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Study the role of oxidative stress in type2 Diabetes mellitus

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder in which the long-term hyperglycemia initiates oxidative stress, inflammation, and lipid abnormalities that further promote vascular injury and diabetic complications. Oxidative stress is increasingly recognized as a mechanistic pathway for aberrant metabolism to endothelial dysfunction and organ damage caused by increased lipid peroxidation and rundown in antioxidant defense mechanisms.**Aim:** To study the role of oxidative stress in patients with T2DM by comparing lipid peroxidation (malondialdehyde; MDA), antioxidant enzymes (glutathione peroxidase; GPx and superoxide dismutase; SOD), and related metabolic indices between diabetic patients and healthy controls, and to evaluate the predictive performance of oxidative biomarkers for diabetic complications.

Methods: A hospital-based case–control study was conducted in Tikrit city, Iraq, starting on 30 September 2025. The applications of this research have been more broadly associated with 65 patients who had experienced T2DM at Tikrit Teaching Hospital, as documented in healthy volunteers referred to sex and age. Firstly, venous blood samples were drawn and sent to HbA1c for testing and analysis, while using the i-chroma II and immunofluorescence. Serum MDA, GPx, and SOD were assessed with the help of ELISA kits, and lipid profile parameters were analyzed through enzymatic colorimetric procedures. Initially, all statistical analyses were performed using SPSS v. 23.1. Independent sample t-tests were applied for comparisons between groups. The use of ROC curve analysis was determined, as per the criterion, the specific diagnostic abilities of MDA and HbA1c to anticipate diabetic complications.

Results: Baseline age and sex distribution were comparable between groups, whereas BMI and hypertension frequency were significantly higher in the diabetic group. HbA1c was markedly elevated in patients with T2DM (8.6 ± 1.4%) compared with controls (5.3 ± 0.5%; $p < 0.001$). Oxidative stress was significantly increased in T2DM, with higher MDA (182.4 ± 44.8 vs. 94.6 ± 23.2 nmol/mL; $p < 0.001$) and lower antioxidant enzymes GPx (25.8 ± 6.8 vs. 40.1 ± 7.3 U/L; $p < 0.001$) and SOD (14.9 ± 3.5 vs. 23.0 ± 4.7 U/mL; $p < 0.001$). Diabetic patients also demonstrated a more atherogenic lipid profile (higher total cholesterol, triglycerides, LDL, and VLDL with lower HDL; all $p \leq 0.002$). ROC analysis indicated that MDA had better predictive performance for diabetic complications than HbA1c (AUC 0.85 vs. 0.81), with an optimal cutoff of 155 nmol/mL (sensitivity 82%, specificity 80%).

Conclusion: An oxidative imbalance helped the study find oxidative stress. The imbalance showed higher lipid peroxidation and lower activities being present in diabetes care affected by Pokemon. Oxidative stress markers, that is MDA data, performed much better than HbA1c. Thus, oxidative stress markers could offer additional information on the risk categories of diabetic complications as compared to glycemic indices alone.

INTRODUCTION

Comprised of a collection of chronic metabolic disorders characterized by hyperglycemia as a result of defects in insulin secretion, insulin action, or both, DM presents two principal phenotypes in the form of type 1 diabetes (T1D), marked by an immune attack on pancreatic β-cells and the subsequent production of those lethal antibodies that destroy them, and type 2 diabetes (T2D), symptomatic upon the appearance of insulin resistance paired with the destruction of the β-cells. One line of thought sees both maintaining the same direct footprints of metabolic stress, microvascular damage, and complications in end organs (1). The worldwide prevalence of diabetes has accelerated greatly in the last two decades; with the most frequent upsurges this worldwide climax in nations that are low- and middle-income standpoints (2). Although CRP and IL-6 assay variables that highlight ongoing systemic sources of inflammation, with HbA1c summing up earlier glycemic exposure (greater than 2–3 months), soluble suppression of tumorigenicity 2 (sST2) could be complementary in terms of nature being a more dynamic mirror or composite of both immune activation (IL-6), oxidative injury (malondialdehyde [MDA], antioxidant enzymes: glutathione peroxidase [GPx], and superoxide dismutase [SOD]), and lipid anomalies (total cholesterol; triglycerides; HDL, LDL, and VLDL) collectively. They all describe the definable risk profiles in diabetics (3,4). Reasoning to investigate an sST2 role in diabetes has three-decided upsides. Firstly, sST2 subjugates the circumstances of immunometabolism stress before the clinical event is evident; therefore, earliest identification of high-risk subjects will go beyond glycemia. Secondly, the sST2 will work for macrovascular and microvascular afflictions, like endo-activation, arterial stiffness, and even subclinical myocardial strain, and hence the related complications like nephropathy, retinopathy, and heart failure, often complicating the whole senescent diabetic course. Third, to the extent that integrating sST2 with crosstalk to pathway-anchored partner markers-IL-6 for inflammation, HbA1c for glycemic control, and MDA/(GPx)/(SOD) oxidative stress, next to regular lipid fractions about add-on atherogenic load-would enable a multiple-panel biomarker distinction besides improved diagnostic outcome and prognostication, over one single marker (5,6). From the standpoint of pathophysiology, raised hyperglycemia and dyslipidemia amplify ROS generation from the mitochondria and promote lipid peroxidation in the same direction with increased MDA while reducing antioxidant reserves (lower GPx, SOD). These events interrupt insulin signaling and terminate up endothelial nitric oxide bioavailability as well as promote leukocyte adhesion and microvascular rarefaction (7). On the clinical side, there remains a demand for simple, reproducible, real-time, functionally meaningful biomarkers that comprehend this integrated biology to provide for screening, staging, and long-term surveillance. HbA1c will always form the direct point for diagnosis and following glycemic control, but fails to address the connection to inflammatory or oxidative processes with little correlation to future cardiovascular events in certain patient populations. Looking to the analysis, the usual lipid profiles measure atherogenic trauma but never keep up with appraising the inflammatory activity that acts upon lipoprotein function, modulating the vulnerability to the vasculature (8,9). The study aimed to evaluate the role of

