International Journal of Orthopaedics and Bone Disorders
www.orthopedicsjournals.net
Online ISSN: 2664-8954, Print ISSN: 2664-8946
Received Date: 05-11-2019 Accepted Date: 01-12-2019; Published: 08-12-2019
Volume 1; Issue 1; 2019; Page No. 16-27

Diseases of the locomotor system: A basic review

Dr. Siva Rami Reddy E
Faculty of Homoeopathy, Tantia University, Sri Ganganagar, Rajasthan, India

Abstract
Locomotor diseases (basic review) are to understand very easily for graduate, post graduate and post doctoral ayush, dental, medical etc., students. I am explaining main and important diseases in locomotor system in day to day practical life for medical students and professionals. Diseases are lumbar Spondylosis, rheumatoid arthritis, gout, osteoarthritis, sjogrens syndrome, Reiter's disease and osteoporosis.

Keywords: locomotor diseases, causes, clinical features, investigation

Introduction
We have lot of diseases to explain in locomotor system. But only main/few diseases are reviewing for under graduate, post graduate and post-doctoral AYUSH, dental, medical, nursing etc., for entrance and main examination purpose.

Lumbar Spondylosis
It is defined as a "degenerative changes in the discs and lumbar spine are almost universal in the elderly" (or) is a chronic, no inflammatory disease caused by degeneration of lumbar disc and/or facet joints. Its affects approximately 60–85% of adults during some point in their lives. Fortunately, for the large majority of individuals, symptoms are mild and transient, with 90% subsiding within 6 weeks. Pain, defined as pain symptoms persisting beyond 3 months, affects an estimated 15–45% of the population. Degenerative spine changes are remarkably common in population studies. Symmons' *et al* study of individuals aged 45–64 years identified 85.5% of participants to demonstrate osteophytes within the lumbar spine. O'Neill *et al* explored osteophytosis within a UK adult population over age 50 years, finding 84% of men and 74% of women to demonstrate at least one vertebral osteophyte, with increased incidence among individuals with more physical activity, self reported back pain, or higher BMI scores. Despite marked variability within the population, men appear to have more significant degenerative changes than women, both with regard to number and severity of osteophyte formation [1].

Causes
Age: An extensive autopsy study in 1926 reported evidence of spondylitis deformans to increase in a linear fashion from 0% to 72% between the ages of 39 and 70 years.

Hereditary
Genetic factors likely influence the formation of osteophytes and disk degeneration. Spector and MacGregor proposed that 50% of the variability found in osteoarthritis can be attributed to heritable factors. Similarly, twin studies evaluating the progression of degenerative changes in lumbar MRI imaging suggest that approximately half (47–66%) of the variance could be explained by genetic and environmental factors, attributing only 2–10% of variance to physical loading and resistance training.
Occupation: Disk generation has long been associated with certain activities.

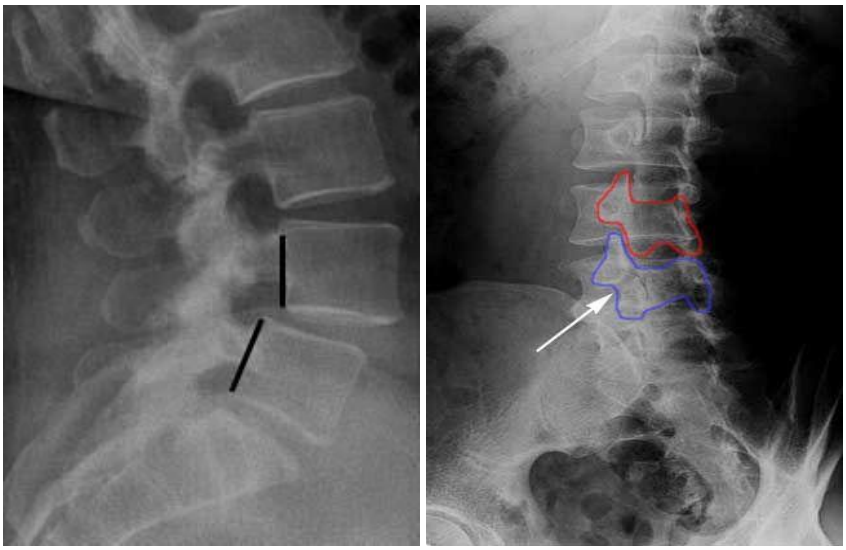


Fig 1: Lumbar Spondylosis

Pathogenesis

The high incidence of simultaneous degenerative changes to the intervertebral disk, vertebral body, and associated joints suggests a progressive and dynamic mechanism, with interdependent changes occurring secondary to disk space narrowing.

1. **Phase (Dysfunction Phase):** It describes the initial effects of repetitive microtrauma with the development of circumferential painful tears of the outer, innervated anulus, and associated end-plate separation that may compromise disk nutritional supply and waste removal. Such tears may coalesce to become radial tears, more prone to protrusion, and impact the disk's capacity to maintain water, resulting in desiccation and reduced disk height. Fissures may become ingrown by vascular tissue and nerve endings, increasing innervation and the disk's capacity for pain signal transmission.
2. **Phase (Instability Phase):** It is characterized by the loss of mechanical integrity, with progressive disk changes of resorption, internal disruption, and additional annular tears, combined with further facet degeneration that may induce subluxation and instability.
3. **Phase (Stabilization Phase):** It continued disk space narrowing and fibrosis occurs along with the formation of osteophytes and transdiscal bridging.

Schneck presents a further mechanical progression, building upon this degenerative cascade of the intervertebral disk, to explain other degenerative changes of the axial spine. He proposes several implications of disk space narrowing. Adjacent pedicles approximate with a narrowing of the superior inferior dimension of the intervertebral canal. Laxity due to modest redundancy of the longitudinal ligaments enables bulging of the ligamentum flavum and potential for spine instability. Increased spine movement permits subluxation of the superior articular process (SAP), causing a narrowed anteroposterior dimension of the intervertebral and upper nerve root canals. Laxity may also translate into altered weight mechanisms and pressure relationships on vertebral bone and joint spaces believed to influence osteophyte formation and facet hypertrophy to both inferior and superior articular processes with risks for projection into the intervertebral canal and central canal, respectively. Oblique orientations of the articular processes may further cause retro spondylo listhesis, with resulting anterior encroachment of the spinal canal, nerve root canal, and intervertebral canal [2].

Clinical features

Postural low back pain is often provoked by prolonged sitting, standing, bending or lifting. Acute episodes with symptoms and signs of nerve root compression are similar to those following acute disc prolapsed.

Investigations

Imaging tests can provide detailed information to guide diagnosis and treatment.

