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A Cross-Sectional Study to Evaluate the Link between Oxidative Stress and Increased Cardiovascular Risks in Prediabetic Patients

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Abstract

Prediabetes is associated with dysglycemia; besides progression to diabetes mellitus, prediabetic patients are at risk for cardiovascular disease (CVD). The current study is intended to evaluate the role of oxidative stress in the development of cardiovascular disease (CVD) in prediabetic patients. Therefore, the biomarkers of oxidative stress were assessed and correlated with possible cardiovascular risk factors, including body mass index (BMI), hypertension, and dyslipidemia. With a total population of 70 individuals, this descriptive study of cross-sectional design was conducted, including 40 patients with pre diabetes and 30 subjects as the control group. Age and BMI were matched between the two groups. The concentrations of reduced glutathione (GSH), malondialdehyde (MDA), lipid profile, and fasting blood sugar (FBS) were analyzed using blood serum samples. Whereas glycated hemoglobin (HbA1c) was measured using whole blood. In comparison to controls, prediabetic patients showed a significant elevation in mean values of systolic blood pressure (SBP), body mass index (BMI), fasting blood sugar (FBG), glycated hemoglobin (HbA1c), total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL), very-low-density lipoproteins (VLDL), and malondialdehyde (MDA), with a significant reduction in mean values of reduced glutathione (GSH) and high-density lipoproteins (HDL). Concentrations of fasting blood sugar (FBS) and glycated hemoglobin (HbA1c) recorded a remarkable positive and significant correlation with MDA and cardiovascular risk factors, and a negative and significant correlation with GSH and HDL. MDA recorded a substantial positive correlation with cardiovascular risk factors in prediabetic subjects, whereas GSH had a considerable negative correlation with cardiovascular risk factors. In conclusion, the findings of this study conclude that prediabetes is closely associated with elevated levels of oxidative stress biomarkers and confirm the link between oxidative stress and increased cardiovascular risks in these patients.

1. Introduction:

Prediabetes is defined as a rise in blood glucose levels beyond normal but below the diagnostic criteria of diabetes, as indicated by impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or a glycated hemoglobin (HbA1c) level of 5.7% to 6.4%. It is a premorbid disease linked to a higher

chance of developing type 2 diabetes mellitus (T2DM) [1].

According to evidence from prospective and observational studies, untreated prediabetes will progress to diabetes and related complications in 25-40% of cases after three to eight years [2]. In addition, prediabetes is associated with oxidative stress, endothelial dysfunction, dysglycemia, dyslipidemia, obesity, pro-thrombotic status, hypertension, insulin resistance, and inflammation, placing prediabetic patients at an increased risk of developing cardiovascular diseases [3].

However, high blood sugar levels result in oxidative stress due to many metabolic processes producing excess free radicals [4]. The possible causes of oxidative stress in patients with hyperglycemia might include alterations of glucose, increased activity of free radical-induced lipid peroxidation, and impaired activities of the defensive system consisting of antioxidant enzymes [5]. Lipid peroxidation, oxidized proteins, and oxidative nucleic acid damage are some of the biomarkers that can be used to estimate oxidative stress [6]. Malondialdehyde (MDA) is the most prominent and well-studied endproduct of lipid peroxidation and is frequently used as an indicator of free radical damage [7].Potential applications for lipid peroxidation products include early diagnosis of disease, monitoring disease development, and assessing the therapeutic efficacy [8].

Both a deficiency in antioxidant defense mechanisms and a proliferation of reactive oxygen species (ROS) are responsible for oxidative stress [9]. In several pathological states, it is known that the change in redox balance in impacted fluids, tissues, or organs results in variations in antioxidant activity. Therefore, the level of oxidative stress has been estimated using measurements of antioxidant content or activity [10].

The most important endogenous antioxidant is glutathione (GSH). It detoxifies reactive oxygen species (ROS), prevents ROS from damaging cells, and protects against oxidative stress. It also acts as a substrate for glutathione peroxidase-1 (Gpx-1), an enzyme whose inadequacy has been linked to a rise in the risk of cardiovascular events and coronary artery disease. However, studies regarding the role of oxidative stress in the prediction of cardiovascular disease in prediabetes are still lacking [8].

