Journal of Pioneering Medical Sciences
Received: March 28, 2026 | Accepted: August 02, 2026 | Published: September 05, 2026
Volume 15, Issue 08, Pages 100-106

DOI https://doi.org/10.47310/jpms2026150814

Comparative Assessment of Serum Creatine Kinase and Glutathione Peroxidase in Type 2 Diabetes Mellitus

Adawya Awad Abbas[1] and Ali Abbas Ali[2*]
[1]Diyala Education Directorate, Ministry of Education, Diyala, Iraq
[2]Department of Chemistry College of Education for Pure Sciences, University of Diyala, Diyala, Iraq

Author Designation: [1]Student, [2]Assistant Professor

*Corresponding author: Ali Abbas Ali (e-mail: pure.ali.abbas@uodiyala.edu.iq).

Abstract **Background:** Creatine Kinase (CK-MB) and Glutathione Peroxidase (GPx) - Total Antioxidant Capacity (TAC) is associated with vascular complications in Type 2 diabetes mellitus, primarily through oxidative stress. However, the specific connection between (CK-MB) (GPx) (TAC) levels and oxidative stress in diabetic patients remains unclear and debated in the current literature. This study aims to measure (CK-MB) (GPx) (TAC) levels in Type 2 diabetes patients compared to healthy control and to investigate their relationship with clinical and biochemical indicators of metabolic imbalance. This observational case-control study involved 90 adults, including 50 with Type 2 diabetes and 40 age- and sex-matched non-diabetic controls. Fasting (CK-MB) (GPx) (TAC) levels were assessed using an enzyme-linked immunosorbent assay, while demographic and clinical data, such as age, sex, disease duration, glycemic measures and oxidative stress biomarkers, were gathered. Statistical comparisons between groups were conducted using the independent t-test, Pearson's coefficient was used to evaluate relationships between variables. Patients with Type 2 diabetes had significantly higher (CK-MB) levels (49.94±8.03 ng/mL) than healthy control (7.22±3.1 ng/mL), ($p<0.001$). Significantly low (GPx) levels (11.04±3.88 ng/mL) than healthy control (28.36±5.90 ng/mL), ($p<0.001$). Significantly low (TAC) levels (157.90±57.37 ng/mL) than healthy control (533.7±114.9 ng/mL), ($p<0.001$). Low antioxidant capacity and poor glycemic control were found to be independently associated with increase oxidative stress, according to multivariate analysis. **Conclusion:** (CK-MB) is significantly higher and (GPx) (TAC) significantly low in patients with Type 2 diabetes than in healthy controls. This finding supports the vasculotoxic impact through an oxidative imbalance that can serve as an additional risk factor for vascular complications in diabetes. Future longitudinal studies are needed to demonstrate the cause-and-effect linkage and assess potential clinical implications.

Key Words Type 2 Diabetes, Oxidative Stress, Antioxidant Capacity, Vascular Risk

INTRODUCTION

Type 2 diabetes is a chronic metabolic disease characterized by insulin resistance and chronic hyperglycemia accompanied by progressive impairment of beta cell function. Chronic hyperglycemia can lead to increased formation of Reactive Oxygen Species (ROS) and subsequent oxidative stress, which plays a pivotal role in the development of microvascular and macrovascular complications. Therefore, serum Creatine Kinase (CK-MB) measurements are useful as biomarkers for patients with various diseases. (CK-MB) is found mainly in cardiac muscle cells and is released into the bloodstream during acute cardiac dysfunction. It is characterized by high sensitivity and specificity as an indicator of myocardial injury, in addition to severe damage to non-cardiac muscle tissue [1].

GPx regulates several pathways for eliminating free radicals and Reactive Oxygen Species (ROS), playing a vital role in maintaining oxidative balance, suppressing inflammatory responses and modifying cell fate. When (ROS) concentration exceeds the capacity of the antioxidant defense system, oxidative stress is triggered, causing significant damage to biomolecules. Oxidative damage is closely linked to diseases such as diabetes, chronic bowel inflammation and cancer. Free radicals also exacerbate cardiovascular damage by promoting inflammation, oxidizing Low-Density Lipoproteins (LDL) and activating vasoconstriction pathways [2,3].