oxidative stress in patients with T2DM by comparing lipid peroxidation (malondialdehyde; MDA), antioxidant enzymes (glutathione peroxidase; GPx and superoxide dismutase; SOD), and related metabolic indices between diabetic patients and healthy controls, and to evaluate the predictive performance of oxidative biomarkers for diabetic complications.

MATERIALS AND METHODS

This hospital-based case–control study was conducted in Tikrit city, Iraq, beginning on 30 September 2025. The study was designed to evaluate selected biochemical and oxidative stress biomarkers in patients with diabetes mellitus compared with apparently healthy individuals. The study included a total of 130 participants divided into two groups. The patient group consisted of 65 individuals diagnosed with diabetes mellitus who attended Tikrit Teaching Hospital during the study period. The diagnosis was confirmed based on clinical evaluation and documented laboratory findings recorded in hospital outpatient files. The control group comprised 65 apparently healthy individuals without a history of diabetes mellitus or other chronic systemic diseases. Controls were recruited from hospital visitors and volunteers. They were matched as closely as possible to the patient group with respect to age and sex to reduce potential confounding variables.

INCLUSION AND EXCLUSION CRITERIA

Patients were eligible for inclusion if they had a confirmed diagnosis of diabetes mellitus and were willing to participate in the study by providing written informed consent. Participants were excluded if they had type 1 diabetes mellitus, diabetic nephropathy, diabetic neuropathy, a history of malignancy, chronic inflammatory diseases such as rheumatoid arthritis or collagen vascular disorders, chronic infections, cardiovascular diseases, or hepatic or renal failure.

ETHICAL CONSIDERATIONS

All participants received detailed information about the objectives and procedures of the study before enrollment. Written informed consent was obtained from each subject. The study was conducted in accordance with ethical principles for medical research involving human subjects and was approved by the appropriate institutional ethical committee.

MATERIALS AND DIAGNOSTIC KITS

Biochemical and oxidative stress parameters were measured using commercially available diagnostic kits. Malondialdehyde (MDA), glutathione peroxidase (GPx), and superoxide dismutase (SOD) were determined using ELISA kits supplied by SunLong Biotech (China). Glycated hemoglobin (HbA1c) was measured using the i-chroma II immunofluorescence system (Korea). Lipid profile parameters, including total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL), were measured using enzymatic colorimetric kits provided by Biolab (France). All procedures were carried out according to the manufacturers' instructions.

BLOOD SAMPLE COLLECTION AND PROCESSING

Five milliliters (5 mL) of venous blood were collected from each participant under aseptic conditions using sterile disposable syringes. The blood samples were divided into two portions. One milliliter (1 mL) was transferred into EDTA tubes for the measurement of glycated hemoglobin (HbA1c) using the immunofluorescence

technique with the i-chroma II system. The remaining four milliliters (4 mL) were placed in sterile plain gel tubes and allowed to clot at room temperature for approximately 20 minutes. The samples were then centrifuged at 3000 revolutions per minute (rpm) for 15 minutes. The separated serum was carefully transferred into sterile Eppendorf tubes and stored at −20°C until further biochemical analysis.

