Chapter-16

Disease of Bones and Joint

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Abstract

Diseases of bones and joints significantly impact mobility and quality of life, with rheumatoid arthritis, osteoporosis, and gout being prevalent conditions. Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by inflammation of the synovial joints, leading to progressive joint damage. The pathophysiology involves the immune system attacking the synovium, causing inflammation and joint destruction. Epidemiologically, RA affects about 1% of the global population, with a higher prevalence in women. Symptoms include joint pain, swelling, stiffness, and fatigue. Diagnosis is based on clinical evaluation, blood tests for rheumatoid factor and anti-CCP antibodies, and imaging studies. Treatment includes anti-inflammatory drugs, disease-modifying antirheumatic drugs (DMARDs), and biologics. Complications can include joint deformity, cardiovascular disease, and increased risk of infections. Prevention strategies focus on early diagnosis and treatment to prevent joint damage. Osteoporosis is a metabolic bone disease characterized by reduced bone density and increased fracture risk. The pathophysiology involves an imbalance between bone resorption and formation, leading to weakened bones. Epidemiologically, osteoporosis is most common in postmenopausal women and the elderly. Symptoms are often absent until a fracture occurs, commonly in the hip, spine, or wrist. Diagnosis is made through bone mineral density testing using DEXA scans. Treatment includes calcium and vitamin D supplementation, bisphosphonates, and lifestyle modifications like weight-bearing exercises. Complications include fractures, chronic pain, and decreased mobility. Prevention focuses on maintaining a healthy diet, regular exercise, and avoiding smoking and excessive alcohol consumption. Gout is a type of inflammatory arthritis caused by the deposition of monosodium urate crystals in the joints due to hyperuricemia. The pathophysiology involves the crystallization of uric acid in the joints, leading to intense inflammation. Epidemiologically, gout affects about 1-2% of the population, more commonly in men and older adults. Symptoms include sudden, severe joint pain, redness, and swelling, often in the big toe. Diagnosis is confirmed by identifying urate crystals in joint fluid and elevated serum uric acid levels. Treatment includes nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, and urate-lowering therapies like allopurinol. Complications can include recurrent gout attacks, chronic gouty arthritis, and kidney stones. Prevention involves managing risk factors such as obesity, hypertension, and diet high in purines, and ensuring proper hydration.

I. INTRODUCTION

Diseases of Bones and Joints

1. **Osteoporosis**
   a. **Definition:** A condition characterized by weakened bones that are more susceptible to fractures. It occurs when the body loses too much bone mass or makes too little bone.
   b. **Risk Factors:** Age, gender (more common in women), family history, low body weight, smoking, excessive alcohol consumption, and certain medications.
   c. **Symptoms:** Often asymptomatic until a fracture occurs. Common fractures occur in the spine, hip, and wrist.
   d. **Diagnosis:** Bone density tests (DEXA scans), X-rays.
   e. **Treatment:** Medications like bisphosphonates, hormone replacement therapy, calcium and vitamin D supplements, and lifestyle changes including exercise and diet.

2. **Rheumatoid Arthritis (RA)**
   a. **Definition:** An autoimmune disorder that primarily affects the joints, causing inflammation, pain, and eventual joint damage.
   b. **Risk Factors:** Gender (more common in women), family history, age, and smoking.
   c. **Symptoms:** Joint pain, swelling, stiffness, and reduced joint mobility. Often affects joints symmetrically.
   d. **Diagnosis:** Clinical examination, blood tests (rheumatoid factor, anti-CCP antibodies), and imaging studies.
   e. **Treatment:** Disease-modifying antirheumatic drugs (DMARDs), biologics, NSAIDs, and corticosteroids.

3. **Osteoarthritis (OA)**
   a. **Definition:** The most common form of arthritis, characterized by the degeneration of cartilage in the joints.
   b. **Risk Factors:** Age, joint injury, obesity, genetics, and repetitive stress on joints.
   c. **Symptoms:** Joint pain, stiffness, and swelling. Typically affects weight-bearing joints like the knees, hips, and spine.
   d. **Diagnosis:** Clinical examination, X-rays, MRI.
   e. **Treatment:** Pain management with NSAIDs, physical therapy, weight management, and joint replacement surgery in severe cases.

4. **Gout**
   a. **Definition:** A form of inflammatory arthritis caused by the accumulation of uric acid crystals in the joints.
   b. **Risk Factors:** High purine diet, obesity, genetic predisposition, and certain medications.
   c. **Symptoms:** Sudden and severe pain, redness, and swelling in the affected joint, often the big toe.
   d. **Diagnosis:** Joint fluid analysis, blood tests to measure uric acid levels, and imaging.
   e. **Treatment:** Medications to lower uric acid levels, pain relief, dietary changes, and lifestyle modifications.

