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Commentary

The Pathological Process and Development of Osteoporosis

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DESCRIPTION

Osteoporosis is a major public health burden that's expected to increase because of the global population's ages. Within the last twenty years, Advanced Glycation End products (AGEs) are shown to be important mediators both in the pathological process and development of osteoporosis and other chronic diseases associated with aging. The growth of AGEs among the bone induces the formation of covalent cross-links with collagen and different bone proteins that affect the mechanical properties of tissue and disturb bone remodelling and deterioration, underlying osteoporosis. On the other hand, the gradual deterioration of the immune system in the time of aging (defined as immunosenescence) is additionally characterised by the generation of a high level of oxidants and AGEs. The synthesis and accumulation of AGEs (both localized within the bone or within the systemic circulation) may trigger a vicious circle (in which inflammation and aging united within the word "Inflammaging") which may establish and sustain the event of osteoporosis. This improves the molecular mechanisms/pathways by that AGEs induce the functional and structural bone impairment typical of Osteoporosis.

Bone diseases to represent a significant socioeconomic issue as recently recognized by the World Health Organization. The development of innovative bone-healing methods has been represented as a requirement for the successful treatments against bone defects. Among the wide spectrum of bone disorders, osteoporosis has emerged as a medical and socioeconomic threat. Though it's accepted more than 8.7 million fractures annually worldwide are caused by osteoporosis, they're typically diagnosed once the primary clinical fracture has occurred as a result of bone loss arises and initially asymptomatic. The lifetime fracture risk of a patient with osteoporosis has been calculable to be within the order of 35%-40%, which is extremely close to the risk for heart disease. Moreover, additionally to pathologic fractures, osteoporosis carries a substantial risk of disability because of serious medical complications. With the aging of the population, the prevalence of osteoporosis is predicted to further increase.

Osteoporosis is characterized by a general impairment of bone mass, strength, and microarchitecture, which will increase the propensity of fragility fractures. This pathological condition, whose aetiology is attributed to varied endocrine, metabolic, and mechanical factors, will occur at any age of life, however, it's preponderantly found in older and diabetic patients. As a skeletal disorder, osteoporosis results from a heterogeneous cluster of abnormal processes resulting in low bone mass and bone microarchitectural disruption. Low bone mass could result from increased bone reabsorption and/or reduced bone formation during remodelling, is usually accepted that the primary encompasses a higher impact on pathology development. Despite that the osteoporotic bone is often mineralized, there's a disruption of trabecular bone loss and microarchitecture and an increased cortical porosity. Recently, grown understanding of the bone remodelling method suggests that factors concerned in inflammation are linked with bone physiology and remodelling, supporting the hypothesis that inflammation considerably contributes to the pathological process of osteoporosis [1].

Bone is a permanently regenerating organ that is regularly revived in a very complicated method of formation and reabsorption. Bone reworking is a physiological method necessary to keep up the standard and strength of the skeleton by removing old bone and replaced it with a new matrix. It happens within the microscopic Basic Multicellular Units (BMU), in the main composed of osteoblasts, osteoclasts, and osteocytes [2]. The normal bone reabsorption and bone formation, are measures primarily mediated by osteoclasts and osteoblasts.

Aging are characterised by a decline of anatomical integrity and performance across multiple organ systems and a reduced ability to respond to stress. The multisystem decline is related to increasing pathology, disease, and a higher risk of death. Among the age-related chronic diseases, osteoporosis represents a major challenge to health care services, significantly with increase in the elderly population worldwide. Indeed, additionally to fractures, osteoporosis carries a significantly risk of incapacity because of serious medical complications thus, with the aging of population, its prevalence is predicted to extend in developed

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countries and consequently its heavy medical, social, financial burdens [3,4]. Osteoporosis is also related to low level of chronic inflammation. This inflammatory state related to immunosenescence is defined as "inflammaging" and is characterised by the generation of oxidative stress mediators (including AGEs) and pro-inflammatory cytokines. AGEs accumulate within the bone tissue, because of its low turnover and also the content of long life-time proteins, like albuminoid. The modification of those proteins by AGEs is clearly involved in the development of osteoporosis. AGEs might also directly increase osteoporosis via their binding to the particular receptor RAGE expressed on bone and inflammatory cell. Thus, osteoblast and osteoclast differentiation, maturation, and performance can be directly influenced by AGEs. Particularly, the compounds were shown to lower the capability of osteoblasts to form normal bone and increase osteoclastogenic potential.

Although scientific evidence extra studies, the use of potent antiosteoporotic medicine (such as BP) needs to be carefully evaluated in clinical conditions characterised by AGE accumulation. In fact, the low bone turnover because of osteoclastogenesis suppression may paradoxically favor the institution of aerophilous harm and chronic inflammation which probably worsening (at least within the 1st days of treatment) the bone tissue stability [5]. We believe that a better understanding of molecular mechanisms by which AGEs trigger impairment in bone operate and structure may enable the identification of more selective treatments to stop and treat adverse "Inflammaging" in osteoporosis.

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