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Article

Assessment of Oxidative Stress Biomarkers and Antioxidant Enzyme Activity in Patients with Type 2 Diabetes

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Abstract: This study aimed to estimate the levels of oxidative stress markers and the activity of antioxidant enzymes in patients with type 2 diabetes mellitus (T2DM), and to compare them with healthy individuals. The study included 120 volunteers divided into two groups: the patient group (60 individuals with T2DM) and a healthy control group (60 individuals).

The following oxidative stress markers were measured: malondialdehyde (MDA) and protein carbonyl (PC), along with the activity of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and the level of reduced glutathione (GSH).

The results revealed a statistically significant increase ($P<0.001$) in MDA and protein carbonyl levels in diabetic patients, accompanied by a marked decrease in the activity of all antioxidant enzymes compared to the control group.

A strong positive correlation ($r=+0.78$, $P<0.001$) was observed between glycated hemoglobin (HbA1c) and MDA levels, indicating that poor glycemic control is associated with accelerated lipid peroxidation and increased cellular oxidative damage. Conversely, a strong negative correlation ($r=-0.72$, $P<0.001$) was found between HbA1c and SOD activity, suggesting that elevated HbA1c is associated with a significant impairment of the enzymatic antioxidant defense system.

These findings contribute to a deeper understanding of the mechanistic link between oxidative stress and diabetic complications, and provide a scientific basis for considering antioxidant-based therapeutic strategies in the management of T2DM.

Keywords: Oxidative stress, Type 2 diabetes mellitus, Superoxide dismutase, Malondialdehyde, Antioxidant enzymes, Glycated hemoglobin.

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1. Introduction

Type 2 diabetes is considered (T2DM)heThe most widespread metabolic disease on the planet, with an estimated number ofpatientsIt affects more than 537 million people worldwide, according to the International Diabetes Federation (IDF, 2021These rates are rising at an alarming rate, especially in middle- and low-income countries, including Iraq and other Middle Eastern countries, making this disease a public health priority[1], [2].

Type 2 diabetes is characterized by insulin resistance and impaired insulin secretion from pancreatic beta cells, and persistently high blood sugar (chronic hyperglycemia). Biochemical and molecular research has showntheIn-depth This chronic increase causes the generation of huge quantities of reactive oxygen species (ROSThis disrupts the delicate balance between the production of free radicals and the body's inability to neutralize them.

This defect is called "oxidative stress" (Oxidative Stress)And heIt is of great interest in diabetes studies, as it represents the primary link between hyperglycemia and the serious

vascular problems of the disease, such as coronary heart disease, diabetic nephropathy, neuropathy and retinopathy [3], [4], [5].

importanceSearch

The importance of this research stems from the following key themes:

1. Epidemiological burden: Type 2 diabetes represents a significant health and financial burden in Iraq and the Arab region, with a prevalence rate ranging between 9-13% of adults.

2. Molecular understanding: Assessing indicators of oxidative stress contributes to elucidating the molecular mechanisms responsible for the emergence of complications, thus providing opportunities for early intervention.

3. Clinical use: These indicators can be used as biomarkers (BiomarkersTo assess the risk of complications and monitor the response to treatment.

4. Therapeutic guidance: The results of this study provide a scientific basis for evaluating the effectiveness of antioxidant treatments.

Oxidative stress: concept and mechanisms

Oxidative stress is a state of imbalance between the generation of reactive oxygen species (ROSActive nitrogenRNSOn the one hand, and the antioxidant capacity on the other hand.Sies, 2015The primary causes of hyperglycemia include the following:

- superoxide anion($O_2^{\bullet -}$): It is formed via the electron transport pathway in mitochondria.
- Water peroxideH_2O_2): is generated from the superoxide reaction thanks to the SOD enzyme.

$O_2^{\bullet -} + 2H^+ \rightarrow H_2O_2 + O_22$

- hydroxyl ion (•OH): The most harmful, it is formed via the Fenton reaction.
- Nitrogen oxide (NO) and its products: play a role in the dysfunction of blood vessels.

Production pathsROS in type 2 diabetes

Recent studies have identified four key axes for the formation of free radicals under conditions of high blood sugar (Brownlee, 2001):

a) Sorbitol pathway (Polyol Pathway)

In cases of hyperglycemia, excess glucose takes an alternative pathway involving two successive enzymatic steps:

Step one: The enzyme aldose reductase catalyzes the reduction of glucose to sorbitol upon consumption.NADPHThis depletes its cellular stores and reduces the replenishment of reduced glutathione (GSHThis weakens the antioxidant defense.

