Distinctive Parameters and Strategies for Bone Related Diseases Management

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INTRODUCTION

Bone reformation is an active and complex process that depends on osteoclastic bone resorption and osteoblast bone supplanting being properly regulated. In fact, these two functions must be clearly linked, not only quantitatively, but also in time and space. The right bone mass could be affected if the connection is broken, leading to many skeletal pathologies. Indeed, the rise in osteoclast function and/or decreased osteoblast development is the product of bone degradation and osteoporosis. In the other hand, several pathologies are correlated with failure of osteoclastic bone resorption, such as osteopetrosis, which seems to be a rare hereditary disorder associated with high bone mass and often associated with diminished function of the bone marrow. Thus, this kind of mechanism of bone is very important to understand to study different type of disorders and their pathogenesis. Further this will also clear the path of new treatment approach for future [1]. In current review, we have discussed advancement in several common and complex disorders which are affected by the different type of bone mechanisms.

Paget's Disease

Paget's disease is the second most usual metabolic disease of bone characterized by a disorganized bone change leading from a defect in osteoclast that leads to increment in bone destruction process. It is simply a condition in which bone remodeling process eventuates too rapidly. causing bone deformities.

Main phases of pathogenesis have included activation of a lytic process in which bone alteration is substantially elevated and increased resorption happens by irregular osteoclast activity. A second mixed period of lytic and osteoblastic activity is dominated by rapid rises in osteoblast activity, resulting in an abnormal bone with sporadic collagen fibre deposition [2].

Osteoporosis

Osteoporosis being a common and silent condition before fracture is chronic and long-term skeletal disorder of bone affecting million people, more frequent in sessile adult and imposing pressure on the affected people, health service and culture around the world. Amenorrhea triggers the deterioration of bone due to which progression of osteoporosis is intensified because of estrogen deficiency [3].

Pathophysiology of Fracture Associated with Osteoporosis

Continuous process of resorption of bone by osteoclast and substitution with new osteoblast made bone enables the restoration and improvement of mechanical strength. As a chronic disorder, the pathophysiology of osteoporosis is dynamic and is the impact of genetic, hormonal, nutritional, life styles and physical contaminants. An excess of re-modeling operations in which re-sorption exceed development can result in its pathophysiology change.