In Iraq, no studies have been established to provide data addressing the link between oxidative stress and an elevated risk of cardiovascular disease in patients with prediabetes. Accordingly, the preliminary aim of the present study is to give an idea regarding this relationship in prediabetes by assessing oxidative stress biomarkers and their correlation with the possible risk factors for cardiovascular disease, including dyslipidemia, obesity, and hypertension.

2. Materials and Methods:

2.1 Subjects and Study Design:

Ln Ranye/Sulaymaniyah, this cross-sectional study was conducted in 2023 between May 1 and July 31. A total of 70 subjects of either gender aged 32–54 years were randomly selected from the prediabetic outpatient clinic at Smart Hospital. At the time of subject recruitment, a questionnaire form was designed to collect health-related information during an in-person interview. Participants were divided into 2 groups: 40 prediabetic and 30 healthy control subjects. According to the American Diabetes Association (ADA) criteria, inclusion criteria were prediabetic patients with a FBG level of ≥ 100 and < 126 mg/dL and HbA1C level of 5.4% to 6.4%. Exclusion criteria were type 2 diabetes, cardiovascular disease (CVD), chronic liver disease, chronic renal disease, chronic inflammatory disease, and malignancy.

2.2 Ethical Approval:

The ethics committee of the Faculty of Science at Raparin University formally approved the study proposal. All individuals provided informed written consent prior to sample collection, and all procedures followed Iraqi regulations and protocols concerning biomedical research.

2.3 Biochemical Measurements:

For biochemical analysis, venous blood samples were taken from all subjects after at least 10–12 hours of overnight fasting under all aseptic precautions. Glycated hemoglobin (HbA1C) was estimated directly using whole blood, and another set of samples was clotted at room temperature for 15 minutes and then centrifuged for 10-15 minutes at 4,000 rpm for serum separation. Glycated hemoglobin (HbA1C), fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) concentrations were measured enzymatically with a chemical autoanalyzer (Cobas Integra 400, Roche Diagnostics, Mannheim, Germany) in according to the manufacturer's instructions.

2.4 MDA and GSH Measurements:

Evaluation of serum MDA and GSH was performed by a High-Performance Liquid Chromatography (HPLC) system with a Smart Line Manager 500-KNAUER, an autosampler 3950, and a Pump 1050. UV absorbance detectors were used to measure the eluted peaks. Highest purity chemicals and reagents were run for quantitative and qualitative analysis of standards under the same conditions as the samples. 1,1,3,3-tetraethoxypropane (TEP), hydrochloric acid (HCL), perchloric acid (HCLO4), sodium perchlorate (NaClO4.H2O), and phosphoric acid (H3PO4) were obtained from Sigma Chemical Company, Istanbul, Turkey. To prepare the MDA standard, 10 μL of 1,1,3,3-tetraethoxypropane (TEP) was dissolved in 10 mL of 0.1M HCL and incubated in the boiling water bath for 10 minutes to give a 2.92 μg/mL stock solution.

After quickly cooling, a series of standard solutions were prepared by dilution with double-distilled water to yield 6 different concentrations of 10, 5, 2, 1, 0.5, and 0.2 μg/mL to get a calibration curve for determination of serum MDA. A GSH

standard was prepared by dissolving 1 mg of standard GSH in 10 mL of double-distilled water to give a 1000 μg/mL stock solution. Different concentrations of the working standard were prepared by further dilution with double-distilled water to get the calibration curve for determination of serum GSH.

For protein precipitation and release of both MDA and GSH, 0.3 mL of 0.5 M perchloric acid (HCLO4) was added to 0.3 mL of serum. Then the sample solution was diluted to 3.0 mL with double-distilled water and vortexed for 20 seconds. After centrifugation for 10 minutes at 3000 rpm, 1 mL of the supernatant was immediately injected into the HPLC system. MDA and GSH were determined separately using an Agilent 1100 series HPLC apparatus (Germany). The analytical column was an ODS-2 C18 reserve phase column with a particular size of 5 μm (Thermo, England).

