Bone & Joint Disorders

Rheumatoid Arthritis (RA)

Definition

- Rheumatoid arthritis is a chronic autoimmune inflammatory disease affecting synovial joints.
- It causes symmetrical joint inflammation, pain, swelling, and progressive joint destruction.
- It may also affect extra-articular organs such as the heart, lungs, and blood vessels.

Etiology

1. **Genetic Factors**
   - Family history increases susceptibility.
   - Certain HLA genes are associated with the disease.
2. **Autoimmune Factors**
   - The immune system mistakenly attacks healthy joint tissues.
3. **Environmental Factors**
   - Smoking.
   - Infections.
   - Hormonal influences.

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Etiopathogenesis

Step 1: Autoimmune Activation

- The immune system becomes abnormally activated.
- Autoantibodies are produced.

Step 2: Synovial Inflammation

- Inflammatory cells infiltrate the synovial membrane.
- Synovitis develops.

Step 3: Pannus Formation

- Abnormal granulation tissue forms over cartilage.

Step 4: Cartilage Destruction

- Enzymes damage cartilage and bone.

Step 5: Joint Deformity

- Progressive joint destruction leads to deformities.

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Clinical Manifestations

- Joint pain and tenderness.
- Morning stiffness lasting more than one hour.
- Swelling of small joints.
- Fatigue.
- Reduced joint movement.
- Joint deformities in advanced disease.

Pharmacotherapy of Rheumatoid Arthritis (RA)

Drug Class	Examples	Actions
NSAIDs	Ibuprofen, Diclofenac, Naproxen	Reduce pain and inflammation; do not prevent disease progression.
Corticosteroids	Prednisolone, Methylprednisolone	Provide rapid anti-inflammatory effects.
Disease-Modifying Anti-Rheumatic Drugs (DMARDs)	Methotrexate, Sulfasalazine, Hydroxychloroquine, Leflunomide	Slow disease progression and prevent joint damage.
Biological DMARDs	Infliximab, Adalimumab	Target inflammatory cytokines; used in moderate to severe disease.
TNF-α Inhibitors	Etanercept	Target TNF-α and reduce inflammation.

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Treatment Goals

- Control inflammation.
- Prevent joint destruction.
- Maintain joint function.
- Improve quality of life.

Osteoarthritis (OA)

- Osteoarthritis is a degenerative joint disorder characterized by progressive breakdown of articular cartilage and structural changes in the underlying bone.
- It is the most common form of arthritis, especially among elderly individuals.
- Osteoarthritis is a chronic degenerative disease of synovial joints characterized by cartilage deterioration and bone remodeling.

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Etiology

A. Primary Osteoarthritis

- Age-related degeneration.
- No specific underlying cause.

B. Secondary Osteoarthritis

- Joint injury
- Obesity
- Congenital abnormalities
- Metabolic disorders

Etiopathogenesis

Step 1: Cartilage Degeneration

- Articular cartilage gradually wears away.

Step 2: Reduced Joint Cushioning

- Friction between bones increases.

Step 3: Bone Changes

- Osteophytes develop.
- Subchondral bone thickening occurs.

Step 4: Joint Inflammation

- Mild inflammation develops.

Step 5: Joint Dysfunction

- Pain and movement restriction occur.

Clinical Manifestations

- Joint pain worsened by activity.
- Morning stiffness lasting less than 30 minutes.
- Crepitus during movement.
- Reduced range of motion.
- Joint enlargement.

Pharmacotherapy of Osteoarthritis (OA)

Drug Class / Therapy	Examples	Actions
Paracetamol	Paracetamol	Used for mild pain relief.

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NSAIDs	Diclofenac, Ibuprofen, Celecoxib	Reduce pain and inflammation.
Topical Agents	Diclofenac gel, Capsaicin cream	Provide local pain relief.
Intra-Articular Corticosteroids	Triamcinolone, Methylprednisolone	Provide temporary symptom relief.

Non-Pharmacological Management

- Weight reduction.
- Physical exercise.
- Physiotherapy.
- Joint protection measures.

Gout

- Gout is a metabolic disorder characterized by deposition of monosodium urate crystals in joints and tissues due to elevated serum uric acid levels.
- Gout is an inflammatory arthritis caused by hyperuricemia and deposition of urate crystals.

Etiology

1. Increased Uric Acid Production

- High purine diet.
- Certain malignancies.

2. Decreased Uric Acid Excretion

- Renal impairment.
- Certain medications.

3. Genetic Factors

- Inherited abnormalities of uric acid metabolism.

Etiopathogenesis

Step 1: Hyperuricemia

- Blood uric acid levels increase.

Step 2: Crystal Formation

- Monosodium urate crystals form.

Step 3: Crystal Deposition

- Crystals accumulate in joints.

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Step 4: Inflammatory Response

- White blood cells react to crystals.

Step 5: Acute Gout Attack

- Severe inflammation develops.

Clinical Manifestations

- Sudden severe joint pain.
- Redness and swelling.
- Warmth over affected joint.
- Commonly affects the great toe.
- Recurrent attacks may occur.

Pharmacotherapy of Gout

Treatment Phase	Drug Class	Examples	Actions
Acute Attack	**NSAIDs**	Indomethacin, Naproxen	Reduce pain and inflammation during acute gout attacks.
Acute Attack	**Colchicine**	Colchicine	Reduces inflammatory response to urate crystals.
Acute Attack	**Corticosteroids**	Prednisolone	Used when NSAIDs are contraindicated.
Long-Term Management	**Xanthine Oxidase Inhibitors**	Allopurinol, Febuxostat	Reduce uric acid production.
Long-Term Management	**Uricosuric Agents**	Probenecid	Increase uric acid excretion.

Osteoporosis

- Osteoporosis is a skeletal disorder characterized by reduced bone mass and deterioration of bone microarchitecture, leading to increased fracture risk.
- Osteoporosis is a condition in which bones become weak, porous, and prone to fractures.

Etiology

1. **Aging**
   - Bone resorption exceeds bone formation.
2. **Postmenopausal Estrogen Deficiency**
   - Major cause in women.
3. **Nutritional Deficiencies**
   - Calcium deficiency.

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