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Evaluation of Plasma Malondialdehyde as a Predictive Biomarker for Oxidative Stress in Patients with Type 2 Diabetes Mellitus: A Case-Control Study.

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Abstract

Background:
Type 2 diabetes mellitus is frequently accompanied by oxidative stress driven by sustained hyperglycemia. Malondialdehyde (MDA), an end-product of lipid peroxidation, can serve as a surrogate marker of systemic oxidative damage.

Objectives:
To estimate plasma MDA and HbA1c levels in adults with type 2 diabetes mellitus and to compare these values with those of healthy controls, and to examine the association between MDA and glycemic status.

Methods:
A hospital-based case-control study was conducted at Government General Hospital, Suryapet, from July 2024 to September 2024. Sixty adults with clinically diagnosed type 2 diabetes mellitus (duration 2-5 years; age 30-60 years) and sixty age- and sex-matched apparently healthy controls were enrolled. Fasting venous blood was collected in EDTA vacutainers. Plasma MDA was quantified using a sandwich ELISA method, and HbA1c was measured by an enzymatic method on an automated analyzer. Group comparisons, Pearson correlation, and ROC analysis were performed.

Results:
Mean HbA1c was significantly higher in cases than controls (8.59 ± 1.72% vs 5.45 ± 0.29%; $p<0.00001$). Plasma MDA was also markedly elevated in cases compared with controls (12.57 ± 2.61 nmol/mL vs 5.92 ± 0.67 nmol/mL; $p<0.00001$). Among cases, plasma MDA showed a strong positive correlation with HbA1c ($r=0.9155$; $p<0.00001$). ROC analysis demonstrated excellent discrimination between cases and controls, with an area under the curve of 0.989. The optimal MDA cutoff of 8.3 nmol/mL showed 98.3% sensitivity and 100% specificity.

Conclusion:
Plasma MDA demonstrated a strong association with glycemic status and excellent diagnostic performance for identifying oxidative stress among adults with type 2 diabetes mellitus. Routine MDA assessment could complement glycemic monitoring for early risk stratification of oxidative complications.

Recommendations:
Optimise glycaemic control and incorporate periodic plasma MDA assessment alongside HbA1c in T2DM follow-up.

Keywords: *Type 2 diabetes mellitus; oxidative stress; malondialdehyde; lipid peroxidation; Glycated hemoglobin; Receiver operating characteristic curve.*
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Introduction
Type 2 diabetes mellitus (T2DM) is a major public health challenge, with a steadily rising prevalence across low- and middle-income countries and substantial downstream morbidity. Beyond hyperglycemia, the clinical burden of T2DM is largely driven by progressive vascular injury that culminates in microvascular and macrovascular complications. Contemporary epidemiologic projections

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emphasize that this expansion will continue over the coming decades, reinforcing the need for biomarkers that capture early pathobiology rather than late organ damage [1].

Oxidative stress is a central mechanistic link between chronic hyperglycemia and tissue injury. Sustained elevations in glucose accelerate reactive oxygen species (ROS) generation through several interrelated pathways, including mitochondrial superoxide overproduction, glucose auto-oxidation, and non-enzymatic glycation reactions. These processes amplify redox imbalance, impair endogenous antioxidant defences, and promote endothelial dysfunction and inflammation [2,3]. Conceptual frameworks also support a close interplay between oxidative stress and inflammation in T2DM progression [4], and contemporary reviews describe ROS as active drivers of insulin resistance and complication pathways rather than simple metabolic by-products [5,6].

Among the measurable outputs of oxidative injury, lipid peroxidation has particular relevance because polyunsaturated fatty acids in cell membranes are highly vulnerable to ROS attack. The peroxidation cascade generates relatively stable aldehydes, of which malondialdehyde (MDA) is widely used as an index of systemic oxidative damage. MDA formation reflects ongoing membrane lipid injury and can also participate in downstream signaling and protein adduct formation, making it a biologically meaningful marker rather than a passive footprint [7].

