

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

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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

pathways. Inflammatory cytokines, inhibitors of Wnt/ β -catenin pathway, up-regulation of bone turnover markers, and mineral metabolism have been suggested to contribute to the etiology of osteoporosis in diabetes patients ([Classification & Diabetes, 2020](#)). The comparative relationship of insulin resistance to these diverse biomarkers has largely not been well characterized, though. Therefore, this study is to estimate the relation of insulin resistance with various types of biomarkers of diabetic Osteoporosis.

2. Methods

2.1 Study Design and Population

In this study, women aged between 40 to 65 years with T2DM diagnosed and treated in the Diabetic and Endocrine Glands Center in Al-Mawany Teaching Hospital and Al-Fayhaa Teaching Hospital in Basra Province, in Iraq, between September 2025 and January 2026. Another group was sampled from private clinics operated by Dr. Mohamed Hassan from Abu-Al-Khaseeb General Hospital.

2.2 Inclusion Criteria

Fifty menopausal women with a diagnosis of T2DM and osteoporosis. Forty healthy female volunteers.

2.3 Exclusion Criteria

Patients who smoke. Individuals receiving anti-osteoporotic or hormonal interventional therapies altering bone metabolism for more than 6 months. People with Type 1 diabetes mellitus, acute infections, and diabetic crises. 4. Those with advanced heart failure, pulmonary diseases or hepatorenal insufficiency. In severely sick cases, the thyroid is dysfunctional, tumors are malignant and other BMD-related conditions are present. Diagnosis of T2DM followed the American Diabetes Association's guidelines ([Ezeldein & Zaky, 2023](#)). Guidelines from the World Health Organization to evaluate the severity of osteoporosis with regard to T-scores achieved by dual-energy X-ray absorptiometry were followed. Written informed consent was obtained from all women participants, based in Basrah province, who participated in the study in accordance with the Helsinki Declaration.

3. Sample Collection Procedure

Blood samples were taken after a 12-h fast and 30 min recovery lie supine, 9:00 AM-10:00 AM. In healthy volunteers and those with osteoporotic T2DM, there is a total of 5 mL of blood collected from the forearm veins. Blood was deposited into EDTA-containing polypropylene tubes and 1 mL was collected to measure HbA1c concentrations before centrifugation on a gel tube (402 × g) for twenty minutes, which was used for serum harvesting. Also, immediately extracted serum was performed for all study parameters, and serum from it was stored in a plain tube, at -70°C until required.

4. Biochemical Analysis

4.1 Methods Used to Analyze the Biochemical Data of Blood Samples Included

- Body Mass index (BMI) was assessed [$\text{BMI (kg/m}^2\text{)} = \text{Weight in kg / Height in m}^2\text{}$] ([Abdul-Redhalsmaiel, 2022](#)).
- HbA1c levels were analyzed with Bio-Rad D-10® HPLC (Bio-Rad Laboratories, CA, USA).
- The serum glucose was analyzed by UV-Vis Spectrophotometer (UV-EMC-LAB, Duisburg, Germany) using Randox kit GL2279/UK.
- The insulin level was measured with ELISA kit with solid phase sandwich technique (BT Lab, Shanghai-E0010Hu/China) with different values of Cathepsin K, Interleukin-6, Sclerostin, Osteocalcin, Dickkopf-1, TNF- α were determined as per the respective kits (BT-Lab).
- Serum calcium level was determined by an automatic method, with Roche kit (Germany). Phosphorus (second Roche kit) levels and magnesium (IFU kit from Italy) were also measured.
- Roche's kits were accessed as the standard of alkaline phosphatase, Linear's from Spain as the standard of parathyroid hormone and Intron's from Korea as the standard of vitamin D.
- Fasting insulin resistance (IR) was analyzed by Applying the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) formula: $\text{HOMA-IR} = (\text{Fasting insulin (}\mu\text{IU/mL)} \times \text{Fasting glucose (mg/dL)}) / 405$.

