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Perspective

# Relationship between Iron Overload and Bone Loss

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## DESCRIPTION

Iron was a fundamental component involved with human physiological functions. Lack of iron can prompt bone loss and afterwards induce osteoporosis. Excess iron in the body affects osteoblasts, and gives rise to osteoporosis by restraining osteoblast proliferation and differentiation, and also osteoclastogenesis. Excess iron aggravated the impacts of ovariectomy on bone mass. Postmenopausal Osteoporosis (PMOP) is a bone metabolism disease, described by progressive bone loss following menopause. Lack of estrogen because of menopause was known to increase bone resorption and speed up bone loss. The iron impacted the bone mass just in the absence of estrogen, and the inhibition of estrogen on iron-induced osteopenia was especially relevant to bone resorption instead of bone development.

Bone markers in serum can be set on for diagnostic and monitoring purposes for PMOP patients. Serum was suggested as test material over urine because of better practicability and lower inter and intraindividual variability. Alkaline Phosphatase (ALP) was emitted by the osteoblasts and develops bone mineralization. Increased serum ALP was thought of as an indication of primarily increased osteoblasts or secondarily as a corrective reaction of increased bone resorption. Bone was made up of 17-20 wt% collagen and different components. The collagen of bone was considered to add to the toughness (energy to break) of the bone, moderating the brittleness of the mineral, and adding to bone strength. Type I collagen as the most ubiquitous collagen constitutes around 30% of all proteins in the extracellular matrix secreted by osteoblasts. PINP occurred in the serum in two structures: as intact, trimeric peptide relating to the separation product of procollagen during the synthesis of type I collagen and as monomeric peptide which was a degradation product of procollagen. Type I collagen discharges CTX into the bloodstream during bone resorption including α-CTX and isomerized β-CTX.

In this analysis of a companion of postmenopausal women, we evaluated the impact of iron overload on characteristics (counting age, long stretches of menopause, Ca, P, BMI, liver and kidney, glucose and lipid metabolism, and inflammatory reaction), BMD, TRACP, ALP, and type I collagen. We found that the relationship between iron overload and bone loss perhaps began from its role in advancing the degradation of type I collagen and afterwards exacerbated the process of bone loss, induced osteoporosis, and fracture without any problem. BMD was the foremost indicator in the clinical diagnosis of osteoporosis. However, grouping based on -values of BMD (femoral neck and lumbar spine) didn't represent the overall data of the samples.

The menopause period of Chinese women were normally around 50 years; frequently the older the case, the more the long periods of menopause and the lower the estrogen level in the body. In this manner, the average age of women with osteoporosis was bigger than normal. As the postmenopausal age increased, their bone mass diminished from normal to osteopenia to osteoporosis. The height and weight of osteoporosis patients both showed a critical downward pattern and eventually caused their BMI index to be fundamentally lower. CRP was a significant indicator of inflammatory reaction. The CRP in Bone Loss and Osteoporosis group was altogether expanded which exhibited that osteopenia or osteoporosis was related to an inflammatory reaction.

Loads of *in vitro* examinations demonstrated the way that iron salts could repress the development of hydroxyapatite crystals in an acellular and nonenzymatic model of calcification which recommended a direct inhibitory impact of iron on bone mineralization, cells, proteins, and enzyme. So the serum Fer in patients with osteoporosis fundamentally increased with the BMD critical decline. The serum iron accumulation may likewise be one of the significant factors prompting inflammatory reaction in patients with osteoporosis, as CRP increase with the rise of Fer. β-CTX and PINP were the corruption results of type I collagen. Serum Fer levels had a beneficial impact on both β-CTX and PINP. Moreover, the complete analysis showed that iron overload reduced bone density by leading to the degradation of type I collagen. The overloaded iron improved the degradation of type I collagen and is joined by bone strength reduction. Advancing collagen degradation might aggregate the process of bone loss under an estrogen-lacking environment.

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