The mobile phase was 3.6 g of sodium perchlorate (NaClO4.H2O) dissolved in 375 mL of double-distilled water containing 0.34 mL of phosphoric acid (H3PO4). The separation process was performed at PH adjusted to 4 with a flow rate of 1.5 mL/min, and the UV detector was set at 254 and 210 nm for detection of MDA and GSH, respectively. The calibration curve was used to compute the concentrations of MDA and GSH, which were represented as μg/mL [11, 12].

2.5 Statistical Analysis:

Data analysis was done using SPSS statistical software version 23 (IBM Corporation, Armonk, NY, USA). The normality of distribution of variables was checked using the kolmogorov–smirnov test. The variation of continuous variables between normal and prediabetic patients was estimated by an independent sample t test and reported as mean $\pm$ standard deviation. A chi-square test was used to determine categorical variables between the two groups. Possible associations between the studied parameters were evaluated using Pearson correlation analysis, and a significance threshold of less than 0.05 was set for the p-value.

3. Result:

Figure 1 illustrates the frequency distribution of the study participants. In this study, a total of 70 subjects were enrolled. Among them, 40 (57.1%) were prediabetic, while 30 (42.9%) were free from pre diabetic conditions. Anthropometric parameters and blood pressure measurements of the study participants are displayed in Table 1. The results clarify non-significant variation in terms of age, sex, family history of DM, and family history of CVD between the two groups. In comparison to the control group, the prediabetic group showed significantly increased mean values of BMI and SBP (29.79 $\pm$ 4.61 kg/m2 vs. 26.49 $\pm$ 4.77 kg/m2, p = 0.035; 128.00 $\pm$ 14.73 mmHg vs. 118.42 $\pm$ 11.19 mmHg p = 0.029). However, considering DBP, the difference between the mean values of both groups remains non-significant.

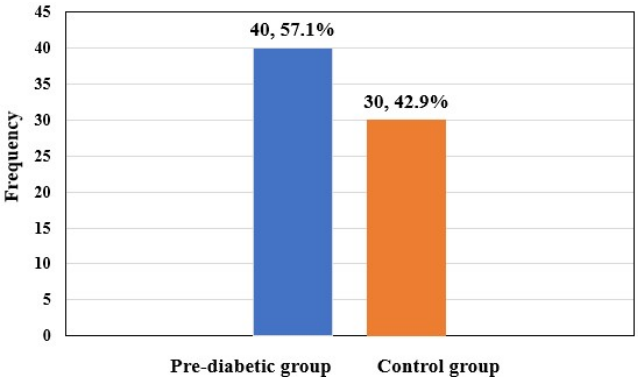

Figure 1. Distribution of the study participants.

Table 1. Principal characteristics of prediabetics and controls.

Variables	Prediabetics (n =40)	Controls (n = 30)	p-value
Age (years) Mean $\pm$ SD	40.10 $\pm$ 6.07	39.789 $\pm$ 5.35	NS
Sex Male Female	14(58.3%) 26(56.5%)	10(41.7%) 20(43.5%)	NS
Family history of DM no. (%) Yes No	17(58.6%) 23(56.1%)	12(41.4%) 18(43.9%)	NS
Family history of CVD no. (%) Yes No	15(60%) 25(55.6%)	10(40%) 20(44.4%)	NS
BMI (kg/sqm) Mean $\pm$ SD	29.79 $\pm$ 4.61	26.49 $\pm$ 4.77	0.035
SBP (mmHg) Mean $\pm$ SD	128.00 $\pm$ 14.73	118.42 $\pm$ 11.19	0.029
DBP (mmHg) Mean $\pm$ SD	80.50 $\pm$ 12.76	73.16 $\pm$ 11.08	NS

BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure. Results are expressed as mean $\pm$S D and (no. %). no = subjects' number, NS=non-significant.