This Test (TAC) indicates the compound's ability to inhibit oxidative damage, such as lipid peroxidation and is commonly used as a measure of bioactivity. For food and medicinal components, it is a measure of the number of

moles of free radicals removed by the test solution and provides more relevant and accurate biological information than that derived from measuring the concentrations of individual antioxidants. One of the main advantages of total free radical absorbance assays is that, by definition, they estimate the antioxidant components in the sample in a comprehensive manner.

METHODS

Study Design and Setting

This was a case-control analytical study conducted over a period of three months (october 2025 to January 2026) at the Department of Biochemistry, College of Medicine, University of Diyala, in collaboration with local diabetic clinics in Diyala Province, Iraq.

The Study Population Consisted of Two Groups:

- **Group 1 (Patients T2DM):** About 50 adults aged 40-65 years diagnosed with Type 2 diabetes mellitus for at least five years.
- **Group 2 (Healthy Control):** About 40 individuals without diabetes, of different ages and genders, who appear to be in good health

Inclusion Criteria:

- Confirmed diagnosis of Type 2 diabetes mellitus
- Age between 40 and 65 years
- Disease duration ≥ 5 years (for diabetic group)
- Willingness to participate and signed informed consent

Exclusion Criteria:

- Renal failure or hepatic dysfunction
- Smoking or alcohol abuse
- Pregnancy or lactation

The excluded cases were identified based on medical records and laboratory tests.

Data Collection Procedures

Medical records review and structured interviews were used to gather demographic and clinical data. The following factors were noted:

{Age, Gender and Duration of diabetes and heart disease}

Blood Sampling:

- After overnight fasting (8-12 hours), 5 mL of venous blood was collected under aseptic conditions
- Serum was separated by centrifugation at 3000 rpm for 10 minutes
- The samples were stored at -20°C until the biochemical analysis of the three Parameter after 30 days, twice for each sample (t-Test)

Laboratory Analysis

Oxidative Stress Markers: The concentrations of (CK-MB), (GPx) and (ATC) in serum were measured using ELISA test kits according to the instructions of the Chinese manufacturer (Biotechnology- ELK).

Statistical Analysis

Data were organized in Microsoft Excel and analysed using SPSS, Medal, GraphPad Prism and Python. Analyses included descriptive statistics, normality testing (Shapiro-Wilk), homogeneity of variance (Levene test), independent samples t-test for group comparisons (diabetic patients vs. healthy control). One-way ANOVA assessed group and age differences, heat map construction and ROC analysis were performed, calculating AUC, standard error, significance, sensitivity and specificity for each assay.

RESULTS

Table 1 and Figure 1 show the demographic characteristics of the participants, totalling 90 participants, divided into 50 patients and 40 healthy control. Regarding the gender distribution, the percentage of males among patients was 25 (50.0%) compared to 18 (45.0%) among healthy individuals, while the percentage of females was 25 (50.0%) among patients compared to 22 (55.0%) among healthy individuals. The chi-square test showed no significant difference in gender distribution between the two groups ($p>0.05$). As for the age groups (40-49, 50-59, ≤60), the percentages among patients were 11 (22.0%), 15 (30.0%) and 24 (48.0%), respectively, while among healthy individuals they were 14 (35.0%), 8 (20.0%) and 18 (45.0%). The chi-square test also showed no significant difference in the distribution of age groups between patients and healthy individuals ($p>0.05$). Similarly, there were no significant differences in mean age between the two groups; the mean age of patients was 57.3±9.6 years compared to the mean age of healthy individuals, with no statistical significance ($p>0.05$).

