



Evaluation of Antioxidant Status in Type 2 Diabetes Patients Attending Madonna University Teaching Hospital

Arunsi Ogbonnaya Mba^{1*}, Onwubiko Dike¹, and Ibeh Rhoda Ifechukwu²

¹Department of Medical Laboratory Science, Abia-State University, Uturu

²Department of Medical Laboratory Science, Madonna University Elele

Corresponding Author: Arunsi Ogbonnaya Mba

Department of Medical Laboratory Science, Abia-State University, Uturu

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ABSTRACT	Original Research Article
Type 2 diabetes mellitus (T2DM) is a chronic metabolic condition characterised by persistent hyperglycemia, insulin resistance and increasing pancreatic β-cell dysfunction. Increased oxidative stress and poor antioxidant defense are critical factors implicated in the development and progression of diabetes problems. This study investigated certain selected antioxidant enzyme activities in patients with T2DM visiting Madonna University Teaching Hospital, Elele, Rivers State, Nigeria. A cross-sectional case-control study design was conducted using 115 participants including 69 patients with T2DM and 46 seemingly healthy non-diabetic controls. All individuals had venous blood samples obtained after an overnight fast. The activities of serum catalase (CAT), superoxide dismutase (SOD) and glutathione peroxidase (GPx) were measured by standard spectrophotometric procedures (Misra and Fridovich for SOD; Sinha for CAT). Data were analysed using the Statistical Package for the Social Sciences (SPSS), version 23.0, and expressed as mean $\pm$ standard deviation. Differences between the diabetic and control groups were assessed using Student's t-test, with statistical significance set at $p < 0.05$. The results showed significantly lower CAT activity in patients with T2DM (65.48 ± 6.70 U/L) compared with controls (84.92 ± 9.96 U/L) ($p < 0.001$). Similarly, SOD activity was significantly reduced in the diabetic group (39.89 ± 2.60 U/L) compared with the control group (63.95 ± 6.22 U/L) ($p < 0.001$). GPx activity was also significantly lower among patients with T2DM (21.33 ± 16.70 U/L) than among controls (26.37 ± 20.02 U/L) ($p < 0.001$). These findings demonstrate a significant reduction in key antioxidant enzyme activities among patients with T2DM, suggesting compromised endogenous antioxidant defense and increased susceptibility to oxidative stress. Assessment of antioxidant enzymes may therefore provide useful biochemical information for understanding oxidative imbalance in T2DM and may complement conventional clinical parameters in the evaluation of disease-related metabolic disturbances. Key words: antioxidant status, type 2 diabetes, madonna university teaching hospital.	Article History Received: 16-07-2026 Accepted: 15-09-2026 Published: 06-10-2026
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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic condition with persistent hyperglycemia, caused by a combination of insulin resistance and the progressive failure of pancreatic β -cell activity. It is one of the world's biggest public health issues and associated with significant morbidity and mortality due to microvascular and macrovascular consequences. The rising prevalence of T2DM is a major issue particularly in developing nations like Nigeria as modifications in lifestyle, urbanisation, dietary habits and other socio-

economic factors continue to add to the growing burden of the illness [1].

Chronic hyperglycemia in T2DM causes a sequence of metabolic and cellular abnormalities that lead to overproduction of reactive oxygen species (ROS) and reactive nitrogen species (RNS). Several metabolic mechanisms have been implicated in hyperglycemia-induced oxidative stress, including enhanced glucose autooxidation, activation of the polyol system, production of advanced glycation end products (AGEs), activation of protein kinase C, and mitochondrial dysfunction [2].

These mechanisms contribute to an elevated cellular oxidant burden, which might overwhelm endogenous antioxidant defenses and result in oxidative stress.

