causes of secondary gout.
- Fresh synovial fluid examination under polarized light microscope for presence of urate crystals (diagnostic). Synovial fluid is inflammatory in nature with predominance of neutrophils.
- Elevated serum uric acid (not diagnostic): may be normal in 30% of patients at time of acute attack. A high level alone is not diagnostic as asymptomatic hyperuricaemia is common
- Blood tests: leucocytosis, raised ESR and CRP (varies with gout severity).
- Radiologic features (Figure 4.2):
  - Soft tissue swelling around the affected joint.
  - In chronic gout: tophi, punched out erosions with sclerotic margin and overhanging edge.



Gout involving the 1st MTPj. Peri-articular swelling with 'punched out' erosion located away from the articular surface with an over-hanging lip appearance.

Large tophaceous deposits

Figure 4.2. Common radiologic features of gout.

TREATMENT

Asymptomatic hyperuricemia: no treatment except if

- Uric acid level > 11 mg/dl

Treatment of acute attack

- NSAIDs in maximum doses.
- Colchicine: 0.5 mg/3 hours for 12 hours.
- Systemic steroids and ACTH: cases with contraindications to NSAIDs and colchicine.
- Effusion in large joints should be aspirated and corticosteroid injected to reduce inflammation.

Treatment of underlying cause

Long term Treatment

Considered when acute attack subsides.

- Patient education: maintain ideal body weight, ingestion of at least 2 liters of fluids per day to prevent renal stones, avoid low dose aspirin.
- Diet: avoidance of high-purine foods e.g. meat and sea-food. Encourage intake of low fat dairy products and vegetable proteins.
- Colchicine (prophylaxis): 0.5-1 mg/day to prevent gout flares.
- Hypouricemic drugs:
  - Allopurinol :
    - Action: inhibits xanthine oxidase enzyme.
    - Dose: 100-300 mg/day.

- Side effects: rash, vasculitis, agranulocytosis.
- Contraindications: acute gout.
- Concurrent treatment: low dose NSIADs or colchicin for at least 4 months.

- Febuxostat :
  - Action: inhibits xanthine oxidase enzyme.
  - Dose: 40-120 mg/day.
  - Contraindications: acute gout.
  - Advantages: more safe than allopurinol in kidney diseases.

- Uricosuric drugs: (probencid, sulphinpyrazone):
  - Side effects: occasionally rash or hepatitis.
  - Contraindications: acute gout.
  - Concurrent treatment: low dose NSIADs or colchicin for at least 4 months.
- Pegloticase: in severe tophaceous gout and cases resistant to hypouricemic drugs.
- Joint aspiration for joint effusion and intra-articular corticosteroid injection for patients with persistent synovitis.
- Prevention of renal stones:
  - Alkalinization of urine (to maintain pH at 6): use sodium or potassium citrate or acetazolamide 500 mg at bedtime.
  - Intake of adequate fluid to produce at least 2 liters of urine daily.

5 LOW BACK PAIN

A very common condition affecting 80% of the individuals at some point in their life time.

CAUSES OF LBP

- Congenital: e.g. spina bifida, scoliosis.
- Traumatic: e.g. lumbar disc prolapse, fracture of the spine, tears or sprain of spinal ligament and/ or muscle.
- Degenerative: e.g. intervertebral disc (lumbar spondylosis), facet joint (osteoarthritis), spinal canal stenosis.
- Postural: e.g. bad posture (sitting, standing), inequality of limb length, high heels, pendulous abdomen.
- Inflammatory: e.g. ankylosing spondylitis, Reiter's disease, psoriatic arthritis, enteropathic arthritis.
- Infection: e.g. non-specific (osteomyelitis), specific (Pott's disease in TB).
- Metabolic: e.g. osteoporosis, osteomalacia, Paget's diseases.
- Neoplasm: benign, malignant (secondaries are more common than primaries).
- Referred (visceral): e.g. peptic ulcer, pancreatitis, pancreatic tumor, pyelonephritis, aortic aneurysm, peritoneal tumor, pelvic disease.
- Psychogenic LBP.

Table 5.1. Red flags for serious diagnosis in LBP

Alarming (Red flag) symptom/sign	Suggested serious diagnosis
Severe persistent pain not changes by position, not improved by rest	Infection, malignancy
Fever, chills, weight loss	Infection, malignancy
Pain worse in walking, radiating to lower limbs, exacerbated by spinal extension and relieved by sitting in flexion	spinal stenosis
Pain and stiffness > 30 minutes, worse in the morning in young adult male	Spondylo-arthropathy
Bilateral radiation of pain, Abnormal neurologic findings, sensory deficit, bowel/ bladder dysfunction, saddle anesthesia, +ve Babiniski, ankle clonus	Cauda equina compression (e.g. Central disc prolapse, rarely cancer)
Acute severe pain with point tenderness, history of Severe trauma (or even minor trauma in osteoporosis).	Fracture

N.B.

Although the most of LBP is mechanical in nature (e.g. disc prolapse, spondylosis, postural), however, the most serious rare causes must be excluded (Table 5.1).

Mechanical LBP

- Over 95% of LBP.
- Due to anatomic or functional abnormality, without underlying inflammatory or neoplastic diseases.
- Pain increases with physical activity and is released by rest and recumbency.
- Causes:
  - Postural (sprain or strain, lumbago, non-specific): 70%.
  - Lumbar spondylosis: 10%.
  - Disc herniation: 6%.
  - Spinal stenosis.
  - Spondylolisthesis.
  - Diffuse idiopathic skeletal hyperostosis.
  - Factures.

1. POSTURAL BACK PAIN (STRAIN AND SPRAIN, LUMBAGO)

- Bad posture is probably the most common cause of persistent back pain.
- Common predisposing factors:
  - Prolonged sitting or standing with leaning forward → flat lordosis.
  - High heeled shoes, pendulous abdomen → exaggerated lordosis.
  - Unequal leg length, asymmetric lifting heavy weight → scoliosis
- Correcting bad postural habits may be difficult for a patient to accept and may need re-inforcement through programs as back school.

2. LUMBAR SPONDYLOSIS

Degenerative joint disease affecting lumbar vertebrae and intervertebral disc causing pain and stiffness, sometimes with sciatic radiation (L4-5, S1,2,3) due to nerve root pressure by associated osteophytes.

Clinical Features

- Pain: midline, radiating to the region of buttock, occasionally sciatica. Pain worse towards end of the dayand often not aggravated by coughing and sneezing.
- Lumbar morning stiffness, inactivity stiffness.
- Diminished spinal mobility.
- Midline tenderness.
- Sensory and motor neurological signs (if there is root compression by osteophytes).

Radiology

- Narrow disc space.
- Osteophytes.
- Evidence of apophyseal osteoarthritis.

3. LUMBAR DISC PROLAPSE

One of the causes of mechanical LBP.

Etiology

Trauma, is usually not a direct one, typically lifting a heavy weight while back unsupported (bending).

Pathology

- Direction of prolapse mainly posterior or postero-lateral.
- Commonest site between L4-5 and L5-S1.

Symptoms (of 1st attack)

- Sudden onset of LBP while patient lifting a heavy object.
- Pain worse by straining (sneezing, coughing).
- Pain increase by movement, relieved by rest.
- Sciatic pain: pain along the course of the affected nerve.

Back signs

- Diminished or obliterated lumbar lordosis.

- Sciatic scoliosis (lateral bending to one side).
- Midline tenderness opposite prolapsed disc.
- Restriction of back movement.

Stretch signs

- +ve straight leg raising test (sciatic stretch):
  - The leg is lifted with knee extended. Sciatic roots are tightened over a herniated disc between 30º and 70º.
  - Indicate sciatic compression i.e. lower lumbar disc prolapse (Figure 5.1).
- +ve femoral stretch test:
  - The knee is flexed and lifted superiorly. Sharp pain that is generated in the anterior thigh is considered a positive test.
  - Indicate femoral nerve compression i.e. high lumbar disc prolapse (Figure 5.2).

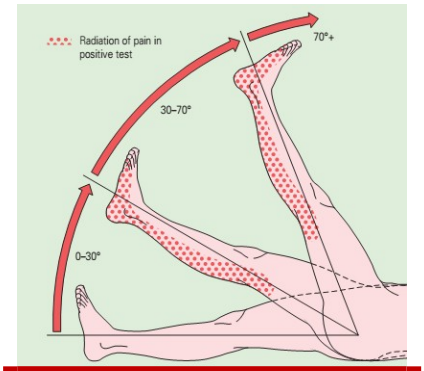


Figure 5.1. Straight leg raising test.

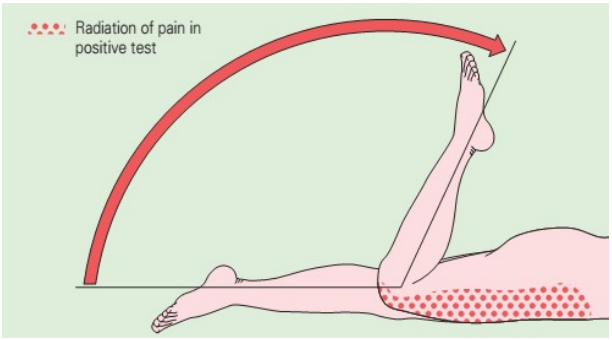


Figure 5.2. Femoral stretch tests.

Neurologic signs

Are usually localizing signs and depend on which root is compressed by the prolapsed material.

Investigations

- Plain x-ray: Narrow disc space, sometimes normal if small disc prolapse.
- CT scan: Localize exactly site of prolapsed disc.
- MRI**:** better imaging of soft tissues.
- Myelography: detect prolapse as filling defect.
- Dsciography.
- Radiculography.

Treatment of Mechanical LBP

Conservative treatment is the main line. Tailored to the specific needs of the individual patient.