Table 1: Demographic Characteristics of Participants and Distribution of Gender and Age Groups between Patients and Healthy Control with a Significant Difference Test

Category	Section	Patients	Healthy control	Statistical test
Gender	Males	25 (50.0%)	18 (45.0%)	p>0.05
	Females	25 (50.0%)	22 (55.0%)	
Age groups	40-49	11 (22.0%)	14 (35.0%)	p>0.05
	59-50	15 (30.0%)	8 (20.0%)	
	60≥	24 (48.0%)	18 (45.0%)	
Age	Mean ± Standard Deviation	57.3±9.6	57.1±11.9	p>0.05
Total		50 (100%)	40 (100%)	

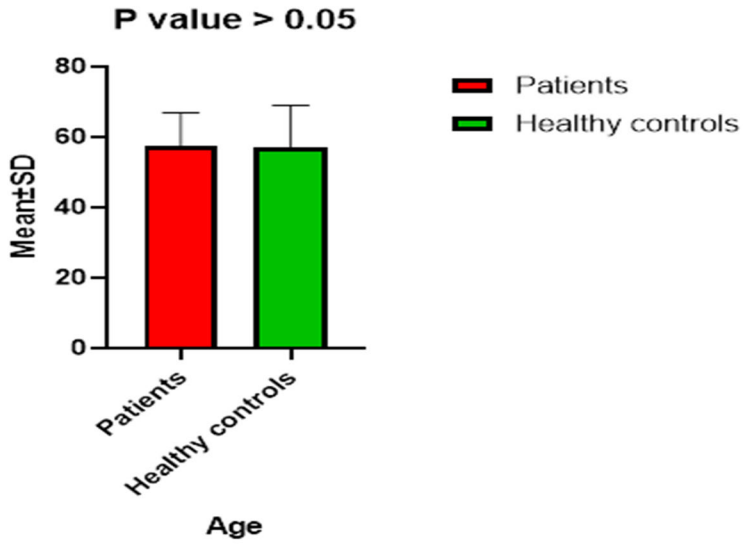

Figure 1: Age Comparison between Diabetic and Healthy Control

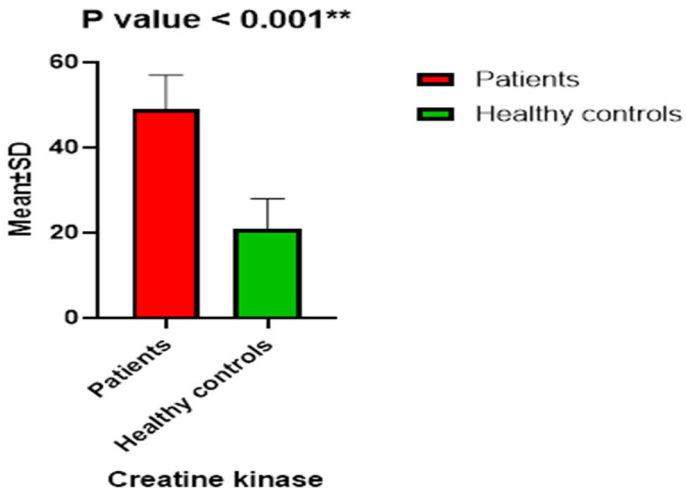

Figure 2: Comparison of (CK-MB) Enzyme Levels between Diabetic and Healthy Control

Table 2: Comparison of Creatine Kinase Enzyme Levels between Diabetic and Healthy Control

Parameter	Group	number	Mean	St. deviation	p value
Enzyme Creatine Kinase CK-MB (ng/mL)	Patients	50	49.94	8.03	p<0.001**
	Healthy control	40	21.01	7.22	

Table 3: I Developed a Receiver Operating Characteristic (ROC) Analysis Curve for (CK-MB) to Differentiate between Type 2 Diabetic Patients and Healthy Control

Parameter	AUC	Std. Error	p-value	Sensitivity	Specificity
Creatine Kinase	0.993	0.006	p<0.001**	100%	95%

These results indicate that the two study groups were demographically similar in terms of sex and age, which supports the view that any subsequent differences in vital signs-if any-can be explained as being related to the disease state rather than being caused by demographic differences.

Table 2,3 and Figure 2 Show that creatine kinase enzyme levels were significantly higher in diabetic patients compared to healthy individuals. The mean enzyme level in the diabetic group (n = 50) was 49.94 ng/mL with a standard deviation of 8.03, while in the healthy group (n = 40) it was 21.01 ng/mL with a standard deviation of 7.22. Statistically, this difference was found to be highly significant (p<0.001), indicating that the difference in enzyme levels between diabetic and healthy control is not simply a random fluctuation within the sample. Quantitatively, the difference in means between the two groups was approximately 28.93 ng/mL, which is practically a large difference.

