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Original Article

The association of vitamin D, vitamin D-binding protein, and ferritin levels in Iraqi men with type 2 diabetes

Mohammed Jawad Kadhim
Department of Chemistry, Institute of Graduate Studies, Near East University, Nicosia, Cyprus
mohammedjawadkd@hotmail.com (corresponding author)

Süleyman Aşir
Department of Chemistry, Institute of Graduate Studies, Near East University, Nicosia, Cyprus

Amer Hasan
Department of Chemistry, College of Science, Al-Mustansiriya University, Baghdad, Iraq

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Abstract:
This study investigated the relationship between vitamin D, vitamin D-binding protein (VDBP), and ferritin in Iraqi men with type 2 diabetes (T2D). A total of 120 participants, comprising 60 diabetic patients and 60 healthy controls, aged 30 years and older, were matched for normal body mass index. Glucose levels were significantly elevated in the diabetic group (p=0.001), confirming impaired glycemic regulation. Liver function markers, including total serum bilirubin (TSB) and glutamate oxaloacetate transaminase (GOT), were also significantly higher (p=0.015 and p=0.009, respectively), suggesting hepatic dysfunction. Although vitamin D3 concentrations did not differ significantly, reduced vitamin D bioactivity may result from liver impairment and significantly lower VDBP levels in diabetic individuals (p=0.001), compromising its transport to target tissues. No significant differences were found in ferritin and renal markers. These findings highlight the potential role of disrupted vitamin D metabolism, particularly in transport and activation—in the pathogenesis of type 2 diabetes (T2D).

Key words:
vitamin D, vitamin D binding protein (VDBP), ferritin, type 2 diabetes

Apstrakt:
Povezanost nivoa vitamina D, proteina koji vezuje vitamin D i feritina kod iračkih muškaraca sa dijabetesom tipa 2
Ova studija je ispitivala odnos između vitamina D, proteina koji vezuje vitamin D (VDBP) i feritina kod iračkih muškaraca sa dijabetesom tipa 2 (T2D). Ukupno je učestvovalo 120 ispitanika — 60 pacijenata sa dijabetesom i 60 zdravih kontrola starijih od 30 godina, sa normalnim indeksom telesne mase. Nivoi glukoze bili su značajno povišeni u grupi sa dijabetesom (p=0.001), što potvrđuje poremećenu regulaciju glikemije. Markeri funkcije jetre, uključujući ukupni serumski bilirubin (TSB) i glutamat-oksaloacetat transaminazu (GOT), takođe su bili značajno viši (p=0.015 i p=0.009), što ukazuje na disfunkciju jetre. Iako koncentracije vitamina D3 nisu pokazale značajne razlike, smanjena biološka aktivnost vitamina D može biti posledica oštećenja jetre i značajno nižih nivoa VDBP kod dijabetičara (p=0.001), što narušava njegov transport do ciljnih tkiva. Nisu pronađene značajne razlike u nivou feritina i bubrežnim markerima. Ovi nalazi naglašavaju potencijalnu ulogu poremećenog metabolizma vitamina D, posebno njegovom transportu i aktivaciji, u patogenezi dijabetesa tipa 2 (T2D).

Ključne reči:
vitamin D, vezujući protein vitamina D (VDBP), feritin, dijabetes tipa 2

Introduction

Type 2 diabetes (T2D) has become one of the most prevalent metabolic disorders in modern society, driven by sedentary lifestyles, poor dietary habits, and urbanization (Kakil & Meena, 2020). Globally, it presents significant public health challenges due to its progressive nature and multi-organ complications (Antar et al., 2023). In Iraq, the rising incidence—particularly among men—underscores the urgent need for deeper investigation into contributing physiological and biochemical factors (World Health Organization, 2020).