- Neck X-ray: An X-ray can show abnormalities, such as bone spurs, that indicate cervical spondylosis. Neck X-ray can also rule out rare and more serious causes for neck pain and stiffness, such as tumors, infections or fractures.
- CT scan: A CT scan can provide more detailed imaging, particularly of bones.
- MRI: MRI can help pinpoint areas where nerves might be pinched.
- Myelography. A tracer dye is injected into the spinal canal to provide more detailed X-ray or CT imaging.
- Electromyography: This test measures the electrical activity in your nerves as they transmit messages to your muscles when the muscles are contracting and at rest.
- Nerve conduction study**:** Electrodes are attached to your skin above the nerve to be studied. A small shock is passed through the nerve to measure the strength and speed of nerve signals [3].

Osteoarthritis

Osteoarthritis also called as degenerative joint disease. It involved more than one disease. Osteoarthritis is the clinical and pathological

outcome of a range of disorders that results in structural and functional failure of synovial joints. Traditionally, it has been

considered a disease of articular cartilage. The current concept holds that osteoarthritis involves the entire joint organ, including the subchondral bone, menisci, ligaments, periarticular muscle, capsule, and synovium. Osteoarthritis is the most prevalent form of arthritis, with an associated risk of mobility disability (defined as needing help walking or climbing stairs) for those with affected knees being greater than that due to any other medical condition in people aged [4, 5].

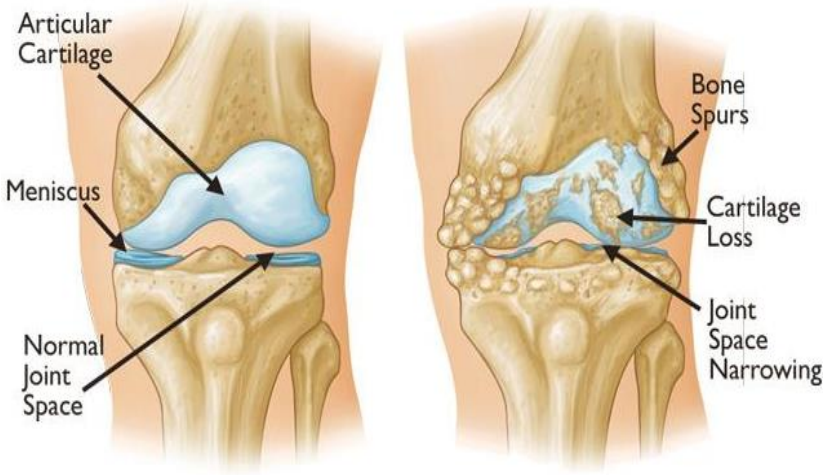


Fig 2: Normal knee and Osteoarthritis knee

Osteoarthritis is classified into two groups. Primary osteoarthritis can be localised or generalised, the latter more commonly found in postmenopausal women, with development of Heberden's nodes. Secondary osteoarthritis has an underlying cause, such as trauma, obesity, Paget's disease, or inflammatory arthritis.

Causes: osteoarthritis etiology is unknown and degenerative joint changes occur in response to a recognizable local or systemic factor. In developmental causes are perthes diseases, slipped capital femorl epiphysis, epiphysiolysis, hip dysplasia, epiphysial dysplasias, Intra articular acetabular labrum. Traumatic causes are intra articular fracture, meniscectomy, occupational e.g. elbows of pneumatic drill operators, hypermobility e.g. Ehlers danlos syndrome, long leg arthropathy. Metabolic causes are al kaptonuria (ochronosis), haemo chromatosis, Wilsons disease, chondro calcinosis. Inflammatory causes are rheumatoid arthritis, gout, septic arthritis, haemophilia. Aspectic necrosis are corticosterioids, sickle cell disease, decompression sickness, SLE and other collageneses. In neuropathic causes are tabes dorsalis, syringomyelia, diabetes mellitus, Peripheral nerve lesions. In endocrine is acromegaly and paget's disease, gaucher's disease.

Pathogenesis

Cartilage is made of water (70%) and a type II collagen framework with proteoglycans and glycosaminoglycans (consisting Mainly of aggrecan and also chondroitin), produced by chondrocytes. Proteoglycans in turn bind to hyaluronate which stabilises the macromolecule. Chondrocytes receive nutrition from the synovium by diffusion and the synovial fluid is circulated by joint movement. It has been postulated that if the joint stops moving (as a result of a fracture or immobility) and chondrocytes lose their source of nutrition, they go into shock and cartilage repair ceases. Metalloproteinases are produced, which catalyse collagen and proteoglycan degradation. The synovium has been shown to be variably inflamed in osteoarthritis producing increased levels of interleukin-1 (IL-1) and tumour necrosis factor-alpha (TNF-α), cytokines that induce nitric oxide and metalloproteinase production. Interleukin-6 (IL-6) and mechanical loading of the joint also induce catabolic cytokine receptors. These bind IL-1 and TNF-α within cartilage causing more destruction. It is thought that the osteophytes and subchondral sclerosis seen in osteoarthritis may be the body's way of trying to compensate for lack of cartilage, although some researchers have found bony changes before cartilage changes in animal models. This sort of abnormal bone is also thought to lead to further degradation of the cartilage surrounding it. Poor synthesis of cartilage building blocks may be caused by dysfunctional forms of insulin like growth factor-1 and transforming growth factor beta, agents which normally promote new cartilage formation [6].