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II. DISEASE OF BONES AND JOINTS

Rheumatoid Arthritis

Introduction

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder that primarily affects the joints. It is characterized by inflammation of the synovial membrane, leading to joint damage and deformities. RA can also affect other organs and systems in the body, making it a complex and potentially debilitating condition.

Pathophysiology

1. **Immune System Dysfunction:** RA involves the immune system attacking the synovial membrane (the lining of the joints). This immune response leads to inflammation and damage.
2. **Inflammatory Cascade:** The inflammation results in the release of cytokines (e.g., tumor necrosis factor-alpha, interleukin-1, and interleukin-6) and other inflammatory mediators that promote further joint damage.
3. **Synovial Hyperplasia:** The synovium thickens and becomes inflamed, forming a pannus (an abnormal tissue growth) that erodes cartilage and bone.
4. **Joint Damage:** Over time, the destruction of cartilage and bone leads to joint deformities, pain, and loss of function.

Epidemiology

1. **Prevalence:** RA affects approximately 1% of the global population.
2. **Gender:** Women are more commonly affected than men, with a ratio of about 2-3:1.
3. **Age:** RA can occur at any age but is most commonly diagnosed between the ages of 30 and 60.
4. **Genetics:** There is a genetic predisposition to RA, with certain genetic markers like the HLA-DR4 allele being associated with increased risk.
5. **Environmental Factors:** Smoking is a known environmental risk factor that can trigger RA in genetically predisposed individuals.

Symptoms and Complications

1. **Symptoms**
   a. **Joint Pain and Swelling**: Commonly affects the small joints of the hands and feet.
   b. **Morning Stiffness**: Lasts for more than an hour and is often worse in the morning.
   c. **Fatigue and General Malaise**: Systemic symptoms like fatigue and a feeling of general unwellness.
   d. **Joint Deformities**: Over time, affected joints may become deformed and lose function.
   e. **Systemic Manifestations**: RA can also cause symptoms in other organs, including the lungs, heart, and eyes.

2. **Complications**
   a. **Joint Deformities:** Progressive damage can lead to joint deformities such as ulnar deviation and swan-neck deformities.
   b. **Bone Loss:** Increased risk of osteoporosis and bone fractures due to chronic inflammation.

c. **Cardiovascular Disease:** Higher risk of developing heart disease due to inflammation.
d. **Lung Disease:** RA can lead to interstitial lung disease and pleuritis.
e. **Infections:** Immunosuppressive treatments increase susceptibility to infections.

Diagnosis

1. **Clinical Evaluation:** Based on symptoms, medical history, and physical examination.
2. **Laboratory Tests**
   a. **Rheumatoid Factor (RF):** An antibody present in many RA patients but not exclusively.
   b. **Anti-Cyclic Citrullinated Peptide (Anti-CCP):** More specific for RA and helps in early diagnosis.
   c. **Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP):** Indicators of inflammation.
3. **Imaging Studies**
   a. **X-rays:** Can reveal joint damage, erosion, and deformities.
   b. **Ultrasound:** Useful for detecting early inflammatory changes and synovitis.
   c. **MRI:** Provides detailed images of joint structures and can detect early inflammatory changes.

Treatment

1. **Medications**
   a. **Disease-Modifying Antirheumatic Drugs (DMARDs):** Methotrexate, sulfasalazine, and leflunomide to slow disease progression.
   b. **Biologic DMARDs:** TNF inhibitors (e.g., etanercept, infliximab), IL-6 inhibitors (e.g., tocilizumab), and other targeted therapies.
   c. **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):** For pain relief and inflammation reduction.
   d. **Corticosteroids**: Short-term use to manage severe inflammation.
2. **Physical Therapy:** Helps maintain joint function and mobility.
3. **Surgical Interventions:** Joint replacement or repair may be necessary in advanced cases.

Complications

1. **Joint Deformities:** Progressive damage can lead to significant joint deformities affecting function.
2. **Osteoporosis:** Increased risk due to chronic inflammation and steroid use.
3. **Cardiovascular Problems:** Higher risk of heart disease and related complications.
4. **Infections:** Increased risk due to immunosuppressive treatments.

Prevention

1. **Early Diagnosis and Treatment:** Prompt treatment can help prevent or minimize joint damage.
2. **Lifestyle Modifications**:
   a. **Smoking Cessation:** Reducing the risk of developing RA.
   b. **Healthy Diet:** Anti-inflammatory diets may help manage symptoms.
   c. **Regular Exercise:** Maintains joint function and overall health.
3. **Regular Monitoring:** Ongoing evaluation and management by a healthcare provider to prevent complications and adjust treatment as needed.

