

Comparison of methylglyoxal and malondialdehyde as biomarkers of oxidative stress in type 2 diabetes mellitus with cardiovascular risk factors in patients

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Abstract

The aim of the study was to compare the plasma concentration of MGO and MDA in patients with T2DM with and without cardiovascular risk factors and examine their relationship with clinical and biochemical variables. The participants consisted of 80 people, 30 with cardiovascular risk T2DM, 30 with no cardiovascular risk T2DM, and 20 healthy controls. The plasma levels of MGO and MDA were determined by the HPLC and TBARS methods, respectively, whereas the level of fasting blood glucose, HbA1c, lipid profile, blood pressure, and BMI were measured as well. The MGO and MDA levels were significantly elevated in diabetic patients with the cardiovascular risk and a positive correlation between the oxidative stress indicators and the HbA1c, fasting glucose, LDL, and triglycerides and a negative correlation between the HDL levels. These results suggest that higher plasma MGO and MDA are the indicators of the presence of high oxidative stress in patients with T2DM, especially those with the cardiovascular risk factors who may have cardiovascular complications, and thus the biomarkers can be used as sensitive ones to predict early occurrence of oxidative stress and cardiovascular complications in patients and thereby the biomarkers can become a sensitive indicator of the early detection of oxidative stress and cardiovascular complications in patients and thereby that can trigger timely intervention strategies to improve patient outcomes.

Keywords: Type 2 diabetes mellitus; Methylglyoxal; Carbonyl stress; Oxidative stress; Cardiovascular risk

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is a leading chronic metabolic condition that is marked by unremitting hyperglycemia, insulin resistance and decreased glucose metabolism, which affects millions of people globally[1]. The effects of chronic hyperglycemia are several biochemical imbalances with oxidative stress being one of the primary stimuli in the pathogenesis of both microvascular and macrovascular complication[2]. The oxidative stress is the result of the lack of balance between the production of reactive oxygen species (ROS) and antioxidant defenses of the body, resulting in the destruction of lipids, proteins, and DNA[3].

Two important oxidative stress biomarkers of T2DM are Methylglyoxal (MGO) and Malondialdehyde (MDA). The reaction of MGO, a reactive dicarbonyl product that is mostly generated during glucose metabolism[4], with proteins, nucleic acids, and lipids leads to the formation of advanced glycation end products (AGEs), which play a role in the endothelial dysfunction, inflammation, and the vascular damage[5]. MDA, which is a large lipid peroxidation byproduct, is a manifestation of oxidative damage of cell membranes and lipoproteins in circulation[6]. Both the markers have been linked with poor glycemic control, lipid abnormalities and elevated risk of cardiovascular disease in diabetic patients[7].

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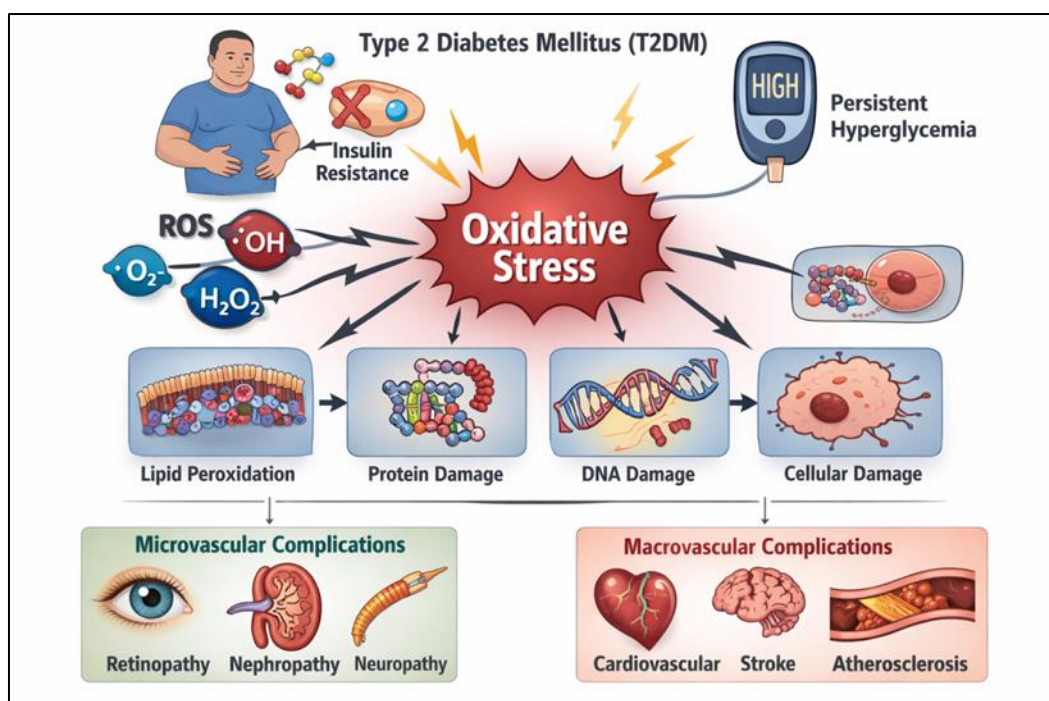