Step two: The enzyme sorbitol dehydrogenase catalyzes the oxidation of sorbitol to fructose upon consumption. $^+$NADProductionNADHPlus, this overfeeds the mitochondrial electron transport chain and increases superoxide anion production.($O_2^{\bullet -}$),This exacerbates oxidative stress.

Sorbitol also accumulates inside the cell due to its limited permeability through membranes, which raises osmotic pressure and damages retinal, nerve, and kidney cells.

b) Advanced glycosyl-end complex pathway (AGEs/RAGE)

Advanced glycosylated products are connected (Advanced Glycation End-products, AGEswith its futureRAGEReleasing inflammatory and oxidative signals that activate an enzymeNADPHOxidases and inflammatory transcription factors such asNF-κBResearch has shownYamagishiHe and his colleagues (2011) found a strong positive correlation between levelsAGEsIndicators of oxidative stress and severity of vascular involvement.

c) Protein kinase activationC (PKC) Activation

High blood sugar increases the production of diglycerol (DAG), which activates protein kinaseCIn its various forms. This activation leads to excessive secretion of suproxenx viaNADPHOxidase and mitochondria, which contribute to the dysfunction of vascular and kidney cells.

d) The hexosamine pathway (Hexosamine Pathway)

A portion of glucose is converted to glucosamine-6-phosphate via the hexosamine pathway, altering gene expression of oxidative factors and reducing enzyme activity.eNOSProducer of blood vessel-protecting nitric oxide.

Antioxidant mechanism: Key enzymes

a) Superoxide dismutase (SOD)

representsSODThe first line of defense against free radicals. It accelerates the conversion of superoxide radicals. ($O_2^{\bullet -}$)It is composed of hydrogen peroxide and oxygen gas. It exists in three forms:Cu/Zn-SODIn matterandIt is also found in the nucleus and in the intermembrane space of mitochondria.Mn-SODIn mitochondria, andEC-SODOutside the cell. Numerous studies have shown (Moussa, 2008;Muttigi et al., 2018A significant decrease in its effectiveness in diabetic patients is inversely proportional to the level of HbA1cAs shown in Table (1).

b) Catalase(CAT)

Peroxisomes accelerates the breakdown of hydrogen peroxide into water and oxygen. Red blood cells, the liver, and the kidneys have high concentrations of it. Research indicates... (Johansen, JS et al. 2005) Its effectiveness is reduced in patients with uncontrolled diabetes, which exposes cells to accumulationH2O2The harmful one.

c) Glutathione peroxidase (GPx)

Most formsGPxIt depends on selenium,accelerates oxidationGSHTo equalizeH2O2And organic peroxides. Its effectiveness is closely linked to the availability of selenium in food. Research shows(2020Rajeswari & Satyanarayana,To reviewGPxIn diabetes, the observed increase in lipid oxidation may be justified.

Table (1) Key antioxidant enzymes

Changes in diabetes	Job	the site	enzyme	No.
↓ 40-60%	Convert $O_2^{\bullet -} \rightarrow H_2O_2$	Cytoplasm / Mitochondria	SOD	1
↓ 45-55%	H_2O_2 decomposes $\rightarrow H_2O + O_2$	Peroxisome/cytoplasm	CAT	2
↓ 35-50%	GSH oxidation to neutralize H_2O_2	Cytoplasm / Mitochondria	GPx	3
↓ 40-60%	GPx and GST/Thiol substrate	inside the cell	GSH	4

Indicators of oxidative stress: biomarkers

a) Malondialediide (MDA)

It is consideredMDAThe most prominent final product of lipid oxidation, estimated by the thiobarbituric acid assay (TBARSThis index represents the standard for measuring oxidative stress in clinical research due to its stability and ease of measurement. Numerous studies have shown (Ceriello et al., 2008;Furukawa et al., 2017Its noticeable increase among diabetic patientsAs shown in Table (2).

b) Protein carbonyl group (Protein Carbonyl, PC)

This index assesses protein oxidation through the formation of carbonyl groups on amino acid residues. A high index indicates oxidation of enzymes and functional proteins, which impairs the activity of metabolic enzymes and ion pumps.

c) Reduced glutathione (GSH)

It representsGSHEssential intracellular thiols and a key substrate for enzymesGPxandGSTIts concentration reflects the overall effectiveness of the antioxidant system; its decrease is associated with increased lipid peroxidation and weakened cellular immune protection.

