**NOVATEUR PUBLICATIONS**
**JournalNX- A Multidisciplinary Peer Reviewed Journal**
**ISSN No: 2581 - 4230**
**VOLUME 10, ISSUE 9, September - 2024**

# NEGATIVE EFFECTS OF DIABETES ON THE BONE SYSTEM

Yoldosheva Roila Jumayevna
Karshi State University Teacher of the Department of Physiology,
Faculty of Chemistry and Biology
Phone: 998 912200569

**Abstract**

The recent increase in the prevalence of diabetes mellitus has made it a major chronic disease that poses a serious threat to human health. The prevalence of osteoporosis has increased significantly in patients with diabetes. Diabetic bone disease is secondary osteoporosis caused by diabetes mellitus. Patients with diabetic bone disease have variable levels of bone loss, low bone mineral density, degradation of bone microarchitecture, and increased bone fragility with the progression of diabetes mellitus, which increases their risk of fracture and reduces the ability to heal after fracture.

**Keywords**: diabetes, bone, osteoporosis, microarchitecture, disease.

## Introduction

Diabetes mellitus is derived from the Greek word diabetes and Latin mellitus meaning sweet. A review of history shows that the term “diabetes” was first used by Apollonius of Memphis in 250-300 BC. Ancient Greek, Indian and Egyptian civilizations discovered the sweet nature of urine in this condition and hence the term Diabetes Mellitus was coined. Mering and Minkowski discovered the role of the pancreas in the pathogenesis of diabetes in 1889. In 1922, Banting, Best and Collip purified the hormone insulin from the pancreas of cows at the University of Toronto, which led to the availability of an effective treatment for diabetes in 1922. Over the years, great work has been done and many discoveries as well as management strategies have been made to address this growing problem. Unfortunately, even today, diabetes is one of the most common chronic diseases in the country and around the world. It remains the seventh leading cause of death in the United States[1].

With age, changes in lifestyle habits and changes in the structure of the diet in humans, diabetes mellitus has become the third most dangerous non-infectious disease after cardiovascular diseases and malignant tumors. Diabetes mellitus is a group of metabolic disorders characterized by chronic hyperglycemia caused by multiple etiologies that are accompanied by insufficient insulin secretion and impaired Effect [1] . People with diabetes may have an increased risk of osteoporosis. This is a disease that causes thin and weakened bones and is more likely to break. Osteoporosis the most common bones include the spine, forearms, ankles.

Osteoporosis is often referred to as a “silent disease” because a person does not notice that his bones have become thinner. Many people do not know that there is osteoporosis until the bone is broken. Osteoporosis patients often break bones when falling from a height while standing, as a result of a simple fall. People with diabetes may have more bone loss, in diabetes it is important to keep blood glucose levels as close to normal as possible.

**Type 1 diabetes:**

Bone-forming cells do not work as well as in people without diabetes. For these reasons, bone mass may be low and the risk of bone fractures may be high[1,2] .

**Type 2 diabetes:**

Patients with Type 2 diabetes mellitus (T2DM) have an increased risk of fragility fractures, despite increased body weight and normal or high bone mineral density. The mechanisms by which T2dm increases skeletal fragility are unclear. Perhaps the combination of factors such as the risk of miscarriage, regional osteopenia and impaired bone quality contributes to an increased risk of fractures. Medications to treat T2DM can also affect the risk of fractures. Thiazolidinediones, for example, accelerate bone loss and increase the risk of fractures, especially in older women. On the contrary, metformin and sulfonylureas do not have a negative impact on bone health and can actually protect against fragility fractures. Animal models show a potential role for incretin hormones in bone metabolism, but there is only limited data on the effects of dipeptidyl peptidase-4 inhibitors and glucagon-like peptide-1 agonists on bone health in humans. Animal models have also demonstrated the role of amylin in bone metabolism, but clinical studies in patients with Type 1 diabetes with an amylin analogue (pramlintide) have not significantly affected bone metabolism. The effects of Insulin treatment on fracture risk are incompatible with some studies showing increased risk, while others have no effect. Finally, although there is limited data on the latest class of drugs for the treatment of T2DM, sodium-glucose joint carrier-2 inhibitors, these drugs do not increase the risk of fractures. Since diabetes is an increasingly common chronic condition that can affect patients for decades, further research on the effects of remedies for the treatment of t2dm on bone metabolism is guaranteed[1,2,3]
Even if the bone mass is the same, there may be a higher risk of bone fractures than people without diabetes. The risk associated with complications of diabetes can increase, such as altered vision or neuropathy (insomnia, itching or pain in the legs).
Blood pressure is very important in the development of diabetic kidney disease. Albuminuria only manifests as a decrease in Nocturnal diastolic blood pressure[1,2,3]