The means of the studied groups based on blood levels of FBG, HbA1c and lipid profile parameters are included in Table 2. When compared to normal individuals, prediabetic individuals had significantly higher mean values of FBG (119.17 ± 3.71 mg/dL vs. 94.63 ± 3.961 mg/dL, p ¡ 0.001), HbA1c (5.79 ± 0.35 mg/dL vs. 4.74 ± 0.36 mg/dL, p ¡ 0.001), TC (181.95 ± 21.71 mg/dL vs.161.62 ± 21.40 mg/dL, p = 0.009), TG (149.25 ± 52.78 mg/dL vs.114.33 ± 22.94 mg/dL, p = 0.020), LDL (116.75 ± 17.98 mg/dL vs. 100.60 ± 23.52 mg/dL, p = 0.023), and VLDL (32.01 ± 10.22 mg/dL vs. 23.36 ± 4.34 mg/dL, p = 0.002). Reversely, there was a considerable drop in HDL levels (39.55 ± 7.09 mg/dL vs. 45.94 ± 6.30 mg/dL, p = 0.022) in the first group in comparison to the second group.

Table 2. Statistical description of FBS, HbA1c, and lipid profile in prediabetes and controls.

Biochemical parameters	Prediabetic (n=40) Mean ± SD	Control (n=30) Mean ± SD	p-value
FBG (mg/dl)	119.17 ± 3.71	94.63 ± 3.96	< 0.001
HbA1c	5.79 ± 0.35	4.74 ± 0.36	< 0.001
TC (mg/dl)	181.95 ± 21.71	163.27 ± 21.40	0.009
TG (mg/dl)	149.25 ± 52.78	114.33 ± 22.94	0.020
LDL (mg/dl)	116.75 ± 17.98	100.60 ± 23.52	0.023
VLDL (mg/dl)	32.01 ± 10.22	23.36 ± 4.34	0.002
HDL (mg/dl)	39.55 ± 7.09	44.94 ± 6.30	0.022

FBG: Fasting blood glucose; TC: Total cholesterol; TG: Triglycerides; LDL: Low-density lipoprotein; VLDL: Very low-density lipoprotein; HDL: High-density lipoprotein. Results are expressed as mean ± SD.

Outcomes for oxidative stress biomarkers are displayed in Table 3. Significant and extreme reductions in serum GSH levels (117.03 ± 31.29 μg/mL vs.172.01 ± 48.49 μg/mL, p ¡ 0.001) and an intense elevation in serum MDA levels (0.26 ± 0.20 vs. 0.107 ± 0.07, p = 0.005) were noticed in the prediabetic group compared to the healthy control group.

Table 3. Statistical description of oxidative stress biomarkers in general population.

oxidative stress biomarkers	Prediabetic (n=40) Mean ± SD	Control (n=30) Mean ± SD	p-value
GSH (μg/mL)	117.03 ± 31.29	172.01 ± 48.49	< 0.001
MDA(μg/mL)	0.260 ± 0.087	0.107 ± 0.07	0.005

GSH: Reduced Glutathione; MDA: Malondialdehyde. Results are expressed as mean ±S

Table 4 signifies the presence of a significant direct correlation between MDA and BMI, SBP, DBP, TC, TG, LDL, and VLDL. Inversely, a significant negative correlation was noted between MDA and HDL.

Table 4. Correlation analysis between serum MDA levels and risk factors for CVD.

Cardiovascular RiskFactors	Pearson Correlation (r)	p-value
BMI (kg/sqm)	0.371*	0.022
SBP (mmHg)	0.375*	0.020
DBP (mmHg)	0.300*	0.030
TC (mg/dl)	0.419*	0.010
TG (mg/dl)	0.313*	0.026
LDL (mg/dl)	0.417*	0.010
VLDL (mg/dl)	0.418*	0.010
HDL (mg/dl)	-0.381*	0.022

* Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Considering reduced glutathione, there was a significant inverse correlation between serum GSH level in pre diabetic group and BMI, SBP, DBP, TC, TG, and VLDL. Also, serum GSH levels decreased with increasing LDL, but no significant correlation was observed. However, GHS levels increased considerably with increasing HDL levels Table 5.The results of the correlation analysis between serum FBG levels with oxidative stress biomarkers and risk factors for CVD are similar to those reported for HbA1c (Table 7 and Figures 4 and 5).

