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Review Article

Antidiabetic Agents and Bone Quality: A Focus on Glycation End Products and Incretin Pathway Modulations

Abstract

Diabetes mellitus is associated with inadequate bone health and quality and heightened susceptibility to fractures, even in patients with normal or elevated bone mineral density. Elevated advanced glycation end-products (AGEs) and a suppressed incretin pathway are among the mechanisms through which diabetes affects the bone. Accordingly, the present review aimed to investigate the effects of antidiabetic medications on bone quality, primarily through AGEs and the incretin pathway. Google Scholar, Cochrane Library, and PubMed were used to examine related studies until February 2024. Antidiabetic medications influence AGEs and the incretin pathway directly or indirectly. Certain antidiabetic drugs including metformin, glucagon-like peptide-1 receptor agonists (GLP-1RA), dipeptidyl-peptidase-4 (DDP-4) inhibitors, α -glucosidase inhibitors (AGIs), sodium-glucose co-transporter-2 inhibitors, and thiazolidinediones (TZDs), directly affect AGEs through multiple mechanisms. These mechanisms include decreasing the formation of AGEs and the expression of AGEs receptor (RAGE) in tissue and increasing serum soluble RAGE levels, resulting in the reduced action of AGEs. Similarly, metformin, GLP-1RA, DDP-4 inhibitors, AGIs, and TZDs may enhance incretin hormones directly by increasing their production or suppressing their metabolism. Additionally, these medications could influence AGEs and the incretin pathway indirectly by enhancing glycemic control. In contrast, sulfonylureas have not demonstrated any obvious effects on AGEs or the incretin pathway. Considering their favorable effects on AGEs and the incretin pathway, a suitable selection of antidiabetic drugs may facilitate more protective effects on the bone in diabetic patients.