Clinical studies consistently report higher circulating or cellular MDA in individuals with T2DM compared with non-diabetic controls, and higher values among those with poorer glycemic control or cardiovascular complications [8,9]. Case-control observations have shown significant associations between MDA and HbA1c, supporting a graded relationship between chronic hyperglycemia and lipid peroxidation [10]. In addition, disease duration has been linked to higher MDA independent of several metabolic covariates, suggesting that cumulative glycemic exposure contributes to oxidative burden over time [11]. Work comparing HbA1c with oxidative markers in the assessment of chronic vascular risk further underscores the clinical value of oxidative stress profiling alongside conventional glycemic indices [12].

Despite this evidence, routine clinical monitoring of oxidative stress remains uncommon in many settings, partly due to variability in assays and uncertainty about clinically useful thresholds. Establishing the discriminative performance of plasma MDA in a defined hospital cohort can help clarify its translational relevance. Therefore, this study aimed to estimate plasma MDA and HbA1c levels in adults with T2DM and to compare these measures with healthy controls, and to evaluate the correlation between MDA and HbA1c as an indicator of oxidative stress associated with chronic glycemia.

Materials and Methods

Study design and setting

A hospital-based case-control study was conducted in the Department of Biochemistry in collaboration with the Department of General Medicine at Government General Hospital, Suryapet, Telangana, India, from July 2024 to September 2024. Government General Hospital, Suryapet, is a large public teaching hospital attached to Government Medical College, Suryapet, and functions under the Director of Medical Education, Telangana. It provides outpatient and inpatient clinical services to patients from Suryapet and surrounding areas, with broad medical, surgical, obstetric, emergency, diagnostic, and laboratory support services. Official district sources describe the hospital as an upgraded district-headquarters-level government hospital attached to the medical college, intended to provide comprehensive and quality healthcare services to the public.

Participants and sampling

A total of 120 participants were enrolled in the study using a purposive sampling technique. The study cohort comprised 60 adults with clinically diagnosed type 2 diabetes mellitus (T2DM), who were categorized as cases, and 60 apparently healthy individuals without diabetes, who served as controls. Eligible cases were adults aged 30-60 years attending the Medicine outpatient department with a disease duration of 2-5 years and willingness to provide written informed consent. Controls were age- and sex-matched individuals without known diabetes and without acute illness at recruitment; glycemic status was screened using HbA1c, and values <6.5% were considered non-diabetic in keeping with widely used diagnostic recommendations [13,14].

Eligibility criteria

Exclusion criteria were applied to reduce confounding by conditions or therapies known to influence oxidative status or HbA1c interpretation. Individuals with type 1 diabetes, gestational diabetes, hypertension, thyroid disorders, iron overload disorders (hemochromatosis, thalassemia, hemosiderosis), chronic kidney disease, chronic liver disease, malignancy, chronic infections or inflammatory disorders, pregnancy or lactation, and critically ill patients

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requiring intensive care were excluded. Participants receiving iron therapy, diuretics, antioxidant supplements, or systemic steroids were also excluded. Current smokers and alcohol users were excluded.

Specimen collection and laboratory measurements

After an overnight fast, venous blood was collected into EDTA vacutainers. Samples were processed promptly; plasma was separated by centrifugation and stored as per kit instructions until analysis. Plasma MDA was quantified using a commercially available sandwich ELISA kit, following the manufacturer's protocol, with calibrators and internal controls run in each batch. HbA1c was measured by an enzymatic method on a Randox RX Modena automated analyzer with routine quality control procedures.

Bias

Potential sources of bias were addressed at different stages of the study. Selection bias was reduced by enrolling clinically diagnosed type 2 diabetes mellitus cases and apparently healthy controls from the same hospital setting, with age- and sex-matching as far as possible. Misclassification bias was minimized by applying predefined inclusion and exclusion criteria and by screening controls for non-diabetic status using HbA1c. Conditions and exposures that could influence oxidative stress or HbA1c interpretation, including chronic kidney disease, chronic liver disease, inflammatory disorders, malignancy, pregnancy, smoking, alcohol use, antioxidant supplementation, steroid therapy, and iron therapy, were excluded. Measurement bias was limited by using standardized venous blood collection, uniform sample processing, ELISA-based estimation of plasma malondialdehyde, and enzymatic HbA1c measurement on an automated analyzer with routine internal quality control. Statistical bias was reduced by applying predefined analytical methods, including the independent samples t-test, Pearson correlation, and receiver operating characteristic analysis. However, residual confounding from unmeasured dietary, lifestyle, and metabolic factors could not be completely eliminated.