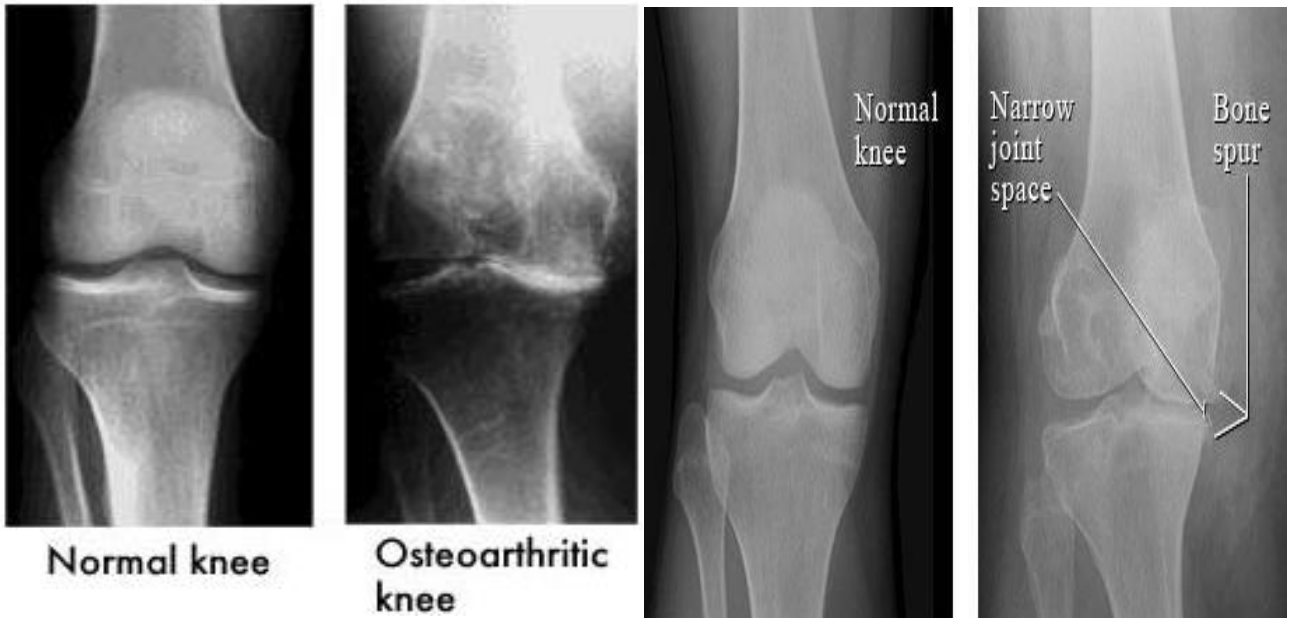


Fig 3: X ray normal knee and osteoarthritis knee

Clinical features

Osteoarthritis most frequently involved are those of the spine, hips, knees and hands. Common patterns of joint involvement include the nodal and non-nodal types of primary generalized osteoarthritis with prominent involvement of the knee and hands (distal interphalangeal joints, proximal interphalangeal joints, carpometacarpal joints of thumbs), as well as osteoarthritis confined to the knee or hips. All symptoms are gradual in onset. Pain is at first intermittent and is provoked by the use of the joint and relieved by rest. As the disease progresses, movement in the affected joint becomes Increasingly limited, initially as a result of pain and muscular spasm, but later because of capsular fibrosis, osteophyteformation and remodeling of bone. Muscle wasting is an important factor in the progress of the disease, as in the absence of normal muscular control the joint becomes more prone to injury. Nodal osteoarthritis occur predominatly in middle aged women. It affects the terminal interphalangeal joints of the fingers, with the development of gelatioonus cysts or bony out growths on the dorsal aspect of these joints (Heberdent's nodes, see fig.4). The onset is sometimes acute, with considerable pain, swelling and inflammation.

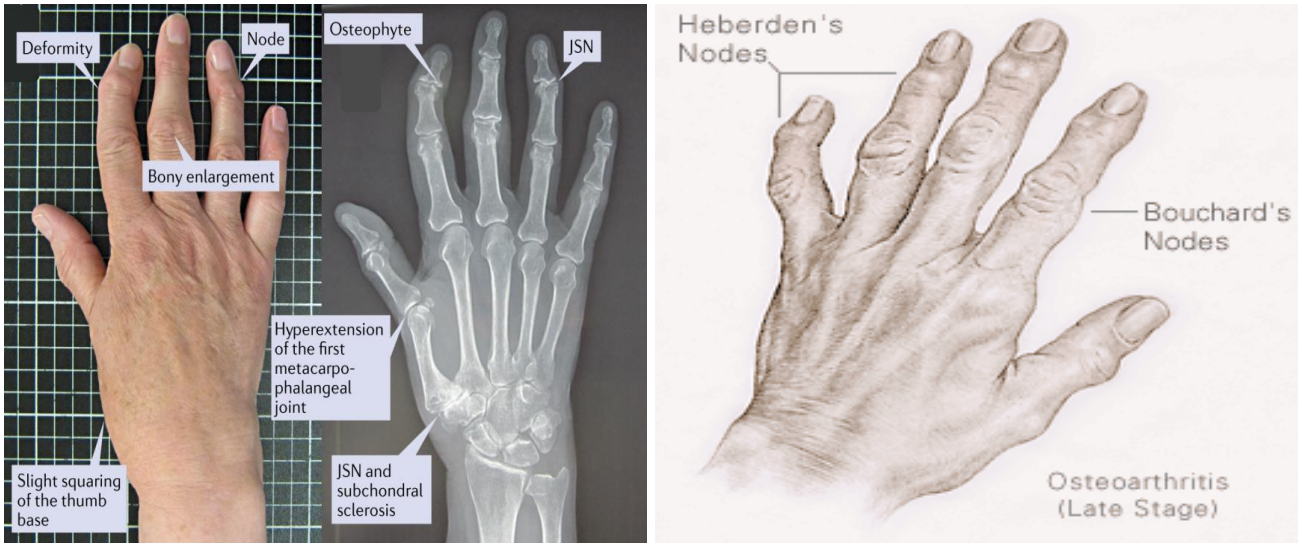


Fig 4: Nodal osteoarthritis

Heberden's nodes seldom cause serious disability. Similar lesions may affect the proximal interphalangeal joints (Bouchard's Nodes) (figure 4), and the disorder also frequently involves the carpometacarpal joints of the thumbs, the spinal apophyseal joints, the hips and the knees.

Investigation

The blood count and ESR are characteristically normal. Several radiograph scoring systems have been employed to assist the measurement of osteoarthritis progression. Other techniques include chondrometry, where minimal inter bone distance is

measured using a special compass magnifying glass calibrated to 0.1 mm. Synovial fluid is viscous and has a low cell count.
Plain radiographs: The following changes may be seen on plain radiographs: Joint space narrowing, Osteophytes, bony cysts and sub chondral sclerosis.

MRI
This is already well established for use in assessing ligament and meniscal tears in the knee. It has no place in routine clinical assessment of osteoarthritis, but may be a specific and sensitive way of quantifying cartilage loss. Currently, magnetic resonance imaging has not proved to be sensitive enough in the detection of preclinical osteoarthritis. Changes in surface morphology and full thickness cartilage defects can be seen, but fibrillation cannot yet be evaluated.

Management
Weight loss-Encourage overweight patients with osteoarthritis of the hip and knee to lose weight through a combination of diet and exercise.
Physical therapy consists of several strategies to facilitate resolution of symptoms and improve functional deficits, including range of motion exercise, muscle strengthening, muscle stretching, and soft tissue mobilisation.
Knee braces and orthotics-For those with instability of the knee and varus misalignment, valgus bracing and orthotics shift the load away from the medial compartment and, in doing so, may provide relief of pain and improvement in function.