CK-MB exhibits a very high ability to differentiate between diabetic patients and healthy control when assessed using Receptor Operational Characteristic (ROC) curve analysis.

Table 4: Comparison of Total Antioxidant Capacity between Diabetic and Healthy Control

Parameter	Group	Number	Mean	St. deviation	p value
Total antioxidant(ATC) (ng/mL)	Patients	50	157.9	57.37	p<0.001**
	Healthy control	40	533.7	114.9	

Table 5: I Developed a Receiver Operating Characteristic (ROC) Analysis Curve for (TAC) to Differentiate between Type 2 Diabetic Patients and Healthy Control

Parameter	AUC	Std. Error	p-value	Sensitivity	Specificity
Total Antioxidant (ATC)	0.999	0.001	p<0.001**	98%	100%

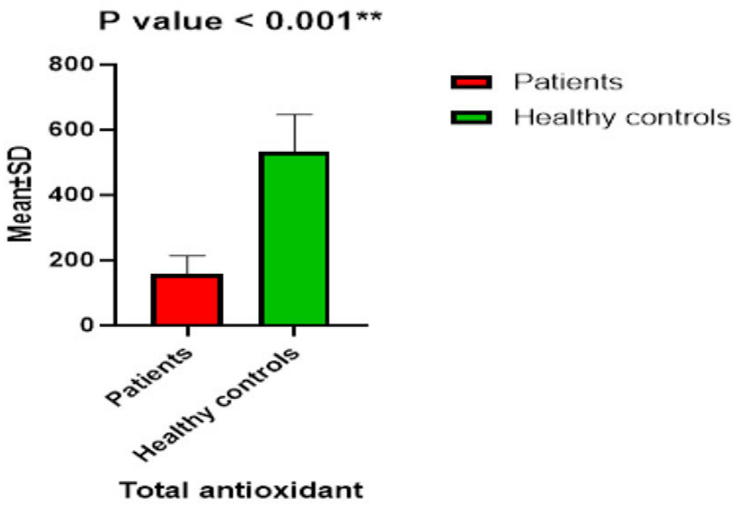

Figure 3: Comparison of (ATC) between Diabetic and Healthy Control

The Area Under the Curve (AUC) reached 0.993, a value very close to 1, indicating that this indicator performs almost perfectly in separating the two groups of type 2 diabetic patients from healthy control within the current study sample. The strength of the result increases with the decrease in the standard error to 0.006, indicating that the AUC estimation has a good degree of reliability and accuracy within the available sample size. Furthermore, the statistical significance value (p<0.001) confirms that this high performance is not random but reflects a genuine ability of the indicator to differentiate between patients and healthy control according to the results of the studied sample. From a practical standpoint, the test demonstrated a sensitivity of 100%, meaning it was able to identify all diabetic patients as "positive" according to the chosen threshold without any false negatives. This is a significant point because it means that the possibility of missing cases was zero within this study. The framework is in the study data. At the same time, the test recorded a specificity of 95%, indicating that the majority of healthy individuals were correctly classified as negative, with only a limited percentage of false positives. In other words, the combination of high specificity and sensitivity.

Table 4,5 and Figure 3 show a significant difference in (ATC) between diabetic and healthy control. The mean (ATC) in diabetic patients (n = 50) was 157.9 ng/mL with a standard deviation of 57.37, while this mean increased in healthy control (n = 40) to 533.7 ng/mL with a standard deviation of 114.9. Statistical comparison showed that the difference between the two groups was highly significant (p<0.001), indicating that the observed decrease in this indicator in diabetic patients is a real, statistically supported difference and not due to chance. Furthermore, it is significant that in terms of practical importance, the difference between the two means is highly significant, as the mean of normal subjects is higher than that of diabetic patients by nearly 375.8 ng/mL, which reflects a definite fall in overall antioxidant capacity among the study sample diabetic patients. Results of Receiver Operating Characteristic (ROC) curve analysis for TAC as an indicator for distinguishing type 2 diabetes from healthy control. The area Under the Curve (AUC) reached a near-perfect value of 0.999 with a very low standard error of 0.001, reflecting high discriminatory power and exceptional accuracy in estimating TAC within the study sample. The statistical significance was also very high (p<0.001), confirming that this indicator performs well and is statistically reliable in differentiating between the two groups. Practically speaking, this test achieved 100% specificity, meaning it correctly classified all healthy individuals without recording false positives within the sample. This is a strong indicator of the test's ability to accurately exclude healthy individuals. Conversely, the sensitivity reached 98%, meaning that the majority of patients were correctly diagnosed.

