

ORIGINAL A R T I C L E

Evaluation of Oxidative Stress Parameters and Antioxidant Status in Diabetes Type 1 Patients at Dhi Qar, Iraq

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Abstract

Background Oxidative stress and low antioxidant defense have a role in the initiation and progression of complications related to type 1 diabetic mellitus (T1DM).

Objective of the study The study aims to evaluate oxidative stress parameters and antioxidant status, which were assessed, compared, and correlated with controls.

Materials and Methods A case-control study design was employed from June 1, 2023, to February 1, 2024; the present study included 100 age ranges (30–75) years of clinically proven T1DM patients selected from Al Hussein Teaching Hospital and 100 controls in the same age range and with no history of T1DM were chosen after undergoing health checkups in the hospital. Measured were for HOMA IR, MDA, CAT, insulin, fasting blood sugar, haemoglobin A1C%, and T-AOC. Both inferential and descriptive statistical analyses were applied to the data.

Results The results showed that MDA was considerably higher in patients than in controls ($P < 0.05$), although T-AOC and CAT were significantly lower in patients than in controls ($P < 0.05$). T1DM patients showed significantly higher HOMA-IR, HbA1c, and insulin levels.

Conclusion Patients with type 1 diabetes exhibit higher oxidative stress and a lower antioxidant level. Future therapy plans could focus on increasing antioxidants and lowering oxidative stress in patients with diabetes mellitus.

Keywords: T1DM; MDA; T-AOC; CAT; Insulin and HOMA IR.

1 Introduction

Free radicals are very reactive chemicals that may harm macromolecules, including proteins, lipids, carbohydrates, and nucleic acids, causing oxidative dam-

age to living things. Under typical physiological circumstances, organisms maintain a balance between producing oxygen-free radicals and using antioxidant defence mechanisms to mitigate and safeguard against the harmful effects of free radicals [1, 2]. Oxidative

stress is a situation that arises from an imbalance in the oxidant/antioxidant equilibrium. Oxidative stress is recognized as a contributing factor to the molecular and cellular damage seen in several clinical illnesses [3, 4]. The most significant metabolic change linked to diabetes is hyperglycemia. The exact mechanism by which hyperglycemia may contribute to the development of coronary heart disease (CHD) is a matter of debate [5]. The development of vascular disease is caused by hyperglycemia in both type I and type II diabetes, which has several consequences on the body, including cellular damage, increased creation of extracellular matrix, and vascular dysfunction [6]. Among the mechanisms responsible for the elevated oxidative stress in diabetes are changes in the quantity and functionality of antioxidant defence systems within the tissues, as well as the production of oxygen-free radicals due to the lack of enzymatic glycosylation (glycation) and the auto-oxidation of glycation products [7]. Enzymatic and non-enzymatic antioxidant mechanisms regulate an unequal rise in reactive oxygen species (ROS) under standard physiological homeostatic settings [8]. The presence of the conventional risk factors listed above, such as diabetes, obesity, and hypertension, raises the amount of ROS [9]. Myeloperoxidase (MPO) and other pro-oxidant enzymes are synthesized more significantly as ROS concentrations rise. Leukocytes, primarily neutrophils and partially monocytes, produce MPO from their azurophilic granules [10]. When made during activation, MPO catalyzes the formation of hypochlorous acid (HOCl) in the presence of halides and hydrogen peroxide. As a result, hypochlorous acid may either react with lipoproteins to oxidize them or combine with superoxides to form hydroxyl radical, an activator of lipid peroxidation [11, 12].

According to epidemiological research that is now accessible, diabetes-type 1 disorders are linked to lower levels of antioxidants and higher levels of oxidants. Nevertheless, little is known about the degree of the imbalance between oxidant and antioxidant enzymes or the total amount of oxidant and antioxidant indicators.

2 Materials and Methods

2.1 Study subjects and samples collection

2.1.1 Study design

The present study included 100 age ranges (30–75) years of clinically proven T1DM patients selected from Al Hussein Teaching Hospital, and 100 controls in the same age range with no history of T1DM were chosen after undergoing health checkups in the hospital. Blood samples were centrifuged for 10–15 minutes

at 3000 rpm, and serum was subsequently separated. Samples were stored at -80°C until the time of analysis. Blood samples were collected from each patient after obtaining written informed consent per the criteria laid down by the scientific and ethical committee of the university. Detailed demographic, anthropometric, and other relevant information was recorded using Proforma. Peripheral blood (4 ml) was collected from all participants and analyzed to obtain the mentioned biochemical parameters.