Gout
Gout is a picturesque presentation of uric acid disturbance. It is the most well understood and described type of arthritis. Gout is a systemic disease that results from the deposition of mono sodium urate crystals (MSU) in tissues. Increased serum uric acid (SUA) above a specific threshold is a requirement for the formation of uric acid crystals. Despite the fact that hyperuricemia is the main pathogenic defect in gout, many people with hyperuricemia do not develop gout or even form UA crystals. In fact, only 5% of people with hyperuriceamia above 9 mg/dL develop gout. Accordingly, it is thought that other factors such as genetic predisposition share in the incidence of gout. The general prevalence of gout is 1–4% of the general population. In western countries, it occurs in 3–6% in men and 1–2% in women. In some countries, prevalence may increase up to 10%. Prevalence rises up to 10% in men and 6% in women more than 80 years old. Annual incidence of gout is 2.68 per 1000 persons [7].

Factors predisposing to hyperuricaemia
Renal failure, drugs (diuretics, low does of aspirin), lead poisoning, hyper parathyroidisim, myxoedema, down's syndrome, lactic acidosis (alcohol, exercise, starvation, vomiting, toxaemia of pregnancy, type 1 glycogen storage disease), unidentified inherited defect, myeloproliferative disorder, lymphoproliferative disorder, hypoxanthine, phosphoribosyl pyrophosphate synthetase overactivity, glucose 6 phosphate deficiency, idiopathic, Deficiency of enzymes involved in purine metabolism leads to overproduction of UA [8].

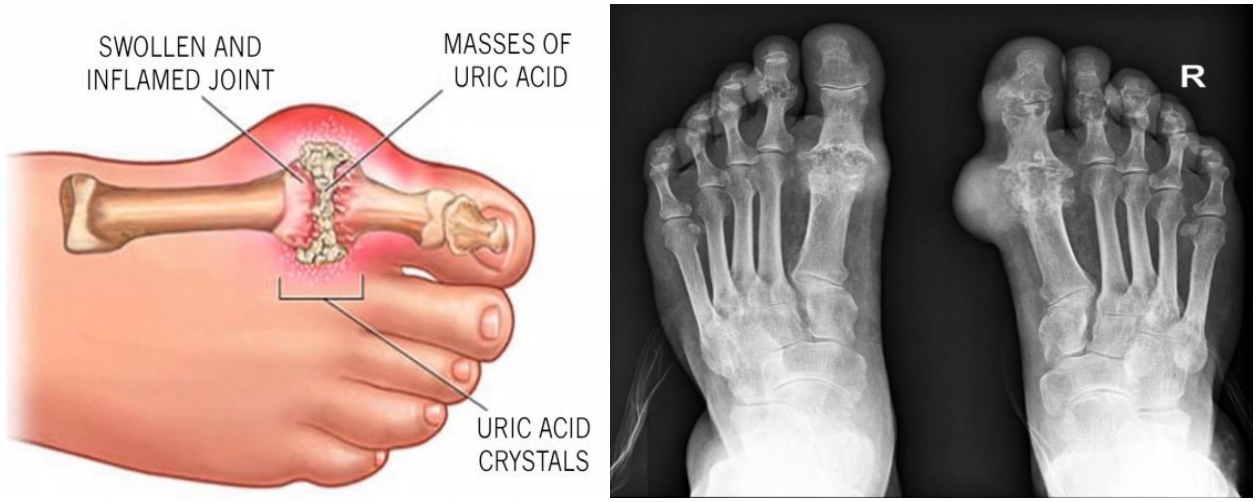


Fig 5: Radiology of the Gout

Pathophysiology
Urate is the ionized form of uric acid present in the body. Uric acid is a weak acid with pH of 5.8. Urate crystals deposition in tissues starts to occur when serum uric acid level rises above the normal threshold. Pathological threshold of hyperuricemia is defined as 6.8 mg/dl. Some factors may affect the solubility of uric acid in the joint. These include synovial fluid pH, water concentration, electrolytes level, and other synovial components such as proteoglycans and collagen. SUA level in the body is determined by the balance between its production either from purine intake in diet or endogenous production by cellular turnover and its excretion by the kidneys and GIT. Increased production
Of UA is responsible for only 10% of cases of gout while the remaining 90% are caused by its renal under excretion. Factors affecting SUA levels include age and gender. SUA is low in children. After puberty, SUA levels start to increase to reach their normal levels. In men, levels are higher than in women. However, SUA levels in postmenopausal women increase to reach men's levels. This explains why gout is usually a disease of middle aged and older men, and postmenopausal women. Rarely, it may happen in children and young adults in some rare inborn errors of

purine metabolism. These enzymatic defects result in increased SUA with consequent production of UA crystals in kidneys and joints [9].

Clinical features

The ankle, knee, small joints of the feet and hands, writs, elbow follow in decreasing order of frequency. The onset may be insidious or explosively sudden, often waking the patient from sleep. The effected joint in hot, red, swollen with shiny overlying skin and dilated veins, painful and tender.

Investigation

The serum urate level is usually raisesd but it is important to appreciate that this does not prove the diagnosis because asymptomatiac hyper uricaemia is very common. Among patients with SUA levels between 7 and 7.9 mg/dL only 0.09% will develop gout every year. As for patients with SUA between 8 and 8.9 mg/dl, 0.4% out of them may develop gout. With hyper uriceamia above 9 mg/dl, only 0.5% of patients may get gout. The gold standard of diagnosis is the identification of MSU crystals in synovial fluid aspirate using polarized light microscopy. In chronic tophaceous gout, the main radiographic features are:

- Tophi which are articular or periarticular soft tissue dense nodules.
- MSU deposits in the cartilaginous part.
- Joint space narrowing in advanced disease.
- Bone erosions are characteristic. They are well circumscribed intraarticular or juxtarticular lesions with overhanging margins. They result from the growth of tophi into the bone, hence are usually seen near tophi.
- Periarticular osteopenia is usually absent and proliferating bone can be seen mostly as irregular spicules.

Doppler US can distinguish between active/hot tophi and inactive/cold ones based on their doppler signal. Conventional CT is not helpful in the diagnosis of acute gout as it can't detect inflammation, synovitis, tenosynovitis and osteitis. This handicap is however, more than counterbalanced by its role in chronic gout. It is able to detect erosions better than Magnetic Resonance Imaging (MRI) or CR. Nuclear Scintigraphy is rarely used for evaluation. Positive results are often found incidentally when a study is performed for other indications.