Statistical analysis

Continuous variables were summarised as mean ± standard deviation. Differences between cases and controls were assessed using the independent samples t-test. Pearson correlation coefficients were computed to evaluate the relationship between HbA1c and MDA within cases and within controls. Diagnostic performance of plasma MDA for discriminating diabetic cases from controls was assessed using receiver operating characteristic (ROC) curve analysis, using HbA1c (≥6.5%) as the reference standard [13,14]. Sensitivity and specificity were calculated across MDA thresholds, the area under the ROC curve (AUC) quantified overall discrimination, and the optimal cutoff was selected using the Youden index. A two-sided p-value <0.05 was considered statistically significant.

Ethical considerations

Written informed consent was obtained from all participants prior to enrolment. Ethical approval was obtained from the Institutional Ethics Committee, Government Medical College, Suryapet. All data were anonymised during analysis and reporting to ensure confidentiality.

Results

Sex-stratified descriptive statistics for age, HbA1c, and plasma MDA are presented in Table 1.

Table 1. Demographic characteristics of cases and controls

Parameter	Sex	Cases (Mean ± SD)	Controls (Mean ± SD)	Unit
Age	Female	50.66 ± 10.97	41.23 ± 9.10	years
Age	Male	48.86 ± 13.96	42.13 ± 5.52	years
HbA1c	Female	8.59 ± 1.72	5.45 ± 0.29	%
HbA1c	Male	8.77 ± 1.89	5.46 ± 0.45	%
MDA	Female	12.57 ± 2.61	5.92 ± 0.67	nmol/mL
MDA	Male	13.34 ± 2.36	6.14 ± 0.52	nmol/mL

Between-group comparisons are summarised in Table 2. Mean HbA1c and plasma MDA were significantly higher among cases than controls ($p<0.00001$ for both).

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Table 2. Comparison of HbA1c and plasma MDA between cases and controls

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	p value
HbA1c (%)	8.59 ± 1.72	5.45 ± 0.29	<0.00001
MDA (nmol/mL)	12.57 ± 2.61	5.92 ± 0.67	<0.00001

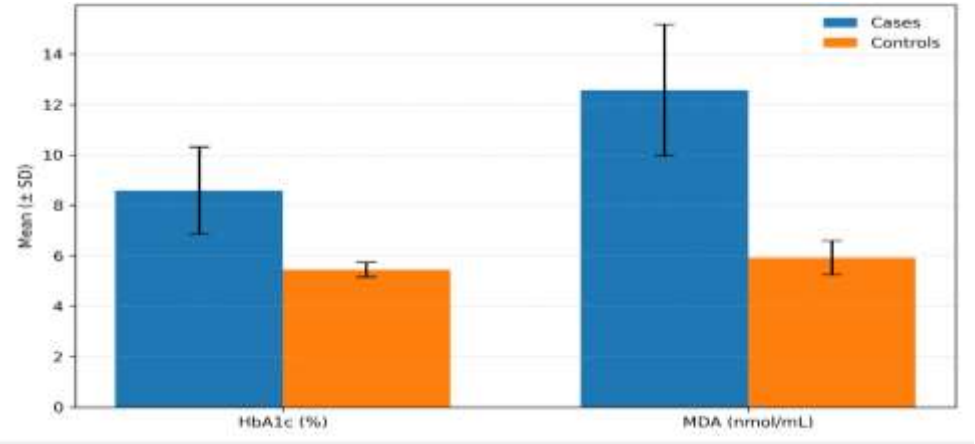

Figure 1: Comparison of HbA1c and plasma MDA between cases and controls

Pearson correlation analysis between HbA1c and plasma MDA is shown in Table 3.

Table 3. Pearson correlation between HbA1c and MDA among cases and controls

Group	Parameter 1	Parameter 2	r value	p value
Cases	HbA1c	MDA	0.9155	<0.00001
Controls	HbA1c	MDA	0.4529	<0.00001

Diagnostic performance of plasma MDA for discriminating diabetic cases from controls by ROC analysis is summarised in Table 4.

