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Opinion Article

Effect of Type 2 Diabetes on Bone Fracture

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DESCRIPTION

Type 2 diabetes is a condition in which the body's ability to control and utilize sugar (glucose) as a fuel is impaired. There will be increase in the level of sugar in the bloodstream as a result of this long-term (chronic) disease. High blood sugar levels can eventually cause problems with the circulatory, neurological, and immunological systems.

When compared to their age-matched healthy peers, men and women with Type 2 Diabetes (T2D) have normal to high Bone Mineral Density (BMD). This observation is likely due to a number of variables. Low Body Mass Index (BMI) is a known risk factor for the development of osteoporosis and fractures, and BMD is directly linked to body weight. When compared to age-matched controls, people who are overweight, such as those with T2D, will have a greater BMD. Increased BMD in people with T2D could be a result of biomechanical adaptation to elevated skeleton loads. In T2D, lean mass, which is generally increased in proportion to weight growth, puts biomechanical pressure on bones and can accelerate bone production. Insulin promotes bone formation, and the hyperinsulinemic episodes associated with T2D may aid in this process. It's worth noting that the enhanced BMD found in men and women with T2D contrasts sharply with the minimal BMD found in people with Type 1 Diabetes (T1D). T1D is an autoimmune disease characterized by insulin insufficiency, which is frequently accompanied by nutritional deficiencies, low body weight, and other variables that affect bone formation and lower BMD.

A higher risk of fracture is linked to Type 2 Diabetes (T2D). This may seem counterintuitive, given that persons with T2D have higher bone mineral density than people without the disease. Hyperglycemia, on the other hand, is linked to lower levels of circulating bone turnover markers, and histomorphometry shows that patients with T2D have less bone formation than people without the disease. The increased risk of fracture has thus been postulated to be attributable to the development of microfractures produced by inadequate bone turnover. Sclerostin, a bone-forming inhibitor produced by the Wnt pathway, has been found to be higher in T2D patients than in controls. Hyperglycemia stimulates the production and release of sclerostin in osteocytes, which may limit bone growth and indirectly bone resorption, according to *in vitro* studies.

Histomorphometry revealed that mice with T2D have less bone turnover, and the osteocytes in these bone samples showed pro-inflammatory alterations when compared to non-diabetic mice. Changes in blood glucose levels can alter bone turnover either directly or indirectly. Hyperglycemia stimulates the production and release of sclerostin in osteocytes, which may limit bone growth and indirectly bone resorption, according to *in vitro* studies. The levels of bone turnover indicators are inversely related with plasma glucose levels in diabetic patients.

The plasma glucose level in diabetics might fluctuate throughout the day, and this phenomenon of glycemic variability may influence the levels of bone turnover markers; however, the association between glycemic variability and bone turnover has not been explored. Bone turnover marker levels and glycemic variability may be influenced by physical activity.

Glycemic regulation for end-organ protection has significant consequences for the skeleton, effectively reducing falls and renal osteodystrophy. Vitamin D deficiency should be tested in both men and women with T2D. To avoid osteomalacia and subsequent hyperparathyroidism, vitamin D should be supplemented as needed. The need of a calcium- and vitamin-D-rich diet, as well as weight-bearing exercise, should be discussed with T2D patients, as it is with individuals with metabolic bone disease. Medications that may have a negative influence on the skeleton should be prescribed with prudence, and the risks and advantages of starting the drug should be carefully assessed. There is no evidence in the literature that using antiresorptive or anabolic medication to prevent future fractures in people with T2D and normal BMD will prevent future fractures.

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