Figure 1 Type 2 Diabetes mellitus (T2DM)

The connection between oxidative stress and cardiovascular complications in T2DM is clearly known. Overproduction and build-up of ROS and MGO and MDA enhance endothelial dysfunction, arterial hardening, inflammation, and atherosclerosis, predisposing to coronary artery disease, myocardial infarction, and heart failure[8].

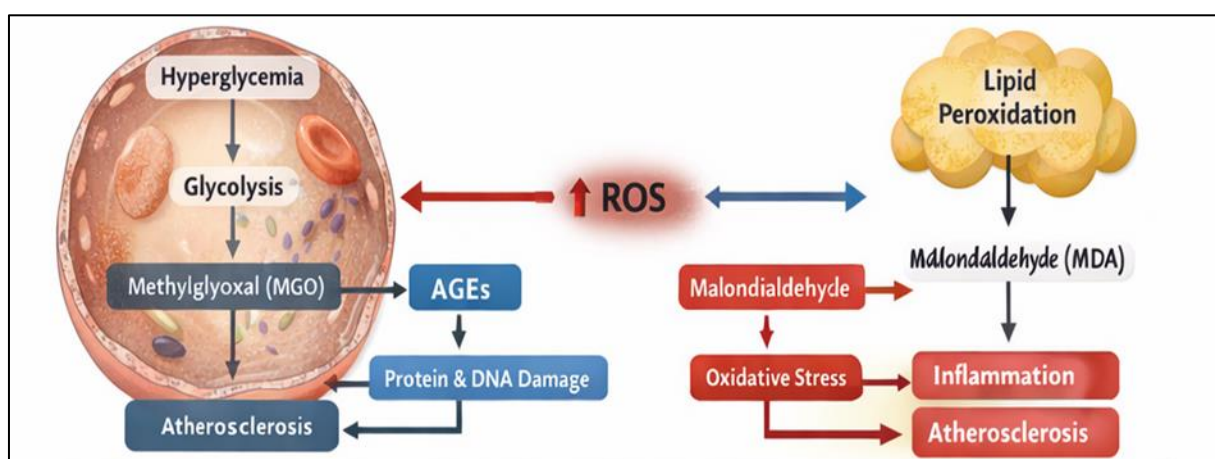


Figure 2 Metabolic pathways of MGO and MDA in diabetes

Considering the paramount role that oxidative stress plays in the pathophysiology of diabetic cardiovascular complications, the study will be conducted to test plasma levels of MGO and MDA in T2DM patients, with or without cardiovascular risk factors and also examine the relationship of MGO and MDA with the clinical and biochemical parameters[9]. The knowledge of such associations could have an indication of early occurrence of oxidative stress and direct prompt intervention measures to mitigate cardiovascular risk among diabetic patients[10].

2. Materials and Methods

2.1. Study Design

To identify the role of plasma methylglyoxal (MGO) and malondialdehyde (MDA) as biomarkers of oxidative stress in patients with type 2 diabetes mellitus (T2DM) who had and did not have cardiovascular risk factors, the analytical cross-sectional design was used in this study. A control group of healthy individuals with a matching in terms of age and sex was also provided. The cross-sectional design also permitted concurrent comparison of the groups at one time, which was used to identify the possible relationship between oxidative stress biomarkers and cardiovascular risk parameters among T2DM patients.

