PREVALENCE OF OSTEOPOROSIS IN ADULT PATIENTS WITH TYPE 1 DIABETES MELLITUS AND APPROACHES TO OPTIMIZING ITS TREATMENT

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Annotation: This article examines the prevalence of osteoporosis in adult patients with Type 1 diabetes mellitus and discusses modern approaches to improving its treatment. The relationship between metabolic disorders, decreased bone mineral density, and fracture risk is analysed. Particular attention is given to early diagnosis, preventive strategies, and comprehensive therapeutic management.

Key words: Type 1 diabetes mellitus, osteoporosis, bone mineral density, fracture risk, treatment optimization.

Type 1 diabetes mellitus (T1DM) is a chronic endocrine disease characterised by autoimmune destruction of pancreatic beta cells, resulting in absolute insulin deficiency and long-term metabolic disturbances. Although the disease is mainly associated with complications affecting the cardiovascular, nervous, renal, and visual systems, increasing scientific attention has recently been directed toward skeletal complications, particularly osteoporosis. Osteoporosis is a systemic metabolic bone disorder characterised by decreased bone mineral density (BMD), deterioration of bone microarchitecture, and increased fragility leading to a higher risk of fractures. In adult patients with Type 1 diabetes mellitus, osteoporosis has become an important clinical and public health concern due to its high prevalence and serious consequences. The relationship between T1DM and osteoporosis is complex and multifactorial. Chronic hyperglycaemia negatively affects bone metabolism through several mechanisms, including impaired osteoblast activity, increased oxidative stress, accumulation of advanced glycation end products, and disruption of calcium homeostasis. These changes lead to reduced bone formation and deterioration of bone quality. In addition, insulin itself

plays an anabolic role in bone metabolism; therefore, insulin deficiency contributes directly to decreased bone mass. Several epidemiological studies have demonstrated that adults with Type 1 diabetes have lower bone mineral density compared to healthy individuals of the same age and sex. Furthermore, patients with T1DM are at significantly higher risk of fragility fractures, especially hip and vertebral fractures. Such fractures often result in disability, reduced quality of life, and increased mortality rates. The burden of osteoporosis in diabetic patients therefore extends beyond musculoskeletal complications and creates substantial socioeconomic consequences. Age, duration of diabetes, glycaemic control, body mass index, physical inactivity, smoking, vitamin D deficiency, and diabetic complications are considered major risk factors for osteoporosis in patients with T1DM. Long-standing diabetes is particularly associated with progressive skeletal deterioration. Moreover, diabetic nephropathy and neuropathy may indirectly increase fracture risk by affecting mineral metabolism and increasing the likelihood of falls. Despite the growing recognition of diabetic osteoporosis, the condition often remains underdiagnosed and undertreated in clinical practice. In many healthcare systems, screening for bone health is not routinely included in diabetes management protocols. As a result, osteoporosis is frequently identified only after fractures occur. Early detection through bone mineral density assessment and laboratory evaluation is therefore critically important.

Modern treatment strategies for osteoporosis in patients with Type 1 diabetes mellitus involve a comprehensive approach combining pharmacological therapy, lifestyle modification, nutritional support, and glycaemic optimisation. Adequate calcium and vitamin D intake, regular weight-bearing exercise, smoking cessation, and proper metabolic control play central roles in prevention and treatment. Pharmacological agents such as bisphosphonates, denosumab, and anabolic therapies may also be considered in high-risk individuals. The growing prevalence of diabetes worldwide highlights the necessity of improving preventive and therapeutic approaches to diabetic osteoporosis. Understanding the