Rest
- Days to few weeks.
- Kept to minimum and early mobilization should be encouraged.
- Early referral to physical therapy is essential.

Instructions
- Positioning:
- Sleep on firm matrix ➔ avoiding back sagging.
- Setting: increase disc pressure ➔ minimize in disc prolapse.
- Standing: avoid prolonged standing.
- Weight reduction.

Medication
- Analgesics and NSAIDs: during acute attack, infrequent courses.
- Muscle relaxants.
- Anticonvulsants.
- Anti-depressants

Physical Therapy
- Physical agents: (e.g. SWD, US, TENS). Advantages:

  - Local anti-inflammatory effect.
  - Decrease pain and muscle spasm.
  - Decrease fibrosis and adhesions.

- Exercise program
  - Cornerstone of conservative treatment and prevention.
  - Benefits:
    - Support vertebral column.
    - Restore normal curves of the spine.
    - Decrease intradiscal pressure.
    - Decrease load on facet joints and open intervertebral foramina.
    - Restore strength, flexibility, function.
    - Reduce pain.

- Traction:
  - Help suction of prolapsed disc.
  - Stretch vertebral ligaments, support the disc.

- Others e.g. local injection, manipulation, acupuncture

- Surgical treatment: indications
    - Progressive muscle weakness.
    - Sphincteric disturbance.
    - Failure of conservative treatment after 12 weeks with severe persistent pain.

Psychogenic LBP

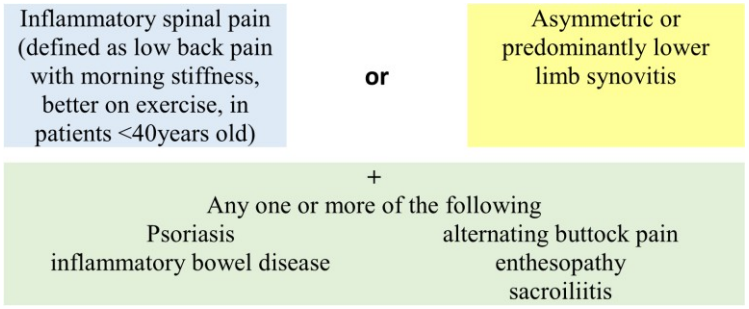
- Psychogenic illness may be manifested as LBP.
- Diagnosis is usually based on:
  - Detection of inadequate personality and psychological illness.
  - An organic disease is excluded.
- Symptoms usually very diffuse and not follow anatomic distribution.
- The description of pain is exaggerated.
- Patient is usually highly demonstrative, hands used to point out various painful areas.
- No root signs could be detected and patient is hesitating about areas of paraesthesia.

Inflammatory Spondylo-arthritis (Inflammatory LBP)

- Spodylo-arthritis include: ankylosing spondylitis, enteropathic arthritis, psoriatic arthritis and reactive arthritis.

- The European Spondylo-arthropathy Study Group Criteria for Spondylo-arthropathy are shown in Table 5.3.
- Characterized by:
    - Predilection for axial skeletal involvement and inflammation at sites of bony insertions of tendons and ligaments (enthesitis)
    - Negative tests for RF, anti-CCP antibody and ANA.
    - Has a strong association with the HLA-B27.

Figure 5.3. The European Spondyloarthropathy Study Group Criteria for Spondyloarthropathy



N.B. Table 5.2 represent the differences between Inflammatory and mechanical LBP.

Table 5.2. Comparison between the mechanical versus inflammatory back pain.

	Mechanical	Inflammatory
Example	Disc prolapse	Ankylosing spondylitis
Onset	Acute	Insidious
Age	Any age	Usually < 35 years
Effect of exercise	Worsen pain	Improve pain
Morning/inactivity stiffness	+	+++
Pain radiation	Anatomical (L4, L5, S1)	Diffuse
Sensory/motor deficit	+	-
Other system is involved	-	+
Sleep disturbance	+	+++
Scoliosis	+	-
Decrease ROM	Asymmetric	Symmetric
Spinal tenderness	Localized	Diffuse
Sacroiliac/hip involvement	-	±

Ankylosing Spondylitis (AS)

- A chronic systemic inflammatory disease affecting the sacroiliac joints (SIJ), the spine, and, frequently the peripheral joints.
- Sacroiliitis is a hall mark of the disease.

EPIDEMIOLOGY

- AS occurs in:
    - 0.2% of the general population
    - 2% of the B27 +ve population

- 20% of B27 +ve individuals with an affected family member.

- Onset of AS usually begin in late adolescence or early adulthood. Onset after age 45 years is uncommon
- Male:female ratio ranging from 2 to 5:1.

MUSCULOSKELETAL MANIFESTATIONS

Back Pain

- Usually the first symptom of AS.
- Insidious onset of LBP and/or buttock pain that persists for > 3 months
- Pain awakens the patient from sleep, is accompanied by morning stiffness. Pain and stiffness typically improve by exercise.
- Fatigue often accompanies inflammatory back pain.

Enthesitis

- Enthseitis (Inflammation at attachments of tendon or ligament to bone) is a characteristic feature of AS.
- In AS, the initial inflammatory process involves the enthesis, followed by a process that results in new bone formation or fibrosis
- Common sites include:
  - enthesitis at the calcaneal attachments of the Achilles tendon, usually accompanied by Achilles tendon bursitis
  - Plantar fascia: causes disabling heel pain.

Peripheral arthritis

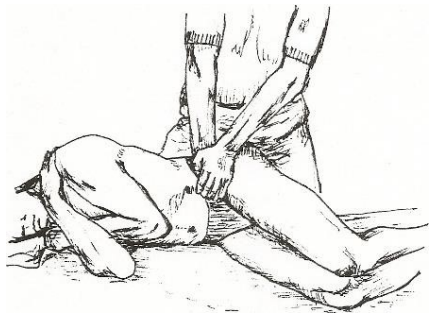
- Occurs in 30% of patients with AS.
- Typically this is asymmetrical oligo-arthritis affecting leg joints, most commonly the knee.
- Hip involvement in AS indicates poor prognosis

PHYSICAL EXAMINATION

Sacroiliac Joint provocative tests

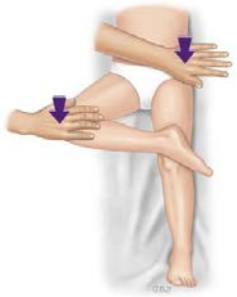
These tests produce pain in patients with sacroiliac joint disease (Figure 5.4).

- **Pelvic compression.** With the patient lying on one side, compression of the pelvis should elicit sacroiliac joint pain.
- **Gaenslen's test.** With the patient supine, a leg is allowed to drop over the side of the examination table while the patient draws the other leg toward the chest. This test elicit SIJ pain on the side of the dropped leg.
- **Patrick's test.** With the patient's heel placed on the opposite knee, downward pressure on the flexed knee with the hip now in flexion, abduction, and external rotation elicit contralateral SIJ tenderness.



Pelvic compression test

Gaenslen's test



Patrick's test

Figure 5.4. Sacroiliac Joint provocative tests

Tests to assess spinal mobility

- **Modified Schober test** –Detects limitation of forward flexion of the lumbar spine: Place a mark at the level of the posterior superior iliac spine (dimples of Venus) and another 10 cm above in the midline. With maximal forward spinal flexion with extended knees, the measured distance should increase from 10 cm to at least 15 cm.
- **Occiput-to-wall test.** Assesses loss of cervical range of motion. Normally with the heels and scapulae touching the wall, the occiput should also touch the wall. Any distance from the occiput to the wall represents a forward stoop of the neck due to cervical spine involvement with AS. The tragus to-wall test could also be used.
- **Chest expansion.** Detects limited chest mobility. Measured at the fourth intercostal space in men and just below the breasts in women, normal chest expansion is approximately 5 cm. Chest expansion less than 2.5 cm is abnormal.

EXTRA-MUSCULOSKELETAL MANIFESTATIONS

Ocular inflammation

- Occurs in up to 40% of AS patients.
- Usually acute anterior uveitis (iritis).

- Typically causes pain, photophobia and, if untreated, impairment in visual acuity.
- Typically, it is unilateral and recurrent.

Cardiopulmonary manifestations

- Aortic insufficiency (3 – 10%)
- Cardiac conduction defects
- Pulmonary fibrosis.

DIAGNOSIS

The diagnosis is based on the modified New York criteria (Table 5.3). Radiographic assessment is a key element of these criteria.

INVESTIGATIONS

x-ray

- SIj - classical changes in the SIjs include erosions in the joint line, pseudo-widening, subchondral sclerosis and finally ankylosis, reflected as obliteration of the SIJ.
- Spine - may reveal squaring and shiny corners of the vertebral bodies and, later, syndesmophytes and facet - joint fusion.

MRI

As radiographic sacroiliitis often develops late. Patients without sacroiliitis on plain radiograph usually have inflammation detected on MRI.

HLA-B27

HLA - B27 is rarely the definitive factor for diagnosis, but when the clinical suspicion is high, the test has high sensitivity and specificity.

Table 5.3. The modified New York criteria for ankylosing spondylitis (1984)

A. Diagnosis	B. Grading
1. Clinical criteria a. Low back pain and stiffness >3months with improvement on exercise, not relieved by rest b. Limitation of spinal motion in both sagittal and frontal planes c. Limitation of chest expansion 2. Radiologic criteria Sacroiliitis: Grade >2 bilaterally or Grade 3–4 unilaterally	1. Definite ankylosing spondylitis if the radiologic criterion is associated with >1 clinical criterion 2. Probable ankylosing spondylitis if: a. the three clinical criteria are present b. the radiologic criterion is present without any signs or symptoms satisfying the clinical criteria
To diagnose AS: one radiologic criterion + at least one clinical criterion is required	

Management

1. Patient education: encourage exercise, stop smoking.

2. Physiotherapy.

3. NSAIDs: first line for pain and stiffness.

4. Glucocorticoid:

- Local injection e.g.: planter fasciitis.
- Patients with axial disease should not receive long-term treatment with systemic steroid.