Table 5. Correlation analysis between serum GSH levels and risk factors for CVD.

Cardiovascular Risk Factors	Pearson Correlation (r)	p-value
BMI (kg/sqm)	-0.330*	0.036
SBP (mmHg)	-0.257*	0.042
DBP (mmHg)	-0.286*	0.038
TC (mg/dl)	-0.371*	0.047
TG (mg/dl)	-0.383*	0.033
LDL (mg/dl)	-0.336	0.060
VLDL (mg/dl)	-0.352*	0.044
HDL (mg/dl)	0.351*	0.031

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

The results indicate the presence of a remarkable positive and significant correlation of HbA1c with MDA, BMI, SBP, DBP, TC, TG, LDL, and VLDL. Conversely, a notable, significant, and negative correlation was demonstrated between HbA1c and both GSH and HDL (Table 6 and Figures 2 and 3).

Table 6. Correlation analysis between serum HbA1c levels and risk factors for CVD.

Cardiovascular Risk Factors	Pearson Correlation (r)	p-value
BMI (kg/sqm)	0.453**	0.004
SBP (mmHg)	0.556**	0.000
DBP (mmHg)	0.411**	0.009
TC (mg/dl)	0.556**	0.000
TG (mg/dl)	0.437**	0.008
LDL (mg/dl)	0.493**	0.002
VLDL (mg/dl)	0.550**	0.001
HDL (mg/dl)	-0.493**	0.002

**Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).

Table 7. Correlation analysis between serum FBG levels and risk factors for CVD.

Cardiovascular Risk Factors	Pearson Correlation (r)	p-value
BMI (kg/sqm)	0.455**	0.004
SBP (mmHg)	0.380*	0.017
DBP (mmHg)	0.319*	0.048
TC (mg/dl)	0.419*	0.011
TG (mg/dl)	0.346*	0.039
LDL (mg/dl)	0.401*	0.015
VLDL (mg/dl)	0.461**	0.005
HDL (mg/dl)	-0.430**	0.009

**Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).

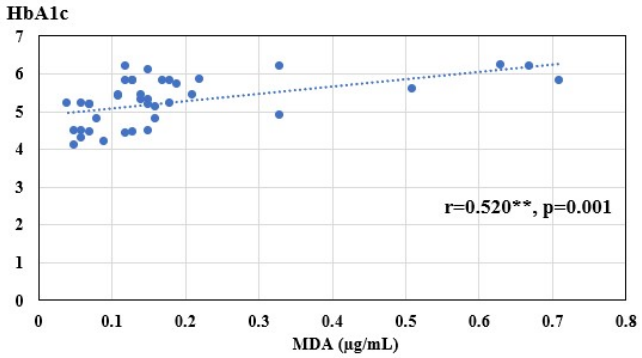

Figure 2. Correlation analysis between HbA1c and malondialdehyde levels in patients.

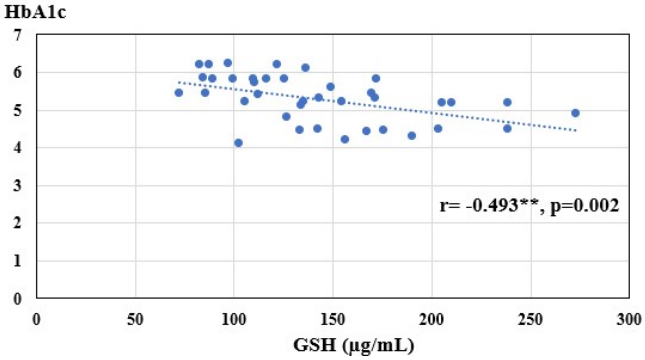

Figure 3. Correlation analysis between HbA1c and malondialdehyde levels in patients.