Oxidative stress refers to a disturbance in the equilibrium between the generation of reactive species and the capacity of biological systems to neutralize these reactive molecules or repair the consequent cellular damage [3]. In T2DM, oxidative stress is considered an important factor to both the development and progression of metabolic abnormalities. Excessive ROS can interfere with insulin signaling, cause insulin resistance, impair pancreatic β -cell function, and accelerate inflammatory processes [4]. Since pancreatic β -cells have very weak antioxidant capability, they may be particularly vulnerable to oxidative injury. Consequently, oxidative stress may lead to increased β -cell malfunction and further loss of glycemic control, generating a pathological loop that maintains diabetes-related metabolic abnormalities [5]

Both ROS and RNS contribute to oxidative damage in T2DM. For example, superoxide anion can react with nitric oxide to generate peroxynitrite, a highly reactive oxidant capable of destroying lipids, proteins, and nucleic acids [6]. Increased oxidative activity may therefore result in lipid peroxidation, protein oxidation, and DNA damage. Biochemical compounds such as malondialdehyde (MDA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and protein carbonyls have been explored as indicators of oxidative damage and have been observed to increase in patients with diabetes [7]. These alterations are consistent with a close association of persistent hyperglycemia with the increased oxidative burden.

The body has an integrated antioxidant defence system of enzymatic and non-enzymatic antioxidants to prevent the damaging effects of reactive species. Antioxidants are chemicals that prevent or limit oxidative damage, by scavenging reactive species, blocking their synthesis or mending oxidatively damaged cellular components. The enzymatic antioxidant system is composed of superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx). Important non-enzymatic antioxidants are glutathione, vitamin C, vitamin E and other dietary and endogenous antioxidant substances [8].

SOD is one of the earliest enzymatic defence mechanisms against superoxide radicals, catalysing the conversion of superoxide radicals to hydrogen peroxide and molecular oxygen. Hydrogen peroxide formed from this reaction is then detoxified mainly by CAT and GPx. Catalase catalyses the conversion of hydrogen peroxide to water and molecular oxygen, particularly at relatively high levels of hydrogen peroxide, while GPx lowers hydrogen peroxide and lipid hydroperoxides with reduced glutathione as the electron donor. Hence, the activities of these enzymes are coordinated to maintain

cellular redox equilibrium and prevent oxidative damage.

In T2DM, the prolonged hyperglycaemia and increased generation of reactive species may overwhelm the antioxidant defences, leading to changes in the activity or concentrations of endogenous antioxidants. Studies have reported reductions in antioxidant enzymes and non-enzymatic antioxidants in subjects with diabetes although the magnitude and direction of these changes may vary according to glycaemic control, duration of disease, nutritional status, age, medication use and presence of diabetic complications [9]. To summarise, the simultaneous presence of enhanced oxidant production and inadequate antioxidant defence may promote the development of diabetes-associated tissue injury.

Therefore, the association between oxidative stress and T2DM is thought to be bidirectional. Persistent hyperglycemia causes oxidative stress through numerous metabolic pathways, whereas oxidative stress can further decrease insulin signaling and β -cell function, ultimately contributing to deteriorating metabolic control. This interaction may play a crucial role in the aetiology of diabetic sequelae, including nephropathy, retinopathy, neuropathy, and cardiovascular disease [10]. Evaluation of antioxidant defenses may subsequently provide valuable information regarding the oxidative component of diabetes and its possible impact to disease development. Of the antioxidant enzymes, SOD, CAT and GPx are of particular importance as they function sequentially in the detoxification of reactive oxygen species. Thus, evaluation of their activities might give an insight into the ability of the endogenous antioxidant system to respond to the increased oxidative burden associated with T2DM. The lower activity of these enzymes may suggest the lack of antioxidant defence and greater susceptibility of tissues to oxidative damage. However, various factors such as dietary condition, age, medications, length of the disease, glycaemic management and the existence of comorbidities can alter antioxidant enzyme activity. Therefore, their interpretation should be in the broader clinical and biochemical context of the individual [11]. The increasing prevalence of T2DM in Nigeria continues to be a major public health concern with the attendant need to investigate the biochemical abnormalities. Standard clinical examination usually includes blood glucose, glycated haemoglobin, lipid profile and other metabolic markers but measurement of antioxidant defence may add information about oxidative imbalance in the patients studied. Thus, determination of the activities of SOD, CAT and GPx among individuals with T2DM may lead to a better understanding of the association between diabetes and oxidative stress in the local population [12].