2.2. Study Population

The total number of participants who had given consent was 80 who were divided into the following groups: 30 T2DM patients with cardiovascular risk factors, 30 T2DM patients without cardiovascular risk factors, 20 healthy controls. The sample of participants was gathered at outpatient clinics and community health centers, and informed was taken as the ethical guidelines.

The diabetic patients who were included suffered between 40 and 70 years of age, with a confirmed T2DM diagnosis not less than one year, and a stable metabolic and cardiovascular state with no recent acute illness. The exclusion criteria were the presence of type 1 diabetes, gestational diabetes, chronic kidney or liver disease, recent infections or inflammatory disease, and taking of antioxidant supplements or drugs that impacted oxidative stress. The healthy volunteers who had never experienced diabetes, cardiovascular disease or other chronic illnesses was used as the control group.

2.3. Sample Collection and Handling

Each participant had their venous blood samples taken (5-7 mL) after a period of 8-12 hours of overnight starvation. To analyse serum, blood was put in anticoagulant-free tubes whereas in analysing plasma, blood was put in EDTA-containing tubes. Samples were centrifuged immediately after collection at 3000 rpm and 10 minutes at 4 °C to separate serum and plasma. Aliquots were moved to sterile and labeled polypropylene tubes and stored at -80 °C until the time of analysis to keep the biomarkers stable as well as prevent pre-analytical oxidative degradation. Handling operations were conducted with exposure to light and air as little as possible in order to minimize artificial oxidation.

2.4. Materials and Chemicals

- Methylglyoxal (MGO), and derivatization reagents such as o-phenylenediamine (OPD) were acquired by Sigma-Aldrich (USA).
- Malondialdehyde (MDA) standard and thiobarbituric acid (TBA) to measure the TBARS were acquired in Merck (Germany).
- HPLC-grade solvents acetonitrile and water were purchased at Fisher Scientific.
- All laboratory products such as microcentrifuge tubes, syringes were sterile and pyrogen free.

The following instruments were used: a high-performance liquid chromatography system (HPLC, Agilent 1260 Infinity) with a UV detector, a spectrophotometer (Shimadzu UV-1800) to measure TBARS and a refrigerated centrifuge.

2.5. Measurement of Methylglyoxal (MGO)

HPLC was employed to measure plasma MGO after chemical derivatization[11]. In summary, derivatization was conducted in which the plasma samples were reacted with o-phenylenediamine (OPD) under controlled conditions to produce a stable quinoxaline derivative, which increased detection sensitivity. A C18 reverse-phase column was used to inject the derivatized samples into the HPLC system and separate them using a gradient elution of acetonitrile and water. The detection was done at 315 nm with a UV detector. Plasma levels, which are in $\mu\text{mol/L}$, were found by a standard calibration curve plotted using known concentrations of MGO. These samples were repeated twice each and internal controls were also included to maintain accuracy and reproducibility.

2.6. Measurement of Malondialdehyde (MDA)

The TBARS assay was performed to measure serum MDA and is founded on the interaction of MDA with thiobarbituric acid through acidic and high-temperature conditions producing a pink-colored chromogen[12]. The samples of serum were combined with TBA reagent and heated in a water bath at temperatures of 95°C -60 minutes. The absorbance of

the colored complex was then recorded using a spectrophotometer at 532 nm after the data had cooled down. The concentration of MDA was estimated with the help of a prepared standard curve containing known concentrations of MDA, and the concentration was expressed in nmol/mL. The measurements were all done in duplicate and there were blanks and controls in each run to ensure that the analysis was analytically valid.

2.7. Assessment of Clinical and Biochemical Variables

To clearly evaluate the status of metabolic and cardiovascular parameters of the participants, the study examined the fasting blood glucose (FBS) and glycated hemoglobin (HbA1c) as the measures of glycemic control. The level of total cholesterol, LDL, HDL, and triglycerides were determined through an automated technique of enzyme analysis[13]. Sphygmomanometer was measured as a calibrated device to measure blood pressure, and body mass index (BMI) was calculated as weight in kilograms were divided by square of height in meters (kg/m^2). These clinical and biochemical variables were taken to investigate correlations with oxidative stress biomarkers (MGO and MDA), which gave the opportunity to see how the metabolic dysregulation correlates with cardiovascular risk in T2DM patients.