Rheumatoid arthritis

It is the most common form of chronic inflammatory joint disease. Rheumatoid arthritis (RA) is an inflammatory rheumatic disease with progressive course affecting articular and extra-articular structures resulting in pain, disability and mortality. Persistent inflammation leads to erosive joint damage and functional impairment in the vast majority of patients. The course of disease may be also different according to the presence or absence of several variables including genetic background, frequency of swollen joints, autoantibody in the serum and the severity of inflammatory process. The initial presenting features of early RA do not substantially differ from other inflammatory arthritis. So prior to definite diagnosis patients with early RA are usually classified as undifferentiated arthritis which difficultly can be discriminated from other inflammatory arthritis. Up to now, early RA was denoted to patients with disease duration of less than 2 years preferentially less than 12 months but currently most rheumatologists are willing to see the patients with symptom duration of less than 6 weeks. At present, "early" rheumatoid arthritis is regarded as patients with symptom duration < 3 months as early disease. Rheumatoid arthritis can involve most synovial joints, but rarely the DIPs or the thoracic, lumbar and sacral spine. The most commonly affected joints include the MCP and PIP joints of the hands, wrists and MTP joints of the feet. Joint destruction begins early in the disease with erosive changes often seen after only six months. The clinical exam can disclose synovial thickening and swelling, indicators of joint inflammation [10, 11].

Causes

Some people appear to have genetic factors that make it more likely. One theory is that bacteria or a virus triggers RA in people who have this genetic feature. In RA, the immune system's antibodies attack the synovium, which is the smooth lining of a joint. When this happens, pain and inflammation result. Inflammation causes the synovium to thicken. Eventually, if left untreated, it can invade and destroy cartilage — the connective tissue that cushions the ends of the bones. The tendons and ligaments that hold the joint together can also weaken and stretch. The joint eventually loses its shape and configuration. The damage can be severe.

Pathophysiology

The earliest changes is swelling and congestion of the synovial membrane and the underlying connective tissues, which become infiltrated with lymphocytes (especially CD4 T cells), plasma cells and macrophages. Effusion of synovial fluid in to the joint space takes place during active phases of the disease. Hypertrophy of the synovial membrane occurs, with the formation of lymphoid follicles resembling an immunologically active lymph node. Inflammatory granulation tissue (pannus) spread over and under the articular cartilage, which is progressively eroded and destroyed. Latterly, fibrous or bony ankylosis may occur. Muscles adjacent to inflamed joints atrophy and there may be focal infiltration with lymphocytes. Subcutaneous nodules consist of a central area of fibrinoid material surrounded by a palisade of proliferating mononuclear cells. Similar granulomatous lesions may occur in the pleura, lung, pericardium and sclera. Lymph nodes are often hypeplastic, showing many lymphoid follicles with large germinal centres and numerous plasma cell in the sinuses and medullary cords. Immunofloresence show that the plasma cells in the synovium and lymph nodes synthesise rheumatoid factors [12].

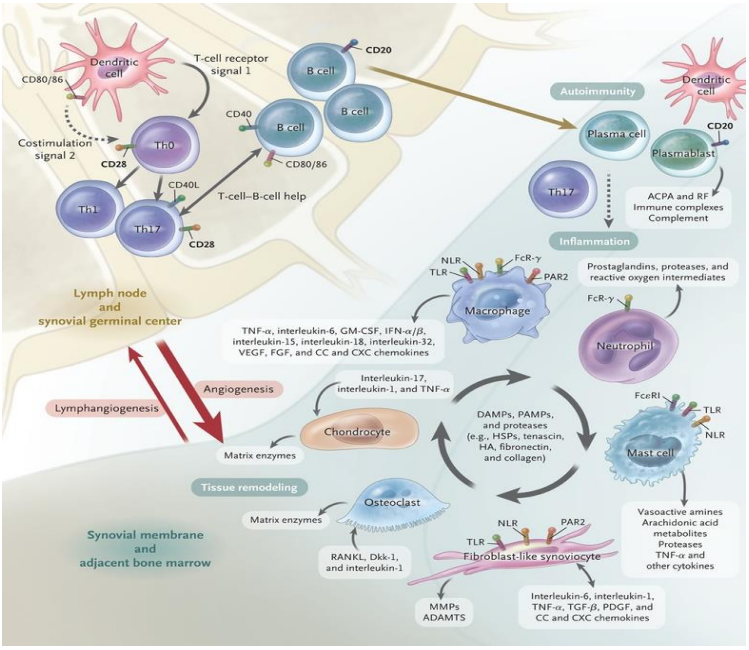


Fig 6: Pathology of rheumatoid arthritis

Clinical features

Early symptoms of rheumatoid arthritis may appear as vague pain with gradual appearance without classic symptoms of joint swelling or tenderness. These unusual symptoms are usually non-specific, and may persist for prolong period. Early articular manifestations of rheumatoid arthritis may be indistinguishable from other Rheumatic diseases. Prolong duration of morning stiffness with arthralgia, or arthritis in a limited number of joints may be a clue for considering rheumatoid arthritis diagnosis. Involvement of small joints of the hands or feet with swelling and tenderness particularly symmetric pattern of involvement along with positive compression test is highly suggestive of rheumatoid arthritis.

