Dijlah Journal of Medical Sciences (DJMS) Vol. 3, No. 2, May, 2026, pp. 28-33
P-ISSN: 3078-3178, E-ISSN: 3078-8625, paper ID: 03

ORIGINAL ARTICLE

Vitamin D3 Deficiency and Its Relationship with Oxidative Stress in Type 2 Diabetes Mellitus: A Case-Control Study (Iraq-Baghdad)

Nesreen Ahmed Nasser[1], Raghad Ahmed Mohammed[2], Saddam Jaber Khudiar[3] and Diyar Adel Lateef[4]
Department of Chemistry and Biochemistry, College of Medicine, Al Nahrain University, Baghdad, Iraq

Article Info

Article history:

Received November, 07, 2025
Revised November, 23, 2025
Accepted January, 15, 2026

Keywords:

Superoxide Dismutase (SOD),
Vitamin D3,
Type 2 Diabetes Mellitus,
Oxidative Stress.

ABSTRACT

The pathophysiology of diabetes involves oxidative stress. Damage from severe oxidative stress can reduce pancreatic beta-cell activity. In type 2 diabetes, antioxidant status imbalance and inflammation work together. Superoxide dismutase (SOD), an enzyme contained in most foods, may minimize intestinal barrier oxidative damage, breaking the hyperglycemia-oxidative damage cycle. This study examined Vitamin D Deficiency and Oxidative Stress Marker (SOD) in Type 2 Diabetes Mellitus Patients. This case-control study examined the associations between selected parameters and T2DM in fifty case and fifty control of either sex patients newly evaluated by an expert using medical evaluation and validated through laboratory analyses at a private lab (Huda Al-Rahman) analysis following the removal of alternative disorders through clinical history, analytical testing, and physical examination. This investigation was place from October 2024 to February 2025. Clinical, biochemical, and hematological tests showed the control group was healthy. Patients with type 2 diabetes' mean ± SD SOD levels (302.02 ± 9.05 U/Ml) were discovered to be substantially greater (P<0.000) than Controls (100.1+4.41 U/Ml) The mean ± SD D3 Levels of type 2 diabetes patients (13.7 ± 4.07 ng/ml) were found to be significantly lower (P<0.000) than Controls (35.6+14.7 ng/ml). This study found that type 2 diabetes individuals with higher SOD levels may have hemostatic indicators.

Corresponding Author:

* Nesreen Ahmed Nasser
Department of Chemistry and Biochemistry, College of Medicine, Al Nahrain University, Baghdad, Iraq
Email: Nenanena1987ahmednena@yahoo.com

1- INTRODUCTION

A huge issue in health care around the world is type 2 diabetes mellitus (T2DM), a metabolic illness. Worldwide, the incidence of diabetes was 2.8% in 2000 and is projected to rise to 4.4% in 2030, according to the World Health Organization [1]. The functions of vitamins in living systems are crucial. Vitamin D has a crucial role in maintaining normal bone metabolism and calcium and phosphorus homeostasis [2]. Cardiovascular disease, cancer, autoimmune disorders, psoriasis, MS, and type 2 diabetes are just a few of the non-skeletal medical issues that vitamin D may impact [3]. There appears to be a negative correlation between the frequency of type 2 diabetes and circulating vitamin D levels. Actually, some research has linked vitamin D insufficiency to an increased likelihood of type 2 diabetes [4]. According to some research, vitamin D may influence insulin secretion and sensitivity, which in turn affects glucose tolerance [5]. In addition, glycated hemoglobin (HbA1c) levels in gestational diabetes mellitus appear to be inversely associated to vitamin D levels, suggesting that vitamin D affects glucose homeostasis [6].

Potentially contributing to the formation of type 2 diabetes and its consequences, oxidative stress (OS) is a key risk factor for both [7]. OS is linked to a rise in ROS generation and a decline in antioxidant system efficiency [8]. Excessive ROS generation in diabetic patients can result from continuous long-term hyperglycemia, and there is evidence that type 2 diabetes is significantly associated with OS [9]. Enzymatic antioxidants like superoxide dismutase (SOD) and catalase (CAT) and nonenzymatic antioxidants like vitamins A and E make up the standard defensive system against reactive oxygen species (ROS) [10]. Risk for type 2 diabetes and diabetic complications can be predicted by testing blood antioxidant defenses [11].