5. Statistical Analysis

SPSS was used for statistical analysis with software package (IBM Corporation, New York, USA). Group analyses for

Significance differences were analyzed using One-way ANOVA for group analyses with one-way ANOVA. The Receiver Operating Characteristic curve showed sensitivity to one minus specificity and area under the curve (AUC) for this function. We can also estimate sensitivities and specificities with 95% CI. A $p < 0.05$ was regarded as statistically significant, a $p < 0.01$ as highly statistically significant and an AUC value on either of the extremities as reasonable diagnostic ability, thus drawing the conclusion that different groups are diagnostic. Osteocalcin ($p < 0.01$) and T-score ($p < 0.01$) were significantly but negatively correlated with clinical parameters of patients with osteoporosis and type two diabetes. Furthermore, HOMA-IR showed strong positive correlations with TNF- α , IL-6, Dkk-1, SOST, and CTSK and very significant values ($p < 0.01$) in postmenopausal women with osteoporosis and Type Two Diabetes Mellitus. Findings of the AUC analysis reveal a measure of biomarkers including HOMA-IR, Insulin, HbA1c, Glucose, TNF- α , IL-6, ALP, Dkk-1, SOST, CTSK and PTH that could have a predictive value (AUC=0.97, 0.93, 1.00, 1.00, 0.95, 0.99, 0.90, 0.96, 0.97, 0.86 respectively). Conversely, VIT. D, Osteocalcin, Ca, Phosphorus, Mg and T-score Levels, that all show suspected negative predictive biomarkers for T2DM and osteoporosis patients (AUC=-0.96, -1.00, -0.82, -0.96, -0.70, -1.00 respectively), as displayed in Figure II and Table IV.

Table 1: Demographic Factors for the Current Study

The Characteristics		Patients	Healthy Control
Total subject No.		50	40
Age (Years) (Mean $\pm$ SD)		60.22 $\pm$ 7.70	59.10 $\pm$ 7.92
T2DM Duration (Years) (Mean $\pm$ SD)		10.09 $\pm$ 2.00	-
Osteoporosis Duration (Years) (Mean $\pm$ SD)		3.68 $\pm$ 1.30	-
Demographic Area	Urban	43	45
	Rural	7	5
Educational Background	Learned	38	20
	Illiterate	12	20
Smoking Habits	Positive	0	0
	Negative	50	40
Food Habits	Vegetarian	1	2
	Non -Vegetarian	49	38
Employment Status	Employed	30	25
	Non-Employed	20	15

Data are presented as mean $\pm$ SD, SD: Standard Deviation

Table 2: Levels of all Parameters in Osteoporosis and Type 2 Diabetes Patients and the Control Group. The Values are the Mean $\pm$ SD.

Parameters	Osteoporosis and type 2 diabetes patients No = 50						Healthy Control No = 40	
	Mean ± SD	Median	SE	Range	95% CI			Mean ± SD
					Lower	Upper		
BMI (kg/m²)	31.23 ± 3.17	31.04	0.52	24.03-40.27	30.17	32.29	32.02±5.08	
HbA1c (%)	8.86±1.47**	8.6	0.20	7.5-15.0	8.44	9.28	5.13±0.47	
Glucose (mg/dL)	227.60±40.01**	225	5.66	160-310	216.23	238.9	98.28± 9.89	
Insulin(uIU/mL)	16.64±5.60**	16.6	0.79	6.24-29.11	15.05	18.24	7.78 ± 2.12	
HOMA-IR	4.53± 1.28**	4.57	0.18	1.00-6.85	4.17	4.90	1.68± 0.62	
TNF-α (ng/L)	7.90± 2.53**	8.04	0.35	3.20-14.16	7.17	8.62	3.68± 0.93	
IL-6 (ng/L)	6.16± 1.78**	6.39	0.25	2.97-11.44	5.65	6.67	2.11±0.88	
ALP (U/L)	125.30± 38.17**	127.05	5.39	50.0-254.8	114.5	136.1	73.81± 16.99	
DKK1 (ng/mL)	2.58± 0.74**	2.63	0.11	0.50-4.09	2.37	2.80	1.13 ± 0.34	
SOST (ng/mL)	36.29±6.96**	36.04	0.98	22.97-54.83	34.31	38.26	19.41±5.86	
CTSK (ng/mL)	2.85± 0.96**	2.92	0.13	1.04-4.75	2.58	3.13	1.64±0.48	
PTH (pg/ml)	113.40± 36.98**	109	5.23	52-180	102.9	123.9	42.15± 13.68	
VIT.D (ng/ml)	18.67± 5.15**	19	0.72	8.5-27.5	17.22	20.15	33.93± 7.33	
Osteocalc. (ng/mL)	9.03± 2.47**	8.6	0.35	4.5-13.5	8.33	9.73	23.42±4.26	
Ca(mg/dL)	8.69± 0.49**	8.67	0.06	7.54-9.69	8.55	8.83	9.23± 0.27	
Phosphor. (mg/dL)	3.10± 0.50**	3.2	0.07	2.00-3.80	2.96	3.24	4.20± 0.42	
Mg (mg/dL)	1.99±0.26**	1.97	0.03	1.50-2.44	1.92	2.07	2.18± 0.23	
T-score	-2.98± 0.31**	-2.90	0.04	-3.6-(-2.5)	-3.1	-2.9	-0.20 ± 0.26	

