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Oxidative Stress and Antioxidant Status in Gestational Diabetes Mellitus: A Comparative Study

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ABSTRACT:

Background: Gestational diabetes mellitus (GDM) is associated with increased oxidative stress and impaired antioxidant defence. This study compared oxidative stress and antioxidant status between women with GDM and healthy pregnant controls.

Materials and Methods: This hospital-based comparative study included 60 pregnant women, comprising 30 women with GDM and 30 healthy controls. Glycaemic parameters, lipid profiles, superoxide anion, superoxide dismutase (SOD), and ascorbic acid levels were evaluated and compared.

Results: Women with GDM had significantly higher fasting blood glucose (117.65 ± 7.43 vs 81.12 ± 8.15), HbA1c (6.88% vs 4.81%), and triglycerides (244.58 ± 17.95 vs 176.65 ± 13.21) compared with controls (all $p<0.001$). Superoxide anion levels were significantly elevated (189.58 ± 10.50 vs 141.38 ± 9.82; $p<0.001$), whereas median SOD (4.20 vs 5.71) and ascorbic acid (0.40 vs 0.69) were significantly reduced (both $p<0.001$). No significant correlations were observed between HbA1c and oxidative stress or antioxidant markers.

Conclusion: GDM was associated with increased oxidative stress, reduced antioxidant status, and significant metabolic abnormalities, suggesting an important role of oxidant–antioxidant imbalance in its pathophysiology.

INTRODUCTION

Gestational diabetes mellitus (GDM) is a common metabolic complication of pregnancy characterized by hyperglycaemia first detected during gestation that does not meet the diagnostic criteria for overt diabetes mellitus [1]. It develops primarily due to increased insulin resistance and inadequate compensatory insulin secretion from pancreatic β-cells. GDM is associated with adverse maternal and neonatal outcomes, including preeclampsia, fetal macrosomia, neonatal hypoglycaemia, operative delivery, and an increased long-term risk of type 2 diabetes mellitus. Besides disturbances in glucose metabolism, oxidative stress and impaired antioxidant defence mechanisms have emerged as important contributors to its pathophysiology [2]. Pregnancy is physiologically associated with increased metabolic activity, oxygen consumption, and placental mitochondrial function, resulting in enhanced production of reactive oxygen species (ROS). Under normal conditions, ROS participate in cellular signalling, placental development, and regulation of maternal–fetal interactions [3]. Their concentrations are maintained through enzymatic and non-enzymatic antioxidant defence systems. Oxidative stress develops when excessive ROS generation overwhelms antioxidant capacity, causing oxidative damage to lipids, proteins, and nucleic acids. Although moderate oxidative activity is a normal physiological adaptation during pregnancy, excessive oxidative stress has been implicated in pregnancy-related metabolic complications, particularly GDM [4]. In women with GDM, maternal hyperglycaemia promotes ROS generation through mitochondrial dysfunction, glucose auto-oxidation, activation of protein kinase C, and increased formation

of advanced glycation end products. These mechanisms may impair insulin signalling, contribute to pancreatic β-cell dysfunction, and aggravate insulin resistance. Increased oxidative stress may also affect placental function and vascular endothelial integrity, thereby contributing to adverse pregnancy outcomes. Consequently, oxidative imbalance represents an important mechanism linking maternal hyperglycaemia with metabolic and vascular dysfunction [2,5,6].The antioxidant defence system protects biological tissues against oxidative injury through enzymatic and non-enzymatic mechanisms. Superoxide dismutase (SOD) is a major enzymatic antioxidant that catalyses the conversion of superoxide radicals into hydrogen peroxide and molecular oxygen. Ascorbic acid (vitamin C), an important water-soluble non-enzymatic antioxidant, neutralizes free radicals and maintains cellular redox balance [7]. Alterations in SOD activity and ascorbic acid concentrations may indicate impaired antioxidant capacity among women with GDM. Therefore, simultaneous assessment of superoxide anion levels, SOD activity, and ascorbic acid concentrations may provide valuable information regarding oxidative imbalance associated with maternal hyperglycaemia [8]. Previous investigations have demonstrated increased oxidative stress and altered antioxidant enzyme activity in women with GDM compared with normoglycemic pregnant women. However, considerable variations have been reported in antioxidant concentrations and enzyme activities, potentially due to differences in gestational age, glycaemic control, nutritional status, maternal characteristics, and laboratory methods [9,10]. Although dietary and antioxidant interventions have been investigated, their clinical benefits remain uncertain [11,12]. Further comparative studies are therefore required to clarify the relationship between oxidative stress and antioxidant status in GDM.The present study aimed to evaluate and compare oxidative stress and antioxidant status among women with gestational diabetes mellitus and healthy pregnant controls by assessing superoxide anion levels, superoxide dismutase activity, and ascorbic acid concentrations.