The first defense against ROS is superoxide dismutase. This decomposes superoxide anion into oxygen and hydrogen peroxide, which CAT uses to disintegrate further into oxygen and water increasing OS due to hyperglycemia [12]. Type 2 diabetics with low SOD values may be more susceptible to OS. CAT, another antioxidant enzyme, breaks down H2O2 into O2 and water. Deficit of CAT raises the risk of type 2 diabetes and impairs β-cell function by protecting them from ROS damage. Low CAT activity in type 2 diabetics is consistently related with type 2 diabetes, and hyperglycemia downregulates CAT expression [13]. Research on antioxidant enzymes has yielded conflicting results. To demonstrate, T2D patients had lower, unchanged, or higher SOD and CAT levels than controls.

2- MATERIALS AND METHODS

Fifty cases and fifty control of either sex patients with newly diagnosed type 2 diabetes mellitus (T2DM) were the subjects of this case-control study. The patients were chosen at random and confirmed their diagnosis with laboratory tests at a private lab (Huda Al-Rahman) in Baghdad, after the specialists had ruled out other possible diagnoses using the patients' medical histories and physical examinations. In addition, 50 individuals of a different age were randomly chosen to serve as a control group in this research. From October 2024 to February 2025, this investigation was conducted. The health of the control group was verified using hematological, biochemical, and clinical tests. All actions followed predetermined ethical guidelines. The College of Medicine / Al-Nahrain University Ethics Committee of Medical Research has given its approval to the study's protocol. Before the study began, all subjects gave their verbal consent.

2.1 Sample collection

After dividing the 10 ml of fasting blood samples into 2.5 ml of EDTA and 7.5 ml of total volume, the samples were left to coagulate for 30 minutes before being centrifuged for 15 minutes between 2500 and 3500 pm. In preparation for the biochemical analysis, the subjects' sera were segregated and partitioned into many portions, which were then placed into multiple plain plastic tubes. From the day of storage at (-80 Cº) until the day of analysis, the sera of both the control and patient groups were kept. Thawing the frozen sera at room temperature has prepared the sera for measurement. Use of the enzyme-linked immunosorbent assay (ELISA) method allowed for the estimation of the investigated parameters.

2.2 Statistical analysis

The Statistical Patch for the Social Sciences (SPSS vi.18) was used to conduct the statistical study. The mean ± SD was used to express the findings of the biochemical testing. Additionally, two means were compared using the student t-test. For statistical purposes, a p-value less than or equal to 0.05 was deemed significant. We used linear regression analysis to look for connections between the lab results and the continuous variables.

3- RESULTS AND DISCUSSION

In this study, 50 cases and 50 controls of either sex were included. In table 1 Shows the comparison of SOD, D3, FBS & HbA1c level between cases and controls. Compared to controls (302.02±9.05), cases had significantly reduced SOD levels (100.1±4.41) with a p-value of 0.0001*. The case group had significantly lower D3 levels (13.7±4.07) compared to the control group (35.6±14.7), and the case group had much higher FNB levels (245.2±4.87) than the control group (108.8±1.78). When comparing cases (8.08±1.02) to controls (5.17±0.72), the HbA1c level was significantly higher (p=0.001) in the former group.

Table (1): The comparison of SOD, D3, FBS & HbA1c level between cases and controls

	Groups	**Mean +Std. Deviation**	**P value**
AGE	patients	27.8 +8.6	0.531
	control	33.9 +11.28	
SOD	control	100.1±4.41	0.000
	Patients	302.02±9.05	
D3	patients	13.7±4.07	0.004
	controls	35.6±14.7	
HBA1C	patients	8.08±1.02	0.001
	controls	5.17±0.72	
FBS	patients	245.2±4.87	0.001
	controls	108.8±1.78	

Correlation coefficient

As D3 increased, SOD decreased, with a correlation coefficient of -0.820 and a p-value less than 0.000. Moreover, d3 exhibited a strong negative correlation (r= 0.712, p<0.000).