2.2 Kits

The kits used in this study are shown in Table 1.

Table 1: Distribution of demographical and employment data for health care providers.

No	Kit	Company	Country
1	Glucose kit	Biosystem	Spain
2	Hemoglobin A1C-Direct Total Antioxidant Capacity	Biosystem	Spain
3	(T-AOC) Colorimetric Assay Kit	Elabscience	USA
4	Human Insulin ELISA Assay	Elabscience	USA

2.3 Methods

HOMA IR, MDA, CAT, insulin, fasting blood sugar, haemoglobin A1C (HbA1c)%, and T-AOC.

3 Measurement of malondialdehyde

Malondialdehyde concentrations in plasma samples were measured using high-performance liquid chromatography (HPLC) with fluorescence detection as described by Öhrvall et al. [13]. To execute a thiobarbituric acid reaction, 200 μL of the plasma sample, 750 μL of 0.15 M phosphoric acid, 300 μL of water, and 250 μL of 42 mM thiobarbituric acid were mixed. The reaction mixture was allowed to cool on ice after a 60-minute incubation period in a boiling water bath. The malondialdehyde–thiobarbituric acid combination was extracted with methanol, and 20 μL of the mixture was put into an HPLC column (LiCrospher 100 RP-18, Merck, Darmstadt, Germany; 250×4 mm). The mobile phase included methanol–50 mM phosphate buffer (2:3). Fluorescence was seen with an excitation length of 532 nm and an emission wavelength of 553 nm.

3.1 Determination of human insulin

This ELISA kit uses the Sandwich-ELISA principle.

3.2 Determination of the homeostatic model assessment for insulin resistance

The Homeostatic model assessment of insulin resistance (HOMA-IR) was calculated with the following formula (glucose levels in mg/dL, insulin levels in μ IU/mL) [14]:

$$HOMA - IR \equiv \frac{Glucose \times Insulin}{405}$$

3.3 Statistical analysis

Microsoft Office Excel 2013 and GraphPad Prism 9.2.0 were used to gather, analyze, and present the data. Numbers were used to represent categorical data, while mean Standard Error Mean was used to convey numerical data. An unpaired t-test and a one-way ANOVA were used to compare the mean values across the different groups for regularly distributed variables. A chi-square analysis was performed on the qualitative data. Bivariate correlation was carried out using Pearson's correlation coefficient. When the P-value was less than 0.05, it was deemed significant.

4 Results

The research aimed to evaluate antioxidant levels and oxidative stress indicators in individuals with type 1 diabetes. T1DM patients had assessments for MDA, CAT, antioxidants, fasting blood sugar, haemoglobin A1C (HbA1c)%, insulin, and HOMA IR. The results

were contrasted with the values obtained for the control group (individuals in the same age range). In all, 100 T1DM patients and 100 controls participated in the research. Gender-wise, there were 30 (30%) and 70 (70%), respectively, female and male, patients out of 100, and 50 (50%) and 50 (50%) subjects out of 100 controls. The patient and control groups had mean $\pm$ SD ages of 54.2 (with an age range of 30-70) and 52.3 (with an age range of 30-70), respectively. Baseline characteristics of T1DM patients and controls are presented in Table 2.

Table 2: Baseline characteristics of T1DM patients and control group.

Baseline characteristics		Control (n=100)	T1DM (n=100)
Gender	Age	52.3 (30-70)	54.2(30-70)
	Male	50 (50 %)	70 (70 %)
	Female	50 (50 %)	30 (30 %)
	BMI (kg/m ²)	23.21 $\pm$ 2.17	31.24 $\pm$ 3.24

4.1 Biochemical analysis

4.1.1 Type 1 diabetes mellitus measurements, diagnosis

The following metrics were compared between individuals with T1DM and the control group: insulin, HOMA IR, HbA1c%, and fasting blood sugar. Insulin, HOMA IR, haemoglobin A1C (HbA1c)%, and fasting blood sugar were much higher in T1DM patients than in controls. Are shown in Table 3.