There is growing evidence for the association between oxidative stress and T2DM. However, different populations and clinical contexts have shown variance in

the activity of antioxidant enzymes. These differences could be due to variance in genetic background, eating patterns, environmental exposures, disease duration, treatment and glycaemic state. Therefore, demographic specific investigations of antioxidant defence among patients with T2DM are needed.

In view of this, the present study was done to test selected antioxidant enzyme activities- SOD, CAT and GPx in patients with type 2 diabetes mellitus attending Madonna University Teaching Hospital, Elele, Rivers State, Nigeria. The findings may add to the understanding of oxidative imbalance among T2DM patients and provide locally relevant information that may help to promote further study on biochemical markers linked with diabetes and its consequences.

MATERIAL AND METHOD

Study Area

This study will be conducted at Madonna University Teaching Hospital, Elele, located in Ikwerre Local Government Area of Rivers State, Nigeria. The hospital serves as a major healthcare facility providing medical services to both urban and rural populations within the region.

Research design

This study adopted a cross-sectional case-control design to evaluate antioxidant status in type 2 diabetic patients and compared findings with non-diabetic individuals serving as controls.

INCLUSION CRITERIA

1. Confirmed diagnosed cases of type 2 diabetes
2. Non-diabetic patient as control

EXCLUSION CRITERIA

1. Types 1 diabetic patients

Sample size calculation

The sample size for this study will be determined using the formula:

$$n = \frac{z^2 P(1 - P)}{d^2}$$

Where:

n = required sample size

Z = standard normal deviation at 95% confidence interval (1.96)

P = prevalence of type 2 diabetes is 4.07% = 0.0407 (Madonna University teaching hospital elele, rivers states, Nigeria)

d = margin of error (0.05)

Substituting into the formula:

$$n = \frac{1.96^2 \times 0.0407(1 - 0.0407)}{0.05^2}$$

$$n = 60$$

Therefore, the calculated sample size is 60 diabetic patients. Using a control-to-case ratio of 2:2,

approximately 60 non-diabetic individuals will be recruited as controls, giving a total sample size of 120 participants.

Ethical Consideration

Ethical approval was obtained from the Ethics Committee of Madonna University Teaching Hospital. Informed consent was obtained from all participants before sample collection. Confidentiality of participants' information was strictly maintained throughout the study.

Sample Collection

A total of 5–10 mL of venous blood were collected aseptically from each participant after an overnight fast of at least 8 hours. The blood sample was divided into EDTA and plain tubes.

The EDTA sample was used for plasma analysis, while the plain tube was allowed to clot and centrifuged to obtain serum. Samples were processed within 30 minutes of collection and stored at -20°C until analysis.

Laboratory assay

The following antioxidant parameters were analyzed using standard laboratory methods:

Superoxide dismutase (SOD)

Catalase (CAT)

Glutathione peroxidase (GPX)

aEstimation of catalase activity: It was done spectrophotometrically and was based on a principle in which Catalase reacts with hydrogen peroxide to produce water and oxygen molecules. The decrease in absorbance of hydrogen peroxide is then measured spectrophotometrically at 240 nm and is proportional to catalase activity.

b Estimation of Glutathione Peroxidase (GPx) Activity: It was done spectrophotometrically and was based on a principle in which Glutathione peroxidase (GPx) catalyzes the reduction of hydrogen peroxide (H₂O₂) to water by oxidizing reduced glutathione (GSH) to oxidized glutathione (GSSG). The oxidized glutathione is subsequently reduced back to GSH by glutathione reductase in the presence of nicotinamide adenine dinucleotide phosphate (NADPH). During this reaction, NADPH is oxidized to NADP⁺, resulting in a decrease in absorbance at 340 nm. The rate of decrease in absorbance is directly proportional to the GPx activity in the sample.

c Estimation of superoxide dismutase (SOD) activity: It was done by Auto-oxidation method and was based on a principle in which SOD inhibit the autooxidation of epinephrine, superoxide (O₂⁻) radical, which is produced by the xanthine oxidase reaction, at an alkaline Ph of 10.2, causing the oxidation of epinephrine to adrenochrome which later give rise to (O₂⁻) which increases with increased Ph and epinephrine concentration.