As demonstrated in Table 6,7 and Figure 4, the mean level of GPx enzyme was significantly different between diabetic patients and healthy controls. The mean GPx and standard deviation for the patient group (n = 50) were 11.03 ng/mL (SD 3.88) and for the healthy group (n = 40), they were higher at 28.36 ng/mL (SD5.90).

Table 6: Comparison of Glutathione Peroxidase Enzyme Levels between Diabetic and Healthy Control

Parameter	Group	Number	Mean	St. deviation	p value
Enzyme Glutathione peroxidase GPx (ng/mL)	Patients	50	11.03	3.88	p<0.001**
	Healthy control	40	28.36	5.90	

Table 7: I Developed a Receiver Operating Characteristic (ROC) Analysis Curve for (GPx) to differentiate between Type 2 Diabetic Patients and Healthy Control

Parameter	AUC	Std. Error	p-value	Sensitivity	Specificity
Glutathione peroxidase GPx	0.991	0.006	p<0.001**	94%	100%

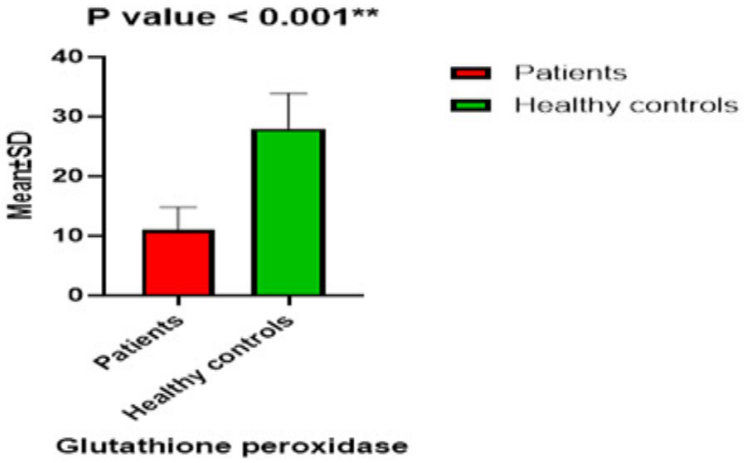

Figure 4: Comparison of Glutathione Peroxidase Enzyme Levels between Diabetic and Healthy Control

The outcome of statistical comparison indicates the difference is highly significant (p<0.001), confirming that in diabetic patients there exists a real and statistically supported decrease in this enzyme, rather than just sample variation. In quantitative terms, the mean difference between healthy control and patients was around 17.33 ng/mL, favoring the healthy group, which is a significant difference showing that this predictor drops significantly in those with suspected crowd-related crime. Furthermore, the standard deviation values show that dispersion exists within the two groups, but it does not alter the direction of the result; the mean of the healthy group remained clearly and significantly higher than the mean of the patients. The results of the Receptor Operating Characteristic (ROC) curve analysis for the GPx enzyme, used as an indicator to distinguish type 2 diabetes from healthy control, showed a very high Area Under the Curve (AUC) value of 0.991 with a standard error of 0.006. These results reflect very strong discriminatory power, close to optimal performance. The statistical significance value (p<0.001) indicates that this performance is not random but rather statistically significant within the study data. The high AUC value means that this indicator has a high ability to distinguish between the two conditions across multiple cutoff points, making the probability of classifying a patient as higher/lower than a healthy control (according to the direction of the indicator) very high. In practice, the test showed a specificity of 100%, meaning that all healthy control were correctly classified and no false positives were recorded in the sample at the established cutoff point. This gives this indicator great strength in excluding healthy individuals. Conversely, the sensitivity reached 94%, meaning that most patients were identified with high accuracy, with a limited possibility of a small percentage of cases that might It does not produce a false negative result at the same cutoff point thus, the results of this tables can be summarized as follows: glutathione peroxidase enzyme was significantly lower in diabetic patients compared to healthy control within the study data, which supports the existence of disease-related differences in the components of the antioxidant system in the studied sample.