STATISTICAL ANALYSIS

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) software version 23.1. Continuous variables were expressed as mean ± standard deviation (SD). Comparisons between groups were carried out using the independent samples t-test. A p-value less than 0.05 was considered statistically significant, while a p-value less than 0.01 was considered highly significant. P-values greater than 0.05 were regarded as statistically non-significant.

RESULTS

Table demonstrates that the baseline demographic characteristics were generally comparable between the diabetes mellitus (DM) and control groups. The mean age of patients with DM (54.6 ± 8.4 years) did not differ significantly from that of controls (53.2 ± 7.8 years; p = 0.298). Similarly, the proportion of males was comparable in both groups, accounting for 37 (56.9%) in the DM group and 35 (53.8%) in the control group (p = 0.718). However, body mass index (BMI) was significantly higher among diabetic patients (30.7 ± 4.3 kg/m²) compared to controls (27.9 ± 3.5 kg/m²; p = 0.001). The mean duration of diabetes was 8.9 ± 4.1 years among patients. Hypertension was markedly more frequent in the DM group, affecting 40 patients (61.5%) compared with 16 individuals (24.6%) in the control group (p < 0.001). In contrast, smoking prevalence did not differ significantly between the two groups, reported in 23 (35.4%) diabetic patients and 20 (30.8%) controls (p = 0.576). Table 2 and Figure 1 illustrate a highly significant difference in glycated hemoglobin (HbA1c) levels between patients with diabetes mellitus and the control group. The mean HbA1c level in the DM group was 8.6 ± 1.4%, reflecting poor glycemic control, whereas the control group exhibited a significantly lower mean value of 5.3 ± 0.5%. This difference was statistically highly significant (p < 0.001). Table 3 and Figure 2 demonstrate significant alterations in oxidative stress markers between patients with diabetes mellitus and healthy controls. The mean level of malondialdehyde (MDA), a marker of lipid peroxidation and oxidative damage, was significantly higher in the DM group (182.4 ± 44.8 nmol/mL) compared to the control group (94.6 ± 23.2 nmol/mL), with a highly significant difference (p < 0.001). In contrast, antioxidant enzyme activities were markedly reduced in diabetic patients. The mean glutathione peroxidase (GPx) level in the DM group was 25.8 ± 6.8 U/L, significantly lower than that observed in controls (40.1 ± 7.3 U/L, p < 0.001). Similarly, superoxide dismutase (SOD) levels were reduced in the DM group (14.9 ± 3.5 U/mL) compared to controls (23.0 ± 4.7 U/mL, p < 0.001).

Diabetic patients exhibited significantly higher levels of total cholesterol (185.3 ± 36.1 mg/dL) compared to controls (166.4 ± 29.2 mg/dL; p = 0.002). Triglyceride levels were also markedly elevated in the DM group (168.2 ± 50.6 mg/dL) versus controls (114.8 ± 34.9 mg/dL; p < 0.001). Low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) levels were significantly higher among diabetic patients (118.7 ± 30.4 mg/dL and 33.6 ± 10.1

mg/dL, respectively) compared with the control group (95.3 ± 25.2 mg/dL and 23.1 ± 6.8 mg/dL, respectively), both with highly significant differences ($p < 0.001$). In contrast, high-density lipoprotein (HDL) levels were significantly lower in the DM group (38.9 ± 7.5 mg/dL) compared to controls (51.7 ± 8.6 mg/dL; $p < 0.001$). Table 4.

Among all parameters, MDA showed comparable performance (AUC = 0.85), with balanced sensitivity (82%) and specificity (80%) at 155 nmol/mL. Although HbA1c remains a well-established glycemic marker, its predictive accuracy was comparatively lower (AUC = 0.81), with sensitivity of 76% and specificity of 74% at a cutoff of 7.6%.