Previous relevant studies

Table (2) Previous studies

Key findings	Indicators	The group	The researcher and the year
↑MDA ↓SOD ↓GPx	MDA, SOD, CAT, GPx	80 patients / 40 officers	Mahboob et al. (2005)
Strong HbA1c-MDA binding	MDA, GSH, HbA1c	100/50	Fathallah et al. (2016)
↓GPx precedes complications	GPx, CAT, PC	70/35	Rajeswari et al. (2020)
OS pre-complication	All OS	Comprehensive systematic review	Asmat et al. (2016)
↓Nrf2 = ↑Multiplications	Nrf2, MDA, SOD	150 graduated	Bhatt et al. (2023)

Objectives and assumptions

Study objectives

This study aims to measure and evaluate indicators of oxidative stress (such asMDA and PC) and the activity of antioxidant enzymes (SOD, CAT, GPx) in the blood serum of diabetic patients, and an analysis of the statistical relationship between these variables and glycated hemoglobin (HbA1c) levels [6], [7], [8].

The sub-objectives are:

1. Measure the levels of malondialdehyde (MDA) and carbonyl protein (PC) in the blood, as these are indicators of oxidative stress.
2. Determining the activity of SOD, CAT, and GPx enzymes In blood plasma and red blood cells.
3. Measuring reduced glutathione levels(GSH), because it expresses the strength of non-enzymatic antioxidant defense.
4. How to observey correlationlevelsHbA1c with indicators of oxidative stress and enzyme activitythatFights oxidation.
5.Search forThe duration of diabetes and blood sugar control affect oxidative stress levels..
6. ComparisonResults among patientsThose who haveBlood sugar levels are under controlHbA1c) less than 7%andNot controlledHbA1c Equal to or greater than 7%.(

Research hypotheses

Basic hypothesis(H1)betweenTThe study found that the level of oxidative stress was significantly higher ((P<0.001) in patients with type 2 diabetes,ThisIt increases with the duration of the illness and poor blood sugar control..

Second hypothesis(H2) Antioxidant enzyme activity is lowerwhenThe levelHbA1chigher

2. Materials and Methods

the study

Comparative analytical cross-sectional study (Cross-sectional Analytical StudyIt was completed at Al-Karkh Teaching Hospital andinEndocrinology clinics in Baghdad, during the period from October 2024 to April 2025. The study received approval from the Scientific Research Ethics Committee, and all participants signed the informed consent form.

Study participants

1. Listing criteria:

- Individuals diagnosed with type 2 diabetes based on the American Diabetes Association's criteria (ADA, 2023).
- Their ages range from 35-65 years.
- The illness period is no less than one year.
- Full acceptance of the contribution.
- All patients are taking oral medication only (metformin, sulfonylurea, or both).

2.Exclusion criteria:

- Pregnancy and breastfeeding.
- Long-term inflammatory conditions and malignant tumors.
- Sudden kidney or liver failure.
- Smokers and those who consume alcoholic beverages.
- Taking vitamins or antioxidants during the past three months.
- Any individual who scored below a certain level was excluded from the control group.HbA1c is equal to or greater than 5.7%, or shows any disturbance in glucose metabolism according to the American Diabetes Association (ADA, 2023) criteria.
- Insulin users were excluded to avoid its effect on indicators of oxidative stress.

3.Sample size and distribution

The total number of participants reached 120 individuals, divided as follows:

Table (3) Sample distribution by gender and group

females	Males	number	The group	No.
28	32	60	Type 2 diabetes patients	1
29	31	60	Health control	2
57	63	120	total	3

4. Sample collection

Venous blood samples were taken after a 10-12 hour fast, depending on theroad Next:

- 5 ml in a tube containingEDTATo measureIndicators of oxidative stress and we prepare an extract of red blood cells.
- 3 ml in a milliliter tubeand forSodium fluoride" (Sodium Fluoride) to control fasting blood sugar levels.andTo prevent glucose breakdown in blood collection tubes
- 5 ml in an empty tubetoWe isolate the serum and measure glycated hemoglobin, lipids, and kidney and liver function.

The samples were centrifuged in a centrifuge at 3000 rpm for 10 minutes at 4°C.