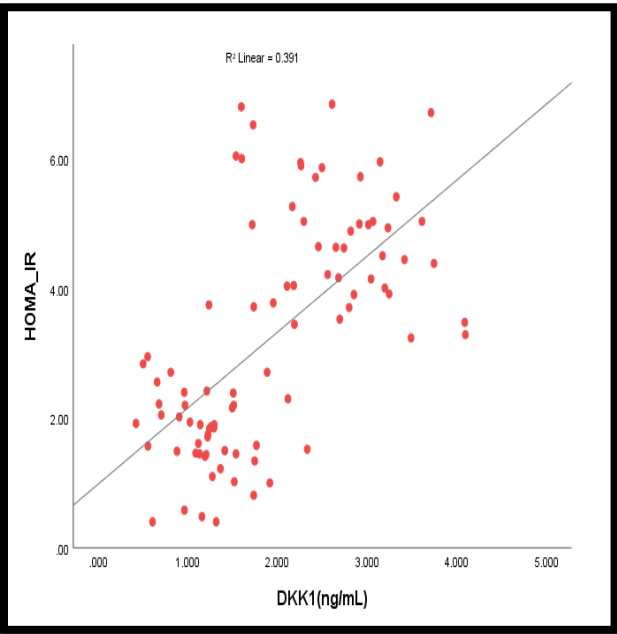
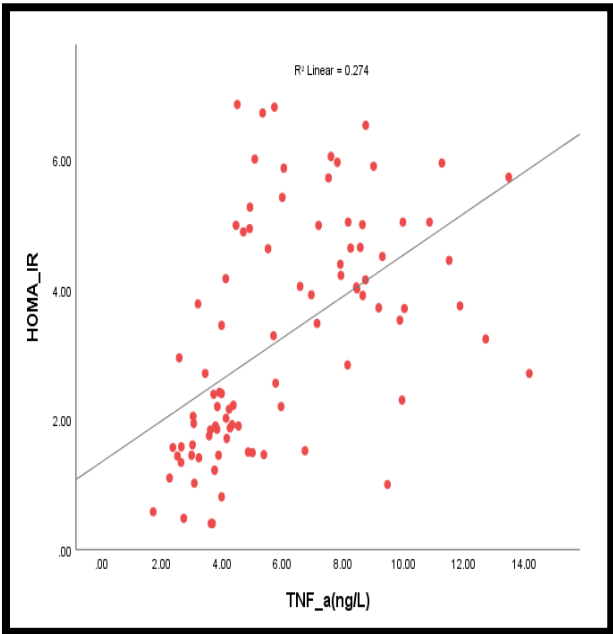
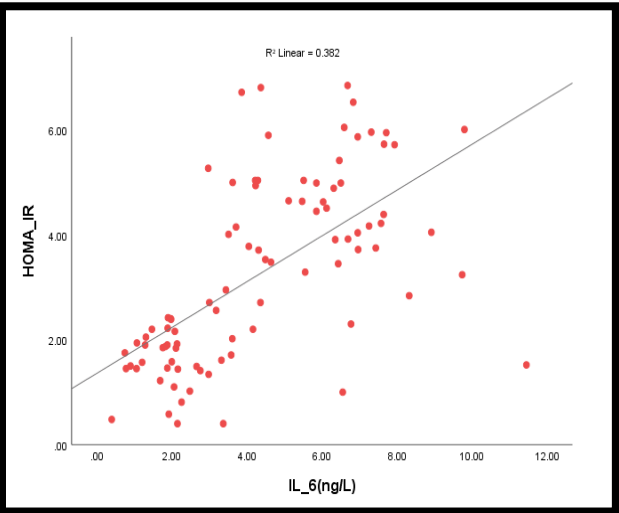
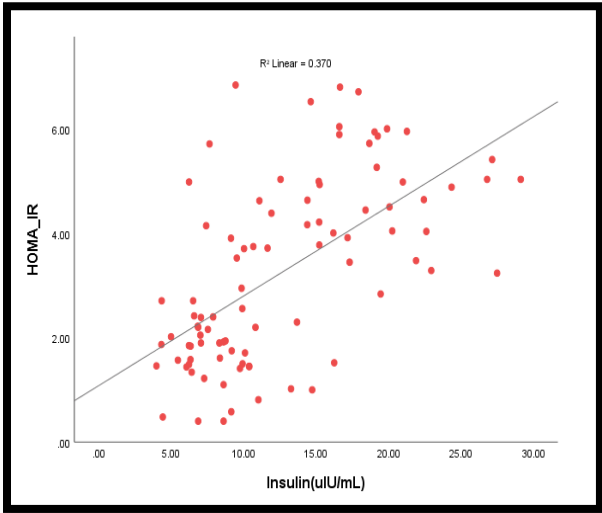
Data are expressed as mean $\pm$ standard deviation (SD). SE denotes Standard Errors; n indicates the number of subjects;