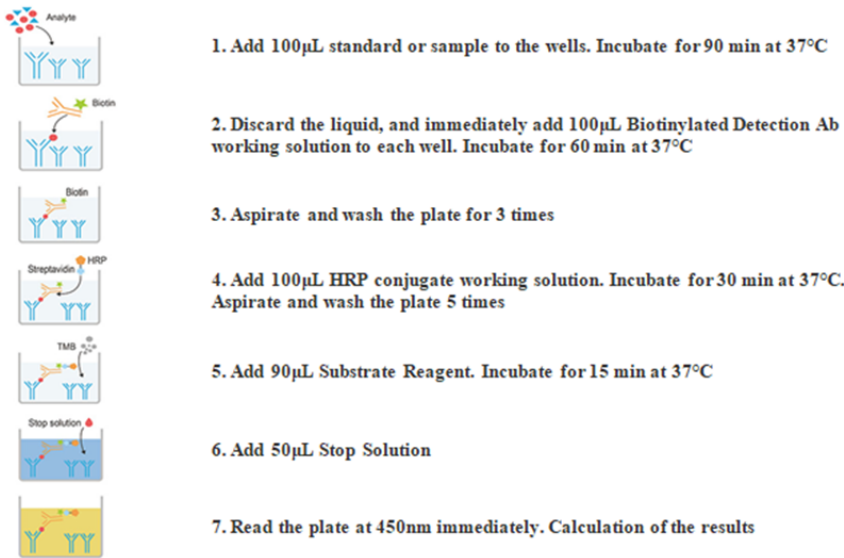
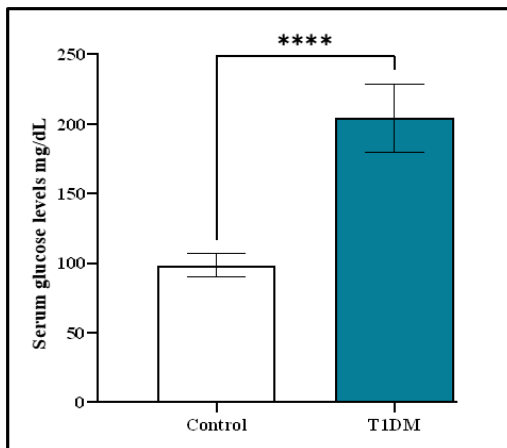
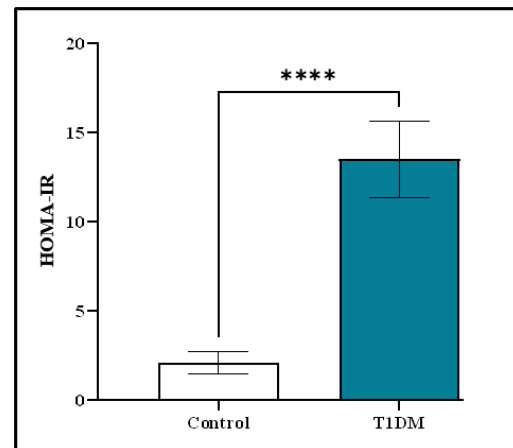
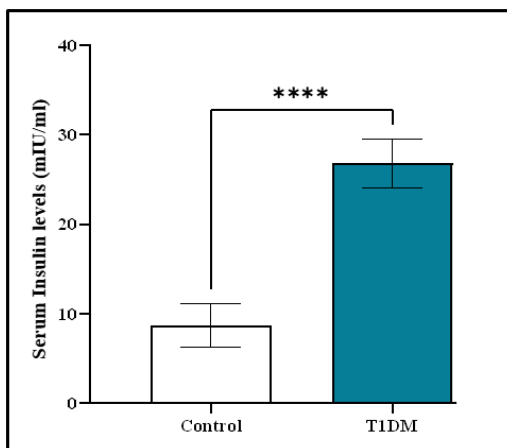
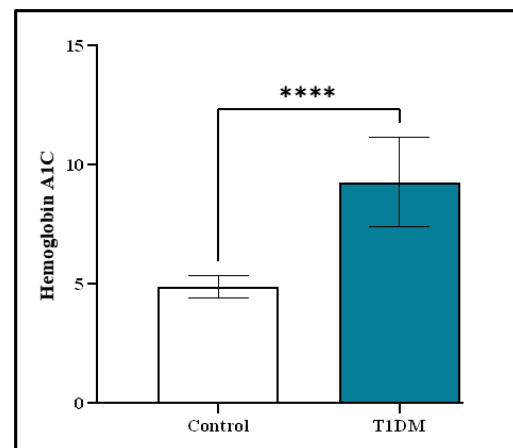


Figure 1: Assay procedures summary.