Unity	Principle	The method	Measurement
nmol/mL	Interact withTBA at 532 nm	TBARS – Ohkawa (1979)	(MDA) Measurement of malondialdehyde
unit/ml	Inhibition of adrenaline oxidationpH 10.2	Misra & Fridovich (1972)	Measuring superoxide dismutase activity (SOD)
kilo unit/L	DecreaseH_2O_2 at 240 nm	Aebi (1984)	Measuring catalase activityCAT)
unit/L	DecreaseNADPH at 340 nm	Paglia & Valentine (1967)	Measuring glutathione peroxidase activity (GPx)
micromol/mL	interactionDTNB at 412 nm	Beutler et al. (1963)	Measurement of reduced glutathione (GSH)
nmol/ml	interactionDNPH at 370 nm	Levine et al. (1994)	Carbonyl protein measurementPC)
%	Cation exchange chromatography	HPLC – Bio-Rad D10	Measuring glycated hemoglobin (HbA1c)HbA1c)

Afterwards, the serum and plasma were isolated and stored at -80°C.Until it beganThe tests.

***Table (4) Analytical methodologies adopted for measuring indicators of oxidative stress, antioxidant* enzymes**

5. Analytical methods

The analysis was performed using softwareSPSS (version 26). The following statistical methods were used: descriptive statistics (mean ± variance), separate-samples t-test, one-way ANOVA for subgroups, Pearson correlation coefficient, and multiple linear regression analysis. P<0.05 was considered the threshold for statistical adequacy [9], [10], [11].

3. Results and Discussion

1.Demographic and clinical characteristics of the participants

Table (5)Demographic and clinical characteristics of the participants

valueP	The officer(n=60)	patients(n=60)	variable	No.
(g.m.) 0.132	50.1 ± 7.9	52.4 ± 8.7	Age (years) ± deviation	1
<0.001*	25.1 ± 3.1	29.8 ± 4.2	Body Mass Index (kg/m²)	2
<0.001*	88.3 ± 9.2	187.4 ± 45.6	Fasting blood sugar (mg/dL)	3
<0.001*	5.1 ± 0.4	8.9 ± 1.6	HbA1c (%)	4
—	—	7.3 ± 4.8	Duration of injury (year)	5
<0.001*	120.3 ± 9.8	138.5 ± 14.2	Systolic blood pressure (mm/Hg)	6
		26 (43.3%)	Treatment:Metformin	7
		34 (56.7%)	Treatment:Metformin + Sulfonylurea	8

*: Statistically significant difference (P<0.05) g . m Non-essential

* It was verified that all members of the control group fall within the normal range ofHbA1c (<5.7%), and no individual was recorded as being in the pre-diabetic range.

2.Indicators of oxidative stress

1.Malondaldehyde level (MDA)

The results of the malondialdehyde test showed (MDA) A statistically significant increase in type 2 diabetes patients compared to the control group (P<0.001The average level of this hormone in all patients was 6.84 nmol/ml compared to 2.31 nmol/ml in the control group, representing a 2.96-fold increase. This ratio was even higher in the uncontrolled patients.HbA1c ≥7%Those who recorded an average of 7.98 nanomoles/ml, which is 3.45 times the level of the control group, reflecting the close direct relationship between poor blood sugar control and increased oxidative stress.

Table (6)The effect of blood sugar control (HbA1c) on serum MDA concentration

valueP	Percentage increase compared to the control	standard deviation	middleMDA (nmol/mL)	The group	No.
	2.96 times	±1.92	6.84	patients(n=60)	1
<0.001*	-	±0.64	2.31	The officer(n=60)	2
	3.45 times	±1.74	7.98	patientsHbA1c ≥7% (n=52)	3
0.003*	1.82 times	±0.98	4.21	patientsHbA1c <7% (n=8)	4

2.carbonyl protein (PC)

We observed that the level of carbonyl protein increased significantly, reaching an average of 4.82 ± 1.23 nmol/ml in patients, while it was only 1.68 ± 0.42 nmol/ml in the healthy group.Table (7)This difference is very significant, and it means that there is strong oxidation of the proteins.WhichIt affects the function of vital enzymes and cell structure.

Figure (1) clearly illustrates a comparison between the levels of oxidative stress in the two groups, showing the significant difference between them.