Table 4. ROC analysis of plasma MDA for discriminating diabetic cases from controls

Measure	Value	Cutoff (nmol/mL)	Sensitivity (%)	Specificity (%)
AUC	0.989	8.3	98.3	100

Overall, plasma MDA demonstrated a strong positive correlation with HbA1c in cases (r=0.9155; p<0.00001) and excellent discriminative accuracy (AUC=0.989).

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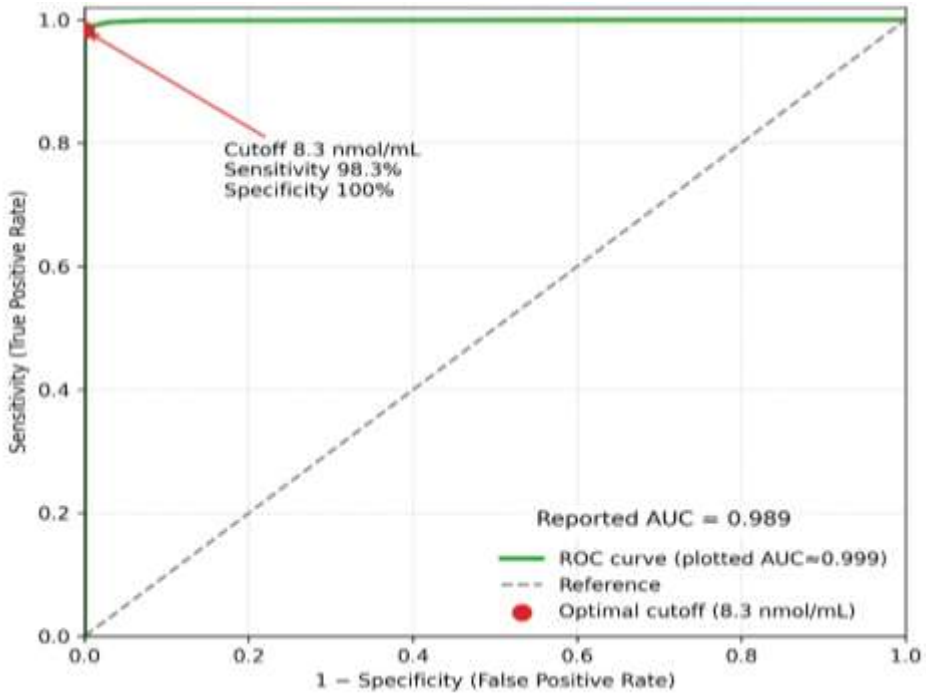

Figure 2: ROC analysis of plasma MDA for discriminating diabetic cases from controls

Discussion

The present case-control study demonstrated substantially higher plasma MDA concentrations in adults with T2DM compared with non-diabetic controls, indicating enhanced lipid peroxidation and systemic oxidative stress in the diabetic state. This observation is biologically plausible because chronic hyperglycemia increases reactive oxygen species generation through mitochondrial and non-mitochondrial routes, and sustained redox imbalance contributes to endothelial dysfunction and metabolic dysregulation [2,3]. Reviews of oxidative biology in diabetes further support that oxidative stress interacts with inflammatory signaling and accelerates complication pathways [4-6]. Lipid membranes, rich in polyunsaturated fatty acids, are particularly susceptible to oxidative attack, and MDA represents a relatively stable downstream aldehyde formed during lipid peroxidation [7].

The magnitude of MDA elevation observed in this cohort is consistent with prior reports showing increased lipid peroxidation products in T2DM, including studies in patients with microvascular and cardiovascular complications [8,9]. The correlation analysis provides direct statistical support for a graded relationship between chronic glycemic exposure and oxidative injury. Among cases, HbA1c showed a strong positive correlation with plasma MDA ($r=0.9155$; $p<0.00001$), indicating that patients with poorer long-term glycemic control had higher lipid peroxidation burden. This supports a dose-response pattern, where sustained hyperglycemia intensifies oxidative stress through increased reactive oxygen species generation and membrane lipid peroxidation. Earlier evidence also reported that higher HbA1c levels were associated with increased MDA and reduced antioxidant enzyme activity in patients with type 2 diabetes mellitus [10]. In the control group, HbA1c also showed a statistically significant but weaker correlation with MDA ($r=0.4529$; $p<0.00001$), suggesting that within the normoglycemic range, glycemia contributes only partly to oxidative variability. Therefore, non-glycemic factors such as dietary antioxidant intake, adiposity, physical activity, subclinical inflammation, and metabolic reserve could also influence MDA levels among healthy individuals. The much stronger correlation observed in cases emphasizes that hyperglycemia is a dominant driver of oxidative stress in type 2 diabetes mellitus.