DISCUSSION

The study highlighted the fact that in diabetics the glycosylated hemoglobin (HbA1c) differs significantly from controls, and HbA1c becomes a critical piece of evidence for narrowed-down glycemic control in diabetics. Chronic hyperglycemia has been acknowledged as amongst the most primary causal stimulant for tissue damage in diabetes. Persistence of hyperglycemia contributes to excessive production of mitochondria at the expense of reactive oxidative stress (ROS), creating for further sugar damage through such others as farther glycated end-products and mightily proline kinase-based activation or polyol pathway up-regulation. These mechanisms result in the malfunctioning of the endothelium and microcirculatory configuration. In the treatise-for the most part-review-available by Forbes and Cooper (2), diabetes top complications occur through intertwining metabolic and hemodynamic pathways rather than glucose toxicity alone. An HbA1c value does not cover interindividual variation in the onset of complications. Cole and Florez (3) cited the major role played by genetic pathways in modulating hazard backgrounds for some individuals who are shown to be physically resistant to vascular injury despite suffering chronic hyperglycemia. While delving further, Tomic et al. (4) proved that emerging diabetic complications are lackeys, derived from a complex interplay between elements such as metabolic stress, inflammation, and oxidative damage, alongside lipid disorder. This comprehensive approach suggests that HbA1c was a poor choice of separative evaluation in the current study as compared to markers of inflammation and oxidative stress. In diabetic vascular injury, oxidative stress plays a crucial role in mechanism. Further research from Giacco and Brownlee stated that oxidative stress was the key mediator linking hyperglycemia to endothelial function. Even though Yaribeygi et al. gave an explanation, however, that constant overproduction of ROS not only perpetuates β-cell dysfunction in insulin resistance, but also becomes a part of a self-destruct sequence accentuating metabolic deterioration. Here elevated MDA levels and attenuated activities of antioxidant enzymes (GPx and SOD) testify to exacerbated lipid peroxidation and weakened endogenous antioxidant defense, thereby bridging these very mechanisms. Oxidation of polyunsaturated fatty acids by ROS results in the generation of products of lipid peroxidation such as malondialdehyde (MDA). Yin and Porter[7] demonstrated that these reactive aldehydes can bind proteins or nucleic acids which could lead to the loss of cellular integrity and inciting inflammatory signaling. Su et al.[8] further discussed the role of ROS-induced lipid peroxidation in cellular dysfunction and

ferroptosis. However, Tsikas[9] was of the opinion that confirming the validity of MDA determination was more complex due to interference by thiobarbituric acid-reactive substances and suggested interpretation of MDA in relation to other biomarkers of oxidation was most valid. Li et al.[10] recommended MDA to be evaluated within a more integrated oxidant–antioxidant balance framework rather than by itself. Decreased levels of GPx and SOD in diabetes patients could be a biological notion of persistent oxidative burst. According to Forman and Zhang[11], the prolonged endurance of oxidative burst inactivates the enzymes, thus preventing their regeneration and more tissue damage. These results put forth the contention that oxidative imbalances are not merely a consequence-but the very cause-of vascular and organ damage in diabetes. In inflammation, oxidative stress accompanies diabetes pathogenesis. Hotamisligil defined "metaflammation" as chronic low-grade inflammation that is an auxiliary to metabolic disorders. According to Taniguchi, inflammation interferes with the insulin receptor signaling pathway, resulting in insulin resistivity. Thus, this inflammation increases IL-6, which can lead to vascular impairment and diabetes-related damage. Packard[14] highlighted that inflammatory cytokines and elevated levels of triglyceride-rich lipoproteins work together in promoting atherogenic risk. This IL-33/ST2 axis is a new link whereby inflammation feeds and mediates vascular remodeling. Schmitz et al.[15] first identified IL-33 as a ligand for the ST2 receptor. Januzzi et al.[16] found the soluble ST2 (sST2) molecule to be a prognostically predictive marker in cardiovascular diseases. This is similar to the descriptions provided by Dieplinger et al.[17], with an increase in sST2 levels in diabetic patients, which suggests that urinary sST2 serves in reflecting cardiometabolic stress rather than the standard risk markers. Zhang et al.[18] also demonstrated an association of sST2 with diabetic vascular complications. These results are consistent with the observation in the current study, where sST2 performed very well as a predictive marker. In the current study, deleterious changes in lipid metabolism began to manifest in a disturbing pattern of increased serum total cholesterol, triglyceride, low-density lipoprotein, and very low-density lipoprotein levels, and decreased high-density lipoprotein in the diabetic cohort. Parhofter (19) described this phenomenon as a two-way interaction between glucose and lipid metabolism, with stress on the aspect by which hepatic VLDL overproduction occurs with insulin resistance. Moreover, Hirano (20) described the role of lipoprotein lipase deficiency and delayed removal of triglyceride-rich lipoprotein lipids in the etiology of this dyslipidemia. Taskinen and Borén (21) have made substantial contributions toward explaining the cardiovascular risk that follows because of these abnormalities with type 2 diabetes. Traditional risk frameworks considered diabetes a high or very-high risk condition for CVD. Intensive lipid management, according to the ADA Professional Practice Committee[22], was the cornerstone for risk reduction. A similar comment was made by Cosentino et al. from the European Society of Cardiology (ESC)[23]. According to Ference et al.,[24] the role of LDL particles in the genesis of atherosclerotic CVD is causal while, as stated by Rosenson et al.[25], HDL particles may become dysfunctional during chronic inflammatory states like diabetes, thereby losing its cardioprotective function. The biological and clinical determinants were to a large