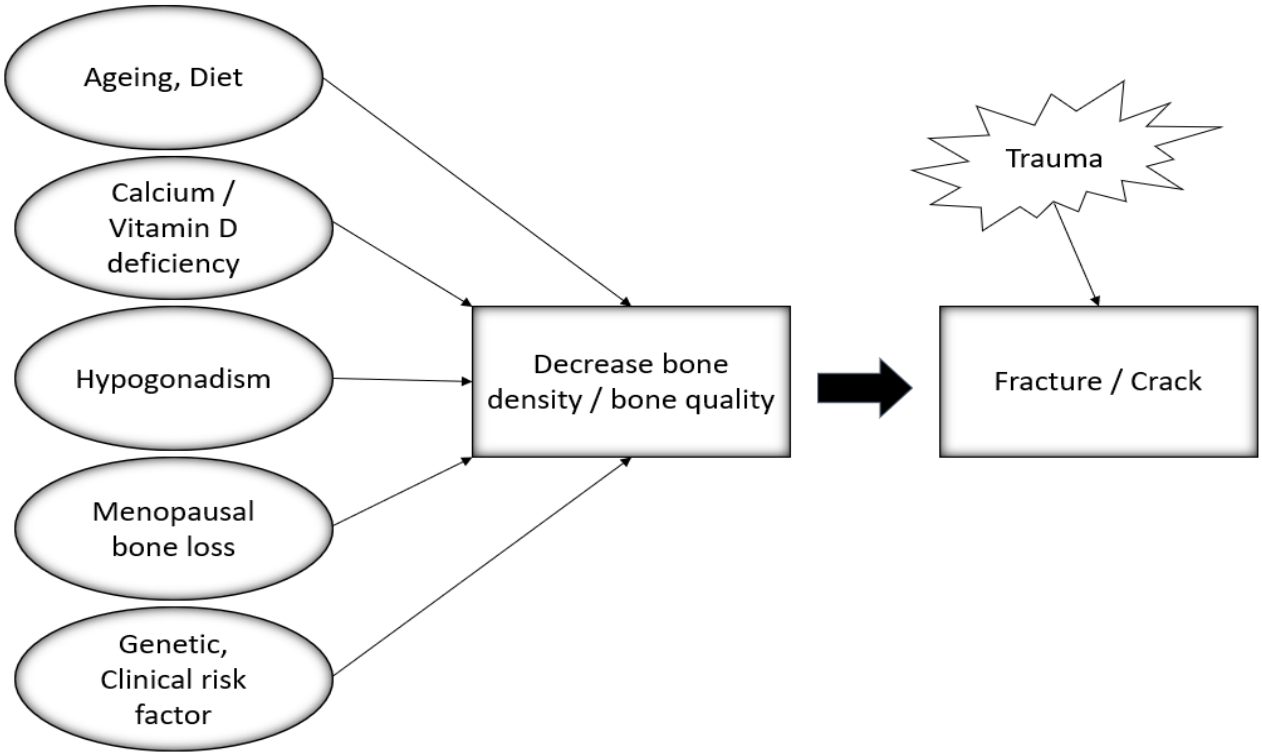

Figure. Pathogenesis of osteoporosis.

At present, anti-resorptive agents, including selective oestrogen receptor modulators (SERMs), bisphosphonates, oestrogen, and, are the drugs of choice for osteoporosis. Parathyroid hormone (PTH) and dietary calcium and vitamin D are other drugs. New medications, such as tissue-selective oestrogen receptor complexes (TSECs), are still commonly used.

Gout

Gout is an autoimmune type of arthritis caused by uric acid crystallisation between the joints, and hyperuricemia is also connected with it. Usually, acute gout is irregular and is one of the most painful conditions encountered by human beings. After years of acute gout, chronic gout normally begins, while tophi might indeed be part of the original stage.

Molecular Pathogenesis

The increased uric acid in blood circulation reacts with sodium and form sodium urate crystals (tophi), these crystals deposits into soft tissue and joints causing inflammation followed by pain. Basically, it happens due to overproduction of uric acid with uric acid excretion [4].

Osteomyelitis

The word "osteomyelitis" corresponds to bone marrow infection; Induced by an infectious organism “Staphylococcus aureus” that contributes to local bone degradation, necrosis and new bone deposition. In patients with acute peripheral bone osteomyelitis symptoms like, weight loss, weakness, fever, and irregular warmth, inflammation and edema typically occur. Osteomyelitis calls for prolonged use of antibiotic therapy. Surgical debridement may also be appropriate for osteomyelitis. An X-ray can reflect osteomyelitis-characteristic alterations, but often not after more than 3 weeks after the first risk increases [5].

Osteoarthritis

Osteoarthritis (OA) the most predominant, persistent, degenerative and the severe joint condition with unlikely regression and restoration of compromised tissues which rise in frequency with age and influence the most individuals and is the main causes of incapacitation.

Different inflammatory mediators have actually been shown to include synovial fluid in OA, including plasma proteins or C-reactive protein, approved as an OA pathogenesis marker), prostaglandins (PGE2), leukotrienes (PGE2), (LKB4), cytokines (IL1β, IL15, IL21, IL6, IL18, IL17, TNF), growth factors (VEGFs, TGFβ, NGFs, FGFs), constituents of nitric oxide and other supplements.

Both of these elements, which contribute to cartilage degradation contributing to the degradation of proteoglycan and collagen, can induce matrix metalloproteinases as well as other hydrolytic enzymes locally.

Various class of drugs like Analgesic, NSAIDS, Intra-articular corticosteroids, Glucosamine sulphate and Hyaluronic acid derivatives were used for their treatment [6].

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