5. Conventional synthetic DMARDs:

- Should not be used in pure axial disease.
- Sulfasalazine may be considered in patients with peripheral arthritis.

6. Biological DMARDs:

- Should be considered in patients with persistent diseae activity despite conventional treatment.
- Current practice is to start with TNF inhibitor.
- IF TNF inhibitor fails, switch to another TNF inhibitor or IL-17 inhibitor.
- Considered tapering of biological DMARDs in patients with sustained remission.

7. Surgical treatment:

- For patients with disability and radiological evidence of damage.
  - Total hip arthroplasty, spinal corrective osteotomy in specialized centers.

6 OSTEOPOROSIS

OP is a systemic skeletal disease characterized by low bone mass and micro - architectural deterioration of bone tissue that results in a high risk of fracture.

PATHOPHYSIOLOGY

- The human skeleton is composed of 20% trabecular bone and 80% cortical bone.
- Bone undergoes a continual process of resorption and formation (10% of the adult skeleton is remodeled per year).
- Irreversible bone loss results from imbalance between the rates of resorption and formation.
- Trabecular bone is the more metabolically active type, and osteoporotic fractures are more common at sites that contain >50% trabecular bone.
- Trabecular thinning and perforation occurs particularly in situations of increased bone turnover, e.g. after the menopause.
- Post-menopausal estrogen deficiency leads to accelerated bone loss (predominantly loss of trabecular bone).
- This typically results in fractures of vertebral bodies, neck of the femur and distal forearm.

CLASSIFICATION OF OSTEOPOROSIS

- Primary OP - postmenopausal and age-related bone loss
- Secondary OP
    - Due to underlying disease or drug use (Table 6.1).
    - Accounts for 40% of osteoporosis in women and 60% of cases in men.

ASSESSMENT OF OSTEOPOROSIS

1. Assessment of Future Fracture Risk

- Presence of risk factors indicate requirement for treatment.
- Clinical risk factors used for the assessment of fracture probability
    - Age
    - Sex

Table 6.1. common causes of secondary OP

Endocrine	Gastrointestinal	Rheumatologic	Malignancy	Drugs	Others
Thyrotoxicosis Primary hyper-parathyroidism Cushing's syndrome Hypogonadism, including anorexia nervosa Diabetes type I	Malabsorption syndrome Inflammatory bowel disease Liver disease	Rheumatoid arthritis Ankylosing spondylitis	Multiple myeloma Cancer - treatment - induced bone loss	Glucocorticoids Anticonvulsants Heparin Insulin PPI Warfarin SSRI Cyclosporin	Prolonged immobility Organ transplantation COPD

- Low body mass index (≤19kg/m^2)
- Previous fragility fracture
- Parental history of hip fracture
- Current glucocorticoid treatment (any dose, 3 months or more)
- Current smoking
- Alcohol intake
- Patients with diseases or taking drugs that cause secondary OP

2. Plain Radiographs

- The assessment of bone mass on plain radiographs is unreliable. Suspected osteopaenia require confirmation by bone densitometry prior to any therapeutic decisions.
- Can be done to detect fracture and to exclude other causes of bone aches (e.g metastasis).

3. Bone Densitometry

- BMD measured by DEXA is the most reliable determinant of risk of fracture.
- Measurements should be targeted to individuals likely to be at increased risk of OP.
- The WHO diagnostic thresholds for bone mineral density is shown in Table 6.2.

Table 6.2. The WHO diagnostic thresholds for bone mineral density

Diagnosis	Bone mineral density T score (SD units)
Normal	≥ −1
Osteopaenia	< −1 but > −2.5
Osteoporosis	≤ −2.5
Severe osteoporosis	≤ −2.5 + one or more fragility fractures

4. Identify the Underlying Causes of Osteoporosis

TREATMENT

The ultimate goal of osteoporosis management is to reduce the future risk of fracture.

I. Lifestyle Modification

To optimizing peak bone mass and reducing bone loss

- Regular and weight-bearing Exercise e.g. walking or aerobics
- Dietary calcium in adequate amounts
- Sun exposure
- Avoid smoking and alcohol consumption

II. Calcium and Vitamin D supplement

Vitamin D (800 units daily) and calcium (1000–1200 mg daily) to maintain normal level with all therapies for osteoporosis.

III. Anti-Resorptive Agents

1. Bisphosphonates

- Due to poor absorption, these agents must be taken on an empty stomach 1 hour before breakfast or in the middle of a 4 - hour fast.
- Alendronate and risedromate: once - weekly preparations tablet.
- Ibandronate: once - monthly tablet.
- Zoledronate: once - yearly infusion (glomerular filtration rate should be ≥ 30)

N.B: teratogenic effect should be avoided during childbearing period.

2. Denosumab

- A new anti-resorptive therapy
- It is a monoclonal antibody directed against RANK ligand (RANK-L). Activation of RANK by RANKL promotes the maturation of pre-osteoclasts into osteoclasts. Denosumab inhibits this maturation of osteoclasts by binding to and inhibiting RANKL and therefore inhibits bone resorption.
- Recommended for treatment of women with postmenopausal osteoporosis**.**
- Dose: 60 mg subcutaneous every 6 months.

3. Hormone or Oestrogen Replacement Therapy (HRT) and Raloxifene

- HRT is suitable to control climacteric symptoms, or in women under 50 who have undergone an early menopause.
- Raloxifene: a synthetic agents has an oestrogen like action on bone and lipids, but without effect on breast and endometrial tissues (less risk of breast cancer).
- Side effects – thromboembolic events.

4. Calcitonin

- Subcutaneous injections or a nasal preparation.
- This agent has analgesic properties that may be useful in the acute management of vertebral fracture.

IV. Formation - Stimulating Agents

- Parathyroid hormone (Teriparatide)
  - Increase bone formation and improve bone mass and structure, particularly in trabecular bone and thus reduce risk of fracture.
  - Expensive agents and their use is limited to patients with severe, progressive osteoporosis despite exposure to antiresorptive therapy.
  - 20 mg/SC for maximum 2 years.

V. Pain Relief

- Analgesics
- Physical measures

VI. Fall Prevention

- Eliminate predisposing factors e.g. postural hypotension or drowsiness due to drugs, visual disturbance.
- Provide patients with appropriate walking aids.
- Eliminate hazards e.g. loose rugs and cables from the path of the patients.

VII. Rehabilitation

- Physiotherapy: exercise.
- Occupational therapy.

VIII. Surgery

- Fixation of fracture
- Spinal decompression

7 APPROACH TO A PATIENT WITH ARTHRITIS

INFLAMMATORY VERSUS NON-INFLAMMATORY

Inflammatory disorders

Characterized by:

- Systemic symptoms (fever, stiffness, weight loss, fatigue).
- Joint stiffness after prolonged rest (morning stiffness), improves with activity, duration of >1 hour.
- Signs of joint inflammation on physical examination (erythema, warmth, swelling, pain).
- Lab evidence of inflammation (elevated ESR, elevated CRP, hypoalbuminemia, normochromic normocytic anemia, thrombocytosis).
- Examples: systemic lupus erythematosus, rheumatoid arthritis, reactive arthritis, infectious (gonococcal arthritis), or crystal induced (gout, pseudogout).

Non-Inflammatory Conditions

Characterized by:

- Absence of systemic symptoms, pain without erythema or warmth, normal lab tests.
- May cause stiffness usually lasting <1 hour
- Joint symptoms increase with use and weight bearing.
- Examples: OA, fibromyalgia, and traumatic conditions.

ARTICULAR VERSUS NON-ARTICULAR

Pain may originate from:

1. Articular structures (synovial membrane, cartilage, intra-articular ligaments, capsule, or juxta-articular bone):
    - Cause deep or diffuse pain that worsens with active and passive movement.
    - Physical examination may show:
        - Deformity
        - Warmth
        - Swelling (bony swelling or soft tissue swelling)
        - Effusion
        - Crepitus.
    - Synovitis (inflammation of the synovial membrane that covers the joint):
        - The joint looks is a boggy, tender and swollen.
        - The joint loses its sharp edges on examination.
2. Periarticular structures (bursae, tendons, muscle, bone, nerve, skin).
3. Non-articular structures (i.e., cardiac pain referred to the shoulder).

ARTHRALGIA VERSUS ARTHRITIS

Arthralgia

- Refers to joint pain without abnormalities on joint examination.

Arthritis

- Indicates the presence of abnormality in the joint (warmth, swelling, erythema, tenderness).

Approach to Monoarthritis

Acute pain or swelling of a single joint (acute monoarthritis) is an emergency condition and requires immediate evaluation for septic arthritis that can rapidly destroy the joint if left untreated.

Common causes of mono-arthritis are:

- Infection
- Crystal-induced arthritis
- Trauma.

The history

- Exclude trauma
- Give clues to other diagnoses such as history of tick bite (Lyme disease), sexual risk factors (GC arthritis), Colitis, uveitis, and urethritis (ReA).

Physical examination

Usually distinguishes between articular and non-articular disorders.

Investigations

- Perform arthrocentesis in patients with acute monoarthritis. Send synovial fluid for:
  - Leukocyte count with differential (>2000/mm^3 suggest an inflammatory process).
  - Gram stain and culture.
  - Crystal analysis. A wet mount of the fluid examined under polarizing microscopy may identify crystals, but the presence of crystals does not exclude infection.
  - Culture other potential sources of infection (throat, cervix, rectum, wounds, blood).
- Synovial biopsy and arthroscopy are sometimes used to diagnose chronic mono-arthritis.
- Radiographs are useful in cases of trauma and may show OA or chondrocalcinosis in calcium pyrophosphate deposition disease.
- A patient with synovial fluid that is highly inflammatory requires empiric antibiotic therapy until the evaluation, including cultures, is completed.

