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Mini Review

Note on Hematological Disorders in Relation to Osteoporosis

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MINI REVIEW

Multiple myeloma is a plasma cell disorder, characterized by bone marrow infiltration with clonal plasma cells, production of monoclonal immunoglobulin and end organ damage including lytic lesions in bone with hypercalcemia, renal impairment and bone marrow involvement with anemia. The clonal proliferation results in classic lytic lesions and reduced bone mass due to increased bone resorption and decreased bone formation. Myeloma cells achieve these effects through the production of several cytokines, such as IL-6 and IL-7, leading to an increase in RANKL production by bone marrow stromal cells and an increased degradation of OPG. This results in an increased RANKL/OPG ratio, the differentiation of osteoclast precursors and enhanced bone resorption [1]. The expression of the Wnt antagonists, Dkk-1 and secreted frizzled protein-2, are increased in myeloma explaining the decrease in osteoblastogenesis through an inhibition of Wnt signaling.

The net effect of these changes is decreased bone formation and increased bone resorption, leading to osteoporosis. Bisphosphonates and targeted therapies for myeloma help reduce osteolysis and the risk of fractures. Denosumab may be an alternative for the prevention of skeletal-related complications in patients with multiple myeloma with impaired renal function since the drug is not metabolized or excreted by the kidneys.

Systemic mastocytosis is a hematologic condition commonly associated with reduced bone mass and osteoporosis, and is present in 9% of bone biopsies from men with idiopathic osteoporosis. Vertebral compression fractures are relatively common in men and women with systemic mastocytosis [2]. Spine X-rays and BMD are warranted in patients with or without skin involvement and particularly in males. Diagnosis is based on an elevated serum tryptase level and increased 24-h urine excretion of N-methylhistamine, but diagnosis requires histologic confirmation demonstrating mast cell infiltration in the bone marrow as well as the finding of c-KIT mutations.

Monoclonal Gammopathy of Uncertain Significance (MGUS) is characterized by an overproduction of monoclonal protein and an imbalance in bone remodeling that leads to diffuse bone loss. MGUS and multiple myeloma are common causes of osteoporosis associated with fragility fractures.

MGUS is characterized by a plasma cell content of <10% in the bone marrow, a Monoclonal (M) protein spike of 30 g/L and no end organ damage (absence of skeletal lytic lesions, hypercalcemia, renal insufficiency, anemia and bone lesions.

MGUS patients are at increased risk for osteoporosis, vertebral and hip fractures, and a higher prevalence of MGUS is found in patients older than 50 years of age who present with hip fractures [3]. Typically, MGUS is associated with increased bone resorption and reduced bone formation, and the Wnt antagonists, Dickkopf-1 (Dkk-1), soluble frizzled related proteins 2 and 3 and sclerostin have been associated with skeletal disease and bone loss.

Current guidelines do not recommend the use of bisphosphonates in patients with MGUS. Alendronate and zoledronic acid cause a significant increase in BMD in patients with MGUS, but no data are available on fracture risk reduction by these agents.

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