Statistical Analysis

Data obtained from this study was analyzed using Statistical Package for Social Sciences (SPSS) version 25.0. Results were expressed as mean $\pm$ standard deviation. Comparison between groups was done using

Student's t-test, and a p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Standard Levels of Antioxidant Enzymes between Test Subjects and Control Subjects

Antioxidant Enzyme	Test Subjects Mean $\pm$ SD	Control Subjects Mean $\pm$ SD	t-value	df	p-value	Remark
Catalase (CAT) (U/L)	65.48 $\pm$ 6.70	84.92 $\pm$ 9.96	-14.561	59	0.000	S
Superoxide Dismutase (SOD) (U/L)	39.89 $\pm$ 2.60	63.95 $\pm$ 6.22	-30.207	59	0.000	S
Glutathione Peroxidase (GPx) (U/L)	21.33 $\pm$ 16.70	26.37 $\pm$ 20.02	-23.833	59	0.000	S

Data are presented in Mean $\pm$ Standard Deviation.

Table 4.1 shows the levels of antioxidant enzymes between test subjects and control subjects. The results revealed statistically significant differences in the activities of Catalase (CAT), Superoxide Dismutase (SOD), and Glutathione Peroxidase (GPx) between the two groups.

For Catalase (CAT), the test subjects recorded a significantly lower mean value of 65.48 $\pm$ 6.70 U/L compared to 84.92 $\pm$ 9.96 U/L in the control subjects, with a highly significant p-value of 0.000. Similarly, Superoxide Dismutase (SOD) activity was significantly reduced in the test subjects, with a mean value of 39.89 $\pm$ 2.60 U/L compared to 63.95 $\pm$ 6.22 U/L in the control subjects.

Glutathione Peroxidase (GPx) activity was also significantly lower in the test subjects, with a mean value of 21.33 $\pm$ 16.70 U/L compared to 26.37 $\pm$ 20.02 U/L in the control subjects. The significant reduction observed in all antioxidant enzymes indicates increased oxidative stress and reduced antioxidant defense mechanisms among the test subjects. The results showed a statistically significant reduction in the levels of Catalase (CAT), Superoxide Dismutase (SOD), and Glutathione Peroxidase (GPx) in the test subjects compared with the control subjects ($p < 0.05$). This indicates increased oxidative stress and reduced antioxidant defense mechanisms among the test subjects.

DISCUSSION

The present study showed considerable reduction in catalase (CAT) activity in test subjects as compared to healthy control controls. As indicated in Table 4.1, the mean CAT activity was significantly lower in the test group (65.48 $\pm$ 6.70 U/L) than in the control group (84.92 $\pm$ 9.96 U/L) and the difference was statistically significant ($p < 0.001$). Catalase is a key enzyme antioxidant that protects cells from oxidative damage by catalysing the breakdown of hydrogen peroxide (H₂O₂) to water and molecular oxygen. This process is necessary to limit the accumulation of hydrogen peroxide and to prevent its conversion to more reactive oxidant species.

The lower CAT activity in the test individuals could represent a weakened antioxidant defence mechanism linked to increased oxidative burden. Chronic formation of ROS (reactive oxygen species) can impose a substantial burden on endogenous antioxidant systems, leading to a decrease in antioxidant capacity. However, reduced CAT activity should not be interpreted as direct evidence of enzyme consumption or depletion, since antioxidant enzyme activity may also be modulated by altered enzyme synthesis, cofactor availability, post-translational modification, and nutritional status and disease-related metabolic changes [13].