Approach to Polyarthritis

- Polyarthritis is one of the most common problems in rheumatology.
- The number and pattern of joint involvement suggest the diagnosis.
- History and examination:
  - Differentiate polyarthritis from non-articular causes of generalized joint pain.
  - Disorders of periarticular structures (tendons, bursae) cause joint pain but usually involve a single joint.
  - Myopathies occasionally cause widespread pain, but muscle weakness is the primary symptom.
  - PMR causes shoulder and pelvic girdle pain with morning stiffness, but there is usually no arthritis on examination; weakness is not a feature of this disease.
- Neuropathies, primary bone diseases (Paget's disease), and fibromyalgia can also cause widespread pain but are distinguished by history and physical examination.

8 DIFFERENTIAL DIAGNOSIS OF COMMON ARTHROPATHIES

Arthropathy	*Characteristic features*	*Coexisting disorders*	
Rheumatoid arthritis	• F:M ratio = 3:1. • Most often starts at age of 40-60 years (any age can be affected). • Symmetrical Polyarthritis often starting at MCPj, PIPj and wrists (usually sparing DIPj). • Joints are swollen, painful, stiff and tender. • Morning stiffness > 1hour.		• X-rays: rheumatoid changes. • RF +ve in 80% of cases. • ESR usually raised (during activity)
Osteoarthritis	• Middle-aged or elderly. • 1ry or 2ry affects weight bearing joints: knee, hip. • Pain and inactivity stiffness.	• Old injury to joints may have been present	• ESR not elevated. • X-rays: characteristic OA changes.
Gout	• Acute pain, swelling, redness in MTPj or ankle (less common in knee). • 90% males. • Onset at night	Possibly • Renal disease (in 2ry gout) • Hypertension • Obesity	• Raised serum uric acid. • Urate crystals in synovial fluid.
Rheumatic arthritis (Rheumatic fever)	• Age: 5-15 years. • Migratory polyarthritis affecting large joints, fleeting in character • Effusions are common. • No residual joint damage	• Sore throat 1-3 weeks prior to the attack • Carditis, fever, S.C. nodules, erythema marginatum, chorea.	• ESR and CRP elevated in all active cases • ASO titer: raised • Blood picture: anemia, leucocytosis
Calcium Pyrophosphate arthropathy (pseudogout)	• Males = females. • Knee commonest site. • Acute pain and swelling.		• Pyrophosphate crystals in joint fluid. • X- rays helpful in diagnosis.
Systemic lupus erythematosus	• 90% females. • Marked variation in symptoms referred to any system. • Patients often more ill than arthritic and often febrile.	• Sometimes Antiphospholipid syndrome.	ANA + ve. ESR raised. Anti ds-DNA + ve. Often anaemic.
Polymyositis	• Proximal muscle pains, weakness and tenderness. • May complain of joints pain with morning stiffness.		• Muscle biopsy: inflammatory muscle infiltration. • EMG: abnormal. • ↑ Serum creatin kinas, and other enzymes.
Dermato-myositis	• Heliotrope rash around eye. • Proximal muscle weakness and tenderness.	• Malignant disease may be present.	• Biopsy. • Muscle enzymes. • EMG.
Progressive systemic sclerosis (Scleroderma)	• Tight fingers, blanched fingers and face. • Raynaud's phenomenon • Often dysphagia. • More common in females.	• Dysphagia • Raynaud's phenomenon • Pulmonary hypertension	• Skin Biopsy
Psoriatic arthropathy	• Patchy polyarthritis with DIP joints often involved. • Sometimes spondylitis with sacro-iliac joint affection.	• Psoriasis sometimes slight. • Nail changes of psoriasis.	RF -ve.

Ankylosing spondylitis	• Mostly young males. Axial arthritis • Stiffness and pain in spine and girdle joints (hips and shoulders). Iritis in 30%		• RF- ve • HLA B27 +ve in 95% • of cases. • Sacroiliac joint involvement evident in x- ray.
Enteropathic arthropathy	• Asymmetrical polyarthritis often • associated with relapse of colitis.	• Ulcerative colitis or • Crohn's disease.	• RF – ve.
Polymyalgia rheumatica	• • Pains and morning stiffness in • girdle muscles (shoulders and hips). • Patient usually >60 years of age.	• Low grade fever. • Fatigue Weight loss • Arteritis with risk of blindness.	• ESR > 50 in 1st hour. • RF –ve.

9 CAUSES OF POLYARTHRITIS

	Cause	Characteristics
1. Inflammatory	Viral arthritis	Very acute, self-limiting
	Rheumatoid arthritis	Symmetrical, small and large joints, upper and lower limbs.
	Spondylo-arthropathies	Asymmetrical, large > small joints, lower > upper limbs, spondylitis, sacroiliitis.
	Lupus	Symmetrical, small > large joints, joint damage uncommon
	Chronic gout	Distal > proximal joints, preceded by acute attack
	Juvenile idiopathic arthritis	Symmetrical, small and large joints, upper and lower limbs
	Chronic sarcoidosis	Symmetrical, small and large joints
	Scleroderma and polymyositis	Rare, small and large joints
	Hypertrophic osteoarthropathy	Rare, large > small joints, clubbing
2. Non-inflammatory	Generalized osteoarthritis	Very common, Symmetrical, small and large joints, Heberden's nodes, only a few joints symptomatic at any one time
	Hemochromatosis	Rare, small and large joints
	Acromagally arthropathy	Rare, mainly large joints, spine

Anti-Rheumatic Drugs

Analgesics

- Drugs used to relieve pain only and have no anti-inflammatory effects.
- e.g. salicylates in small doses, paracetamol, nefopam (acupan), glafinene.

Non-steroidal anti-inflammatory Drugs (NSAIDs)

- Group of drugs have an analgesics and anti-inflammatory effects.
- They are similar to corticosteroids in some characters but have no steroid ring in their chemical structure.
- They are less potent and have less side effects than corticosteroids.

Classification

- There are more than 100 types of NSAIDs in the market.
- The classification is based on the acid component (Figure 14).
- The drugs in each class tend to have similar side effects.
- If a drug in one classification is ineffective, try a different structural compound instead of repeatedly using drugs from same structural group.

Mechanism of action

- Mainly via anti-prostaglandin effect by inhibition of cyclooxygenase enzyme.
- Prostaglandins are important mediators of inflammation and pain but they also have many other physiological effects e.g. protective for the stomach, ↓ gastric HCl secretion, ↑ renal blood flow, bronchodilatation.

Side effects

Most of them are due to inhibition of prostaglandins

- Hypersensitivity reaction.
- GIT: dyspepsia, heart burn, gastritis, peptic ulcer, even perforation and GIT bleeding (may occur even if given by injection or supp).
- Liver: transient elevation of liver enzymes.
- Respiratory: bronchospasm and aggravation of bronchial asthma in susceptible patients.
- Renal: ↓ renal blood flow in renal impaired patients.
- CVS: salt and water retention.
- CNS: headache (especially indomethacin).
- Blood: salicylates inhibit platelet function → bleeding tendency and potentiate the effect of anticoagulant.
- Joints: long term use → enhances degeneration process.

Figure 14. Structural classification of NSAIDs

Carboxylic acids					Enolic acids		Non acidic compounds
Salicylic acids	Acetic acids		Propionic acids	Fenamic acids	Pyrazolones	Oxicams	
	Phenyacteic acids	Carbo- and heterocyclic acids					
Aspirin Difunisal Trisalicylate Salicylate	Diclofenac	Etodolac Indomethacin Sulindac Tolmetic Ketorolac	Flurbiprofen Ketoprofen Oxaprozin Ibuprofen Naproxen Fenoprofen	Mefanamic	Phenyl-butazone	Piroxicam Meloxicam	Nabumetone

COX-2 selective inhibitors	
Celecoxib Rofecoxib Meloxicam Nimesulide Paracoxib	Etodolac Lumiracoxib Valdecoxib Deracoxib Etoricoxib

Contraindications

- Absolute: Hypersensitivity to drug.
- Relative contraindications
  - Peptic ulcer.
  - Bleeding tendency.
  - Bronchial asthma.
  - Hepatic impairment.
  - Renal impairment.
  - Pregnancy and lactation.

Precautions for the use of NSAIDs

- Do not prescribe NSAIDs when they are not necessary: in degenerative joint diseases, (no evidence of inflammation) simple analgesics may do the same function with much less side effects.
- Prescribe one NSAID: combination of two or more NSAIDs increase side effects with no better efficacy.
- Use NSAIDs at the lowest possible effective dose, never exceed therapeutic dose.
- Use NSAIDs for shortest time needed.
- Select proper group for the patient: response to NSAIDs varies from patient to patient (individual variation).
- Beware of high risk patients.
- Maintain close supervision.
- Consider the use of other modalities (e.g. Physical therapy) which are effective in treating local pain and inflammation without causing side effects of NSAIDs and decreases the need for them.

Disease Modifying anti-rheumatic Drugs (DMARDs)

Group of drugs used in systemic rheumatic diseases to minimize disease activity and progression (Table 8).

Synonyms

- Specific anti-rheumatic drugs
- Slow Acting Anti-Rheumatic Drugs (SAARDs): their effect takes 4-8 weeks to appear
- 2nd line anti-inflammatory drugs.

Mechanism of action

One or more of the following

- Inhibition of lysosomal enzymes.
- Inhibition of phagocytosis.
- Inhibition of prostaglandins.
- Inhibitory effect on immune system
- Reduction of immune complex formation.
- Sulphasalazine has antimicrobial activity.
- Immunosuppressive drugs has anti RNA and anti DNA properties.