Table 3: Comparison of mean values of the studied biomarkers among the control group and patients with T1DM.

Characteristic	Control n=100	Diabetes n=100	P value
Serum Glucose levels (mg/dl)			
Range	83.33 - 121.7	179.9 - 303.2	<0.0001
Mean $\pm$ SD	98.49 $\pm$ 8.069	204.2 $\pm$ 24.27	****
Insulin (mIU/ml)			
Range	4.11 - 11.9	13.6 - 31.2	<0.0001
Mean $\pm$ SD	8.67 $\pm$ 2.479	26.81 $\pm$ 2.758	****
HOMA IR			
Range	1 - 3	6.7 - 20.14	<0.0001
Mean $\pm$ SD	2.105 $\pm$ 0.6128	13.52 $\pm$ 2.142	****
Hemoglobin A1C			
Range	4.11 - 5.93	7.06 - 13.6	<0.0001
Mean $\pm$ SD	4.87 $\pm$ 0.4809	9.258 $\pm$ 1.875	****

**Figure 2:** Estimation of serum concentrations of glucose (mg/d).**Figure 4:** Estimation of serum concentrations of HOMA IR.**Figure 3:** Estimation of serum concentrations of insulin (mIU/ml).**Figure 5:** Estimation of serum concentrations of hemoglobin A1c.

4.1.2 Oxidative stress and antioxidant status

The antioxidant enzyme CAT and the oxidative stress-related enzyme MDA are measured and compared in

T1DM patients and controls. Table 4 demonstrated that the MDA ratio was much higher in T1DM patients than in controls and that T-AOC and CAT were significantly lower in T1DM patients than controls.

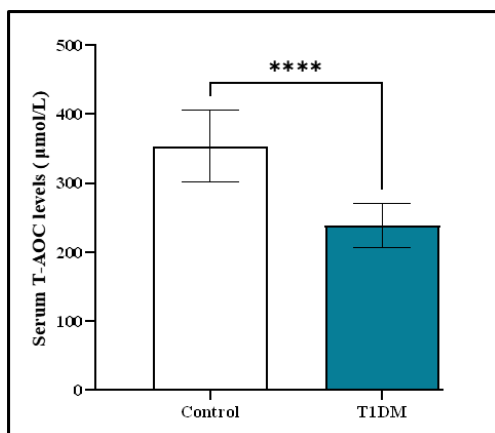


Figure 6: Estimation of serum concentrations of Total Antioxidant Capacity (T-AOC) (U/mL).

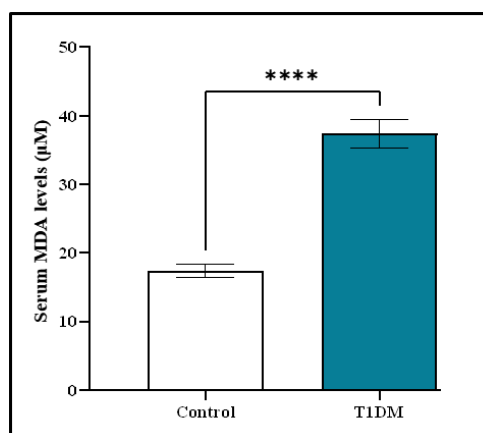


Figure 7: Estimation of serum concentrations of malondialdehyde (MDA) (μM).

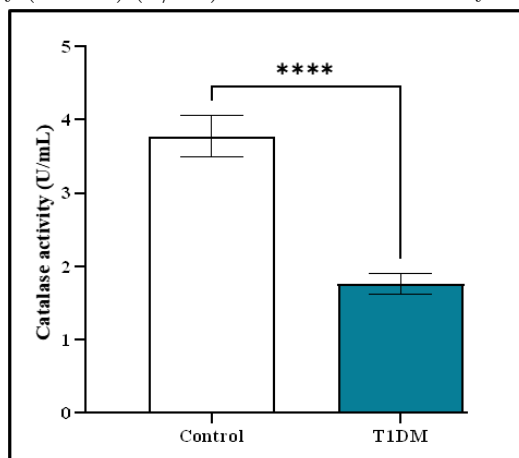


Figure 8: Estimation of Catalase activity U/ml.

Table 4: T-AOC, MDA, and CAT in T1DM patients and control subjects.