The observation of decreased CAT activity is consistent with prior reports of changes in the antioxidant enzymes under oxidative stress-associated situations. According to the report [14], chronic oxidative and inflammatory processes may be related with changes in the endogenous antioxidant defence. The relevance of enzyme antioxidants such as catalase in shielding biological systems from ROS-mediated damage was emphasized [15]. The decrease in CAT activity found in the present study may therefore be an indication of impaired capacity to effectively remove hydrogen peroxide and other oxidant species.

Decreased CAT activity might lead to a prolonged presence of hydrogen peroxide in cells. In the presence of transition metals, accumulated hydrogen peroxide can participate in processes that generate extremely reactive hydroxyl radicals, which can trigger lipid peroxidation and damage proteins, membranes and nucleic acids. Thus, the decreased CAT activity found may be one of the contributing factors for creating an environment conducive for oxidative cellular harm. However, since the present study did not explicitly test hydrogen peroxide or particular markers of oxidative damage, this interpretation should be taken as a plausible mechanism rather than direct evidence [16].

A similar pattern was also seen for superoxide dismutase (SOD). The mean SOD activity was significantly lower in test subjects (39.89 $\pm$ 2.60 U/L) compared to control subjects (63.95 $\pm$ 6.22 U/L) with a

statistically significant difference ($p < 0.001$). Superoxide dismutase (SOD) is a vital initial line enzymatic antioxidant defence that catalyses the dismutation of superoxide anion ($O_2\bullet$) to hydrogen peroxide and molecular oxygen. The generated hydrogen peroxide is detoxified by enzymes such as catalase and glutathione peroxidase [17]. The drastically lower SOD activity in the test group suggested a diminished potential to scavenge superoxide radicals. This may contribute to buildup of superoxide and other downstream reactive species. An increased oxidative burden may lead to a decrease in antioxidant activity, but other factors may affect SOD activity, including altered enzyme expression, dietary inadequacies, metabolic irregularities and disease-related alterations. The present data is therefore best regarded as evidence of impaired antioxidant defence rather than clear proof of enzyme depletion [18].

The conclusion is in agreement with the observations of [19] who reported abnormalities in the endogenous antioxidant systems in situations associated with increased oxidative stress. Same here. It covered the impact of excess ROS production in oxidative damage and the role of antioxidant enzymes in reducing such damage. Thus, the loss in SOD activity demonstrated in the present study may reflect an insufficient enzymatic protection from superoxide-mediated oxidative events [20].

Reduced SOD activity may also promote the survival of superoxide radicals, which can combine rapidly with nitric oxide (NO) to generate peroxynitrite ($ONOO^-$), a highly reactive nitrogen species. Peroxynitrite can oxidise proteins, lipids and nucleic acids and may interfere with normal cellular signalling and metabolic activities. Therefore, the significantly decreased SOD activity in the test individuals provides further evidence for a changed redox balance and a possibly increased sensitivity to oxidative injury [21].

The present investigation also indicated a significant decrease in the activity of glutathione peroxidase (GPx) in the subjects of the study. The mean GPx activity was 21.33 ± 16.70 U/L in the test group as compared to 26.37 ± 20.02 U/L in the control group ($p < 0.001$). Glutathione peroxidase is an important component of the cellular antioxidant defence system. It catalyses the reduction of hydrogen peroxide and lipid hydroperoxides to less reactive products by utilising reduced glutathione (GSH) as an electron donor. This activity of GPx is important for preservation of cellular membranes and other macromolecules from oxidative damage.

Reduced GPx activity in the test individuals could imply compromised glutathione-dependent antioxidant defence. However, the lower GPx activity does not in itself suggest depletion of glutathione stores as the GSH levels were not explicitly assayed in this

current investigation. Altered GPx activity may be due to numerous causes including modified enzyme expression, selenium availability, nutritional condition, metabolic changes in disease and increased oxidative burden.

This result is in agreement with [22] who stressed the relevance of antioxidant enzymes and glutathione-dependent systems in maintaining the cellular redox equilibrium. It has pointed out oxidative stress as a significant factor in the deterioration of antioxidant defences and cellular damage. Thus, the decreased GPx activity found in this study may be a marker of changed antioxidant environment and loss of the ability to effectively remove hydrogen peroxide and lipid hydroperoxides [23].