2.8. Statistical Analysis

The SPSS v26 was used to analyze the data. Continuous variables appear in the form of mean $\pm$ SD and categorical variables in form of percentages. ANOVA was used to compare groups and Pearson or Spearman to evaluate correlations. $p < 0.05$ was taken to be important.

3. Results

3.1. Study Population

Eighty patients were used in the study (30 T2DM patients who were exposed to cardiovascular risk factors, 30 T2DM patients who were not exposed to cardiovascular risk factors, and 20 healthy controls). The average age was 54.7 ± 6.1 years and the distribution of sex was equal among groups. Every participant had attended the study with no sample being left out.

3.2. Clinical and Biochemical Characteristics

Table 1 summarized clinical and biochemical characteristics. The groups were similar in terms of age and sex ($p > 0.05$). The T2DM patients exhibiting cardiovascular risks had a significant high FBS, HbA1c, LDL and triglycerides and a low HDL cholesterol. These variations give the background of the analysis of the oxidative stress biomarkers.

Table 1 Clinical and Biochemical Characteristics of Study Participants

Variable	T2DM with CV Risk	T2DM without CV Risk	Healthy Controls	p-value
Age (years)	55.3 ± 6.1	54.8 ± 5.9	53.7 ± 6.5	0.68
Sex (M/F)	16/14	15/15	10/10	0.95
FBS (mg/dL)	165.2 ± 22.5	142.8 ± 18.4	92.5 ± 8.1	<0.001
HbA1c (%)	8.3 ± 1.2	7.1 ± 0.9	5.4 ± 0.3	<0.001
LDL (mg/dL)	135.6 ± 18.9	118.7 ± 15.4	95.2 ± 12.7	<0.001
HDL (mg/dL)	38.5 ± 6.2	44.1 ± 5.5	50.2 ± 7.1	<0.001
Triglycerides (mg/dL)	210.4 ± 35.6	180.7 ± 28.3	120.5 ± 20.4	<0.001
BMI (kg/m^2)	29.8 ± 3.5	27.1 ± 2.9	24.2 ± 2.5	<0.001

The results of Table 1 have been presented as shown in Figure 3 that graphically demonstrates the variations in the clinical and biochemical parameters across the study groups.