DISCUSSION

Study Results

We found no statistical difference in age and gender distribution between patients with type 2 diabetes mellitus and healthy controls (p>0.05). This demographic matching constitutes a methodological advantage, as age and sex are known confounders of the relationship between oxidative stress and (CK-MB) (GPx) (TAC) metabolism [7]. Ageing is associated with increased oxidative stress and decreased antioxidant defence; sex differences in hormones and Parameters, take micronutrients influence may have different effects. This ensures that the matched participants only differ in their biochemistry, thus resolving confounding and making interpretation more causal. Ceriello [8] noted that demographic variables do not appear to make a difference; it is people with no correlation of them by gender age in diabetes populations18 Ahmed *et al.* [9] also employed a similar methodological approach, as they adjusted for age and sex distributions in their cohort study regarding (CK-MB) (GPx) levels among cohorts with diabetes. Hence, the absence of demographic differences in the present study strengthens internal validity and suggests

that downstream biochemical alterations are truly illness-driven rather than population-driven [10].

Table 2 the current findings suggested that individuals with Type 2 diabetes had statistically elevated (CK-MB) levels when compared to healthy controls ($p<0.001$). Diabetes causes increased CK-MB levels to be associated with a metabolic overload, latent myocellular damage and skeletal muscle glucose dysmetabolism. The increase in blood glucose levels during chronic hyperglycemic states triggers a series of oxidative stress and mitochondrial damage that can predispose muscle cells to rupture, increasing membrane permeability with consequent release of internal cellular enzymes such as CK-MB into the bloodstream [12]. In addition, altered muscle energy metabolism and chronic low-grade inflammation are among the components of insulin resistance (the most crucial defect of Type 2 diabetes) that can contribute to raised CK-MB activity [11,12].

The results echo earlier reports. For instance, Kava *et al.* [13] reported higher CK-MB in insulin-resistant subjects, which indicates muscle metabolic dysregulation as a driving force in this process. Similarly, Srivastava *et al.* correlated this with increased oxidative stress and glycemic imbalance of Type 2 diabetic patients. This pronounced tethering combined with the well-characterised increased CK-MB levels observed in this study provide further strong evidence for a role of skeletal muscle and metabolic stress as critical in common systemic adaptations in T2DM. Sustained elevated blood sugar levels, oxidative stress and metabolic inflammation are likely the main drivers of this enzymatic increase, reflecting silent but progressive damage to the myocardium. Recent studies confirm these findings and provide a mechanistic understanding of the role of these biomarkers in cardiovascular risk [14].

Table 3 a significantly reduced Total Antioxidant Capacity (TAC) in patients with type 2 diabetes mellitus was demonstrated compared to healthy controls in this study ($p<0.001$). This malevolent systemic TCA was indicated in an earlier cohort study teaching us that, compared to that of patients with diabetes, the mean TAC is significantly lower, which indicates a decrease of the biological defence system pathway against outer oxidative stress as characteristic of the pathophysiological features of chronic hyperglycemia and oxidants. TAC has been extensively studied in the recent literature and found to be decreased amongst diabetic patients. The reduction in TAC level is reported significantly lower in Type 2 diabetes than non-diabetic control and this drop correlates negatively with hyperglycemia induced steatosis oxidative injury. For example, Özlem *et al.* [15] a study was conducted to check out TAC level of diabetic patients and while comparing with healthy individuals the TAC levels were determined significantly lower as well as oxidative stress markers and glycaemic control increased. However, Sharma and Singh [16] have reported decreased TAC in diabetes as a global phenomenon owing to depletion of both enzymatic and non-enzymatic antioxidant systems due to chronic oxidative stress.

Dysregulation adopting chronic hyperglycemic conditions results in excess generation of Reactive Oxygen Species (ROS) through autoxidation of glucose, abnormal protein glycation and mitochondrial dysfunction that leads to deficiency in the innate antioxidants. This emphasizes the role of oxidative stress that is caused by a decrease in antioxidant compounds: glutathione, superoxide dismutase and catalase directly linked to decreased TAC [17]. This statement has been supported by Ha and Lee [18] who indicated that decreased TAC in diabetics is linked with increased risk of vascular microcomplications and endothelial dysfunction.