Range refers to the difference between the highest and lowest values within the dataset; 95% C.I represents the confidence intervals (lower and upper bounds); p-value categories are defined as N.S ($p > 0.05$), S ($p < 0.05$), and HS ($p < 0.01$), which reflect the significance level compared to the respective control value.

Table 3: Correlation Coefficient of HOMA-IR with other Clinical Parameters in Women with Osteoporosis and Patients with Type 2 Diabetes

The Correlation of HOMA-IR vs. other Variables	Correlation Coefficient (r)
	Osteoporosis and Type 2 Diabetes Patients
Insulin (uIU/mL)	0.615**
TNF-α (ng/L)	0.626**
IL-6 (ng/L)	0.665**
DKK1 (ng/mL)	0.629**
SOST (ng/mL)	0.634**
CTSK (ng/mL)	0.565**
Osteocalcin (ng/mL)	-0.720**
T-score	-0.704**

Spearman's correlation analysis provides a correlation coefficient denoted as r, which indicates the strength of the correlation. A negative value (-) signifies an inverse relationship, while a positive value (+) indicates a proportional relationship. Results are considered significant if p is less than 0.05 and highly significant when p is equal to or less than 0.01.



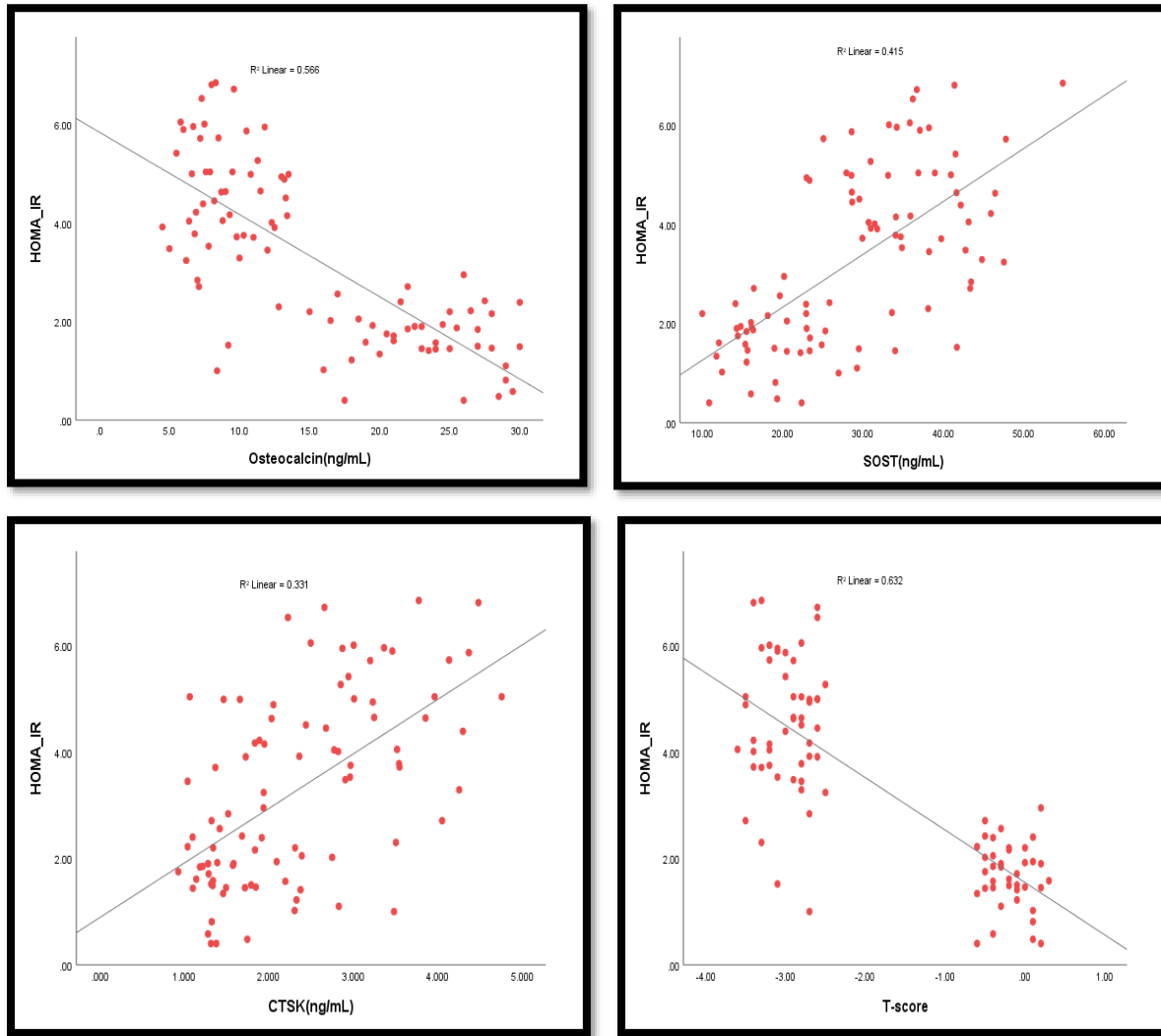