Figure (1): Comparison of levelsMDA, carbonyl protein and GSH between diabetic patients and the control group (P<0.001)

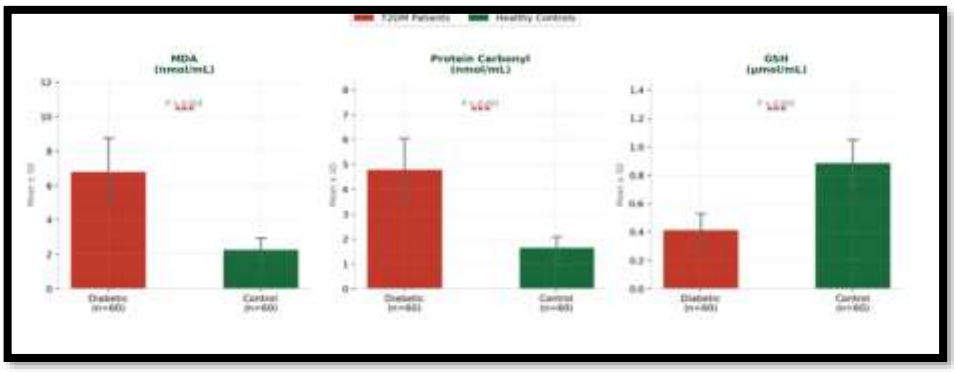


Table (7)The effect of pathological condition on oxidative balance: a study of changes in concentrationsMDA and PC

P-value	Change	The officer(mean±SD)	patients(mean± SD)	Index	No.
*<0.001	↑ 196%	2.31 ± 0.64	6.84 ± 1.92	MDA (nAnnumol/ ml)	1
*<0.001	↑ 187%	1.68 ± 0.42	4.82 ± 1.23	PC (nanomol/ml)	2

3.Antioxidant enzyme activity

Figure (2) showsTable (8)Reduced activity of all antioxidant enzymes in patients compared to the control group:

Figure (2): Enzyme activitySODandCATandGPxand levelGSHAs a percentage of the control groupP<0.001)

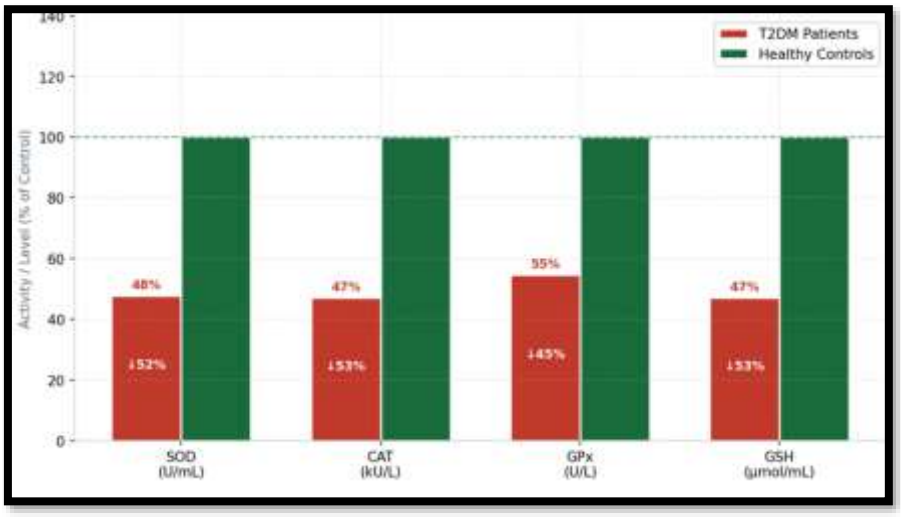


Table (8)Evaluating the efficiency of the antioxidant defense system in patients compared to healthy individuals

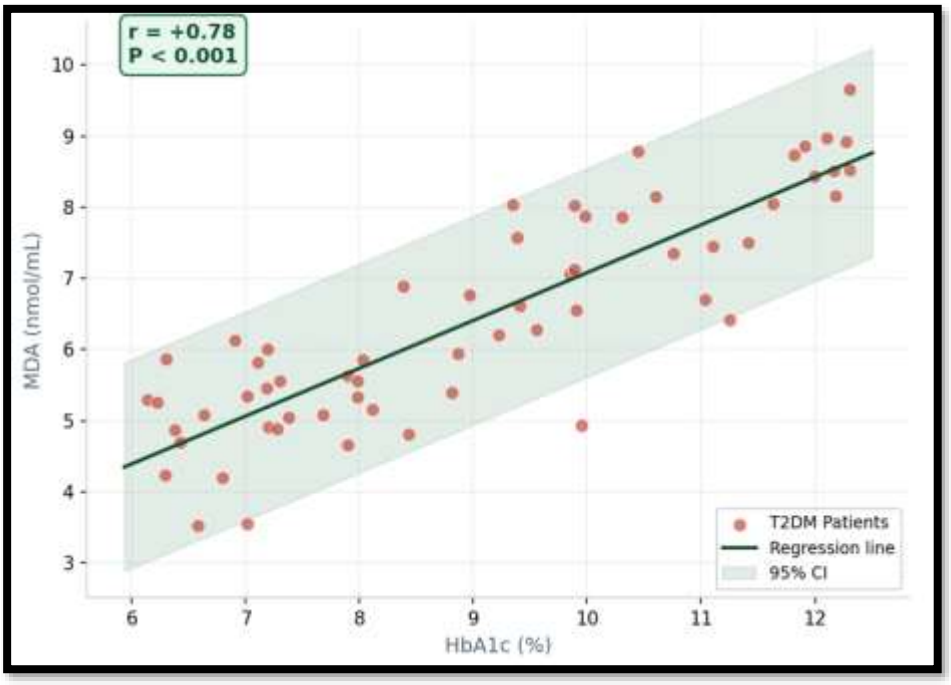
P-value	The decrease%	The officer(mean±SD)	patients(mean±SD)	Vital indicators	No.
*<0.001	↓ 52%	1.42 ± 0.29	0.68 ± 0.18	SOD units/ml	1
*<0.001	↓ 53%	8.74 ± 1.63	4.12 ± 1.08	CAT (kW/L)	2