**Hyperglycemia**

Hyperglycemia is the most obvious clinical picture of diabetes, and excess free sugar in the body negatively affects many tissues and cells. High levels of extracellular free sugars have a direct inhibitory effect on cellular activity of osteoblasts and Osteocytes. Osteoblasts exposed to high glucose levels decrease proliferation capacity, slow extracellular matrix synthesis, and subsequently slow maturation and mineralization. Thus, the hyperglycemic environment is harmful for bone formation and maintenance of bone mass. In contrast, osteoblasts show increased apoptosis and accelerated aging in high glucose environments. The osteocyte lacuno-canalicular system formed by osteoblasts develops microstructure disorders associated with diabetes; as a result, abnormal bone remodeling and changes in mechanical properties ultimately accelerate the progression of diabetic bone disease. In addition, the high glucose environment promotes the activation of osteoclasts and increases their bone resorption function. The final products of complex glycation (AGE) are formed by non-enzymatic glycosylation reactions between aldehyde groups of reversible sugars and amino acids, including carboxymethyl lysine, pentosidine, and acetonide. Abnormal accumulation of ages can be associated with a decrease in

the mechanical properties of the cortical bone, and abnormal interconnection of Ages with collagen in the bone matrix leads to a decrease in collagen elasticity, resulting in increased bone fragility. In addition, AGEs can inhibit osteoblast differentiation and reduce alkaline phosphatase expression, thereby reducing their bone-forming effect.

**Biomechanics**

One of the main functions of bone in the human body is mechanical support and protection. All bones perform these functions by bearing a variety of loads in various combinations, including compression, tension, bending, and twisting. Since bone is a dynamic tissue, it responds to external and internal mechanical stimuli, which in turn affects bone regeneration and the overall quality of its tissue. The structure of the bone, its type and the magnitude of the applied load affect its effect on these forces . Specifically, there are two types of bone: cortical or compact bone is denser, and trabecular or cancellous bone has more porosity and a complex structure of trabeculae [8]. Both types of bones respond very differently to forces. Trabecular bone is mainly found in areas that require efficient load distribution, such as joint regions and the body of vertebrates. On the other hand, cortical bone is found in areas that require strong structural support, such as the outer axis of long bones.

**Conclusion**

Diabetes mellitus negatively affects the strength of the composite to bind to dentin. 2. Type 1 diabetes mellitus significantly reduces the cutting strength of composite resins to dentin. 3. Regardless of whether the participants were diabetic or not, the shear bond strength of the universal adhesive was high when applied to dentin using self-etch mode compared to the general etch mode in all groups

**References**:

1.Diabetes, Amit Sapra; Priyanka Bhandari, state Pearls, 2024-January
2. "A narrative review of diabetic bone disease: Characteristics, pathogenesis, and treatment" , Bo Wu, Zhaoyu Fu, Xinyu Wang, Pengcheng Zhou, Qifan Yang, Ye Jiang, and Dong Zhucorresponding author * Front Endocrinol (Lausanne). 2022; 13: 1052592. Published online 2022 Dec 14. doi: 10.3389/fendo.2022.1052592
3.Osteoporosis in diabetes mellitus: Possible cellular and molecular mechanisms, Kannikar Wongdee and Narattaphol Charoenphandhu, World J Diabetes. 2011 Mar 15; 2(3): 41–48. Published online 2011 Mar 15. Doi: 10.4239/wjd.v2.i3.41PMCID: PMC3083906PMID: 21537459
4. The Effect of Type 2 Diabetes on Bone Biomechanics, Lamya Karim, PhD,1 Taraneh Rezaee, MS,1 and Rachana Vaidya, MS1Curr Osteoporos Rep. Author manuscript; available in PMC 2020 Oct 1.Published in final edited form as:Curr Osteoporos Rep. 2019 Oct; 17(5): 291–300. Doi: 10.1007/s11914-019-00526-w PMCID: PMC6819250NIHMSID: NIHMS1536849PMID: 31392668