Biological agents

Table 8. Examples of DMRADs

Drug	Dose	Main side effects	Monitoring
Methotrexate (MTX)	7.5-30 mg/week orally, IM or SC injection. After 20mg is reached, no further MTX is absorbed orally: shift to injection Folic acid 5 mg once /week should be administrated	Oral ulcers, hepatic toxicity, bone-marrow suppression, pneumonitis, teratogenicity	CBC and liver enz at baseline and monthly for 3 months, then every 3 months
Leflunomide	100mg orally/day for 3 days, then 10-20 mg daily	GIT upset, hepatic enz elevation, neutropenia, teratogenicity, hypertension	As MTX
Sulphasalazine	Maximum dose allowed is 2gm/day in two divided doses. Start at 500mg and increase by 500mg each week.	GIT upsets, hepatic enz elevation, reversible azospermia. Neutropenia	As MTX
Antimalarials	Hydroxy-chloroquine: 200 – 400 mg/day. Chloroquine 250mg/day	Retinopathy, Skin rash, Myopathy	Fundus ex. Before use and then every 6 months
Azathioprine	50-100mg /day orally.	Bone marrow suppression, Allergic hepatitis.	As MTX
Cyclo-phosphamide	50-100mg /day orally	Bone marrow suppression, ↑ incidence of infection, teratogenicity, infertility	Regular CBC

(Anticytokine therapy)

Anti-TNF-α

- TNF is a major inflammatory mediator in RA and a potent inducer of IL-1.
- TNF-α and IL-1 are considered to be master cytokines in RA.
- Anti-TNF therapy shows great efficacy in RA patients. However, it is not effective in all patients, nor does it fully control the arthritic process in affected joints of good responders.
- Indications: RA, spondyloarthropathy, polyarticular JIA.
- Precautions: chest x-ray and tuberculin test (to avoid activation of TB), hepatitis b scereening.
- Examples: etanercept, infliximab, adalimumab (Table 9).

Anti-IL-1

- E.g. Anakinra: IL-1 receptor antagonist, given subcutaneously in a dose of 100 mg daily.
- Toxicities include injection-site reactions and pneumonia.

Anti-IL-6

- Indications: systemic JIA.
- E.g. tocilizumab.

B cell depletion

- Indications: SLE, vasculitis.
- E.g. rituximab.

Miscellaneous Anti-rheumatic Drugs

Muscle Relaxants

- Indicated in conditions associated with muscle spasm.

Colchicine

- In gouty arthritis, sarcoidosis, and familial Mediterranean fever.

Nerve tonics

- e.g. drugs containing vit B1, B6 and B12 used in diseases associated with neuralgias e.g. sciatica, brachialgia.

Hypouricaemic drugs

- Decrease serum uric acid level in chronic gout.

Tricyclic antidepressants, gabapentin, pregabalin

- Used in fibromyalgia, neuropathic pain.

Table 9. Examples of Biologic agents

Drug	Dose	Mechanism	Common adverse effects
Etanercept (Enbrel)	Subcutaneously 25 mg twice weekly	TNF-α soluble receptor (TNF-α blocker)	Injection site reaction, upper respiratory infection (URI), development of antibodies to drug
Infliximab (Remicade)	Intravenously at 0, 2, 6 weeks, then every 2 months 3-5 mg/kg	TNF-α blocker	Injection site reaction, hypotension, rash, URIs, reactivation of TB, development of autoantibodies.
Adalimumab (Humira)	Subcutaneously 40 mg every 2 weeks	TNF-α blocker	URIs, injection site pain, headache, rash, sinusitis, autoantibodies
Anakinra	Subcutaneously 100 mg daily	IL-1 receptor antagonist	injection-site reactions and pneumonia

Physical Therapy and Rehabilitation

Rehabilitation

Rehabilitation means the restoration of the maximum possible function of an organ or part of the body.

The rehabilitation program is individualized according to patient needs. This requires proper evaluation.

Rehabilitation program include:

- Rest: during active stages and acute exacerbations. The amount and type of rest vary with joint involvement and severity of the disease.
- Medical treatment: according to the cause of the disease.
- Physical therapy.
- Splints and walking aids.
- Surgical treatment when indicated.

Physical Therapy

It is the use of physical agents in treatment of the musculoskeletal disorders.

Various forms of physical agents play an important role in the treatment of musculoskeletal disorders

Physiotherapy may be prescribed alone or in conjunction with medical and other measures to get therapeutic benefits without the hazardous effect of the anti-inflammatory drugs.

Therapeutic benefits

- Relief of pain and stiffness.
- Relief of muscle spasm
- Restoration of movement.
- Increase muscle strength.
- Prevent deformity.
- Restoration of maximal functional capacity.

Indications

1. Rheumatic conditions
- Osteoarthritis.
- Rheumatoid arthritis.
- Cervical spondylosis and disc prolapse.
- Lumbar spondylosis and disc prolapse.
- Joint pain or stiffness e.g. frozen shoulder syndrome.
- Soft tissue rheumatism e.g. tendinitis, bursitis.
- Sciatica.

2. Neurologic and neurosurgical conditions
- LMNL e.g. Bell's palsy, neuropathy.
- UMNL e.g. hemiplegia, paraplegia, monoplegia.
- Post-operative e.g. laminectomy, repair of nerve lesions.

3. Pediatric conditions
- Erb's palsy
- Torticollis
- Cerebral palsy
- Spina bifida

4. Orthopedic conditions
- Post-plaster stiffness of joints.
- Correction of deformities.

5. Sports injuries
- Sprains, tears etc.

6. Gynecologic conditions
- Short wave diathermy in pelvic tubal adhesions (in infertility).
- Post-partum abdominal muscle weakness.

7. Others
- Cardiac rehabilitation.
- Respiratory rehabilitation.
- Rehabilitation of peripheral vascular diseases.
- Geriatric rehabilitation.

Forms of physical therapy

- Heat therapy.
- Cold therapy.
- Electrotherapy.
- Exercise therapy.
- Massage.
- Others e.g. traction, suspension, laser etc.

Cold Therapy

The external use of cooling for therapeutic purposes.

Forms

- Various forms of ice
- Frozen gel packs

Therapeutic effects

- Sedative effects on sensory nerves → pain relief.
- Reduction of spasticity and spasm.
- Vasoconstriction of blood vessels which reduce swelling and bleeding (used in mechanical trauma).
- Cryotherapy improves inflammation, more effectively in the acute phase than in the chronic phase.

Heat Therapy

Forms of heat therapy

Superficial heat

- For heating of superficial tissues (at a depth of about 0.5 cm beneath the skin) e.g. superficial ligaments, small joints, tendons, muscles.
- e.g. infra-red rays

Deep heat

- For heating superficial and deep structures at a depth may reach 5 cm under the skin e.g. large joints, bulky muscles.
- e.g. short wave diathermy, ultrasonic waves, microwave.

Therapeutic effects

1. **Anti-inflammatory effects**

   Dilatation of arterioles and capillaries → increase blood flow → increase O2 supply, food stuffs, antibiotics, WBCs → removal of waste products → resolution of inflammation and healing.

2. **Analgesic effect**

   Application of heat to peripheral nerves → increase pain threshold in the area supplied by the nerve without affecting the motor function.

3. **Effect on muscles**

   Muscle relaxation, decrease muscle spasm.

4. **Mechanical effect**

   The to-and-fro movements of the ultrasound waves through the tissue particles causing micro-massage which soften adhesions

5. **Biologic effect of US and laser**

   Stimulate growth of tissue → tissue repair and healing (e.g. ulcers and bed sores).

Electrotherapy

Forms

- Faradic current
- Galvanic current
- TENS

Therapeutic uses

- Facilitate muscle contraction when it is hindered by pain, weakness or denervation e.g. isometric contraction of the quadriceps in patients with RA or after knee surgery or knee effusion and lower motor neuron lesions.
- Training of new muscle action after tendon transplantation.
- Analgesic effect: low frequency current for stimulating afferent sensory component of peripheral nerves for relief of pain (acute or chronic) e.g. back pain, neck pain, OA, RA, neuralgia.

Exercise therapy

Passive exercise

- Accomplished only by therapist or apparatus
- Used to maintain body mobility and prevent contracture when muscles are weak.

Active exercise

Active assistive:

- Accomplished by active contraction by the patient with the assistance of the therapist or mechanical devices
- Used as first step in the muscle re-education for weak muscles.

Active exercise

- Accomplished by patient without assistance
- Used to improve function and strength.

Active resisted

- Accomplished by the patient with various additional resistance either manual or mechanical depending on the muscle power.
- Used to develop muscle power

Stretching

- Accomplished by forced motion to restore normal range of motion which is limited due to loss of elasticity of soft tissues.

Massage

Therapeutic effects

- Assists blood and lymph drainage → ↓ swelling.
- Decrease adhesions between muscle fibers.
- Decrease pain sensation.
- Muscle relaxation.

Spinal Traction

A technique that utilizes a traction force of sufficient magnitude and duration applied to the spine to produce separation of the vertebrae, facets and increase size of foramina.

Used mainly for cervical and lumbar spine.

Techniques

- Manual: force applied by therapist hands. Used in cervical traction only (Figure 15).
- Mechanical: administered using pulley and free weight system (Figure 15).
- Motorized: mechanical traction applied by motorized system, administered in continuous or intermittent periods.
- Gravity: hanging upside down.
- Auto-traction: uses specially designed device that self-administers.