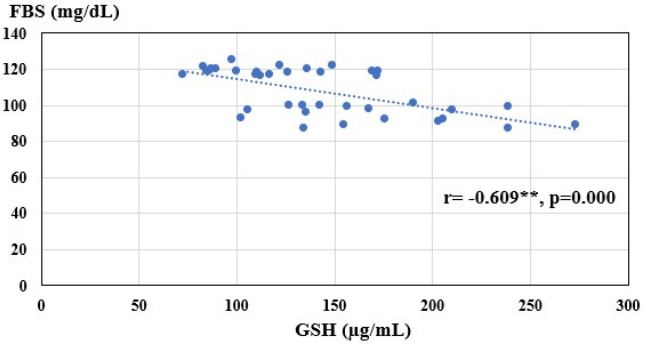

Figure 4. Correlation analysis between FBS and reduced glutathione levels in patients.

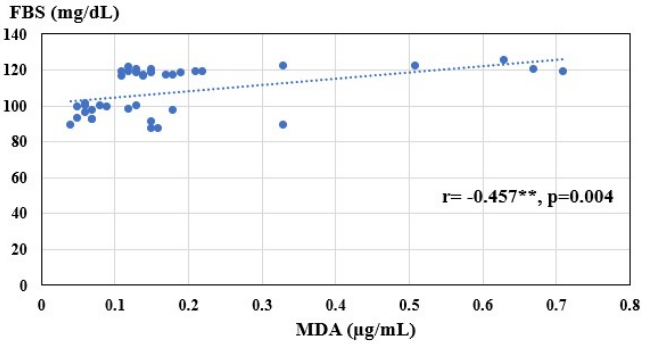

Figure 5. Correlation analysis between FBS and malondialdehyde levels in patients.

4. Discussion:

Many experimental and clinical studies have suggested that type 2 diabetes and its complications, a major risk factor for CVD, are correlated with elevated oxidative stress biomarkers such as protein, lipid, and nucleic acid oxidation products [13, 14], whereas evidence from a recent review article by Bigagli and Lodovici (2019) demonstrated that fewer data in the prediabetic stage are currently available [15].

Accordingly, this study evaluates serum levels of oxidative stress and their possible relationship with other studied biochemicals and parameters, with a focus on comparing the case and control groups. Moreover, the study intends to shed light on the relationship between oxidative stress and increased CVD risk by noting the variation in the levels of the possible CVD risk factors (dyslipidemia, BMI, hypertension) with the change in oxidative stress levels among patients with prediabetes.

Lipid abnormalities induced by insulin resistance can strongly raise the risk of CVD in prediabetes [16]. The study results revealed a significant link between dyslipidemia and prediabetes. Lipid profile parameters recorded statistically considerable differences between the two groups, with prediabetes having lower levels of HDL and higher levels of TC, TG, LDL, and VLDL compared to the control group. These results have been supported by Chakraborty et al., Amawi and Alkhatib, and Raut et al. [17, 18, 19] who demonstrated that the prevalence of dyslipidemia in prediabetic subjects was substantially higher compared with the control group. Furthermore, BMI, SBP, and DBP were significantly elevated in prediabetic subjects in comparison with controls. These findings come in line with those suggested by other studies that revealed the presence of a higher rate of BMI, SBP, and DBP in prediabetic subjects, making them more susceptible to an increased risk for CVD [3, 20].

Considering oxidative stress biomarkers, malondialdehyde (MDA), a biomarker related to oxidative stress that is formed as a result of lipid peroxidation introduced by ROS activity, was shown to have a favorably greater mean value in the serum of the prediabetic group compared to the healthy control group. In India, the outcomes of the study performed by Mahat et al. (2019) [8] confirmed the presence of a significant elevation in MDA levels with a depression in antioxidant concentration in the prediabetic group compared to the normal glucose group. Furthermore, most researchers concur with the study findings and reported increased lipid peroxidation product levels in blood samples of prediabetic and type 2 diabetic patients [21, 22, 23].