*<0.001	↓ 45%	34.2 ± 6.9	18.7 ± 4.8	GPx (unit/L)	3
*<0.001	↓ 53%	0.89 ± 0.16	0.42 ± 0.11	GSH (µmol/ml)	4

4. Correlation analysis betweenHbA1cIndicators of oxidative stress

Figure (3) illustratesTable (9)The strong positive relationship between glycated hemoglobin and levelMDA:

Figure (3): The positive correlation betweenHbA1cand levelMDAIn the blood of diabetic patientsr = +0.78, P < 0.001)



And Pearson's correlation analysis revealed the following relationships:

Table (9) The relationship between glycated hemoglobin and levelMDA:

relationship direction	valueP	Correlation coefficient(r)	relationship	**No.**
Strong positive	*<0.001	+0.78	MDA- HbA1c	**1**
Strong negative	*<0.001	-0.72	SOD - HbA1c	**2**
Strong negative	*<0.001	-0.68	GSH - HbA1c	**3**
Moderately positive	*<0.001	+0.65	MDA - Duration of illness	**4**
Very strong negative	*<0.001	-0.81	SOD -MDA	**5**

5. Effect of injury duration on oxidative stress indicators

Figure (4) showsTable (10)The upward trend ofMDA and the gradual decrease of SOD with increasing duration of infection:

Figure (4): Gradual changes in levelsMDA and SOD according to the duration of diabetes (ANOVA: P<0.001)

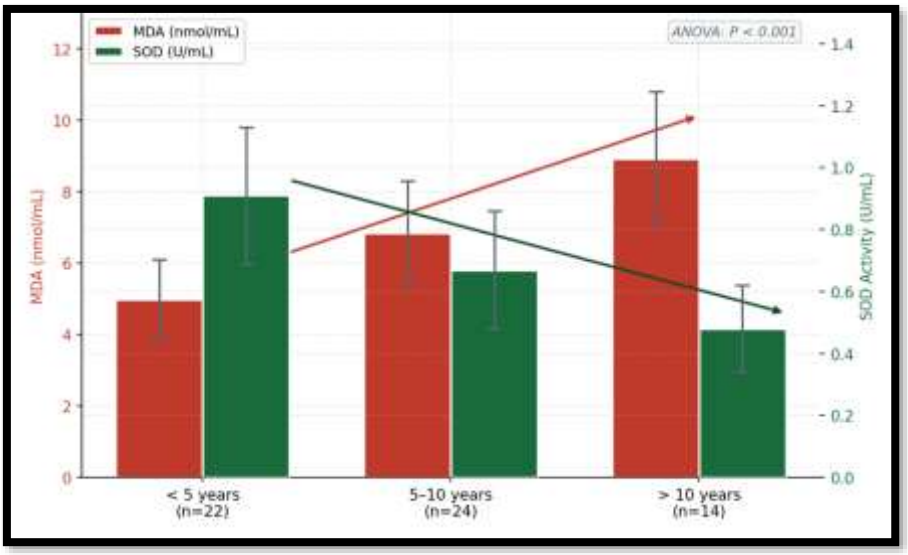


Table (10) The upward trend ofMDA and the gradual decrease of SOD with increasing duration of infection