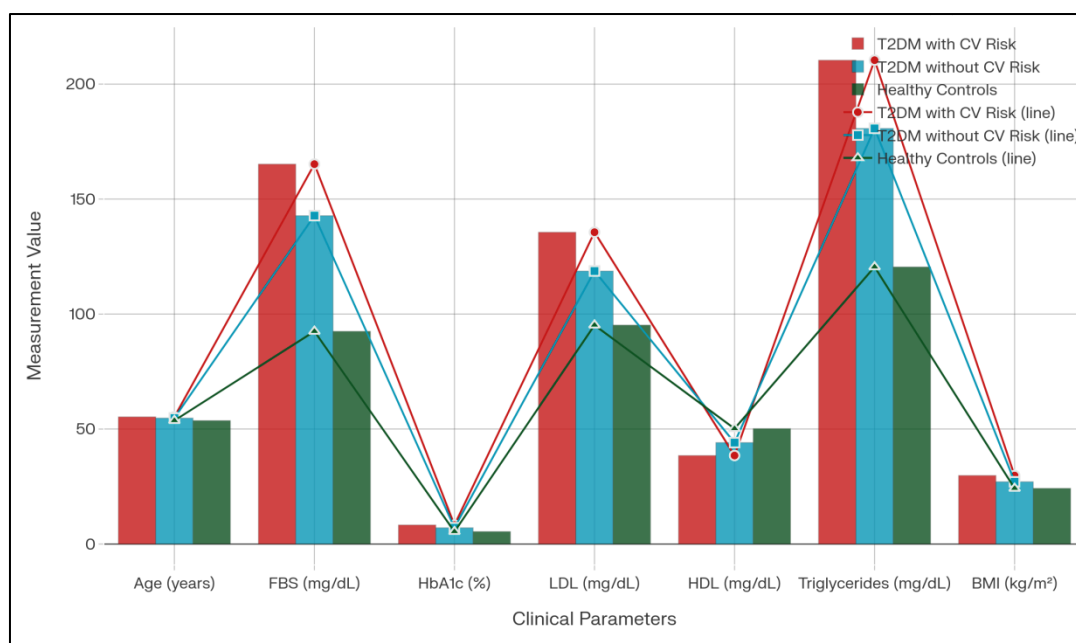


Figure 3 Clinical and Biochemical characteristics of study participants

3.3. Oxidative Stress Biomarkers (MGO and MDA)

The Table 2 shows plasma MGO and serum level of MDA. The two biomarkers were significantly increased in T2DM patients with cardiovascular risk factors ($p < 0.001$). T2DM patients without cardiovascular risk were found to have moderate elevations.

Table 2 Plasma MGO and Serum MDA Levels

Biomarker	T2DM with CV Risk	T2DM without CV Risk	Healthy Controls	p-value
MGO ($\mu\text{mol/L}$)	3.85 ± 0.45	2.90 ± 0.38	1.75 ± 0.25	<0.001
MDA (nmol/mL)	5.12 ± 0.68	4.05 ± 0.55	2.80 ± 0.40	<0.001

Figure 4 presents the mean levels of MGO and MDA, with the showcased increased level of oxidative stress being in T2DM cardiovascular risk patients.

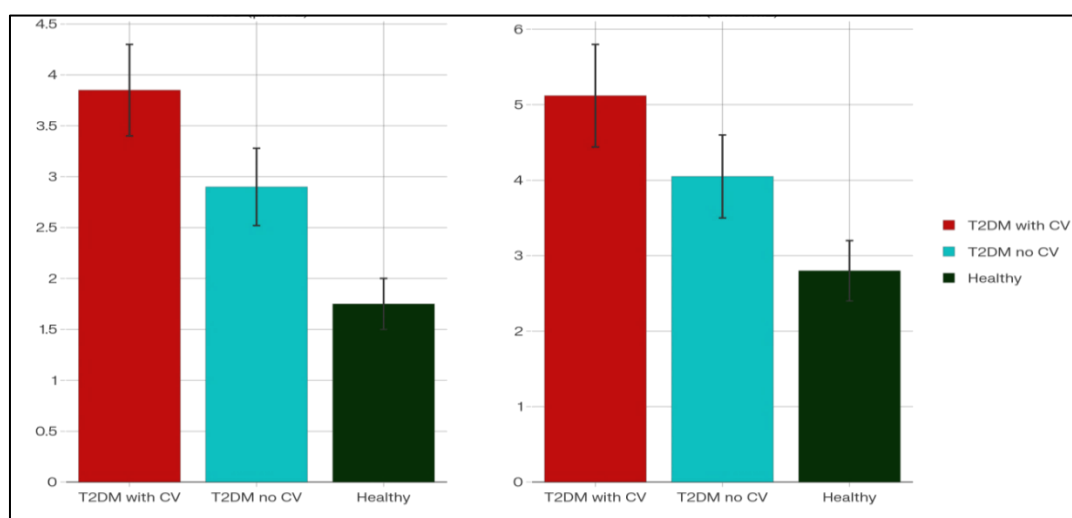


Figure 4 Plasma MGO and serum MDA levels in study groups

3.4. Correlation of the Biomarkers of Oxidative Stress with the Clinical Variables

Table 3 provides correlation coefficients between the biomarkers of oxidative stress and clinical variables. MGO and MDA were both positively associated with FBS, HbA1c, LDL, triglycerides and BMI and negatively linked with HDL.

Table 3 Correlation Coefficients

Variable	MGO (r)	MDA (r)	p-value
FBS	0.62	0.58	<0.001
HbA1c	0.65	0.60	<0.001
LDL	0.49	0.45	0.002
HDL	-0.38	-0.35	0.01
Triglycerides	0.55	0.50	<0.001
BMI	0.41	0.39	0.005

These associations are depicted in Figure 5 as the graphical representation of the connection between MGO and HbA1c.