Osteoporosis

Introduction

Osteoporosis is a metabolic bone disorder characterized by reduced bone mass and deterioration of bone tissue, leading to increased bone fragility and susceptibility to fractures. It is often termed a "silent disease" because it progresses without symptoms until a fracture occurs.

Pathophysiology

1. **Bone Remodeling Imbalance:** Osteoporosis results from an imbalance between bone resorption and bone formation. Osteoclasts (cells that break down bone) become more active or osteoblasts (cells that build bone) become less active, leading to decreased bone density.
2. **Bone Microarchitecture:** The loss of bone mass affects the microarchitecture of bone tissue, making bones more porous and less structurally sound.
3. **Hormonal Changes:** In postmenopausal women, decreased estrogen levels contribute to increased osteoclast activity and bone loss. In men, reduced testosterone levels can similarly affect bone density.

Epidemiology

1. **Prevalence:** Osteoporosis affects approximately 1 in 3 women and 1 in 5 men over the age of 50.
2. **Gender:** Women are more commonly affected due to postmenopausal hormonal changes, which lead to faster bone loss.
3. **Age:** The risk of osteoporosis increases with age, as bone density naturally decreases.
4. **Genetics:** Family history of osteoporosis or fractures increases risk.
5. **Lifestyle Factors:** Factors such as physical inactivity, smoking, and excessive alcohol consumption contribute to the risk.

Symptoms and Complications

1. **Symptoms**
   a. Often asymptomatic until a fracture occurs.
   b. Possible early signs include back pain, loss of height, and a stooped posture.

2. **Complications**
   a. **Fractures:** Common sites include the spine, hip, and wrist. Fragility fractures occur with minimal trauma.
   b. **Kyphosis:** Compression fractures in the spine can lead to a forward-bending posture.
   c. **Chronic Pain:** Fractures and deformities can lead to chronic pain and reduced mobility.
   d. **Functional Impairment:** Increased risk of falls and reduced ability to perform daily activities.

Diagnosis

1. **Bone Density Testing**
   a. **Dual-Energy X-ray Absorptiometry (DXA):** The most common and reliable method for measuring bone mineral density (BMD). T-scores from DXA scans are used to diagnose osteoporosis.

2. **Clinical Evaluation**
   a. **Medical History:** Includes risk factors, family history, and previous fractures.
   b. **Physical Examination:** Identifies height loss, spinal deformities, and other physical signs of osteoporosis.

3. **Laboratory Tests**
   a. **Serum Calcium and Vitamin D Levels:** To rule out secondary causes of bone loss.
   b. **Bone Turnover Markers:** Can indicate the rate of bone resorption and formation.

Treatment

1. **Medications**
   a. **Bisphosphonates:** Alendronate, risedronate, and zoledronic acid inhibit bone resorption.
   b. **Selective Estrogen Receptor Modulators (SERMs):** Raloxifene mimics estrogen's effects on bones.
   c. **Hormone Replacement Therapy (HRT):** Estrogen therapy, though used less frequently due to risks.
   d. **Parathyroid Hormone Analogues:** Teriparatide stimulates bone formation.
   e. **Denosumab:** A monoclonal antibody that inhibits osteoclast activity.

2. **Lifestyle Modifications**
   a. **Diet:** Adequate intake of calcium and vitamin D.
   b. **Exercise:** Weight-bearing and muscle-strengthening exercises to improve bone strength and balance.
   c. **Fall Prevention:** Measures to reduce the risk of falls, such as home safety modifications and balance training.

3. **Surgical Interventions**:
   a. **Fracture Repair:** Surgery may be needed to repair severe fractures.

Complications

1. **Fractures:** High risk of fractures due to reduced bone strength, leading to potential disability and decreased quality of life.
2. **Decreased Mobility:** Fractures and pain can limit physical activity and independence.
3. **Chronic Pain:** Persistent pain from fractures or deformities.
4. **Increased Risk of Falls:** Impaired balance and mobility can lead to more frequent falls.

Prevention

1. **Bone Health**
   a. **Diet:** Ensure adequate intake of calcium (1,000-1,200 mg/day) and vitamin D (800-1,000 IU/day).
   b. **Exercise:** Engage in regular weight-bearing and resistance exercises to strengthen bones and improve balance.

2. **Lifestyle Changes**
   a. **Avoid Smoking:** Smoking accelerates bone loss.
   b. **Limit Alcohol Consumption:** Excessive alcohol can affect bone health and increase fracture risk.

3. **Medications:** For individuals at high risk, medications may be used to prevent bone loss before significant damage occurs.
4. **Regular Screening:** DXA scans for those at risk or over the age of 65 to monitor bone density.

Osteoporosis is a manageable condition, especially with early detection and a proactive approach to treatment and prevention. Addressing risk factors and maintaining bone health through lifestyle choices and appropriate medical interventions can significantly reduce the impact of osteoporosis.