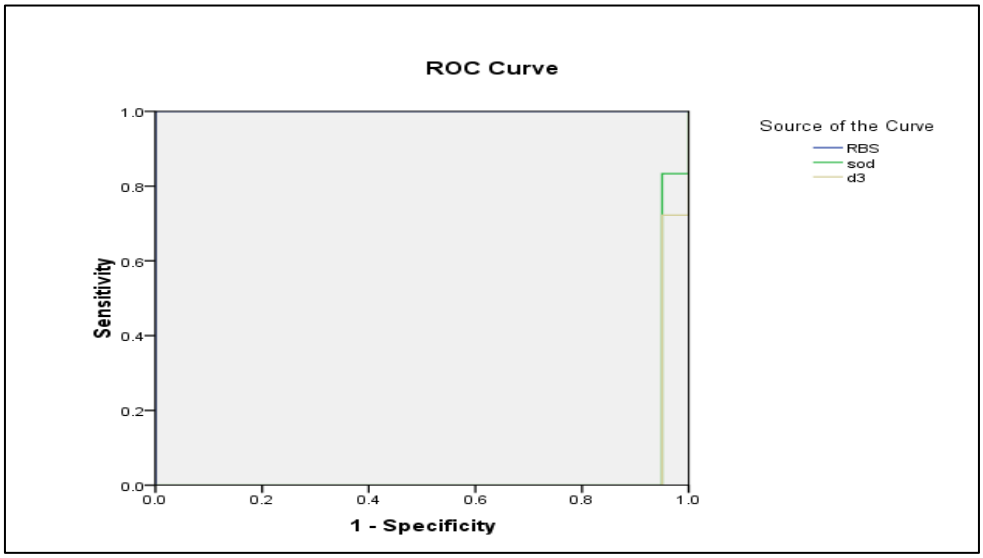
Table 2 Correlation coefficient between SOD and D3 parameters

		sod	**d3**
GROUP	Pearson Correlation	-.820**	-.712**
	Sig. (2-tailed)	.000	.000

Figure 1 compares D3 and SOD AUC and ROC curves to normal controls and type 2 diabetic patients. The AUC for D3 was 0.036 (80% confidence range) while for SOD it was 0.042 (88% confidence interval).

Area Under the Curve

Test Result Variable(s)	**Area**
RBS	1.000
sod	.042
d3	.036



Type 2 diabetes is characterized by insulin resistance and β-cell dysfunction. The condition worsens over time and causes changes in insulin secretion. Diabetes mellitus type 2 has many causes. Whether they function as triggers or as protectors, environmental elements are crucial [14]. Vitamin D insufficiency is one of the environmental variables that could contribute to the development of type 2 diabetes. Vitamin D is involved in many bodily processes, including immune system activity, bone remodeling, and mineral homeostasis regulation [15]. Type 2 diabetics exhibited considerably lower serum 25(OH)D levels than controls. Similar findings have been seen elsewhere. Numerous times Need et al [16] found that vitamin D-rich patients had lower FBS. Various studies show that diabetics and the general population have similar 25(OH) D levels [17, 18].

The presence of Vitamin D receptors (VDR) on pancreatic β-islet cells suggests a function for Vitamin D in T2DM [19]. The biologically active metabolite of Vitamin D, 1, 25-dihydroxy Vitamin D[1,25(OH)2D], increases insulin synthesis and secretion in these cells via the VDR. [20] VDR and Vitamin D binding protein gene single-nucleotide polymorphisms were associated to glucose intolerance and insulin secretion in several investigations [21, 22]. Subjects who consume over 800 IU vitamin D daily may have a lower overall risk of type 2 diabetes [23], and this provides more evidence that vitamin D has a role in this disease [24, 25]. The A other, related theory on vitamin D's role in preventing type 2 diabetes was advanced, however, based on its powerful immunomodulatory effects. There are VDRs in the majority of immune cell types, and 1, 25(OH) 2D regulates the synthesis of both the immunostimulatory and immunosuppressive interleukin (IL)-12 and IL-10, respectively. The β-cells are protected from cytokine-induced cell death by vitamin D [26, 27].

And other cause of coagulation system problems is elevated oxidative stress. This study found that compared to controls, individuals with type 2 diabetes mellitus had significantly higher serum levels of superoxide dismutase ($P<0.000$). Despite contradictory findings on SOD activity in diabetes, this study found that people with type 2 diabetes had higher levels of SOD activity than those without the disease; related research has shown in their study, [28, 29, 30].