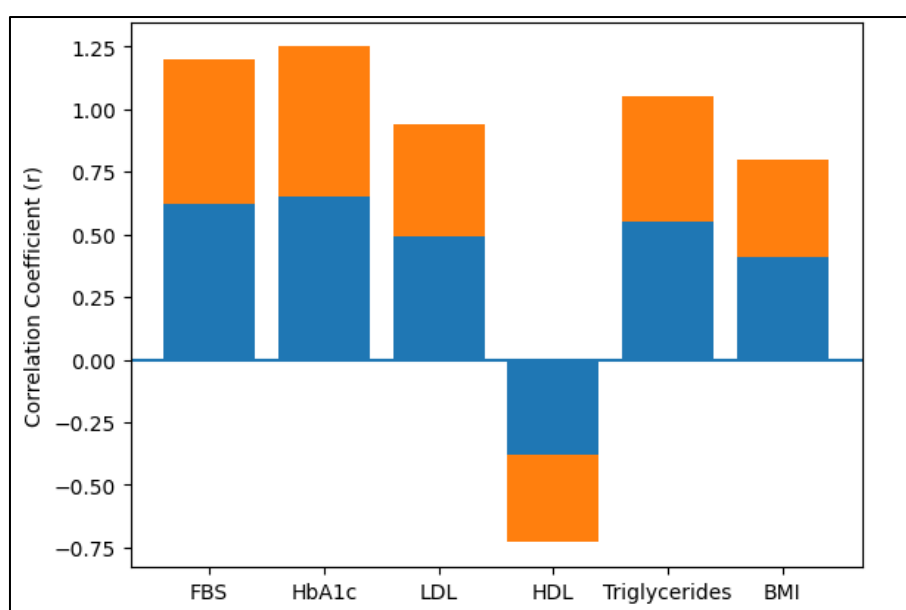


Figure 5 Correlation of oxidative stress markers with metabolic parameters

3.5. Oxidative Stress Biomarkers by Sex

Table 4 summarizes the plasma MGO and serum MDA by sex. There were no significant differences between the males and females in any of the two T2DM groups.

Table 4 MGO and MDA Levels by Sex

Group	Sex	MGO ($\mu\text{mol/L}$)	MDA (nmol/mL)	p-value
T2DM with CV Risk	Male	3.92 ± 0.42	5.18 ± 0.65	0.45
T2DM with CV Risk	Female	3.78 ± 0.48	5.05 ± 0.70	
T2DM without CV Risk	Male	2.95 ± 0.40	4.10 ± 0.52	0.50
T2DM without CV Risk	Female	2.85 ± 0.36	4.00 ± 0.57	

Table 6 results are depicted in Figure 4, which indicates that sex has no significant effect on the biomarkers of the oxidative stress in the population under study.