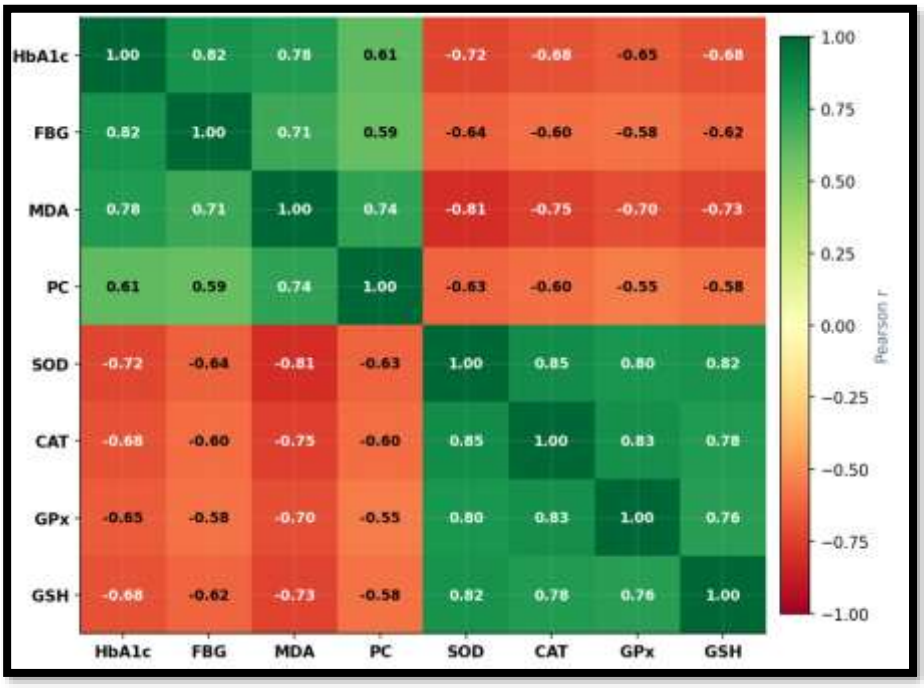
number	GSH(micromoles/ml)	SOD(unit/ml)	MDA (mol/ml)	Duration of injury	No.
22	0.61 ± 0.14	0.91 ± 0.22	4.98 ± 1.12	< 5 years	1
24	0.41 ± 0.11	0.67 ± 0.19	6.84 ± 1.45	5-10 years	2
14	0.28 ± 0.08	0.48 ± 0.14	8.92 ± 1.87	> 10 years	3

The table shows a linear upward trend at the levelMDAand a gradual decrease in activitySODand levelGSHWith an increasing duration of diabetes (ANOVA: P<0.001).

6. Global correlation matrix

Figure (5) illustrates the complete statistical relationships between all the variables assessed in the study:

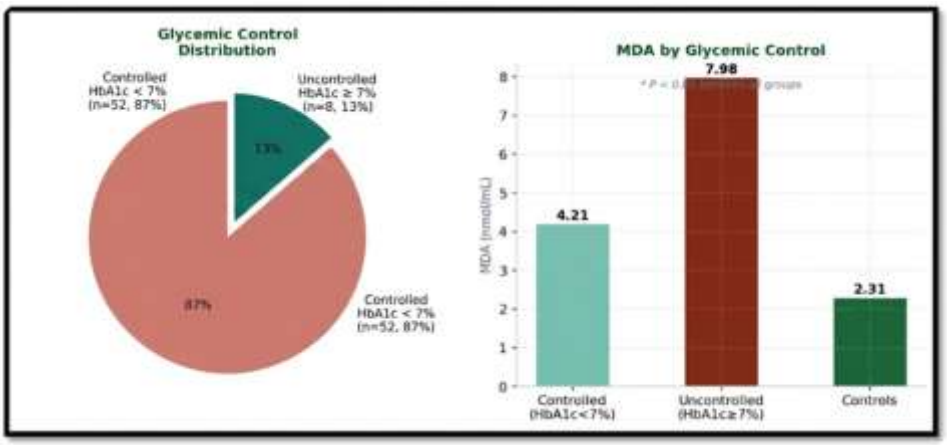
Figure (5): Correlation matrix between oxidative stress indices, antioxidant enzymes and clinical variables



7.Comparison between controlled and uncontrolled patients

Figure (6) compares the levelMDAIn controlled cases (HbA1c <7%and uncontrolledHbA1c ≥ 7%And the control group:

Figure (6): Distribution of cases of glycemic control and levelMDAAccording to the degree of controlHbA1c



discussion

1.Oxidative stress in diabetes: confirmation and explanation

It should be noted that the selection of members of the control group was based on strict criteria that included verifying the integrity of glucose metabolism, as all members underwent measurement.HbA1c and fasting blood sugar were measured, and anyone who showed any indication of a metabolic disorder was excluded, which enhances the reliability of the comparison between the two groups and the accuracy of the conclusions drawn.