4- CONCLUSION

Antioxidant enzymes improve cellular free radical scavenging and reduce ROS damage. Since overall SOD activity has increased, the organism may be adapting by creating more superoxide anion ($O2^-$) and hydrogen peroxide ($H2O2$). Diabetics may enhance SOD activity to adjust for oxidative damage, given this disagreement.

REFERENCES

[1] Wild, S., Roglic, G., Green, A., Sicree, R., & King, H. (2004). Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. Diabetes Care, 27(5), 1047–1053. https://doi.org/10.2337/diacare.27.5.1047

[2] Heaney, R. P. (2004). Functional indices of vitamin D status and ramifications of vitamin D deficiency. The American Journal of Clinical Nutrition, 80(6 Suppl), 1706S–1709S. https://doi.org/10.1093/ajcn/80.6.1706S

[3] Holick, M. F. (2006). High prevalence of vitamin D inadequacy and implications for health. Mayo Clinic Proceedings, 81(3), 353–373. https://doi.org/10.4065/81.3.353

[4] Lim, S., Kim, M. J., Choi, S. H., Shin, C. S., Park, K. S., Jang, H. C., et al. (2013). Association of vitamin D deficiency with incidence of type 2 diabetes in high-risk Asian subjects. The American Journal of Clinical Nutrition, 97(3), 524–530. https://doi.org/10.3945/ajcn.112.048496

[5] Lau, S. L., Gunton, J. E., Athayde, N. P., Byth, K., & Cheung, N. W. (2011). Serum 25-hydroxyvitamin D and glycated haemoglobin levels in women with gestational diabetes mellitus. Medical Journal of Australia, 194(7), 334–337. https://doi.org/10.5694/j.1326-5377.2011.tb03000.x

[6] Pejin, R., Popović, D., Tanackov, I., Bjelica, A., Tomic-Naglic, D., Jovanovic, A., & Stokic, E. (2019). The synergistic action of antioxidative enzymes—Correlations of catalase and superoxide dismutase in the development and during the treatment of type 2 diabetes. Srpski Arhiv za Celokupno Lekarstvo, 147, 286–294.

[7] Darenskaya, M. A., Kolesnikova, L. I., & Kolesnikov, S. I. (2021). Oxidative stress: Pathogenetic role in diabetes mellitus and its complications and therapeutic approaches to correction. Bulletin of Experimental Biology and Medicine, 171, 179–189.

[8] Hou, Y., Lin, M., Qiu, X., He, M., Zhang, Y., & Guo, F. (2021). Effect of type 2 diabetes mellitus in retinopathy patients on MDA, SOD activity and its correlation with HbA1c. Brazilian Archives of Biology and Technology, 64, e21200075.

[9] Ghasemi-Dehnoo, M., Amini-Khoei, H., Lorigooini, Z., & Rafieian-Kopaei, M. (2020). Oxidative stress and antioxidants in diabetes mellitus. Asian Pacific Journal of Tropical Medicine, 13(10), 431–438.

[10] Haddad, N. I. A., Nori, E., & Ali, S. H. (2016). The effect of type two diabetes mellitus on superoxide dismutase (SOD) activity and its correlation with HbA1c in Iraqi patients. International Journal of Engineering Research, 2, 7–15.

[11] Pieme, C. A., Tatangmo, J. A., Simo, G., Biapa Nya, P. C., Ama Moor, V. J., Moukette Moukette, B., et al. (2017). Relationship between hyperglycemia, antioxidant capacity and some enzymatic and non-enzymatic antioxidants in African patients with type 2 diabetes. BMC Research Notes, 10, 141.

[12] Omoruyi, F., Sparks, J., Stennett, D., & Dilworth, L. (2020). Superoxide dismutase as a measure of antioxidant status and its application to diabetes. In Diabetes (2nd ed., pp. 409–417). Academic Press.

[13] Fei, Z., Gao, W., Xu, X., Sheng, H., Qu, S., & Cui, R. (2021). Serum superoxide dismutase activity: A sensitive, convenient, and economical indicator associated with the prevalence of chronic type 2 diabetic complications, especially in men. Free Radical Research, 55(3), 275–280.

[14] Palomer, X., González-Clemente, J. M., Blanco-Vaca, F., & Mauricio, D. (2008). Role of vitamin D in the pathogenesis of type 2 diabetes mellitus. Diabetes, Obesity and Metabolism, 10(3), 185–197.