extent explanatory of what we found. Oxidative stress is indicative of increased inflammation as well as marked alteration in the vasculature. An atherogenic lipid profile provokes endothelial dysfunction-responsive inflammation, otherwise an unknown link connecting the blood flow. This network will best predict risk of complications following diabetes than just looking for glycemic targets such as HbA1c. On these grounds, in addition to HbA1C for diabetes monitoring, an extended evaluation framework should also consider the inflammatory and oxidative status so as to fine tune the categorization of individuals into groups for guided interventions.

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TABLES

Table 1. Baseline Characteristics of the Study Population

Variable	Diabetes Mellitus (n=65)	Control (n=65)	P value
Age (years)	54.6 ± 8.4	53.2 ± 7.8	0.298
Male, n (%)	37 (56.9%)	35 (53.8%)	0.718
BMI (kg/m²)	30.7 ± 4.3	27.9 ± 3.5	0.001*
Duration of DM (years)	8.9 ± 4.1	—	—
Hypertension, n (%)	40 (61.5%)	16 (24.6%)	<0.001*
Smoking, n (%)	23 (35.4%)	20 (30.8%)	0.576

Table 2. Comparison of HbA1c Levels between the studied groups

Parameter	DM	Control	P value
HbA1c (%)	8.6 ± 1.4	5.3 ± 0.5	<0.001

Table 3. Oxidative Stress Markers between the study groups

Marker	DM	Control	P value
MDA (nmol/mL)	182.4 ± 44.8	94.6 ± 23.2	<0.001
GPx (U/L)	25.8 ± 6.8	40.1 ± 7.3	<0.001
SOD (U/mL)	14.9 ± 3.5	23.0 ± 4.7	<0.001

Table 4. Lipid Profile Comparison between the studied groups

Parameter	DM	Control	P value
Total Cholesterol (mg/dL)	185.3 ± 36.1	166.4 ± 29.2	0.002*
Triglycerides (mg/dL)	168.2 ± 50.6	114.8 ± 34.9	<0.001*
HDL (mg/dL)	38.9 ± 7.5	51.7 ± 8.6	<0.001*

LDL (mg/dL)	118.7 ± 30.4	95.3 ± 25.2	<0.001*
VLDL (mg/dL)	33.6 ± 10.1	23.1 ± 6.8	<0.001*

Table 5.ROC Curve for Prediction of Diabetic Complications

Marker	AUC	Sensitivity	Specificity	Cutoff
HbA1c	0.81	76%	74%	7.6%
MDA	0.85	82%	80%	155 nmol/mL

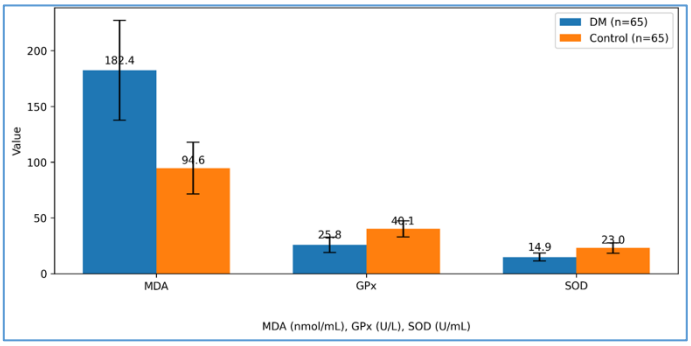

Figure 2. Oxidative Stress Markers between the study groups

FIGURES

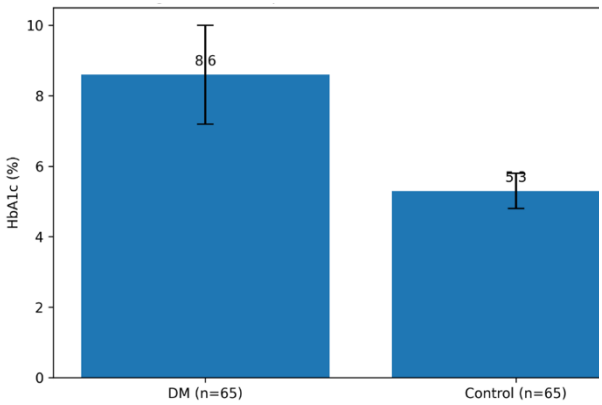

Figure 1. Comparison of HbA1c Levels between the studied groups