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A Comparative Study of Insulin Resistance, Meta Inflammatory, and Bone Turnover Markers in Postmenopausal Women with Osteoporosis and Type 2 DM Patients

Atyaf Zuhair Mansoor Alali, Adnan Jassim Mohammed Al-Fartosy*

Department of Chemistry, College of Science, University of Basrah, AL wofod St., Basra, Iraq

ABSTRACT

Background: Insulin resistance plays a central role in the pathophysiology of T2DM and may critically influence bone metabolism. This study aimed to explore the comparative levels of insulin resistance with Meta inflammatory and bone turnover markers in type 2 diabetic women with osteoporosis. Methods: In a cross-sectional study, 50 women with T2DM and osteoporosis and 40 healthy controls, aged 45 to 75 years, were evaluated for bone turnover markers using dual-energy X-ray absorptiometry (DXA) scans at the spine and hip. T-score of ≤−2.5 was defined as osteoporosis, and score −2.49 to −1.0 as osteopenia at either site. Correlation of Insulin resistance with inflammatory cytokines, Wnt pathway inhibitors, bone turnover markers, minerals, and vitamin D was evaluated. Diagnostic performance was assessed using ROC curve analysis. Results: Non-significant changes ($p>0.05$) were seen in the level of BMI. HOMA-IR, Insulin, HbA1c, TNF-α, IL-6, ALP, DKK1, SOST, CTSK, and PTH were significantly increased ($p<0.01$), while vit. D, Osteocalcin, Ca, Phosphorus, Mg, and T-score were significantly decreased ($p<0.01$) in patients with T2DM and osteoporosis, compared to controls. The HOMA-IR displayed excellent diagnostic accuracy and correlated with inflammatory markers (TNF-α and IL-6), Wnt signaling inhibitors (DKK-1 and sclerostin), changes in bone turnover markers and disturbed mineral homeostasis. This link points to insulin resistance as an integral link between metabolic derangement and skeletal fragility. Insulin resistance is a key central pathophysiological link between metabolic dysregulation, chronic inflammation, impeded bone remodeling and osteoporosis in T2DM. Integrating insulin resistance assessment with bone-related biomarkers may enhance early diagnosis and risk stratification.

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1. Introduction

Osteoporosis (OP) is a common bone disease in the world as it involves low BMD and the gradual erosion of bone tissue, leading to bone fragility. Diabetes mellitus (DM) is a metabolic disease comprising increased blood glucose levels as a result of insulin resistance, lowered insulin secretion or both metabolic pathways (Lecka-Czernik, 2017). About 90% of diabetes cases globally occur in type 2 DM (T2DM), a hyperglycemia state (Awad et al., 2015). Low BMD is present in type 1 DM individuals and high or average BMD in type 2DM if analyzed by dual energy X-ray absorptiometry (DXA) (Napoli et al., 2014; Tariq et al., 2020). Separate sources of fracture risk in T2DM: sarcopenia, peripheral neuropathy related balance impairment, diabetic retinopathy, and specific drugs prescribed for diabetes (Classification & Diabetes, 2020). Osteoblast proliferation and differentiation are promoted through insulin and insulin-like growth factor-1 that have anabolic repercussions on bone health. Thus, insulin-mediated signaling resistance suppresses bone formation ability by bone function and restructures bone remodeling processes. In addition, insulin resistance facilitates chronic, low-grade inflammation and oxidative stress while affecting endocrine pathways (Hardy & Cooper, 2009; Ibrahim & Abdelghani, 2018) that together, lead to skeletal degeneration over the course of time. Insulin resistance not only acts directly on osteoblastic activity it maintains this chronic low-grade inflammation, oxidative stress and endocrine dysregulation in motion and continues intact. These disturbances increase bone loss via osteoclastogenesis and suppression of osteoanabolic

CONTACT Adnan Jassim Mohammed Al-Fartosy: adnan.jassim@uobasrah.edu.iq