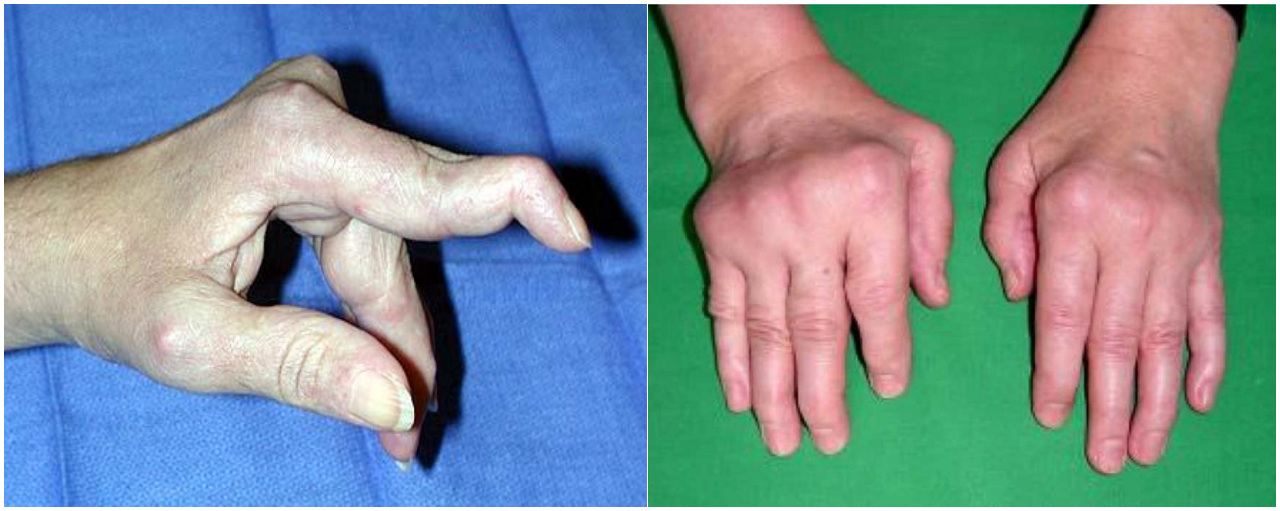


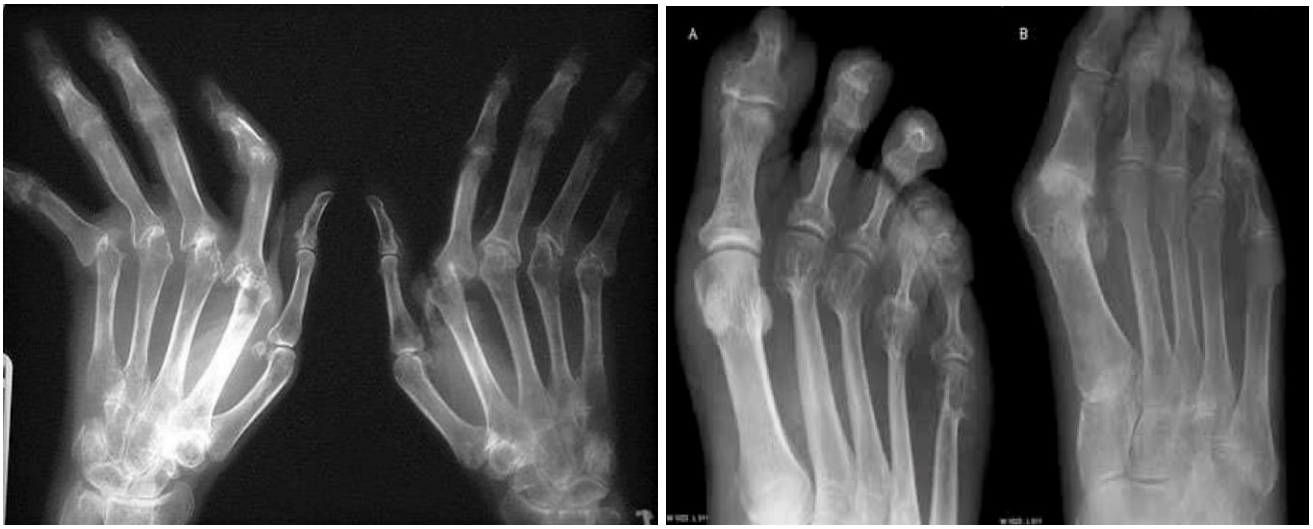
Fig 7: The hand in rheumatoid arthritis

Presence of some clinical features such as polyarthritis, symmetric arthritis,hand arthritis, pain upon squeezing the metcarpophalangeal or metatasophalangeal joints, and morning stiffness greater than 30 minutes can be helpful not only in estimating the future course of arthritis but also in limiting the spectrum of differential diagnosis. Identification of all involved joints by precise clinical examination is essential. Counting the tender and swollen joints, and calculation of disease activity score are logical methods for the determination of disease severity and response to treatment. In rheumatoid arthritis will be there swan neck deformity of the hands, wasting of the small muscles of the hands and synovial swelling at the writs, the extensor tendon sheaths, the metacarpophalangeal and proximal interphalangeal joints (figure 7).

Extra articular manifestation of rheumatoid arthritis

Extra articular manifestation of rheumatoid arthritis are fever, weight loss, fatigue, susceptibility to infection. In musculo

skeletal are muscle wasting, tenosynovitis, bursitis, osetoprosis. In haematological are anaemia, thromcocytosis, eosinophilia. Vasculitis are digital arteritis, ulcers, pyoderma gangrenosum, mononeuritis multiplex, visceral arteritis. In cardiac are pericarditis, myocarditis, endocarditis, conduction defects, coronary vasulitis, granulomatous aortitis. In pulmonary system are nodules, pleural effusion, fibrosing alveolitis, bronchilotis, caphan's syndrome. In neurological system are cervical cord compression, compression neuropathies, peripheral neuropathy, mononeuritis multiplex and also amyloidosis.



Subcutaneous nodules occur in about 20% of patients. They are usually seen at sites of pressure of friction, such as the extensor surfaces of the forearms below the elbow, bilateral posterior of the ankle joint (figure 9).

Figure 8: Radiological feature of rheumatoid arthritis

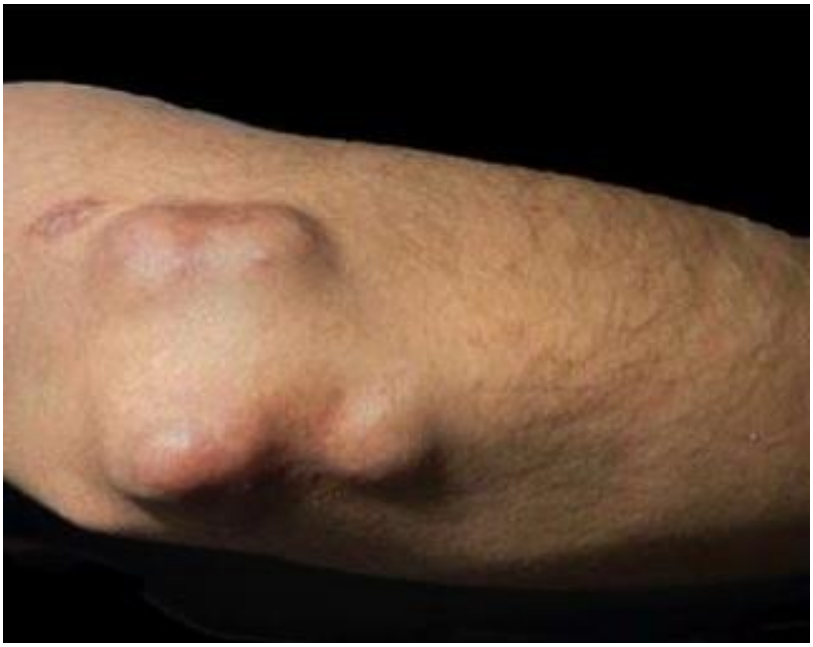


Fig 9: Rheumatoid nodules

Air way manifestation

The presence of airway disease in RA is estimated to affect 20–30% of patients. Manifestations can include cricoarytenoid arthritis, pulmonary fibrosis and small airway disease, typically seen as bronchiolitis obliterans on histopathology, with obstructive abnormalities on lung function testing. Lung disease is more frequent in RA patients who are male, seropositive, Smoke, and have longstanding disease. Some types of RA-associated lung disease are steroid responsive, but some patients have a progressive course leading to end-stage fibrosis and death. In addition to lung disease secondary to RA, patients are also at risk for pulmonary toxicities from RA-related medications, including methotrexate, leflunomide and even anti-TNF medications.