A notable finding of this study is the excellent discriminative performance of plasma MDA, with an AUC

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close to 1.0 and an optimal cutoff offering high sensitivity and specificity. These data support the clinical utility of oxidative stress profiling as a complement to standard glycemic metrics, particularly when the objective is to identify early biochemical perturbations preceding overt complications. Prior work comparing HbA1c with oxidative markers in assessing chronic vascular risk emphasizes that integrating oxidative indices can refine risk assessment beyond glucose control alone [12]. Additionally, evidence that longer T2DM duration independently predicts higher MDA levels highlights the cumulative oxidative burden of prolonged disease and supports longitudinal monitoring approaches [11].

Methodologically, use of an ELISA-based approach for MDA quantification offers practical advantages for routine laboratories, particularly regarding specificity compared with thiobarbituric acid-based assays that can capture non-MDA chromogens [7]. From a clinical perspective, identifying individuals with high oxidative burden can guide intensified lifestyle counselling and optimization of cardiometabolic risk factors, while maintaining HbA1c as the primary target for glycemic management [13].

Generalizability

These findings are most applicable to adults aged 30-60 years with established T2DM of 2-5 years' duration receiving care in a secondary/tertiary hospital setting. The performance estimates for the proposed MDA cutoff reflect the study's case-control spectrum and local pre-analytical conditions. Translation to community screening, older patients, newly diagnosed diabetes, or individuals with significant comorbidities requires validation in broader and more heterogeneous populations using harmonized MDA assays and standardized sample handling.

Conclusion

This study shows that adults with type 2 diabetes mellitus have markedly higher plasma malondialdehyde levels than healthy controls, reflecting increased lipid peroxidation and oxidative stress. Plasma MDA correlated strongly with HbA1c, indicating that poorer long-term glycemic control is linked to greater oxidative injury. ROC analysis demonstrated excellent diagnostic accuracy, and the identified cutoff of 8.3 nmol/mL provided high sensitivity and specificity for distinguishing diabetic cases from controls. Incorporating plasma MDA alongside HbA1c can enhance risk stratification and support earlier preventive strategies focused on limiting oxidative-mediated complications. Serial measurements can help track response to intensified metabolic and lifestyle interventions. Larger prospective studies are needed to confirm thresholds across clinical settings.

Limitations

The study was single-centred and included 120 participants, limiting external validity. Age matching between cases and controls was not exact, and residual confounding by unmeasured lifestyle factors remains possible. The case-control design and short study period prevent temporal inference. Only MDA was assessed as an oxidative marker; antioxidant enzymes and other oxidative indices were not measured. Follow-up for diabetic complications was not performed.

Recommendations

Routine assessment of plasma MDA can be considered as an adjunct to HbA1c in tertiary care diabetes clinics to identify patients with high oxidative burden and to prioritize intensive risk-factor control. Laboratories should standardize pre-analytical handling, calibration, and reporting units to improve comparability across centres. Future research should include multicentric cohorts with larger sample sizes, wider age ranges, and newly diagnosed patients, and should evaluate additional oxidative and antioxidant markers alongside MDA. Longitudinal designs linking baseline MDA to microvascular and macrovascular outcomes will clarify prognostic value and refine clinically actionable cutoffs. Intervention trials should examine whether improving control lowers MDA.

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Abbreviations

T2DM-Type 2 diabetes mellitus;
DM-Diabetes mellitus;
ROS-Reactive oxygen species;
MDA-Malondialdehyde;
HbA1c-Glycated hemoglobin;
ELISA-Enzyme-linked immunosorbent assay;
ROC-Receiver operating characteristic;

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AUC-Area under the curve;
OPD-Outpatient department;
EDTA-Ethylenediaminetetraacetic acid.

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Conflict of interest
The authors declare no conflict of interest.

Author contributions
TPK -Concept and design of the study, results interpretation, review of literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript. **GNP**- Design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript, revision of the manuscript

Data availability
Data Available

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