This increase in serum MDA in prediabetes is a sign of lipid peroxidation, which is a consequence of oxidative stress. MDA is involved in the modification of low-density lipoprotein (LDL), which mediates pathophysiological alterations through nonenzymatic and auto-oxidative glycosylation [3]. Besides that, the absolute level of atherogenic lipoproteins in the blood is a main risk factor for cardiovascular disease, and this oxidative modification of lipids in circulation further increases this risk [24].However, other studies did not find an alteration in lipid peroxidation levels in prediabetes [25, 26].

Additionally, elevated blood glucose levels decrease antioxidative defenses by scavenging enzymes and antioxidative biomolecules [27]. As a result, abnormally high levels of free radicals combined with a decrease in antioxidant defense mechanisms might result in a variety of cellular and enzymatic damage because the antioxidant defense system is important in scavenging free radicals generated by oxidative stress. Glutathione (GSH) is the most common anti-oxidative molecule that detoxifies oxidative molecules and reactive oxygen species (ROS) in the body [28]. The present data underscored that the non-enzymatic glutathione antioxidant in the serum of prediabetes was remarkably lower than controls.

This fact implies that patients with hyperglycemia have impaired glutathione-dependent anti-oxidant scavenging capacity against enhanced lipid peroxidation processes [29]. The results of this study align with prior studies, confirming that diminished glutathione (GSH) levels are prevalent in individuals with prediabetes. These findings suggest a potential connection between reduced GSH and prediabetic conditions, indicating a possible decrease in GSH production or depletion.

This reduction may be attributed to the need to counterbalance the increased free radicals generated during prediabetes as a consequence of hyperglycemia [30, 31]. Nwose et al. (2006) [32] also documented a decline in GSH levels in prediabetes and proposed that the initial phase of response by erythrocytes begins prior to the onset of diabetes. However, Maschirow et al. (2015) did not find this trend in the prediabetic group [33].

According to the results of the correlation analysis, an increase in serum MDA level was significantly and positively correlated with both FBG and HbA1C in the patient group. This may be due to glucose self-oxidation during hyperglycemia, which could produce free radicals that damage lipids [34]. These results are in agreement with those stated

by Agarwal et al. (2016) [3], who reported that oxidative stress markers were elevated and correlated positively with FPG in the prediabetic group.

In another investigation, it was noted that malondialdehyde (MDA) exhibited a positive and notable correlation with fasting blood glucose (FBG) and 2-hour plasma glucose. This implies that oxidative stress is already manifest during the pre diabetic phase, potentially elevating the cardiovascular disease risk among individuals in this stage [8]. Moreover, the findings of this study revealed a notable negative correlation between the concentration of glutathione (GSH) and both fasting blood glucose (FBG) and HbA1c. These results are consistent with other studies confirming the association between hyperglycemia and diminished glutathione (GSH), strengthening the evidence for a link between hyperglycemia, oxidative stress, and an increased risk of cardiovascular disease (CVD) [35]. Finally, a significant positive correlation of MDA and a significant negative correlation of GSH with CVD risk factors in prediabetes further supports the relationship between oxidative stress and increased CVD risk in these patients.

5. Conclusion:

In conclusion, increased MDA levels and decreased GSH activity suggest that there is a relationship between increased oxidative stress and elevated serum glucose levels in prediabetes. Likewise, the results confirm the link between oxidative stress and increased CVD risk in these patients. Hence, these biomarkers should be taken into consideration while assessing the risk of CVD in prediabetes. Because oxidative stress at the initial level of hyperglycemia could be useful targets for health care, not only for preventing the progression of pre diabetes to diabetes but also for decreasing the risk of developing CVD.

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Data Availability Statement: All of the data supporting the findings of the presented study are available from corresponding author on request.

Declarations:

Conflict of interest: The authors declare that they have no conflict of interest.

Ethical approval: The manuscript has not been published or submitted to another journal, nor is it under review.