Insufficient activation of GPx may enhance the susceptibility of cellular membranes to lipid peroxidation. Oxidative alteration of membrane lipids can change membrane fluidity and permeability and may subsequently impact membrane bound enzymes, receptors, ion transport, and cellular signalling.[24] Thus the reduction in GPx activity shown here, in addition to the reduction in CAT and SOD, indicates that several elements of the enzymatic antioxidant defence system were modified in the test participants.

To summarise, the results show considerable decrease in CAT, SOD and GPx activities in the test patients in comparison to healthy controls. The concomitant decrease in these three important antioxidant enzymes shows an impairment in the endogenous antioxidant defence system and may indicate an increased vulnerability to oxidative stress. SOD plays an important role as a first-line of defence against superoxide radicals, whereas CAT and GPx are involved in the subsequent detoxification of hydrogen peroxide and related oxidant products. Modification of these complementing enzymatic systems may consequently impair the overall ability of the body to maintain redox homeostasis [25].

The results are generally in line with the known link between oxidative stress and metabolic diseases. Inadequate antioxidant protection along with increased ROS production can lead to oxidative modification of lipids, proteins, and nucleic acids and may contribute to inflammation, endothelial dysfunction, impaired cellular signalling and tissue injury. In type 2 diabetes mellitus, chronic hyperglycaemia and associated metabolic abnormalities can promote ROS generation via several pathways including mitochondrial dysfunction, advanced glycation end-product formation, the polyol pathway and activation of protein kinase C. Therefore, the decrease in CAT, SOD and GPx activities found in this investigation is in agreement with a changed oxidative-antioxidant balance in the diabetes patients.

However, the cross-sectional design of the present investigation does not allow for the

establishment of a causal association between oxidative stress and the illness status from the observed decreases in antioxidant enzyme activity. In addition, the study did not directly measure ROS production or oxidative damage markers such as malondialdehyde, protein carbonyls or 8-hydroxy-2'-deoxyguanosine. So, the results should be taken as indicative of a changed antioxidant defence, rather than as direct evidence of increased oxidative damage [26].

The considerable decrease in CAT, SOD and GPx activity in the test groups suggests an impaired endogenous antioxidant defence. These results support the importance of oxidative imbalance in the metabolic environment associated with the disease condition and provide biochemical evidence that may help to understand the pathophysiological processes involved. Further studies utilising direct markers of oxidative damage, total antioxidant capacity, reduced glutathione, glycaemic indices, disease duration, and clinical complications would provide a more thorough assessment of the relationship between oxidative stress and antioxidant defence.

CONCLUSION

The present study evaluated the activities of three important antioxidant enzymes—catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GPx)—among the test subjects and healthy control subjects. The findings demonstrated significantly lower activities of CAT, SOD, and GPx in the test group compared with the control group, with all observed differences reaching statistical significance ($p < 0.001$).

The reduced activities of these antioxidant enzymes suggest an alteration of endogenous antioxidant defense and an imbalance in redox homeostasis among the test subjects. Since CAT, SOD, and GPx constitute complementary components of the enzymatic antioxidant system, their simultaneous reduction may indicate reduced capacity to effectively neutralize reactive oxygen species and limit oxidative injury.

The findings therefore support the involvement of oxidative stress and impaired antioxidant defense in the metabolic disturbances associated with the disease condition. However, because this was a cross-sectional study and direct markers of oxidative damage were not measured, the findings should not be interpreted as establishing a causal relationship between reduced antioxidant enzyme activities and disease progression.

Assessment of antioxidant enzyme activities may provide useful biochemical information about the antioxidant status of individuals with the disease and may complement conventional clinical and biochemical assessments. Further research incorporating additional oxidative stress biomarkers, antioxidant parameters, glycaemic indices, disease duration, treatment status, and clinical complications is recommended to provide a more

comprehensive understanding of oxidative imbalance in affected individuals.

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