Recent studies have highlighted a strong correlation between vitamin D deficiency and the onset of type 2 diabetes (T2D) (Zhao et al., 2024). Vitamin D plays a pivotal role in regulating immune responses, promoting calcium absorption, and supporting the differentiation and protection of various cell types (Janoušek et al., 2022). As a fat-soluble hormone, its bioavailability and metabolic function heavily rely on vitamin D binding protein

(VDBP), which transports vitamin D metabolites throughout the bloodstream (Wu et al., 2023; Abu-Shana et al., 2024). Evidence suggests that inadequate levels of vitamin D can impair glucose metabolism and insulin sensitivity, two key processes in the pathophysiology of T2D (Zhang et al., 2020). Furthermore, deficiencies in vitamin D have been linked to reduced pancreatic β-cell activity, which compromises insulin secretion and exacerbates insulin resistance (Szymczak-Pajor & Śliwińska, 2019). Despite Iraq's abundant sunshine, many individuals—especially older males—exhibit low serum levels of vitamin D, a paradox that may reflect underlying metabolic or organ-specific dysfunction, such as impaired liver hydroxylation or kidney conversion of inactive vitamin D into its biologically active form (Salim et al., 2023).

The current study aims to evaluate the levels of serum vitamin D, VDBP, and ferritin in Iraqi males diagnosed with T2D, compared to healthy controls matched for age, body mass index (BMI), and lifestyle factors. The study excludes individuals with existing kidney, liver, or thyroid disorders, ensuring a focused analysis on diabetes-related variables. Through this investigation, we aim to generate region-specific biochemical data that may guide preventive, diagnostic, and therapeutic approaches. An enhanced understanding of vitamin D's role and transport mechanisms could provide novel strategies for managing type 2 diabetes (T2D), particularly in populations with limited access to specialized care. Moreover, findings may serve as a foundation for future research into the interplay between micronutrient metabolism and chronic disease.

Materials and Methods

This study employed a comparative design to investigate biochemical variables associated with T2D, with a focus on vitamin D, VDBP, and ferritin. A total of 120 Iraqi male participants, all aged ≥30 years, were carefully selected based on stringent inclusion criteria. Among them, 60 were clinically diagnosed with T2D and were managing their condition with oral hypoglycemic medication. The control group consisted of 60 healthy males who were matched in age and maintained a BMI between 19 and 24 kg/m². All individuals underwent preliminary interviews to confirm eligibility and received written information outlining study details. Informed consent was obtained from each participant in accordance with research ethics protocols. Blood samples were collected from each subject after an overnight fast ranging from 8 to 12 hours, at Al-Qanah Medical Laboratory in Baghdad, Iraq, between 15 January and 1 March 2025. Samples were processed promptly following standard clinical guidelines.

Laboratory analyses

Approximately 5 mL of blood was drawn from each participant using sterile needles. The collected blood was placed in gel tubes designed to separate serum through centrifugation. After separation, the serum was transferred using micropipettes into cuvette tubes for analysis.

Serum biomarker levels were assessed using standardized clinical analyzers and kits. Insulin, ferritin, and vitamin D concentrations were determined using the Cobas e 411 analyzer and diagnostic kits manufactured by Roche (Germany). Vitamin D binding protein (VDBP) levels were measured via immunoassay kits obtained from Shanghai Coon Koon Biotech Co., Ltd (China).

Glucose levels were evaluated using glucose-specific test kits, while renal function indicators—blood urea and serum creatinine—were analyzed using renal function kits. Liver function biomarkers, including total serum bilirubin (TSB), glutamate pyruvate transaminase (GPT), glutamate oxaloacetate transaminase (GOT), and alkaline phosphatase (ALP), were quantified using liver function kits. All biochemical reagents for glucose, renal, and hepatic assessments were provided by ELITech Clinical Systems SAS (France).

Participant height and weight were recorded using the Anker Eufy Smart Scale P2 Pro, which includes an integrated tape measure for height calculations. BMI was computed using the equation: BMI = Weight in Kilograms /Height in Square Meters. Insulin resistance was measured using the HOMA-IR equation: HOMA-IR = [Glucose (mg/dL) × Insulin (mU/L)] ÷ 405. For reference, 18 mg/dL ≈ 1 mmol/L. A HOMA-IR value ≥2.5 typically indicates insulin resistance, as it is associated with impaired insulin sensitivity (Crook & Godsland, 1998; Porchia et al., 2024). It should be noted that individual factors such as age, weight, and health status may influence reference values (Polonsky & Burant, 2015). As for determining VDBP concentrations, absorbance was measured using a uQuant Bio-Tek Microplate Spectrophotometer (USA). Temperature stability and sample preservation were maintained using a laboratory refrigerator and incubator.