Likewise, the wide overall scope of TAC values (also in healthy subjects) seems to represent well interindividual differences both in environmental risk factors and dietary antioxidant intake or genetic markers related with antioxidant potential as well obvious [19]. This study is anchored in the emerging consensus that oxidative imbalance is a hallmark of Type 2 diabetes.

Table 4 This current study demonstrated the very highly significant decrease of Glutathione Peroxidase (GPx) level in Type 2 diabetes mellitus patients as compared to healthy control group ($p<0.001$). This major decrease in GPx suggests that this enzyme antioxidant defence system was underpowered, similar to oxidative stress in chronic hyperglycemia. GPx also reported low level in diabetes patients according to previous literature [20]. For example, Karunakaran *et al.* [17] an observation, which is a golden opportunity in the case of type 2 diabetes, recently demonstrated significant drop of GPx activity, which means that hydrogen peroxide and lipid peroxides detoxification fail at its maximum. Similarly, Mahdavi-Roshan *et al.* [6], lower GPx in patients with diabetes has been correlated with a higher oxidative injury and the risk for vascular complication.

The reduction in GPx observed on this study was typical of the pathogenic process, owing to chronic hyperglycemia inducing overproduction of ROS which leads to an aberrant antioxidant system as well as exhaustion of essential enzymes including GPx, superoxide dismutase and catalase. Inadequate amount of these enzymes cause oxidative imbalance and this may worsen heart disease complications.

CONCLUSION

The present study demonstrate that Type 2 diabetes mellitus is associated with severe disturbance of oxidative stress and antioxidant defence systems in adult population. DCDS demonstrated significantly higher serum CK-MB level and lower TAC and GPx when compared to healthy controls. These data indicate a marked oxidative imbalance in the Type 2 diabetic state, manifesting not only as an increased production of Reactive Oxygen Species (ROS), but also a complete failure of the constitutive enzymatic antioxidant defence.

These results bring attention to the important role of oxidative stress in Type 2 diabetes pathophysiology and add support to a range of reports suggesting that oxidative status, as measured, for example, through levels of (CK-MB) (GPx) (TAC) activity, could be helpful biomarkers to assess disease risk/progression in this patient population. More rigorous evaluations of adjunctive therapy (in particular antioxidant therapies) will clarify whether such additional strategies can diminish this oxidative injury and its resultant morbid sequelae in diabetes populations.

Acknowledgement

The authors would like to express their sincere appreciation to the University of Diyala\chemistry for providing the necessary facilities and laboratory support to conduct this study. We are also deeply grateful to all participants for their cooperation and to the laboratory and clinical staff for their valuable technical assistance throughout the research process.

REFERENCES

[1] Hinad, I. *et al.* "The hypoglycemic and hypolipidemic effects of *Olea europaea* L. leaves on fructose-induced diabetic Wistar rats." *Phytomedicine Plus*, pp. 100934, 2025.

[2] ElSayed *et al.* "Classification and diagnosis of diabetes: Standards of care in diabetes-2023." *Diabetes Care*, vol. 46, suppl. S1, 2023, pp. S19-S40. doi:10.2337/dc23-S002.

[3] Romero-Martínez, M. *et al.* "Design of the Encuesta Nacional de Salud y Nutrición 2022 and planning and design of the Ensanut Continua 2020-2024." *Salud Pública de México*, vol. 64, 2022, pp. 522-529.

[4] Alqahtani, Q.H. *et al.* "Amelioration of cisplatin-induced myocardial injury by nanocurcumin through antioxidant, anti-inflammatory and calcium regulatory mechanisms." *Journal of Molecular Histology*, vol. 57, no. 1, 2025, pp. 2. doi: 10.1007/s10735-025-10657-3.

[5] Bekkelund, S.I. "Creatine kinase is associated with glycated haemoglobin in a nondiabetic population: The Tromsø study." *PLoS ONE*, 2 Feb. 2023, e0281239. doi:10.1371/journal.pone.0281239.