Figure 1: Correlation Coefficient of CTLA-4 vs. Other Clinical Parameters (Insulin, TNF- α , IL-6, DKK1, SOST, CTSK, Osteocalcin, T-Score) in Osteoporosis and Type 2 Diabetes Patients

Table 4: ROC and AUC Analysis of the Measured Biomarkers for the Diagnosis of Osteoporosis and Type 2 Diabetes Patients

Parameter	Cut-off Concentration	Sensitivity %	Specificity %	AUC	95% CI of AUC	P-Value
BMI (kg/m ²)	37.71	70	100	0.46	0.93-0.99	0.495
HbA1c (%)	6.8	100	100	1.00	0.93-0.99	0.0001
Glucose (mg/dL)	137.5	100	100	1.00	0.93-0.99	0.0001
Insulin (uIU/mL)	11.06	98	82	0.93	0.93-0.99	0.0001
HOMA_IR	3.10	90	100	0.97	0.93-0.99	0.0001
TNF- α (ng/L)	4.41	94	85	0.95	0.93-0.99	0.0001
IL-6 (ng/L)	3.62	96	98	0.99	0.93-0.99	0.0001
ALP (U/L)	92.05	86	85	0.90	0.93-0.99	0.0001
DKK1 (ng/mL)	1.53	96	90	0.96	0.93-0.99	0.0001
SOST (ng/mL)	26.38	94	90	0.97	0.93-0.99	0.0001
CTSK (ng/mL)	2.41	68	95	0.86	0.93-0.99	0.0001
PTH (pg/ml)	64.5	92	100	0.98	0.93-0.99	0.0001
VIT.D (ng/ml)	26.25	94	84	-0.96	0.935-0.995	0.0001
Osteocalcin (ng/mL)	14.30	100	100	-1.00	1.000-1.000	0.0001
Ca (mg/dL)	8.88	70	88	-0.82	0.732-0.908	0.0001
Phosphorus(mg/dL)	3.75	94	85	-0.96	0.936-0.997	0.0001
Mg (mg/dL)	1.97	50	78	-0.70	0.597-0.808	0.001
T-Score	-1.55	100	100	-1.00	1.000-1.000	0.0001

