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Short Communication

The Relationship between Vitamin E and Osteoporosis

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DESCRIPTION

Osteoporosis is a bone disease, characterized by low bone mass and an inflated risk of fractures. It's well accepted that osteoporosis is caused by varied endocrine, metabolic, and mechanical factors. However, recently, there are opinions that there is also an inflammatory part within the etiology of osteoporosis. There's much evidence linking inflammation to osteoporosis. Epidemiological studies have known higher incidence of osteoporosis in varied inflammatory conditions like spondylitis, rheumatoid arthritis, and systemic lupus. This association was conjointly determined clinically whereby the degree of osteoporosis was equivalent to the extent of inflammation. If the inflammation was general, bone loss can occur locally at that site of inflammation. Elderly patients are additional at risk of osteoporosis, and this was believed to be connected to the elevated production of proinflammatory cytokines with aging [1].

The prevalence of inflammation is indicated by the presence of inflammatory markers like cytokines and C-reactive protein. Biochemical studies have an incontestable elevation of proinflammatory cytokines TNF-α and IL-6 in arthritic diseases like gouty arthritis, rheumatoid arthritis, and psoriatic arthritis. A relationship between inflammation and osteoporosis was seen in rheumatoid arthritis, whereby pro-inflammatory cytokines were discharged inflicting bone loss around the affected joints. The level of C-reactive protein, a sensitive marker of general inflammation, was conjointly found to be related to bone mineral density [2]. Inflammation might contribute to bone loss by affecting the bone remodeling process, favoring bone reabsorption activity by osteoclasts instead of bone formation activity by osteoblasts. Bone reabsorption is determined by the balance between 2 cytokines, Receptor Activator of Nuclear Factor κB Ligand (RANKL), and Osteoprotegerin (OPG).

RANKL is crucial for the differentiation and activation of bone cells. Higher RANKL levels were related to lower bone mineral density in men. Administration of serum RANKL to mice promoted bone cell growth and activation, resulting in osteoporosis. On the other hand, OPG antagonizes RANKL by binding with RANKL and preventing it from binding to RANK receptors. By doing that, OPG was ready to inhibit osteoclastogenesis and bone reabsorption. Macrophage colony-stimulating issue (MCSF) is another important determinant of osteoclastogenesis, however, its mechanism to modulate osteoclastogenesis remains not clear [3]. The "upstream" cytokines like IL-1, IL-6, and TNF-α and "downstream" cytokines like RANKL, OPG, and M-CSF compete for a very important role in bone remodeling. An imbalance in their bioactivity might cause bone loss and osteoporosis. Cytokines are small- to medium-sized proteins or glycoproteins with molecular weights ranging from 9 to 50,000 dalton. The biological mediator for many cells and performance at low concentrations between 10^{-10} and 10^{-5} molar.

The cytokine levels increase dramatically throughout inflammation and infection. The measurement of cytokine levels in close vicinity bones like the bone marrow is vital for studies on osteoporosis and different bone diseases. In postmenopausal women, protein production by the peripheral monocytes is related to cytokines secreted by monocytes within the bone marrow. Therefore, cytokine levels within the serum are representative of the local monocytes [4]. Stromal cells and osteoblasts produce interleukin-1, interleukin-6, and tumor necrosis factor-α. These proinflammatory cytokines also are referred to as bone-resorbing cytokines or proosteoclast cytokines as they promote bone cell differentiation and activity. Vitamin E, a potent antioxidant vitamin, was conjointly found to inhibit or suppress protein production. This vitamin E is also responsible for its ability to prevent inflammation and osteoporosis.

In human subjects and animal models, high doses of vitamin E were found to exhibit inflammatory effects by decreasing CRP and inhibiting the release of proinflammatory cytokines. These were evident during a study on patients with coronary artery disease, whereby the CRP and Tumor Necrosis Factor-α (TNF-α) concentrations were found to be considerably lowered with α-tocopherol supplementation. Since vitamin E was also found to inhibit cyclooxygenase-2 activities, it had been thought to be ready to exert anti-inflammatory and anticarcinogenic activities, particularly within the colon [5].

Since osteoporosis is related to inflammation, there is also a possibility that Vitamin E could have some anti-inflammatory

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action. COX-2 is an inducible enzyme expressed throughout inflammation. A RAW cell could be a macrophage-like cell that remodeled into preosteoclasts once RANKL is added. This suggested that vitamin E could act as an anti-inflammatory agent in protective bone against the excessive osteoclastic activity. Like tocotrienol, these anti-inflammatory drugs inhibit COX. As the activation of NFκB is joined to pro-inflammatory cytokines and inflammation, it provides evidence of the anti-inflammatory role of tocotrienol in preventing osteoporosis.

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