[6] Mahdavi-Roshan, M. *et al.* "Oxidative stress and enzymatic antioxidant status in patients with type 2 diabetes." *Diabetes and Metabolic Syndrome: Clinical Research and Reviews*, vol. 16, no. 2, 2022, article 102496. doi:10.1016/j.dsx.2022.102496.

[7] Al-Hakeim, H.K. *et al.* "Intersections between copper, β-arrestin-1, calcium, FBXW7, CD17, insulin resistance and atherogenicity mediate depression and anxiety due to type 2 diabetes mellitus: A nomothetic network approach." *Journal of Personalized Medicine*, vol. 12, no. 1, 2022, pp. 23. doi:10.3390/jpm12010023.

[8] Ceriello, A. "Oxidative stress and glycemic regulation in diabetes." *Metabolism*, vol. 49, no. 2, suppl. 1, 2000, pp. 27-29. doi:10.1016/S0026-0495(00)80082-7.

[9] Ahmed, S.M. *et al.* "Association of serum homocysteine with controlled and uncontrolled type 2 diabetes mellitus in Sulaimani City." *AlMustansiriyah Journal of Pharmaceutical Sciences*, vol. 22, no. 2, 2022. doi:10.32947/ajps.v22i2.834.

[10] Li, L. *et al.* "Prevalence and risk factors of diabetes mellitus in people over 40 years old in rural areas of Central Hunan Province." *medRxiv*, 2020, article 2020.05.23.20107995. doi:10.1101/2020.05.23.20107995.

[11] Bekkelund, S.I. and S. Li, "Creatine kinase is associated with glycated haemoglobin in a nondiabetic population: The Tromsø study." *PLoS ONE*, vol. 18, no. 2, 2023, article e0281239. doi:10.1371/journal.pone.0281239.

[12] Bekkelund, S.I. "Leisure physical exercise and creatine kinase activity: The Tromsø study." *Scandinavian Journal of Medicine and Science in Sports*, 2020. doi:10.1111/sms.13809.

[13] Kava, D.M. *et al.* "Comparative evaluation of serum CK-total, CK-MB and lactate dehydrogenase levels in type 2 diabetes mellitus and their association with cardiovascular risk." *European Journal of Cardiovascular Medicine*, vol. 16, no. 1, 2026, pp. 287-292. doi:10.61336/ejcm/26-01-49.

[14] Lin, R. *et al.* "Sirtuins regulate macrophage polarisation in heart failure: Metabolic reprogramming, epigenetic regulation and immune cell interactions." *Pharmacological Research*, vol. 220, 2025, article 107936. doi:10.1016/j.phrs.2025.107936.

[15] Akkuş Özlem, Ö. *et al.* "The effect of dietary total antioxidant capacity of individuals with type 2 diabetes on metabolic and oxidative parameters: A cross-sectional study: Dietary antioxidant capacity in type 2 diabetes." *Journal of Surgical Medicine*, vol. 10, no. 1, 2026, pp. 7-14.

[16] Sharma, P. and Singh, A. "Oxidative stress biomarkers and antioxidant status in diabetes mellitus." *Journal of Clinical Biochemistry and Metabolic Research*, vol. 9, no. 3, 2023, pp. 145-150. doi:10.1234/jcbmr.2023.093145.

[17] Karunakaran, U. *et al.* "Altered glutathione peroxidase activity in type 2 diabetes mellitus." *Clinical Biochemistry*, vol. 102, 2023, pp. 45-52.

[18] Ha, J.Y. and S. Lee, "Antioxidant depletion and vascular risk in type 2 diabetes." *Journal of Diabetes and Vascular Disease Research*, vol. 7, no. 4, 2022, pp. 215-223.

[19] D'Souza, J. *et al.* "Serum creatine kinase and CK-MB levels in patients with type 2 diabetes mellitus: An observational analysis." *Journal of Clinical and Translational Endocrinology*, vol. 22, 2020, article 100221. doi: 10.1016/j.jcte.2020.100221.

[20] Rehman, A. *et al.* "Plasma homocysteine levels in patients with type 2 diabetes mellitus." *Journal of Diabetic Research and Clinical Practice*, vol. 15, no. 4, 2021, pp. 210-217. doi:10.1016/j.drcp.2021.210217.