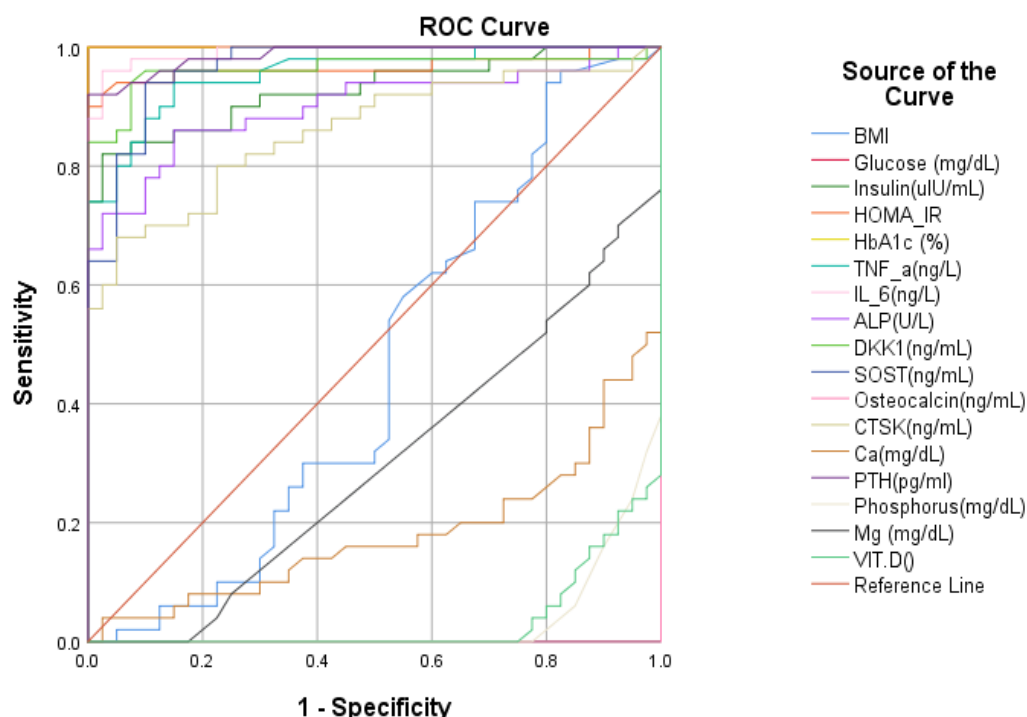


Figure 2: Receiver Operating Characteristic Curve (ROC) for BMI. Level of HOMA-IR, Insulin, HbA1c, TNF-, IL-6, ALP, DKK1, SOST, CTSK, PTH, VIT.D, Osteocalcin, Ca, Phosphorus, Mg, and T-Score Levels in Osteoporosis and Type 2 Diabetes Patients

6. Discussion

All female participants from this study were Basra Province residents themselves, such as patients and health-observers. Most of the volunteer women were urban, had a good educational history and had steady employment; they were not smokers. Study after study demonstrates that, on the other hand, people in the urban areas are very different from people in the rural areas. The differences are as diverse as pollution, environmental, social, psychological, genetic, diet – and many more in the city environment. And working stress can impact the mental well-being of male volunteers. Furthermore, work and family relationships and marriage can act as stressors that could also lead to deterioration in the oxidant/antioxidant imbalance (El Askary et al., 2021). This shows that an increase in insulin secretion leads to decreased insulin sensitivity and β -cell dysfunction. These modified forms may drive hyperglycemia, and fortify the glycation pathway of hemoglobin, which leads to free iron release from glycated proteins (e.g. hemoglobin). So, such a high iron concentration could promote oxidative stress on the cell in this form of biomolecular damage. Excessive iron accumulation also hinders insulin generation and release from the pancreas, and the liver is affected by insulin use. Should skeletal muscle be injured, any accumulation of iron can inhibit the uptake of glucose (Eldieb & Makboul, 2011; Mo et al., 2019). Insulin resistance had also been identified in our study as a central risk factor of osteoporosis and associated with T2DM (Toragall et al., 2019). Inflammatory cytokines were associated with HOMA-IR values indicating that chronic low-grade inflammation is one of the main potential mediators of metabolic decline and is associated with skeletal instability (McClung et al., 2018). When Wnt signaling inhibitors in insulin-resistant patients are upregulated, it supports the suppression of osteoanabolic signaling as a central player in diabetic bone disease. In metabolic disorders, the increased levels of sclerostin and DKK-1 have been positively correlated with lower bone formation, bone strength impairment and higher fracture risk in the patients (Vestergaard, 2007).