The results of the current study confirm a statistically significant increase in oxidative stress levels among patients with type 2 diabetes compared to the control group, which is consistent with the findings of numerous international studies in this field. This marked increase in oxidative stress levels represents...MDA, which reached approximately 2.96 times its level in healthy individuals, is compelling evidence of accelerated lipid peroxidation in cell membranes, particularly in blood vessel cells and tissues susceptible to diabetic complications. This increase is attributed to the synergy of several molecular mechanisms that contribute to the generation of reactive oxygen species (ROS) under conditions of chronic hyperglycemia, most notably: the depletion of NADPH via the polyol pathway, which reduces the stores of reduced glutathione (GSH), and the accumulation of ROS products.yourAdvanced Lycosyl (AGEs) activate the NADPH oxidase enzyme, as well as excessive glucose flow through the mitochondrial electron transport chain, leading to excessive production of the superoxide anion ($O_2^{\bullet-}$) (Brownlee, 2001) [12], [13], [14], [15].

2.Reduced activity of antioxidant enzymes

Our study showed that all measured antioxidant catalysts decreased simultaneously, with the percentage decreasingSOD decreased by 52%, CAT by 53%, and GPx by 45%. The majority of the SOD decrease is attributed to the destruction of thiol groups at the active site, along with a deficiency in essential enzyme-supporting components. The CAT decrease is due to reduced gene expression resulting from a decrease in the Nrf2 pathway under the influence of elevated glucose levels. Similarly, GPx and GSH work closely together; a deficiency in GSH reduces the availability of the precursor to GPx, "leading to a negative feedback loop that progressively and progressively weakens the antioxidant defense system."

3.Connection withHbA1cClinical significance

One of the most important resultsthat It resulted About herThe study was about linking levelsHbA1c levels are clearly markers of oxidative stress. Correlation analysis showed that elevated HbA1c levels are associated with a significant increase in MDA concentration, reflecting increased lipid peroxidation due to poor glucose control.

Conversely, elevated HbA1c is associated with a significant decrease in SOD enzyme activity, indicating a weakening of the antioxidant defense system. These results reveal that glycemic control is not merely about achieving numerical targets, but rather a fundamentally effective means of reducing oxidative damage at the cellular level. This supports medical advice.thatIt emphasizes the importance of reducingHbA1c as part of a comprehensive plan to maintain body health

4.Relationship to duration of injury: Oxidative accumulation

We noticed that levelsMDA levels gradually increase over time, while SOD and GSH levels progressively decrease with continued exposure. This cumulative pattern is consistent with the concept of metabolic memory.Metabolic Memory), which indicates that epigenetic modifications (Epigenetic modificationsThe damage caused by chronic oxidative stress may persist even after blood sugar control improves.Ceriello et al., 2009).

5.The therapeutic importance of the results

These findings provide a solid scientific basis for considering the inclusion of antioxidants in treatment regimens for type 2 diabetes, especially since common antidiabetic drugs like metformin have demonstrated antioxidant activity in addition to their blood sugar-lowering effects. This also opens the door for clinical trials to assess the vitamin's effectiveness.Vitamin E, vitamin C, coenzyme Q10, and curcumin are potential influencers of oxidative stress in this patient group [16], [17], [18], [19], [20], [21], [22].

4. Conclusion

1.Conclusions

1. Individuals with type 2 diabetes experience significantly higher oxidative stress (P<0.001This appears in the rise of the levelMDAAnd the proteocarbonyl.

2. These patients show a general deterioration in the antioxidant protection system with a significant decrease in levelsSODandCATandGPxandGSH.

3. There is a strong positive correlation betweenHbA1cIndicators of oxidative stress and a strong negative correlation with antioxidant enzymes.

4. Oxidative stress gradually increases as the disease duration lengthens, confirming the phenomenon of oxidative accumulation.

5. Oxidative stress indicators are considered promising biomarkers for the imminent assessment of complications.

2. Recommendations

1. Adopting a scaleMDAandSODWithin the system for monitoring diabetic patients as early indicators of complications.

2. Conduct randomized controlled clinical studies to evaluate the potential use of antioxidant supplements.

3. Researching the effect of dietary and physical procedures on oxidative stress among Iraqi patients.

4. Expanding the research to include indicator 8-OHdGand pathNrf2At the molecular level.

5. Coordinating with the health department to adopt awareness plans that link blood sugar control with oxidative protection.

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