Statistical analysis

All data obtained were analyzed using SPSS, MedCalc, and GraphPad Prism for descriptive statistics and inferential comparisons. The Gen5 – BioTek software was used in conjunction with the microplate reader to record optical density and calculate VDBP concentrations using a quadratic fit model. A *p*-value of <0.05 was considered statistically significant across tests. The rigorous design and use of matched samples enhance the validity and reproducibility of the findings, aligning with established research protocols and literature standards.

Results and discussion

The demographic and anthropometric characteristics of the participants are presented in **Tab. 1.** The mean age of the control group was 35.52 years (±2.81), and 35.25 years (±3.24) for the T2D group. No significant difference was observed (p=0.631), indicating age homogeneity between groups. Mean body weight also showed similarity, measured at 65.65 kg (±4.96) in controls and 65.07 kg (±4.78) in T2D patients (*p*=0.513). In terms of height, however, a statistically significant difference was noted (*p*=0.020), with mean values of 1.741 m (±0.047) in controls and 1.717 m (±0.047) in the diabetic group. BMI values were closely aligned, with means of 21.62 kg/m² (±1.26) and 22.06 kg/m² (±1.51) respectively, and the difference was not statistically significant (*p*=0.085), in accordance with the study's inclusion criteria (BMI 19–24 kg/m²). **Fig. 1.** illustrates a near-balanced BMI distribution, with 49% of participants belonging to the control group and 51% to the diabetic group. The second graph shows that participant distribution across ages was evenly split, with both control and diabetic groups comprising 50% of the sample. **Tab. 2.** summarizes T2D biochemical variables. Glucose levels were significantly elevated in the T2D group, with a mean of 152.32 mg/dL (±40.00) compared to 87.67 mg/dL (±5.94) in the control group (*p*=0.001). This confirms the expected glycemic dysregulation in patients with diabetes. Insulin levels showed no significant difference between groups, with values of 12.60 mU/L (±5.23) in the T2D group and 11.63 mU/L (±3.89) in the control group (*p*=0.252). However, insulin resistance was markedly higher in diabetic subjects, with a mean value of 4.72 (±2.34) compared to 2.52 (±0.85) in controls (*p*=0.001), underscoring its role in the pathophysiology of T2D. Ferritin levels did not differ significantly between groups; control group had a mean of 61.32 ng/mL (±16.08),