Cardiac vascular system manifestation

Rheumatoid arthritis patients have a 40% increased risk of mortality as compared to the general population after 20 years of

disease. This increased risk of mortality is primarily attributed to an increased incidence of cardiovascular disease. The propensity for vascular changes is found even in newly diagnosed patients, indicating that common mechanisms may exist linking synovitis resulting in joint destruction with endothelial dysfunction resulting in atherosclerosis.

Bone manifestation

The bones of rheumatoid arthritis patients are affected in both a local and systemic manner. At a local level, factors that stimulate osteoclasts resulting in increased bone resorption are released from inflammatory and fibroblastic pannus cells. Additionally, inflammatory cytokines prevent a compensatory increase in the rate of periarticular bone formation, resulting in net bone loss. This inhibition of osteoblastic activity is via a combination of impaired mineralization and impaired osteoblast differentiation. Bony changes in rheumatoid arthritis patients are not only seen in a periarticular distribution. Rheumatoid arthritis is a known risk factor for osteoporosis, with up to 30% of patients affected by some estimates. The risk of osteoporosis in rheumatoid arthritis patients is greater at the femoral neck than in the spine, but both areas can be involved.

Investigations

Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) provide the best information about the acute phase response. The level of CRP was shown to be significantly correlated with the severity of disease as well as radiographic changes. Auto antibodies such as rheumatoid factor and anti-CCP are very helpful for the diagnosis of rheumatoid arthritis. Anti-CCP antibody demonstrated a comparable sensitivity but a greater specificity than rheumatoid factor for the diagnosis of rheumatoid arthritis.

Radiographic signs of rheumatoid arthritis such as joint space narrowing, erosions and subluxation develop at later stage of rheumatoid arthritis process. Plain radiography is the standard method in investigating the extent of anatomic changes in rheumatoid arthritis patients. However, there are few data regarding the value of conventional radiographic examination in recent-onset arthritis. Synovitis is the early findings of rheumatoid arthritis and is strong predictor of bone erosion. Soft tissue swelling and mild juxtaarticular osteoporosis may be the initial radiographic features of hand joints in early - rheumatoid arthritis. Sonography is also a reliable technique that detects more erosions than radiography especially in early rheumatoid arthritis. In early rheumatoid arthritis, sonography can detect greater number of erosions and in a greater number of patients than can radiography.

Complications

Septic arthritis may complicate rheumatoid arthritis, particularly in patient with long standing nodular seropositive disease.

Sjogren's disease

This is an autoimmune disorder, characterized by lymphocytic infiltration of the salivary and lacrimal gland leading to xerostomia and kerato conjunctivitis sicca [13]. Primary sjogren's syndrome: age of onset in between 40 to 60, male are more than females, HLA-B8/DR3 Clinical features: common clinical features of primary sjogren's syndrome is keratoconjunctivitis sicca, xerostomia, salivary gland enlargement and rare clinical manifestations are anaemia, leucopenia, thrombocytopenia, lymphadenopathy, lymphoreticular, malignancy, hepatomegaly, malignancy, hepatomegaly, hyperglobulinaemic, purpura, vasculitis, neuropathy, myositis, fibrosing alveolitis, glomerulonephritis, renal tubular acidosis [14].

Secondary sjogren's disease are age of onset 40 -60 age, male are more than females. Common clinical features are mild kerato conjunctivitis sicca, dry mouth, and other associated auto immune disorders are systemic lupus erthematosus, progressive systemic, sclerosis, primary biliary cirrhosis, chronic active hepatitis, polymyositis, thryroiditis [15].

Investigations

The salivary flow rate is reduced and reduction in lacrimal secretion can be demonstrated by sue of the schirmer tear test.

Reiter's disease

It is the triad of nonspecific urethritis, conjunctivitis and reactive arthritis that follows bacterial dysentery or exposure to sexually transmitted infection. In 1916, Hans Reiter described the classic triad of arthritis, nongonococcal urethritis, and conjunctivitis (Reiters syndrome, RS) in a Prussian soldier with diarrhea, during the First World War [16, 17].

Clinical features

Symptoms generally appear within 1–3 weeks but can range from 4–35 days from onset of inciting episode of urethritis/ cervicitis or diarrhea. Signs and symptoms usually remit within 6 months. However, a significant percentage of patients have recurrent episodes of arthritis (15–50%), and some patients develop chronic arthritis (15–30%). Cardiac signs such as aortic regurgitation caused by inflammation of aortic wall and valve are rare. Other rare manifestations are central or peripheral nervous system lesions and pleuropulmonary infiltrates [18].

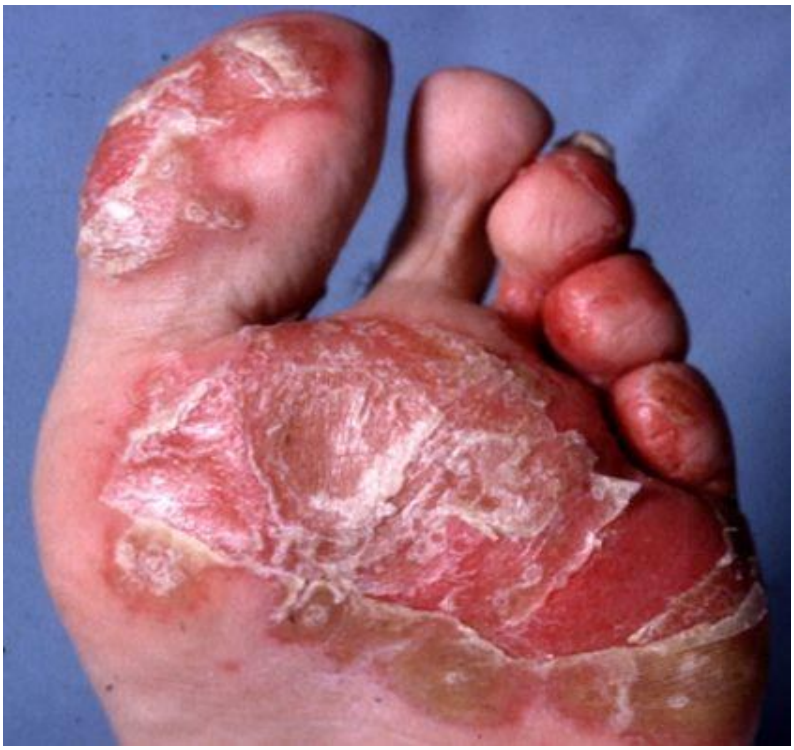


Fig 10: Reiter's disease

It is triggered by bacterial infection that enters via mucosal surfaces usually, (but not always) associated with human leukocyte antigen (HLA)- B27. The syndrome was the first rheumatologic disease noted in association with Human Immunodeficiency Virus. It is most common in individuals aged between 15–35 years; and it is rarely seen in children. The male-to-female post venereal ratio is 5–10:1,while the post-enteric ratio is 1:1. The incidence is estimated at 3.5 per 100,000, and is uncommon among Negroes.