The analysis of the ROC curves indicates that body mass index (BMI) was very ineffective in the diagnosis of individuals with T2D-associated osteoporosis by BMI as a diagnostic criterion showing low AUC (0.46) and non-significant p value. To clarify, this means BMI would not alone be considered a reliable index for skeletal fragility in diabetes. Although body mass index (BMI) and bone mineral content and bone density are correlated across domains, elevated BMI does not necessarily represent bone quality and microarchitecture status. In type 2 diabetics excess adiposity may reduce the bone weakness mechanism of high marrow adiposity, chronic inflammatory state and altered adipokine signaling leading to decreased diagnostic potential of BMI as a diagnostic tool for osteoporosis estimation (Lecka-Czernik, 2017; Napoli et al., 2014). Signs and symptoms of glycemic control, i.e., HbA1c and fasting glucose, were clinically viable (AUC= 1.00,

sensitivity-specificity 100%). These findings support the close link between chronic hyperglycemia and the skeletal derangement in participants with type 2 diabetes. These elevated sugar levels may have a long-term implication of accumulating advanced glycation end products in bone matrix, leading towards collagen cross-linking, bone toughness loss and worsening osteoblast function. So poor glycemic control is a significant contributor to the occurrence of bone fractures, irrespective of the level of mineral density of the bone (Hardy & Cooper, 2009; Starup-Linde & Vestergaard, 2016). Insulin and HOMA-IR showed particularly superior diagnostic abilities (high AUC value, sensitivity and specificity) demonstrating the role of insulin resistance as a key mediator in the pathogenic process of diabetic osteoporosis. And bone metabolism is disturbed by insulin and IGF-1 signaling dysfunction in osteoblast cells, and the inhibition of bone formation promotes oxidative stress, and hyperinsulinemia, in addition to insulin resistance, are facilitators of inflammatory pathways, which disturbed the normal rate of bone regeneration and resorption (Awad et al., 2015; Toragall et al., 2019). TNF- α and IL-6 were found to be the most strongly correlated biomarkers for diagnostics based on AUC values which are almost synonymous. This research determines the impact of chronic low-grade inflammation on bone damage of type 2 diabetes. Pro-inflammatory cytokines promote osteoclast differentiation via the RANK/RANKL axis, further preventing osteoblast maturation and development. During the sustained period of inflammatory state in diabetic patients, bone resorption is stimulated by the body that has maintained a long state of inflammation which ultimately leads to impaired bone quality and decreased bone stability leading to high risk of fracture (Bikle, 2016). The significant diagnostic values showed no consistent positive values for biomarkers of bone turnover such as alkaline phosphatase, osteocalcin, and cathepsin K. Osteocalcin had a negative correlation with health status which was correlated with decreased osteoblast function in type 2 diabetes. High levels of cathepsin K indicate increased osteoclastic resorption in bone, meanwhile. This means that bone remodeling processes are uncoupled in diabetic osteoporosis, with disruption of bone formation and increased bone resorption. We have illustrated with high precision the prominent role of the Wnt signaling inhibitors DKK-1, SOST, and sclerostin in the diabetic bone disease context. These proteins exert a powerful inhibition of Wnt/ β -catenin signaling pathway that disrupts osteoblast differentiation and bone matrix growth. Increased sclerostin expression in type 2 diabetes has been associated with hyperglycemia, inflammation, and insulin resistance in other patient types and hence they are signals of bone anabolism.

Parathyroid hormone has been found to be a diagnostic marker of high diagnostic specificity suggesting a crucial marker for the body to restore calcium and vitamin D homeostasis in response to calcium and vitamin D homeostasis perturbations. Vitamin D and measurements of mineral level (calcium, phosphorus and magnesium) were also strongly inversely associated with disease. If these elements are in short supply, bone mineralization is inhibited and insulin resistance is developed—both of which are causing frailty in the skeleton. The absence of magnesium makes glucose metabolism bad and causes bone strength loss (Gröber et al., 2015). The T-score is the gold standard of osteoporosis diagnosis with diagnostic accuracy of 100%. But T-score alone may underestimate fracture risk in type 2 diabetes people, even though it is highly sensitive and specific. These data offer a unique perspective on bone health and fracture risk in this population (Kanis et al., 2019), when interwoven with densitometric measures and biomarkers for metabolic, inflammatory and bone turnover.

7. Conclusion

Insulin resistance is a significant contributor to osteoporosis in patients with type 2 diabetes mellitus because it influences inflammation, bone remodeling, signaling pathways, and the metabolism of minerals. Metabolic assays such as HOMA-IR, inflammatory and bone markers are useful and could help in predicting the potential for diabetic osteoporosis through a comprehensive way of assessing the skeletal risk.

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