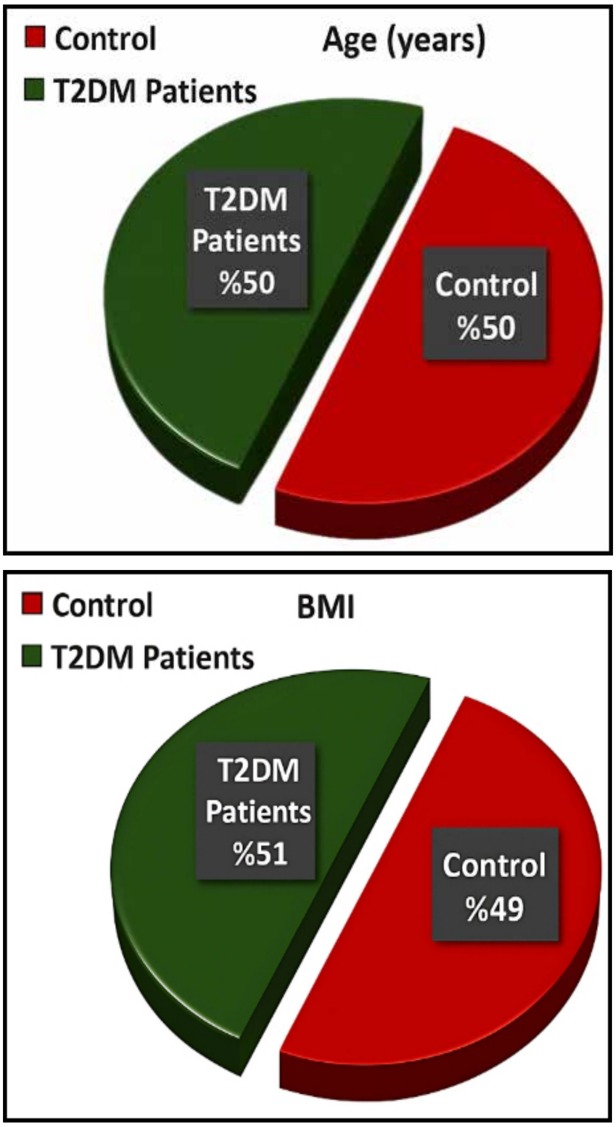

Figure 1. Distribution of age and BMI between T2D patients and the control group

Table 1. The statistical disparities among control and T2D patients for age and anthropometric measurements

	Control Group, N=60		T2D patients, N=60		
Parameters	**Mean**	**SD**	**Mean**	**SD**	***p*-value**
Age (year)	35.52	2.8134	35.25	3.245	0.631
Weight (kg)	65.65	4.957	65.067	4.779	0.513
Height (m)	1.741	0.047	1.717	0.047	0.02*
BMI (kg/m2)	21.618	1.258	22.058	1.508	0.085

*Significant at *p*≤0.05, **Highly significant *p*<0.001, NS: Non-Significant

Table 2. The statistical disparities among control and T2D patients for T2D parameters

	Control Group, N=60		**T2D patients, N=60**		
Parameters	**Mean**	**SD**	**Mean**	**SD**	***p*-value**
Glucose (mg/dL)	87.666	5.942	152.316	40.004	0.001**
Insulin (mU/L)	11.633	3.894	12.601	5.229	0.252
Insuline Resistance	2.516	0.852	4.722	2.338	0.001*
Ferritin (ng/mL)	61.316	16.082	64.648	19.315	0.307

*Significant at $p \leq 0.05$, **Highly significant $p < 0.001$, NS: Non-Significant

while T2D patients had a mean of 64.65 ng/mL (±19.32) (p=0.307), suggesting limited relevance in differentiating disease status.

Tab. 3. presents the serum concentrations of vitamin D3 and VDBP parameters in both study groups. The mean vitamin D3 level in the control group was 26.63 ng/mL (±14.68), slightly higher than in the T2D group, which averaged 22.11 ng/mL (±12.55). Although the diabetic group demonstrated lower levels, the difference was not statistically significant (p=0.072). VDBP levels were significantly reduced in the diabetic group, averaging 135.71 ng/mL (±20.29) compared to 159.66 ng/mL (±28.36) in controls (p=0.001), suggesting impaired vitamin D transport capacity.

Liver function results (**Tab. 4.**) indicate that TSB was significantly higher in T2D patients (0.83 mg/dL ± 0.087) compared to the control group (0.79 mg/dL ± 0.090), with a p-value of 0.015. No significant difference was observed in GPT levels (T2D: 30.42±5.91 IU/L; control: 31.38±6.02 IU/L; p=0.377). However, GOT levels were significantly elevated in T2D patients (29.38±5.52 IU/L) compared to controls (26.73±5.46 IU/L), with a p-value of 0.009. ALP levels did not differ significantly between the groups (T2D: 78.70 IU/L ±9.97; control: 76.63 IU/L ±10.84; p=0.279). Kidney function assessments (**Tab. 5.**) showed no significant differences. Blood urea levels were comparable between T2D (31.85±4.70 mg/dL) and control subjects (32.05±4.25 mg/dL; p=0.807). Similarly, serum creatinine values were consistent, with T2D patients recording 0.76 mg/dL (±0.12) and controls at 0.73 mg/dL (±0.13), yielding a non-significant p-value of 0.185. A substantial elevation in fasting blood glucose was identified in individuals with type 2 diabetes (152.32±40.00 mg/dL) compared to the non-diabetic control group (87.67±5.94 mg/dL), with the difference reaching high statistical significance (p<0.001). This hyperglycemic state is indicative of impaired glucose homeostasis, primarily driven by insulin resistance, a central mechanism in the pathogenesis of T2D. The finding aligns with established literature recognizing persistently elevated glucose levels as a key clinical marker in the diagnosis and progression of type 2 diabetes. Although insulin levels did not differ significantly between the diabetic and control groups (12.60±5.23 mU/L vs. 11.63±3.89 mU/L; p=0.252), this does not reflect practical metabolic function in T2D patients. Despite near-normal circulating insulin levels, the elevated blood glucose observed in the diabetic group suggests underlying insulin resistance. This condition impairs the hormone's ability to facilitate glucose uptake in peripheral tissues, a hallmark of type 2 diabetes. Previous studies have similarly reported that individuals with T2D may exhibit normal or even elevated insulin concentrations due to compensatory β-cell activity. However, the presence of insulin alone is not indicative of glycemic control in the context of resistance. Consistent with this, the current study demonstrated a significant rise in insulin resistance among diabetic participants (4.72±2.34) compared to controls (2.52±0.85), with a p-value < 0.001. These findings reinforce the clinical understanding that insulin resistance plays a central role in the pathogenesis and progression of T2D (American Diabetes Association, 2018). No statistically significant difference in serum ferritin levels was observed between type 2 diabetes patients and the control group (64.65±19.32 ng/mL vs. 61.32±16.08 ng/mL; p=0.307). This result