[15] Taylor, A. V., & Wise, P. H. (1998). Vitamin D replacement in Asians with diabetes may increase insulin resistance. Postgraduate Medical Journal, 74(872), 365–366.

[16] Gagnon, C., Lu, Z. X., Magliano, D. J., Dunstan, D. W., Shaw, J. E., Zimmet, P. Z., et al. (2011). Serum 25-hydroxyvitamin D, calcium intake, and risk of type 2 diabetes after 5 years. Diabetes Care, 34(5), 1133–1138.

[17] Taheri, E., Saedisomeolia, A., Djalali, M., Qorbani, M., & Madani Civi, M. (2012). The relationship between serum 25-hydroxy vitamin D concentration and obesity in type 2 diabetic patients and healthy subjects. Journal of Diabetes & Metabolic Disorders, 11, 16.

[18] Bayani, M. A., Akbari, R., Banasaz, B., & Saeedi, F. (2014). Status of vitamin D in diabetic patients. Caspian Journal of Internal Medicine, 5(1), 40–42.

[19] Need, A. G., O'Loughlin, P. D., Horowitz, M., & Nordin, B. E. (2005). Relationship between fasting serum glucose, age, body mass index and serum 25-hydroxyvitamin D in postmenopausal women. Clinical Endocrinology, 62(6), 738–741.

[20] Kos, E., Liszek, M. J., Emanuele, M. A., Durazo-Arvizu, R., & Camacho, P. (2012). Effect of metformin therapy on vitamin D and vitamin B12 levels in patients with type 2 diabetes mellitus. Endocrine Practice, 18(2), 179–184.

[21] Payne, J. F., Ray, R., Watson, D. G., Delille, C., Rimler, E., Cleveland, J., et al. (2012). Vitamin D insufficiency in diabetic retinopathy. Endocrine Practice, 18(2), 185–193.

[22] Holick, M. F. (2008). Diabetes and the vitamin D connection. Current Diabetes Reports, 8(5), 393–398.

[23] Pittas, A. G., Dawson-Hughes, B., Li, T., Van Dam, R. M., Willett, W. C., & Manson, J. E. (2006). Vitamin D and calcium intake in relation to type 2 diabetes in women. Diabetes Care, 29(3), 650–656.

[24] Saif-Elnasr, M., Ibrahim, I. M., & Alkady, M. M. (2017). Role of vitamin D on glycemic control and oxidative stress in type 2 diabetes mellitus. Journal of Research in Medical Sciences, 22, 22. https://doi.org/10.4103/1735-1995.200278

[25] Singh, S. R., Hijam, D., Dubey, A., Devi, N. O., Jamir, S., Longkumer, C., et al. (2015). Study of oxidative stress status in type 2 diabetic patients. International Journal of Contemporary Medical Research, 2(1), 20–26.

[26] Aouacheri, W., Saka, S., Djafer, R., & Lefranc, G. (2009). Protective effect of diclofenac towards oxidative stress induced by paracetamol toxicity in rats. Annales de Biologie Clinique, 67(6), 619–627.

[27] Bandeira, S. M., Guedes, G. S., da Fonseca, L. J. S., Pires, A. S., Gelain, D. P., Moreira, J. C., et al. (2012). Characterization of blood oxidative stress in type 2 diabetes mellitus patients. Oxidative Medicine and Cellular Longevity, 2012, 819310.

[28] Jawad, G.S., Ghannawi, L.A., Dhaif, S.S., Abdullah, W.H., Al-Jameel, L.S., Thamer, M.A. (2026). Gut Microbiota and Diabetes Mellitus: Insights into Type 1 and Type 2 Diabetes: A Review. Dijlah Journal of Medical Sciences, 3(1): 178-185.

[29] Fu, G., et al. (2018). Shortened activated partial thromboplastin time and increased superoxide dismutase levels are associated with type 2 diabetes mellitus. Annals of Clinical & Laboratory Science, 48(4), 469–477.

[30] Bandeira, S. M., Guedes, G. S., da Fonseca, L. J. S., Pires, A. S., Gelain, D. P., Moreira, J. C., et al. (2012). Characterization of blood oxidative stress in type 2 diabetes mellitus patients: Increase in lipid peroxidation and SOD activity. Oxidative Medicine and Cellular Longevity, 2012, 819310.