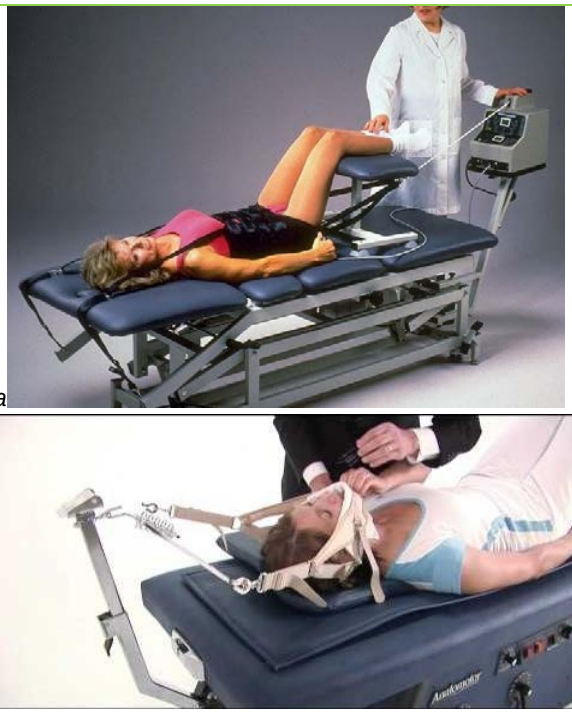


Figure 15: Motorized Traction (a) cervical motorized traction and (b) lumbar motorized traction.

Therapeutic effects

- Prolonged pull on the muscle may lead to paraspinal muscle fatigue which alleviates muscle spasm.
- Enlarge the intervertebral space leading to retraction of herniated disc material.
- Tighten the posterior longitudinal ligament to exert a centripetal force on the annulus fibrosis.
- Widen the intervertebral foramina → relief root compression
- Separate apophyseal joints → relief of pain following degenerative joint space narrowing.

Splints

Aim and types

- Rest splints
  Rest and relief from pain for active joints
- Corrective splint.
  Prevention and correction of deformities.
- Functional splint
  Fixation of damaged joint in good functional position.

Walking aids

- Crutches (Figure 16).
- Canes (Figure 16).
- Walker (Figure 16).
- Wheel chair.

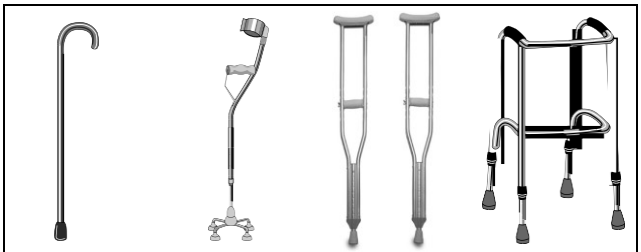
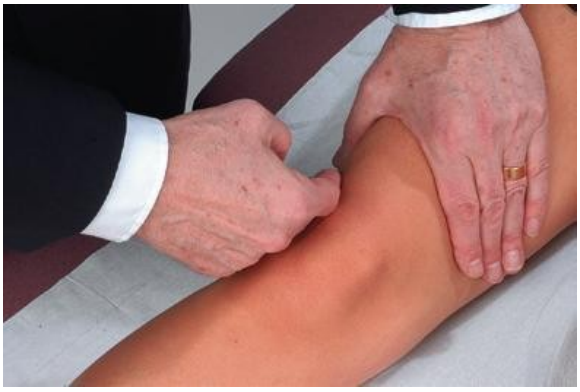


Figure 16: walking aids.

Clinical signs

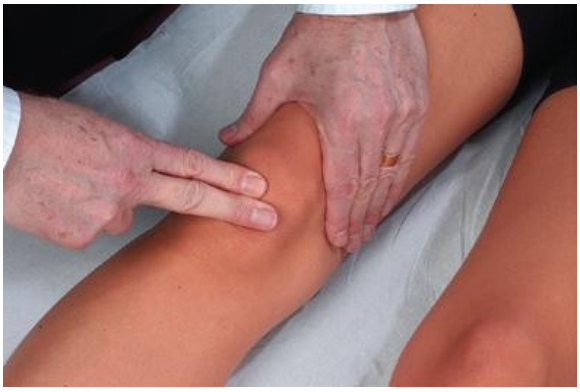
Testing for swelling.

The bulge sign in the knee: The back of the hand gently pushes the fluid from one side of the knee to the other, filling out the "dimples" on either side of the patella. This is most helpful in detecting small knee effusions.

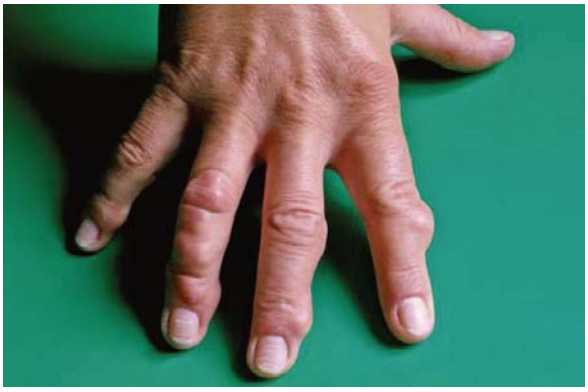


Testing for swelling.

The patellar tap. One hand is used to cup the patella and compress the suprapatellar pouch, and the fingers of the other hand press down on the patella to feel for cross-fluctuation.

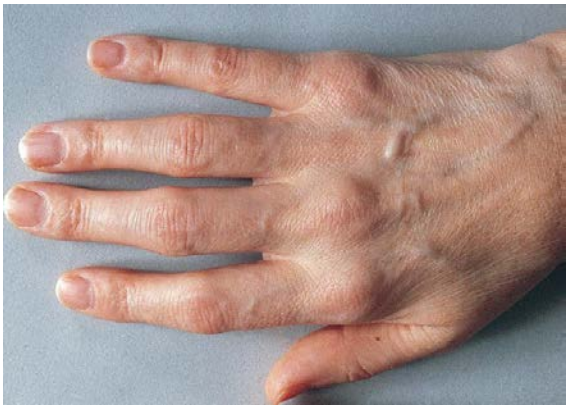


Rheumatoid nodules in a patient with rheumatoid arthritis

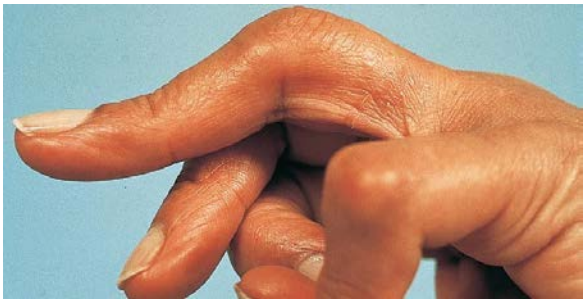


The hand in early RA.

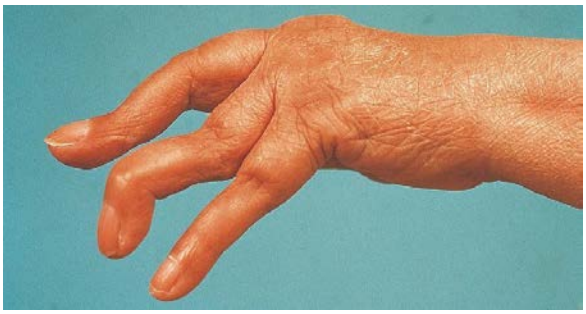
Showing swelling of the MCP and PIP joints.



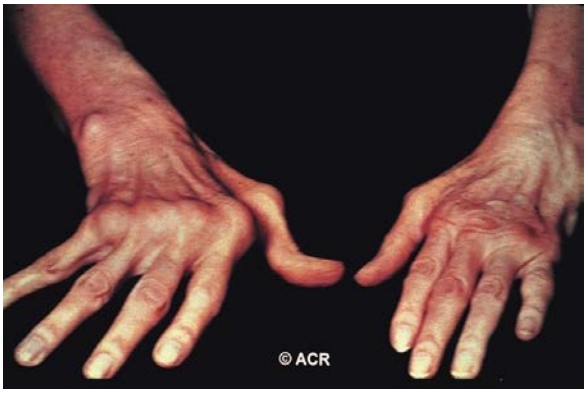
Boutonniere deformity.
PIP flexion and DIP hyperextension.



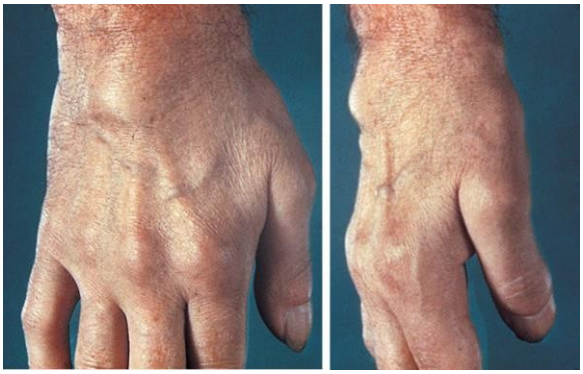
The swan-neck deformity.
PIP hyperextension, and DIP flexion.



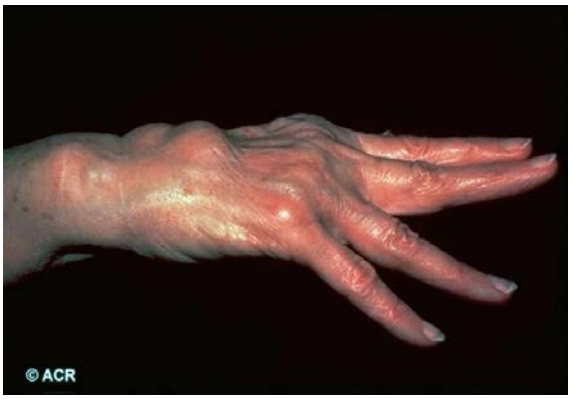
Ulnar deviation of the fingers at level of MCPjs In right hand.