mechanisms underlying skeletal complications in T1DM is essential for developing effective strategies aimed at reducing fracture risk and improving patient outcomes. Therefore, the present article aims to analyse the prevalence of osteoporosis among adult patients with Type 1 diabetes mellitus and evaluate modern approaches to optimising its treatment and prevention. Osteoporosis in patients with Type 1 diabetes mellitus develops as a result of multiple interconnected metabolic, hormonal, and structural abnormalities affecting bone tissue. Unlike postmenopausal osteoporosis, diabetic osteoporosis is characterised not only by reduced bone mineral density but also by impaired bone quality and altered bone microarchitecture. These pathological changes significantly increase fracture susceptibility even in patients with relatively preserved BMD values. One of the principal mechanisms contributing to bone deterioration in T1DM is chronic hyperglycaemia. Persistent elevation of blood glucose levels leads to accumulation of advanced glycation end products (AGEs) in bone collagen. AGEs impair collagen elasticity and reduce bone strength, making skeletal tissue more fragile. Furthermore, AGEs stimulate oxidative stress and inflammatory pathways that negatively affect osteoblast differentiation and activity. Insulin deficiency also plays a major role in the pathogenesis of osteoporosis in T1DM. Insulin is considered an anabolic hormone for bone tissue because it promotes osteoblast proliferation and collagen synthesis. In patients with Type 1 diabetes, lack of endogenous insulin leads to decreased bone formation and impaired skeletal development. This effect is especially important in young adults whose peak bone mass may not be adequately achieved. Another important factor is chronic inflammation associated with autoimmune processes in T1DM. Pro-inflammatory cytokines such as tumor necrosis factor-alpha and interleukin-6 stimulate osteoclast activity, increasing bone resorption. Excessive osteoclast activation disrupts the balance between bone formation and bone breakdown, ultimately leading to progressive bone loss. Vitamin D deficiency is frequently observed in diabetic patients and contributes significantly to osteoporosis development.

Vitamin D plays a fundamental role in calcium absorption and bone mineralisation. Insufficient vitamin D levels impair calcium homeostasis, resulting in secondary hyperparathyroidism and increased bone resorption. Moreover, diabetic nephropathy may worsen vitamin D metabolism due to impaired renal activation of vitamin D.

Calcium metabolism abnormalities are also common in T1DM. Hyperglycaemia-induced osmotic diuresis can increase urinary calcium excretion, reducing calcium availability for bone mineralisation. Over time, chronic calcium loss contributes to skeletal demineralisation and decreased bone density. Diabetic complications additionally influence fracture risk. Peripheral neuropathy affects balance and coordination, increasing the probability of falls. Retinopathy impairs visual function and may further predispose patients to traumatic injuries. Diabetic nephropathy contributes to disturbances in mineral metabolism and bone turnover. Consequently, skeletal fragility in T1DM is not solely related to reduced BMD but also to increased fall risk and impaired bone quality. Hormonal disturbances associated with diabetes may further aggravate osteoporosis. Low levels of insulin-like growth factor-1 (IGF-1), hypogonadism, and altered cortisol metabolism negatively influence bone turnover. In women with poorly controlled diabetes, menstrual irregularities may contribute to reduced estrogen levels and accelerated bone loss. Physical inactivity is another important contributing factor. Many diabetic patients limit physical activity due to fear of hypoglycaemia or complications. Reduced mechanical loading decreases osteoblast stimulation and weakens bone structure. Conversely, regular exercise has been shown to improve bone strength and metabolic control. Nutritional factors also influence skeletal health in diabetic patients. Inadequate intake of calcium, magnesium, protein, and micronutrients may impair bone formation. Smoking and alcohol consumption further exacerbate bone loss by negatively affecting osteoblast function and calcium absorption.