Gout

Introduction

Gout is a type of inflammatory arthritis characterized by sudden and severe pain, redness, and swelling in the affected joints, most commonly the big toe. It is caused by the deposition of monosodium urate crystals in the joints due to high levels of uric acid in the blood.

Pathophysiology

1. **Hyperuricemia:** Gout develops when there is an excessive amount of uric acid in the blood, a condition known as hyperuricemia. Uric acid is a byproduct of purine metabolism, and its excess can result from overproduction or reduced excretion.
2. **Crystal Formation:** High levels of uric acid lead to the formation of monosodium urate crystals, which precipitate in the joints and surrounding tissues.
3. **Inflammatory Response:** The presence of these crystals triggers a strong inflammatory response from the immune system, leading to pain, redness, and swelling. The body's immune cells, particularly neutrophils, are recruited to the site, exacerbating inflammation.

Epidemiology

1. **Prevalence:** Gout affects approximately 1-4% of adults in Western countries. It is becoming increasingly common due to rising obesity rates and changes in diet.
2. **Gender:** More common in men, with a 3:1 ratio compared to women, largely due to differences in uric acid metabolism and excretion.
3. **Age:** Typically manifests in middle-aged adults, with the risk increasing with age.
4. **Genetics:** Family history of gout can increase the risk, suggesting a genetic predisposition.

Symptoms and Complications

1. **Symptoms**
   a. **Acute Attack:** Sudden onset of intense pain, swelling, redness, and warmth in the affected joint, often the big toe (podagra). Attacks can last from a few days to a couple of weeks.
   b. **Intercritical Periods:** Periods between acute attacks where symptoms subside but may recur.
   c. **Chronic Gout:** If untreated, gout can lead to chronic joint pain and the development of tophi (deposits of urate crystals under the skin).

2. **Complications**
   a. **Tophaceous Gout:** Formation of tophi, which can cause joint damage and deformities.
   b. **Kidney Stones:** Elevated uric acid levels can lead to the formation of uric acid stones in the kidneys.
   c. **Joint Damage:** Repeated attacks can lead to permanent joint damage and loss of function.
   d. **Infection:** Joint inflammation can increase the risk of secondary bacterial infections.

Diagnosis

1. **Clinical Evaluation:** Diagnosis is based on symptoms, medical history, and physical examination.

2. **Laboratory Tests**
   a. **Serum Uric Acid:** Elevated levels indicate hyperuricemia but are not definitive for gout.
   b. **Joint Fluid Analysis:** The most definitive test. Synovial fluid from the affected joint is analyzed under polarized light microscopy to identify urate crystals.
   c. **Blood Tests:** To rule out other conditions and assess kidney function.

3. **Imaging Studies**
   a. **X-rays:** May show joint damage or tophi in chronic cases but are not used for initial diagnosis.
   b. **Ultrasound:** Can detect urate crystals and early changes in the joints.
   c. **CT Scan:** Used in certain cases to detect urate crystal deposits.

Treatment

1. **Acute Attack Management**
   a. **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):** Such as ibuprofen or naproxen, to reduce pain and inflammation.
   b. **Colchicine:** Effective for acute gout attacks and can also be used for prophylaxis.
   c. **Corticosteroids:** Such as prednisone, can be used when NSAIDs or colchicine are not suitable.

2. **Long-Term Management**
   a. **Urate-Lowering Therapy**
      - **Allopurinol:** Reduces uric acid production.
      - **Febuxostat:** Another option for lowering uric acid levels.
      - **Probenecid:** Increases uric acid excretion by the kidneys.
   b. **Lifestyle Changes:** Dietary modifications to reduce purine intake (e.g., avoiding red meat, shellfish, and alcohol), weight management, and adequate hydration.
   c. **Medication Adherence:** Ongoing urate-lowering therapy to prevent future attacks and manage hyperuricemia.

Complications

1. **Tophaceous Deposits:** Can lead to chronic pain and joint deformities.
2. **Renal Complications:** Risk of uric acid nephrolithiasis (kidney stones) and potential kidney damage.
3. **Recurrent Attacks:** Failure to manage uric acid levels can lead to frequent and severe attacks.

Prevention

1. **Dietary Modifications**
   a. **Reduce Purine Intake:** Limit consumption of high-purine foods like red meats, organ meats, and certain seafood.
   b. **Limit Alcohol:** Especially beer and spirits, which can increase uric acid levels.
   c. **Increase Fluid Intake:** Helps dilute uric acid and prevent kidney stones.
2. **Maintain Healthy Weight:** Reducing body weight can lower uric acid levels and reduce the risk of gout attacks.
3. **Regular Monitoring:** For individuals with hyperuricemia or previous gout attacks, regular monitoring and management of uric acid levels are crucial to prevent complications.