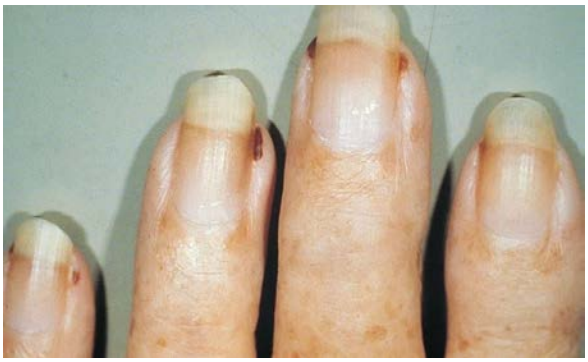
Tenosynovial swelling overlies the metacarpals of the hand. Bulging becomes accentuated with full extension of all the fingers of the hand.



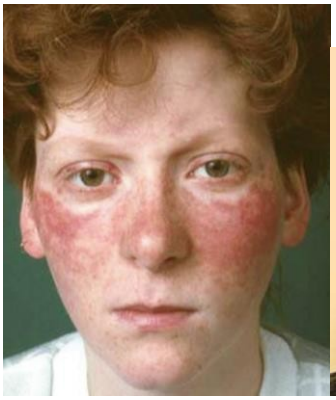
Subluxation of the wrist in severe disease, associated with extensor tenosynovitis and extensor tendon rupture.

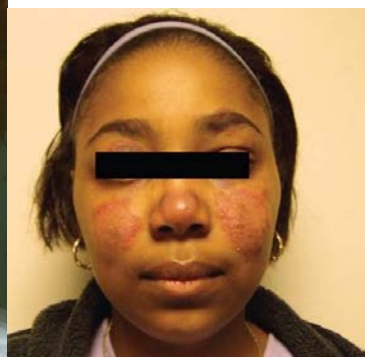


Nailfold infarcts due to vasculitis in a patient with rheumatoid arthritis

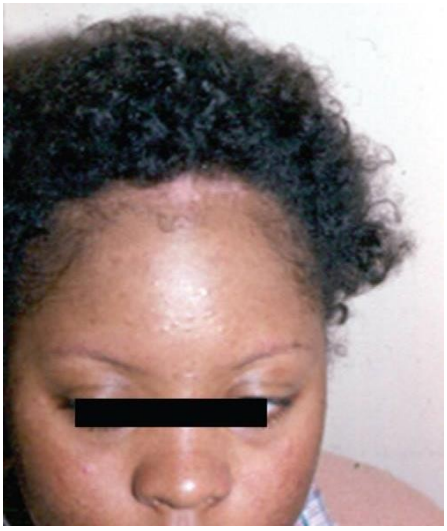


Malar (butterfly) rash in young woman with systemic lupus erythematosus 0ver bridge of nose and cheeks, sparing nasolabial folds.

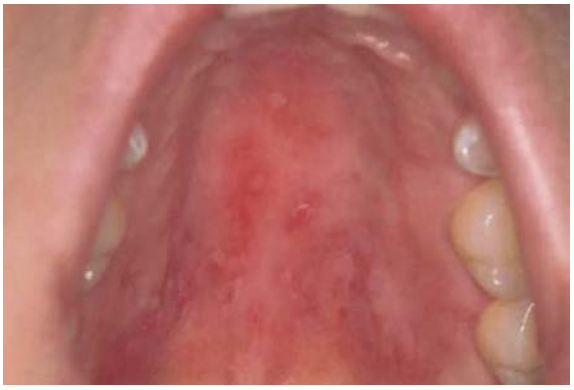




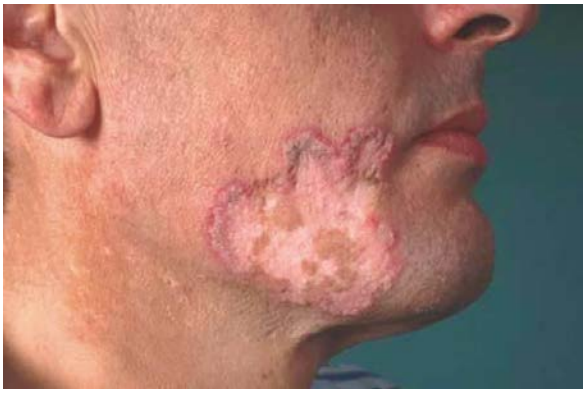
Alopecia affecting the frontal scalp with lupus hairs (receding hair line).



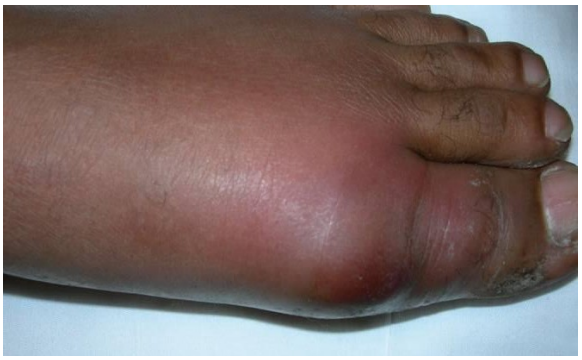
Oral aphthoid lesions and ulcerations of the palatal mucosa in a patient with systemic lupus erythematosus.



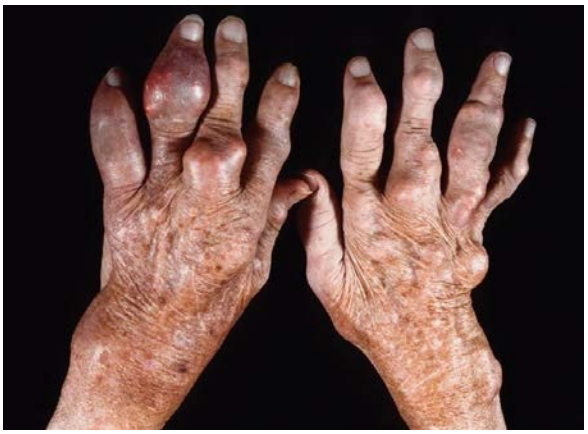
Classic discoid lupus erythematosus of the face. Note central scarring and erythematous hyperkeratotic borders.



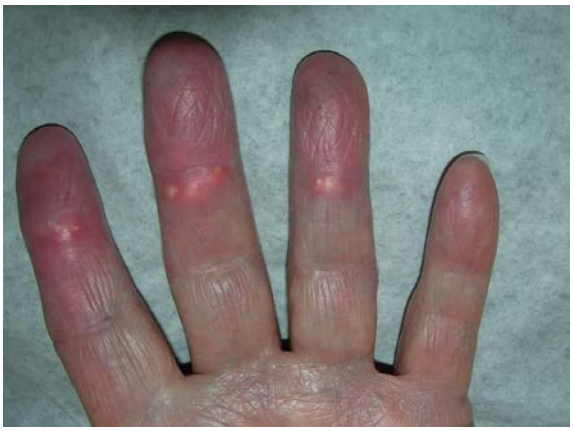
Podagra or acute gout of the first sMTP joint is shown. The hyperintense erythema with a dusky hue is characteristic. The area of inflammation usually extends beyond the area of the involved joint



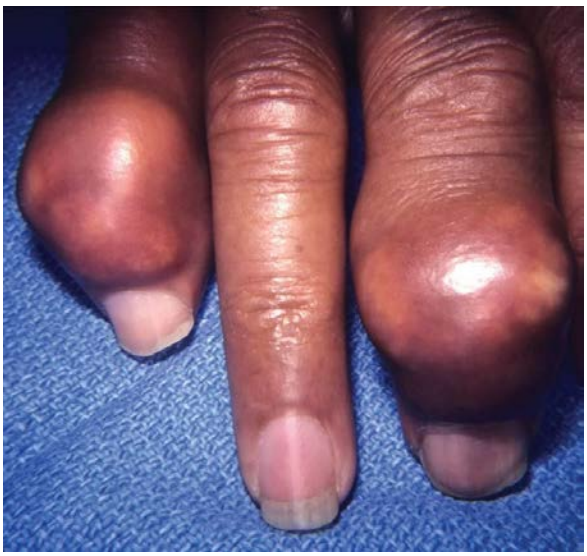
Advanced gout of the hands and wrists demonstrates an asymmetric arthritis with articular and interarticular tophi. The large tophus involving the distal left fourth digit shows very superficial crystalline deposits



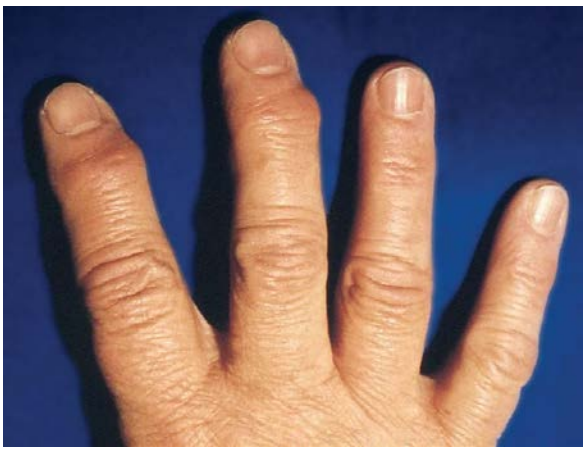
Subcutaneous tophi in the palmar creases of the distal inter-phalangeal joints are an uncommon finding but an easy source of crystals for the diagnostic confirmation of gout.