Characteristic	Control	Diabetes	P value
	n=100	n=100	
Serum T-AOC levels (U/mL)			
Range	233.1 - 497.9	167.2 - 308.2	<0.0001
Mean ± SD	353.6 ± 52.08	238.2 ± 31.84	****
Serum MDA levels (μM)			
Range	11.3 - 31.37	22.48 - 64.62	<0.0001
Mean ± SD	17.38 ± 5.924	37.4 ± 13.16	****
Catalase activity U/ml			
Range	1.38 - 7.95	0.15 - 3.69	<0.0001
Mean ± SD	3.774 ± 1.825	1.76 ± 0.9464	****

5 Discussion

Diabetes mellitus is a long-term, systemic metabolic disorder characterized by changes in the metabolism of fat, protein, and carbohydrates, as well as hyperglycemia. It is believed that oxidative stress is elevated when free radicals are generated and the antioxidant systems are compromised. In recent years, free radicals produced by oxidative stress have been linked to the pathophysiology of IDDM [15–17]. The current research examined how a T1DM patient's antioxidant status and levels changed. Extended periods of high blood sugar levels, which lead to non-enzymatic protein posttranslational modifications called glycation, a chemical interaction between glucose and primary amino groups in proteins, as well as elevated levels of oxygen free radicals as a consequence of glucose autooxidation [18–20].

Our results support previous studies showing that high blood sugar levels lead to diabetes, produce more oxygen free radicals, and speed up the oxidation of fats and proteins (Table 3). The much-increased serum MDA levels suggest that people with diabetes cause more free radical-mediated oxidative damage to proteins and lipids [21–23]. A substantial portion of catalytically inactive or less active versions of enzymes are oxidized proteins, which may have direct metabolic repercussions [24, 25]. Gliesner et al. [21] report that no statistically significant differences were seen for any of the oxidative stress markers (PCG) assessed between patients with DM1 and controls [21].

Additionally, there were no differences in the groups' weight, height, or results from regular metabolic testing like creatininemia and cholesterol levels. The absence of statistically significant differences between DM1 patients and healthy controls indicated that the increased generation of free radicals may be countered by therapy. In diabetes individuals, glycated and oxidized protein proteolytic products were shown to be significantly elevated by Ahmed et al. [26]. Still, glycated and oxidized protein adduct residues increased considerably less rapidly.

The theory that radical-mediated damage plays a role in diabetes illness is further supported by the reduced catalase activity in these individuals. These findings concur with those of others [27–29]. Free radicals are sucked up by circulating red blood cells. As a result, erythrocytes experience an ongoing flow of H₂O₂ and O₂. Given that SOD may catalyze the dissimulation of two superoxide radicals into H₂O₂, it is plausible that this enzyme plays a significant physiological function in thwarting this process.

Subjects with type 1 diabetes showed a substantial decrease in plasma TAOC. At least four investigations have previously demonstrated a deficient total antioxidant defence in persons with type 1 dia-

betes mellitus [5–8]. Studies, however, have also shown no appreciable variation in the antioxidant level between patients and healthy controls [21, 22]. We discovered that people with type 1 diabetes had considerably lower amounts of urate among the individual antioxidants than the controls. Up to two-thirds of the TAOC comes from this water-soluble antioxidant, the leading provider of antioxidant defence [8, 27]. We showed a strong association between TAOC and urate plasma levels to support this. In addition to pointing to a compromised state of protection against oxidation, the apparent irregularity of chain-breaking antioxidant defence in diabetic people may also point to an enhanced antioxidant consumption state brought on by acute radical scavenging. Hypouricemia may also be caused by glucose-induced increased fractional excretion of uric acid [28].

In summary, research suggests that the generation of free radicals and other reactive oxidative species is elevated in cases of hyperglycemia. The type 1 diabetes participants and the nondiabetic control group showed substantial differences in MDA levels. Still, we also detected signs of a reduced antioxidant state.

6 Conclusion

In summary, The antioxidant state and oxidative indicators in T1DM patients, paying particular attention to pro-oxidant and antioxidant enzymes. There was a rise in the oxidative marker MDA. In confirmed cases of type 1 diabetes, antioxidant enzyme levels are decreased by T-AOC and CAT. External antioxidants might be administered to lessen oxidative stress in these individuals. Important pro-inflammatory indicators that are part of essential pathways in atherogenesis should also be thoroughly investigated.

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Conflict of Interest: No conflicts of interest exist between the authors and the publication of this work.

Ethical consideration: The ethical committee approved the study at University of Thi-Qar, Thi Qar, Iraq.

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