Investigations

The ESR is often raised during the acute phase and may remain so long after joint symptoms have settled. The synovial fluid has the characteristics of a low viscosity inflammatory effusion with leucocyte counts as high as 50000/mm^3 but it is sterile on culture.

Osteoporosis

Osteoporosis, defined as low bone mass leading to increased fracture risk, is a major health problem that affects approximately 10 million world people. Osteoporosis is characterized by low bone mass, structural deterioration, and porous bone, which are associated with higher fracture risk. Bone loss related to declining estrogen levels increases fracture risk in postmenopausal women, who make up the majority of osteoporosis cases. Screening and diagnosis use a bone mineral density (BMD) measurement that estimates bone strength. Osteoporosis is the most common bone disease in humans, representing a major public health problem. It is more common in Caucasians, women, and older people. Osteoporosis is a risk factor for fracture just as hypertension is for stroke. Osteoporosis affects an enormous number of people, of both sexes and all races, and its prevalence will increase as the population ages. It is a silent disease until fractures occur, which causes important secondary health problems and even death [19]. It was estimated that the number of patients worldwide with osteoporotic hip fractures is more than 200 million. Osteoporosis is also an important health issue in Turkey, because the number of older people is increasing. The incidence rate for hip fracture increases exponentially with age in all countries as well as in Turkey, which is evident in the FRACTURK study. Bone tissue is continuously lost by resorption and rebuilt by formation; bone loss occurs if the resorption rate is more than the formation rate. The bone mass is modeled (grows and takes its final shape) from birth to adulthood: bone mass reaches its peak (referred to as peak bone mass (PBM)) at puberty; subsequently, the loss of bone mass starts. PBM is largely determined by genetic factors, health during growth, nutrition, endocrine status, gender, and physical activity. Bone remodeling, which involves the removal of older bone to replace with new bone, is used to repair microfractures and prevent them from becoming macrofractures, thereby assisting in maintaining a healthy skeleton. Menopause and advancing age cause an imbalance between resorption and formation rates (resorption becomes higher than absorption), thereby increasing the risk of fracture [20].

Causes

Osteoporosis causes are genetic (low body weight, family history), endocrine (hypogonadism, early menopause, thyrotoxicosis, hyperparathyroidisim), gastro intestinal disease (inflammatory bowel disease, malabosrption, chronic liver disease), inflammatory disease (ankylosing spondylitis, rheumatoid arthritis), inheried (homocystinuria, gaucher's disease, osteogenesis impefecta), life style (diet/calcium intake, exercise/ immobility, high trained athletes), other are anorexia nervosa, myeloma, neoplasia, mastocytosis, pregnancy associated, juvenile. Classification

Osteoporosis can be classified into two main groups by considering the factors affecting bone metabolism:

- Primary osteoporosis.
- Secondary osteoporosis.

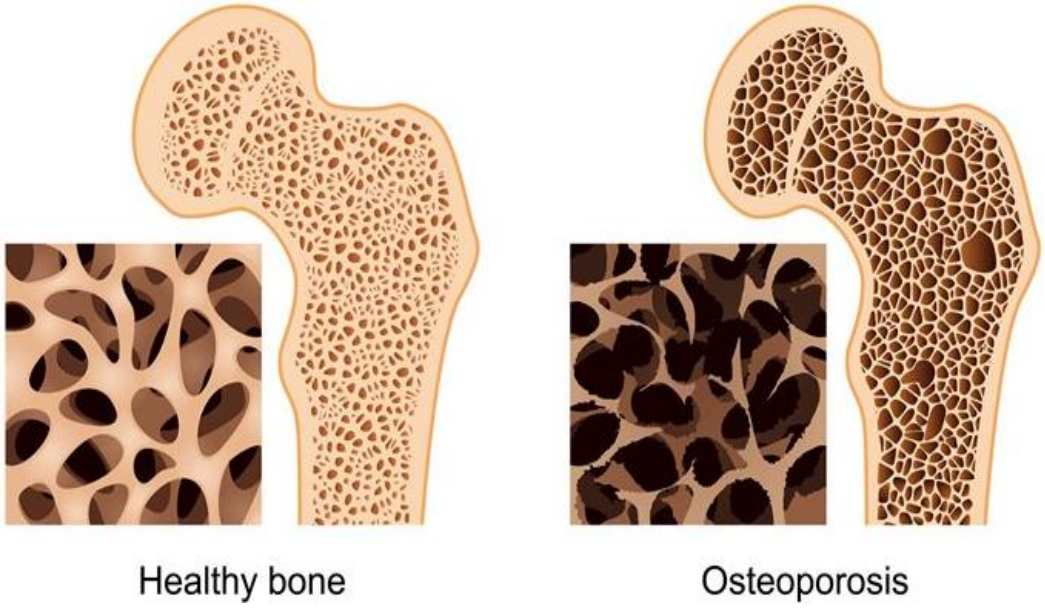


Fig 11: Osteoporosis and health bone

Primary osteoporosis

It is also known as postmenopausal osteoporosis, caused by the deficiency of estrogen, mainly affecting the trabecular bone; therefore, women are more susceptible to osteoporosis than men, as evident by a men/women ratio of 4/5.7.

Secondary osteoporosis

It is also called senile osteoporosis, and it is related to bone mass lost due to the aging of cortical and trabecular bones.

Pathogenesis

Genetic factors are important in the pathogenesis of osteoporosis and family studies suggest that genetic influences account for more than 70% of individual variance in bone mass. The molecular genetic basis by which bone mass is regulated is incompletely defined, but may involve subtle variations in the structure or regulation of genes which are involved in forming bone matrix and regulating bone turnover. Calcium intake is also important in determining the rate of post-menopausal bone loss. It may also occur as a complication of endocrine, inflammatory and neoplastic conditions and drug treatment side effect, substance abuse.

Clinical features

Osteoporosis is a silent disease until the patient experiences a fracture. A recent fracture at any major skeletal site, such as vertebrae (spine), proximal femur (hip), distal forearm (wrist), or shoulder in an adult older than 50 years with or without trauma, should suggest that the diagnosis of osteoporosis needs further urgent assessment involving diagnosis and treatment[21].

Diagnosis

Osteoporosis screening is based on BMD measurement, usually by DXA, which is then used to predict fracture risk. Hip BMD measurement by DXA is the best predictor of future hip fracture risk. In 2011, Nayak *et al* demonstrated through modeling analysis that screening for postmenopausal osteoporosis leads to more quality-adjusted life years compared with no screening. In addition, DXA scans were cost effective, especially when treatment was started for women with a T-score of −2.5 or more negative, with screening repeated every 5 years.

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