Table 3. The statistical disparities among control and T2D patients for vitamin D3 and VDBP

	Control Group, N=60		**T2D patients, N=60**		
Parameters	**Mean**	**SD**	**Mean**	**SD**	***p*-value**
Vita. D3 (ng/mL)	26.632	14.682	22.106	12.554	0.072
VDBP (ng/mL)	159.66	28.362	135.71	20.286	0.001**

*Significant at $p \leq 0.05$, **Highly significant $p < 0.001$, NS: Non-Significant

Table 4. The statistical disparities among control and T2D patients for liver function parameters

	Control Group, N=60		T2D patients, N=60		
Parameters	**Mean**	**SD**	**Mean**	**SD**	***p*-value**
TSB (mg/dL)	0.793	0.09	0.833	0.087	0.015*
GPT (IU/L)	31.38	6.023	30.417	5.907	0.377
GOT (IU/L)	26.73	5.455	29.383	5.521	0.009*
ALP (IU/L)	76.63	10.84	78.7	9.968	0.279

*Significant at $p \leq 0.05$, **Highly significant $p<0.001$, NS: Non-Significant

aligns with the findings, which reported that ferritin concentrations were not associated with glycemic control in T2D patients (Al Argan et al., 2023). Previous research has demonstrated a correlation between elevated ferritin levels and an increased risk of T2D. However, it remains unclear whether this elevation plays a causal role in the onset of diabetes or merely reflects the progression of insulin resistance, a recognized precursor to T2D (Podmore et al., 2016). Additionally, it was found that individuals with T2D exhibited increased ferritin levels, which may be linked to prolonged hyperglycemia, showing a strong positive correlation with glycemic markers such as fasting blood glucose and HbA1c (Bayih et al., 2024). Taken together, these findings suggest that while ferritin may play a role in metabolic disturbance, its diagnostic value in T2D requires further clarification.

Although the observed differences in serum vitamin D3 concentrations between the diabetic group (22.11±12.55 ng/mL) and the control group (26.63±14.68 ng/mL) did not reach statistical significance (p=0.072), this does not preclude a clinical impact. The biological activity of vitamin D3 depends on its conversion to the active form, a process influenced by VDBP, TSB, and liver enzyme levels such as GOT. Disruptions in these factors—common in individuals with T2D—may contribute to reduced levels of active vitamin D3, thereby impairing glucose metabolism and insulin sensitivity. A study reported that vitamin D deficiency is associated with deteriorating glycemic control (Pittas et al., 2007). Furthermore, longitudinal data suggest that low vitamin D status may predict the future onset of T2D (Forouhi et al., 2008). It was also highlighted that vitamin D deficiency is prevalent in diabetic populations and correlates with poorer clinical outcomes, supporting its role in both disease pathogenesis and therapeutic monitoring (Arafat et al., 2020). The current study revealed a highly significant reduction in VDBP concentrations among patients with type 2 diabetes (135.71±20.29 ng/mL) compared to the control group (159.66±28.36 ng/mL), with a p-value of 0.001. This decline in VDBP may compromise the transport efficiency of vitamin D metabolites in the bloodstream, potentially reducing their bioavailability and limiting cellular uptake at target sites. Such disruption could contribute to the metabolic dysfunction associated with T2D, despite the presence of measurable vitamin D levels. Notably, these findings diverge from those reported, which found no significant differences in VDBP levels between individuals with diabetes and those without diabetes. The discrepancy may be attributed to population-specific factors or differences in study design, underscoring the need for further investigation into VDBP's role in vitamin D metabolism and diabetes pathogenesis (Arafat et al., 2020).