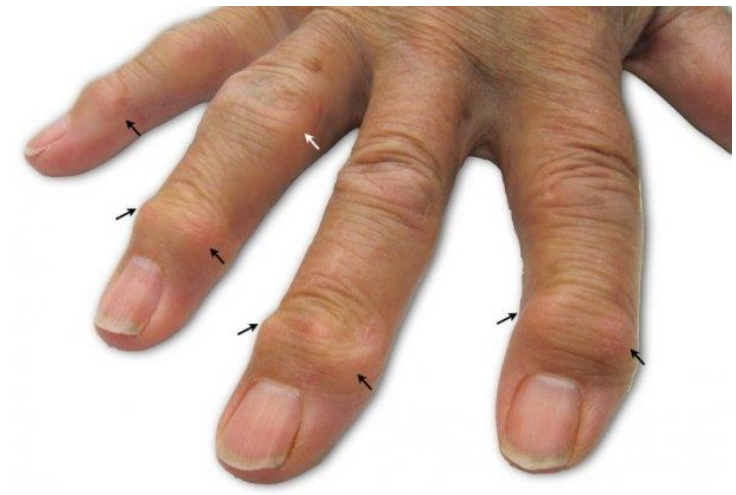
Large tophi involving the distal interphalangeal joints are commonly seen in gouty patients. This is particularly characteristic of late-onset gout.



Osteoarthritis is the most common disorder affecting this segment (Heberden's nodes).



Heberden's node (black arrow) and Bouchard's node (white arrow) in hand osteoarthritis



Hand orthoses may decrease pain and correct deformities. From top to bottom: a resting splint that restricts motion and maintains a functional position, a functional wrist splint that supports the wrist during hand activities, and a silver ring splint that corrects and/or prevents deformities.

Recommended text book :

ABC of Rheumatology, 5th edition April 2018

MCQ

Which of the following is most specific for SLE?
a) Anti-Sm
b) anti Jo 1
c) ANA
d) Anti-La
e) Anticentromere

Q2: A 25 year old woman in her first pregnancy is concerned about her sister's history of a child that died in the neonatal period with complete heart block. Best choice of investigations for this woman?
a) ANA
b) Anti La (SSB) antibodies
c) Anti-phospholipid antibodies
d) Anti-cardiolipin antibodies
e) Anti DNA

Which of the following is <u>not</u> included in the American College of Rheumatology (ACR) diagnostic criteria for SLE?
a) Thrombocytopenia
b) Elevated ANA antibody titre
c) Psychosis
d) Alopecia
e) Photosensitivity

A disproportionate rise in CRP compared to the ESR is typically found in which of the following clinical situations?
a) RA
b) Sepsis in a patient with SLE
c) Gout
d) Cerebral lupus
e) Felty's syndrome

A patient has mild SLE with butterfly rash & Arthralgia. ESR ↑, ANA +ve, renal function normal, platelets mildly ↓. What is the best treatment?
a) Prednisone
b) Hydroxychloroquine
c) NSAID
d) Cyclophosphamide
e) Observe

Which of the following has the least prognostic value in early RA?
a) C-reactive protein
b) Extra-articular affection
c) Radiographic evidence of erosions
d) Decreased peripheral lymphocyte count

Which has the most specificity for the disease matched?
a) Anti-Ro (SS-A) – Sjögren's
b) ANA – SLE
c) Anti-Sm – SLE
d) Rheumatoid factor – Rheumatoid arthritis

Which of the following is the most sensitive to differentiate RA from SLE?

a) Rheumatoid factor
b) Keratoconjunctivis sicca
c) Bilateral knee effusions
d) Nodules over the MCP joint
e) Erosion of the ulnar styloid

Female patient with rheumatoid arthritis and occipital headaches. Next Investigation should be
a) CT of neck
b) Lateral flexion X-ray of cervical spine
c) ESR
d) Myelogram
e) Anti-CCP

Uric acid excretion is
a) increased by low dose aspirin
b) decreased in leukemia
c) Increased in hypertension
d) increased by alcohol consumption
e) largely unaffected by Indomethacin

A 26-year-old woman attended the early arthritis clinic with a 3-month history of an inflammatory polyarthritis affecting her hands and feet. Investigations: haemoglobin 125 g/L (115–165), white cell count 7.3×10^9/L (4.0–11.0), platelet count 350×10^9/L (150–400), ESR 40 mm/1sth (<20), X-rays of hands and wrists periarticular osteopenia. What investigation is most likely to distinguish between persistent and self-limiting arthritis?
a) Anti-citrullinated peptide antigen antibodies
b) Antinuclear antibodies
c) IgA rheumatoid factor
d) IgG rheumatoid factor
e) IgM rheumatoid factor

Which of the following is not a feature in rheumatoid hand
a) Wasting of small muscles of the hand
b) Tenosynovitis
c) Heberden's nodules.
d) Swan neck deformity
e) Z shaped thumb

Which of the following articular regions are unlikely to be involved in rheumatoid arthritis
a) Distal interphalangeal joints
b) Proximal interphalangeal joints
c) Metaocarpophalangeal joints
d) Knee joints
e) Wrist joints

Which of the following radiologic appearance is not associated with in rheumatoid arthritis
a) Marginal erosions.
b) Juxtaarticular osteoporosis.
c) Increased joint space
d) Subluxation
e) Soft tissue swelling

Regarding systemic lupus erythematosus, which of the following is true?
a) It is commoner in males.
b) Phtotsensitivity may occur
c) Complement level C3 and C4 are increased
d) Erosion is common in plain x-ray
e) Antinuclear antibodies are usually negative

The following is not a clinical feature of SLE
a) Depression
b) Alopecia
c) Pleural effusions
d) Extraarticular nodules.
e) Arthralgia

Hyperuricaemia may result from low dose of
a) Methotrexate
b) Aspirin
c) Corticosteroids
d) Anti-TNF α agents
e) Indomethacin

The following drug is used in acute gout
a) Aspirin
b) Probenecid
c) Allopurinol
d) Colchicine
e) Methotrexate

Gout
a) Is associated with calcium pyrophosphate crystals deposited in the cartilage.
b) May cause subcutaneous nodules.
c) Commonly affect 1st metacarpophalangeal joint.
d) Typically has symmetric polyarthritis pattern
e) Associated with HLA-DR

Regarding rheumatoid factor, which of the following is not true?
a) May be present in the absence of rheumatoid arthritis.
b) High titer early in rheumatoid arthritis indicate bad prognosis.
c) Usually present in rheumatoid patients with subcutaneous nodules
d) Absent in normal population
e) One of the criteria of diagnosis of rheumatoid arthritis.

Which of the following is most specific for rheumatoid arthritis
a) Rheumatoid factor
b) Anti CCP
c) Anti ds DNA
d) Anti Sm
e) Anti Ro

The following is associated with poor prognosis in rheumatoid arthritis except
a) Acute onset
b) Bone erosion on x-ray
c) Subcutaneous nodules
d) Low ESR
e) Extraarticular manifestations

Chloroquine
a) Used in treatment of osteoarthriris
b) May cause retinopathy
c) May cause hyperuricaemia
d) Contraindicated in rheumatoid arthritis
e) 2nd line treatment of osteoarthritis

The following is not a feature of Felty's syndrome
a) Associated with rheumatoid arthritis
b) Splenomegaly
c) Dry eye and mouth
d) Neutropenia
e) Rheumatoid factor is usually positive

Which of the following is not a feature of osteoarthritis
a) Heberden's nodes
b) Osteophyte formation
c) Bouchard's nodes
d) Raised ESR.
e) Morning stiffness < 1 hour.

Regarding rheumatoid arthritis, which of the following is not true?
a) Commoner in females
b) Associated with HLA-DR

c) Insidious onset indicate bad prognosis
d) Corticosteroids in high doses is a mainstay of the treatment
e) Pregnancy usually associated with disease remission

Which of the following is not a side effect of NSAIDs
a) Long term use may enhance joint degeneration process.
b) Headache
c) Peptic ulcer
d) Retinal damage
e) Transient elevation of liver enzymes

The following may exacerbate systemic lupus erythematosus
a) Low dose aspirin
b) Exposure to the sun
c) High purine diet
d) Prednisolone
e) Alcohol

Regarding sjogren's syndrome:
a) Associated with systemic lupus erythematosus.
b) Associated with dry eye and mouth
c) Associated with Anti-Sm in most patients
d) Associated with high serum uric acid
e) Osteophyte is a common finding in x-ray

Sulphasalazine
a) Is ineffective in RA.
b) Is effective in osteoarthritis
c) May cause infertility in male
d) May cause neutrophilia
e) Is the first drug of choice in chronic gout

Regarding osteoarthritis
a) Knee joint affection is rare
b) Tophi are common findings
c) There is articular cartilage destruction
d) Obesity is a risk factor
e) Methotrexate is effective

Therapeutic heat
a) Produce capillary and arteriolar vasoconstriction
b) Decrease pain threshold
c) Increase muscle spasm
d) Used in muscle re education
e) Has analgesic effect
f) Stretch adhesions in among muscle fibers

Regarding therapeutic cold, which of the following is not true?
a) Has sedative effect on sensory nerves
b) increase spasticity
c) Reduce swelling
d) Reduce bleeding
e) Used in mechanical trauma

Forced motion used to restore normal range of motion which is limited due to loss of elasticity of soft tissues
a) Passive exercise
b) Active assistive exercise
c) Active resisted exercise
d) Stretch exercise
e) Traction

Patient with osteoarthritis is advised to
a) Sit too long in one position.
b) Use low chairs.
c) Walk for long periods.
d) Over exercise the affected joints.
e) Reduce his bodyweight.

As regards physical therapy, which of the following is not true?
a) It is the use of physical agents in treatment of the musculoskeletal disorders.
b) Heat and cold are forms of physical therapy agents
c) Has no rule in female infertility
d) Good alternative that reduce hazards of NSAIDs
e) Have a role in sports injuries

Splints are not used to
a) Relief pain for active joints
b) Prevent deformities
c) Correct deformities.
d) Place of damaged joint in good functional position.
e) Produce separation of the vertebrae

Red flags for low back pain include the following except
a) Bilateral radiation of pain
b) Bowel or bladder dysfunction
c) Saddle anesthesia
d) Positive stretch test
e) Pain and stiffness > 30 minutes,