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دراسة مقطعية لتقييم العلاقة بين الإجهاد التأكسدي وزيادة مخاطر القلب والأوعية الدموية لدى المصابين بمقدمات مرض السكري

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الخلاصة

ترتبط مقدمات مرض السكري بخلل مستوى السكر في الدم، وهو السبب الرئيسي للإجهاد التأكسدي، مما يساهم في التسبب بأمراض القلب والأوعية الدموية. تهدف الدراسة الحالية إلى تقييم الارتباط بين مقدمات مرض السكري وأمراض القلب والأوعية الدموية من خلال تقييم المؤشرات الحيوية للإجهاد التأكسدي وارتباطها بعوامل الخطر المحتملة للأمراض القلبية الوعائية، بما في ذلك عسر شحميات الدم، ومؤشر كتلة الجسم (*BMI*) ، وارتفاع ضغط الدم. في هذه الدراسة المقطعية تم اختيار 70 فردًا تم تقسيمهم الى 40 مريضًا يعانون من مقدمات مرض السكري و 30 شخصًا كمجموعة ضابطة. تمت مطابقة المجموعتين بالنسبة للعمر ومؤشر كتلة الجسم. تم تحليل نسبة الجلوكوز الصومي في الدم (*FBS*) ، ومستوى الدهون، والمالونديالدهيد (*MDA*) ، ومستويات الجلوتاثيون المختزل (*GSH*) باستخدام عينات المصل. بينما تم قياس الهيموجلوبين السكري (*HbA1c*) باستخدام عينات الدم الكاملة. ولتحليل البيانات، تم تطبيق اختبار ت لعينتين مستقلتين ومعامل ارتباط بيرسون، وتم تحديد مستوى الأهمية عند قيمة p أقل من 0.05 . اظهرت النتائج ارتفاعا معنويا ($P < 0.05$) في قيم متوسط ضغط الدم الانقباضي، مؤشر كتلة الجسم، نسبة الجلوكوز الصومي في الدم (*FBG*) ، نسبة الهيموجلوبين السكري (*HbA1c*) ، الكوليسترول الكلي، الدهون الثلاثية ، الدهون البروتينية واطئة الكثافة ، الدهون البروتينية واطئة الكثافة للغاية، و المالونداياليديهايد في الأشخاص الذين يعانون من مقدمات مرض السكري عند المقارنة مع المجموعة الضابطة، باستثناء نسب الجلوتاثيون المختزل و الدهون البروتينية عالية الكثافة اللذان سجلا انخفاضا معنويا في الأشخاص الذين يعانون من مقدمات مرض السكري. وطبقا لمعامل ارتباط بيرسون فقد سجل الجلوكوز الصومي في الدم (*FBG*) والهيموجلوبين السكري (*HbA1c*) علاقة موجبة مؤثرة مع كل من المؤشرات الحيوية للإجهاد التأكسدي وعوامل الخطر القلبية الوعائية، وعلاقة احصائية سالبة مع كل من الجلوتاثيون المختزل و الدهون البروتينية عالية الكثافة. كما وجدت علاقة موجبة مؤثرة للمالونداياليديهايد مع عوامل الخطر القلبية الوعائية في الأشخاص المصابين بمقدمات مرض السكري، في حين ثبت وجود علاقه احصائية سالبة بين الجلوتاثيون المختزل مع عوامل الخطر القلبية الوعائية. تشير نتائج هذه الدراسة إلى أن مقدمات مرض السكري ترتبط ارتباطًا وثيقًا بمستويات مرتفعة من المؤشرات الحيوية للإجهاد التأكسدي وتؤكد العلاقة بين الإجهاد التأكسدي وزيادة مخاطر الإصابة بأمراض القلب والأوعية الدموية لدى هؤلاء المرضى.

الكلمات الدالة : مقدمات مرض السكري، الإجهاد التأكسدي، مالوندايالديهايد، الجلوتاثيون المختزل، أمراض القلب والأوعية الدموية.

التمويل: لايوجد.

بيان توفر البيانات: جميع البيانات الداعمة لنتائج الدراسة المقدمة يمكن طلبها من المؤلف المسؤول.

اقرارات:

تضارب المصالح: يقر المؤلفون أنه ليس لديهم تضارب في المصالح.

الموافقة الأخلاقية: لم يتم نشر المخطوطة أو تقديمها لمجلة أخرى،كما أنها ليست قيد المراجعة.