Analysis of liver function parameters revealed several biochemical distinctions between diabetic patients and control subjects. TSB levels were modest but significantly elevated in the diabetic group (0.833±0.087 mg/dL) compared to controls (0.793±0.090 mg/dL), with a p-value of 0.015. This aligns with findings that elevated bilirubin may be linked to a higher risk of developing type 2 diabetes. Similarly, GOT levels were significantly higher in T2D patients (29.38±5.52 IU/L) than in the control group (26.73±5.46 IU/L; p=0.009), suggesting potential hepatic stress or dysfunction, which has been implicated in the pathogenesis of diabetes.

Table 5. The statistical disparities among control and T2D patients for renal function parameters

	Control Group, N=60		T2D patients, N=60		
Parameters	**Mean**	**SD**	**Mean**	**SD**	***p*-value**
Urea (mg/mL)	32.05	4.252	31.85	4.697	0.807
Creatinine (mg/mL)	0.73	0.125	0.76	0.121	0.185

*Significant at $p \leq 0.05$, **Highly significant $p<0.001$, NS: Non-Significant

By contrast, no statistically significant differences were found in GPT and ALP levels between the two groups. Nonetheless, previous research indicates that elevated GPT and ALP may arise from liver conditions such as hepatitis or biliary disorders. It may also be linked to abnormalities in bone metabolism or long-term hyperglycemia (Rafaqat et al., 2023). These parameters, relevant in specific metabolic or comorbid contexts.

Importantly, liver function plays a crucial role in the hydroxylation and activation of vitamin D. Any hepatic impairment can disrupt this conversion, potentially reducing active vitamin D availability and contributing to the progression of T2D. The observed elevations in TSB and GOT levels may reflect subtle liver dysfunction in diabetic individuals, underscoring the interconnected nature of hepatic health and vitamin D metabolism.

Renal function analysis revealed no statistically significant differences in urea or creatinine levels between participants with diabetes and those without diabetes. Urea concentrations were comparable between the T2D group (31.85±4.70 mg/dL) and the control group (32.05±4.25 mg/dL; p=0.807). Similarly, creatinine levels showed minimal variation, with the diabetic group averaging 0.76±0.12 mg/dL and the control group averaging 0.73±0.13 mg/dL (p=0.185). These findings suggest that renal impairment was not apparent within the study population.

However, this contrasts with prior research indicating that elevated urea and creatinine are commonly observed in patients with T2D, reflecting progressive kidney damage (Lad & Kava, 2019). A high prevalence of renal dysfunction has also been documented among individuals with diabetes, highlighting the importance of early screening and ongoing monitoring of kidney health in this population (Shahwan et al., 2019).

It is essential to note that the kidneys play a crucial role in converting vitamin D into its active form. Any decline in renal function may compromise vitamin D activation, thereby reduce its physiological effectiveness and increase susceptibility to metabolic disturbances, such as type 2 diabetes.

Conclusion

In conclusion, this study demonstrates that while vitamin D3 levels did not differ significantly between diabetic and non-diabetic Iraqi men, functional disruptions in vitamin D transport and activation—reflected in altered VDBP concentrations and liver functions—may contribute to impaired metabolic regulation in type 2 diabetes. The data affirm the central role of insulin resistance in disease progression and highlight that diminished vitamin D bioavailability, despite normal serum levels, may stem from compromised liver function and reduced VDBP. Together, these findings underscore the importance of evaluating not only vitamin D levels but also its transport and activation pathways in understanding and managing T2D.

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