Osteoporosis has become an increasingly recognised complication among adults with Type 1 diabetes mellitus. Numerous epidemiological studies conducted over recent decades have confirmed that individuals with T1DM demonstrate significantly lower bone mineral density and a considerably higher risk of fractures compared to the non-diabetic population. The prevalence of osteoporosis among diabetic patients varies depending on age, sex, duration of disease, metabolic control, and associated diabetic complications. However, most clinical investigations indicate that osteoporosis occurs more frequently and at an earlier age in patients with T1DM than in healthy individuals. The global increase in diabetes prevalence has simultaneously contributed to a rise in diabetes-associated skeletal disorders. According to recent clinical observations, adults with Type 1 diabetes are approximately four to six times more likely to develop hip fractures than people without diabetes. Vertebral fractures and peripheral skeletal fractures are also significantly more common in this patient group. Importantly, fracture healing in diabetic individuals is often slower and associated with increased complications, further worsening patient outcomes and quality of life. Age is one of the most important determinants influencing osteoporosis prevalence in T1DM patients. Bone mineral density naturally decreases with aging; therefore, elderly diabetic individuals are particularly vulnerable to skeletal deterioration. Nevertheless, osteoporosis may also develop in younger adults with long-standing diabetes because insulin deficiency and chronic metabolic disturbances affect bone metabolism throughout life. Disease duration is another major risk factor. Patients who have lived with Type 1 diabetes for many years often demonstrate more severe skeletal changes. Chronic hyperglycaemia progressively damages bone microarchitecture and reduces bone turnover efficiency. Long-term exposure to elevated glucose levels therefore contributes to cumulative bone loss and increased fracture susceptibility. Poor glycaemic control is strongly associated with osteoporosis progression. Elevated HbA1c levels indicate persistent hyperglycaemia, which promotes formation of advanced glycation end products

and oxidative stress within bone tissue. Several studies have shown that patients with poorly controlled diabetes have significantly lower bone mineral density compared to patients maintaining stable metabolic control. Early diagnosis and comprehensive management of osteoporosis in adult patients with Type 1 diabetes mellitus are essential for reducing fracture risk, improving quality of life, and preventing long-term disability. Since diabetic osteoporosis develops gradually and may remain asymptomatic for many years, timely screening and preventive strategies play a critical role in clinical practice. Modern treatment approaches involve both pharmacological and non-pharmacological interventions. Optimisation of glycaemic control remains one of the central therapeutic goals. Stable blood glucose levels reduce oxidative stress, minimise formation of advanced glycation end products, and improve bone metabolism. Effective insulin therapy therefore indirectly contributes to preservation of bone density. Adequate intake of calcium and vitamin D is essential for maintaining bone mineralisation. Many patients with Type 1 diabetes demonstrate vitamin D deficiency, which negatively affects calcium absorption and skeletal strength. Nutritional correction and supplementation can significantly improve bone metabolism and reduce osteoporosis progression. Physical activity also plays a fundamental role in osteoporosis management. Regular weight-bearing and resistance exercises stimulate osteoblast activity, improve muscle strength, and enhance skeletal stability. Exercise additionally improves insulin sensitivity and metabolic control, creating dual benefits for diabetic patients. Preventive strategies are equally important in reducing osteoporosis prevalence among diabetic patients. Smoking cessation, limitation of alcohol consumption, healthy nutrition, and maintenance of normal body weight contribute significantly to bone protection. Early identification of high-risk individuals through screening programmes allows timely intervention before fractures occur. Overall, successful management of osteoporosis in adult patients with Type 1 diabetes mellitus requires a multidisciplinary and comprehensive approach combining early diagnosis, metabolic optimisation,

lifestyle modification, pharmacological treatment, and preventive care. Integrated therapeutic strategies can significantly reduce fracture risk and improve long-term clinical outcomes in this vulnerable patient population.

Conclusion

Osteoporosis is a significant and increasingly recognised complication in adult patients with Type 1 diabetes mellitus. Chronic hyperglycaemia, insulin deficiency, metabolic disturbances, and diabetic complications contribute to decreased bone mineral density and increased fracture risk. Early diagnosis through bone density assessment and laboratory investigations is essential for timely intervention and prevention of severe skeletal complications. Comprehensive treatment approaches including optimal glycaemic control, adequate calcium and vitamin D intake, regular physical activity, and pharmacological therapy can significantly improve bone health and reduce fracture incidence. Preventive strategies and patient education also play an important role in long-term disease management. Overall, integrated multidisciplinary care is necessary to optimise osteoporosis treatment in patients with Type 1 diabetes mellitus and improve their quality of life.

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