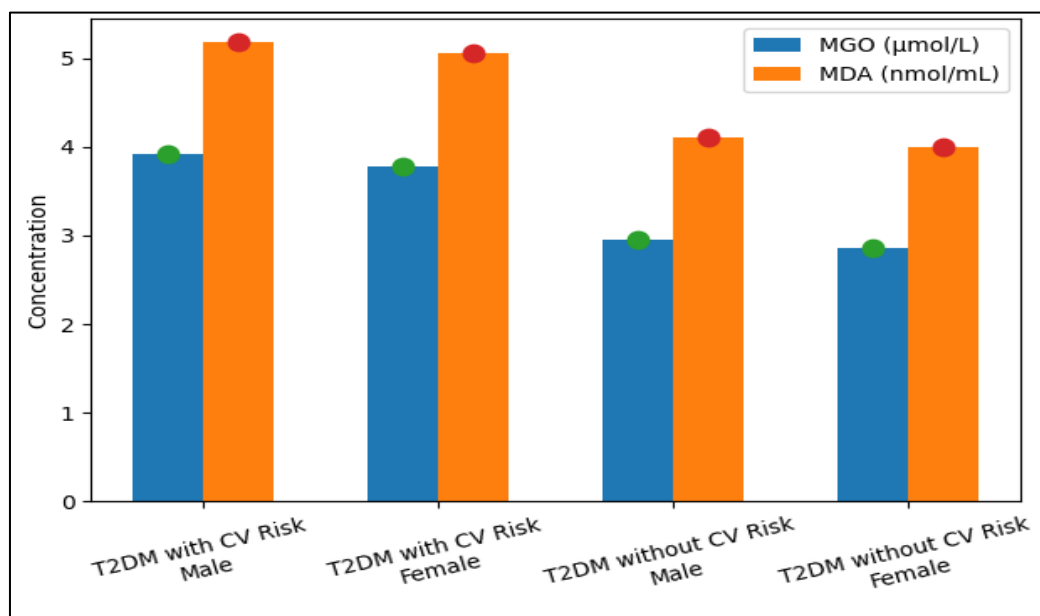


Figure 6 Sex-Based comparison of MGO and MDA levels across study groups

4. Discussion

Oxidative stress is one of the key processes that connect T2DM to the onset of cardiovascular complications, which is a manifestation of disequilibrium in the production of reactive oxygen species (ROS) and antioxidant responses of the body[14]. Chronic hyperglycemia increases the formation of reactive metabolites (metabolites of glucose) including methylglyoxal (MGO), which is a highly reactive dicarbonyl molecule in T2DM[15]. MGO is highly binding to proteins, nucleic acids and lipids and can generate advanced glycation end-products (AGEs) that are involved in endothelial dysfunction, vascular inflammation, and arterial stiffness[16]. This cascade increases oxidative injury and creates a pro-atherogenic condition, which is a mechanistic connection between dysregulated glucose homeostasis and CV risk[17].

A product of lipid peroxidation, malondialdehyde (MDA) is an indication of oxidative damage to cell membranes and lipoproteins and its deposition further increases vascular damage and inflammatory reactions[18]. MGO as MDA combined gives a more global outlook of the carbonyl and lipid-based oxidative stress, which takes into consideration several dimensions of the cellular damage in connection to diabetes and cardiovascular risk factors[19]. These interdependences of hyperglycemia, dyslipidemia, and oxidative stress demonstrate that T2DM is associated with complex metabolic disruptions and the need to measure the oxidative stress biomarkers as the early signs of vascular dysfunction[20].

Not only do these biomarkers indicate current oxidative damage, but they also can be used to present prognostic information[21]. High levels of oxidative stress have been reported to reduce the bioavailability of endothelial nitric oxide, stimulate vascular smooth muscle proliferation and trigger inflammatory pathways, all of which enhance atherogenesis and predispose myocardial infarction, stroke, and heart failure[22]. Moreover, the oxidative stress has a synergistic interaction with the traditional risk factors like obesity, hypertension, and dyslipidemia which increase cardiovascular risk[23]. This reinforces the importance of combining the measurement of the oxidative stress with the traditional clinical parameters to provide a full risk stratification[24].

Therapeutically, the role of oxidative stress in T2DM can be used as an intervention opportunity[25]. These approaches should aim at MGO accumulation, boosting of endogenous antioxidant defenses and lifestyle changes, which can reduce vascular injury and enhance long-term outcomes[26]. Pharmacologic treatments such as drugs that induce the glyoxalase system or lipid peroxidation inhibitors are promising therapies to prevent or slow diabetic complications[27].

It is also proposed that the scientific evidence indicates that these oxidative stress biomarkers can be used as surrogate endpoints in clinical studies, which would give an idea about the effectiveness of the interventions that are aimed at changing the redox balance[28]. MGO and MDA measurements have the potential to support preventive measures in due time and direct individual treatment plans by detecting the initial signs of the biochemical anomalies prior to their manifestations in clinical environments[29].

On the whole, the interdependence of carbonyl stress, lipid peroxidation, and cardiovascular risk factors in T2DM depicts a complex pathophysiological network where oxidative stress is an outcome and a producer of metabolic and vascular dysfunction[30]. This further supports the argument that further investigations are required to clarify the underlying molecular mechanisms and to maximize the use of biomarkers in risk prediction and to develop specific interventions to minimize oxidative load and enhance patient outcome in type 2 diabetes[31].

5. Conclusions

The conclusion made is that oxidative stress is at the centre of the pathogenesis of cardiovascular complication in Type 2 Diabetes Mellitus patients and biomarkers like Methylglyoxal (MGO) and Malondialdehyde (MDA) are highly sensitive biomarkers of measuring this stress. Regular observation of such indicators can also assist in the early detection of patients at higher cardiovascular risk, which would allow them to take preventive and curative measures to increase metabolic regulation and decrease the risk of developing cardiovascular complications in the future.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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