MATERIALS AND METHODS

This hospital-based comparative case-control study was conducted over 24 months in the Department of Biochemistry, Faculty of Medicine and Health Sciences, SGT University, Gurugram, Haryana, India, in collaboration with the Department of Obstetrics and Gynaecology. The present analysis included 60 pregnant women aged 20–40 years, selected from the original study population of 120 participants. They were divided into two groups: Group I, comprising 30 healthy pregnant women, and Group II, comprising 30 women with gestational diabetes mellitus (GDM). GDM was diagnosed according to the applicable American Diabetes Association criteria.

Inclusion and Exclusion Criteria

Pregnant women aged 20–40 years with confirmed GDM or normal pregnancy who provided written informed consent were included. Women with liver or kidney disease, acute or chronic drug or alcohol intoxication, or those receiving hypolipidemic medications, hormone replacement therapy, or steroid treatment were excluded.

Sample Collection and Biochemical Analysis

Following 12–14 hours of overnight fasting, approximately 5 mL of venous blood was collected under aseptic precautions. Serum/plasma was separated using standard laboratory procedures and stored appropriately until analysis. Fasting plasma glucose, haemoglobin, total protein, and lipid profile parameters, including total cholesterol, triglycerides, HDL-C, and LDL-C, were assessed. Glucose and lipid parameters were measured using an ERBA XL 360 automated biochemical analyser.

Assessment of Oxidative Stress and Antioxidant Status

Oxidative stress and antioxidant status were evaluated by measuring superoxide anion, superoxide dismutase (SOD), and ascorbic acid. SOD was estimated using a commercially available sandwich ELISA kit (ELK Biotechnology, Catalogue No. ELK5615), with absorbance measured at 450 nm. Ascorbic acid was determined using a colorimetric assay based on ferric ion reduction, with ascorbate oxidase correction and absorbance measurement at 545–600 nm. Superoxide anion was assessed as an oxidative stress marker; the specific assay used requires confirmation from the original laboratory protocol.

Statistical Analysis

Data were analysed using IBM SPSS Statistics version 26. Continuous variables were expressed as mean ± standard deviation or median (interquartile range), as appropriate. The original study used one-way ANOVA with post-hoc comparisons or non-parametric alternatives across four groups. For the present two-group comparison, independent-samples t-tests or Mann–Whitney U tests were considered appropriate according to data distribution. A p-value less than 0.05 was considered statistically significant.

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Ethical Considerations

The study was approved by the Institutional Ethics Committee of SGT University, Gurugram. Written informed consent was obtained from all participants, and confidentiality was maintained throughout the study.

RESULTS

The present study included 60 pregnant women, comprising 30 healthy pregnant women and 30 women with gestational diabetes mellitus (GDM). Both groups were compared regarding anthropometric characteristics, biochemical parameters, glycaemic profile, lipid profile, oxidative stress, and antioxidant status. The mean body weight was significantly higher among women with GDM compared with healthy pregnant women (69.14 ± 6.49 vs 59.21 ± 6.21 kg; p<0.001). However, no statistically significant differences were observed in height, haemoglobin, or total protein levels between the two groups (Table 1 and Figure 1).

Women with GDM demonstrated significantly higher fasting blood glucose (117.65 ± 7.43 vs 81.12 ± 8.15), postprandial blood glucose (163.20 ± 11.03 vs 108.72 ± 10.63), and median HbA1c (6.88% vs 4.81%) compared with healthy pregnant women (all p<0.001) (Table 2).Women with GDM exhibited significantly higher total cholesterol (249.12 vs 213.01), triglycerides (244.58 ± 17.95 vs 176.65 ± 13.21), LDL-C (159.94 ± 13.24 vs 119.07 ± 11.23), and VLDL-C (48.92 ± 3.59 vs 35.32 ± 2.64). Conversely, HDL-C (41.13 ± 4.80 vs 55.18 ± 5.77) and the HDL/LDL ratio (0.26 ± 0.04 vs 0.47 ± 0.07) were significantly lower in the GDM group (all p<0.001) (Table 3).Mean superoxide anion levels were significantly elevated among women with GDM compared with healthy pregnant women (189.58 ± 10.50 vs 141.38 ± 9.82; p<0.001), indicating increased oxidative stress in the GDM group (Table 4).Women with GDM showed significantly reduced median superoxide dismutase (SOD) levels [4.20 (3.92–4.48) vs 5.71 (5.45–5.96)] and ascorbic acid levels [0.40 (0.34–0.46) vs 0.69 (0.65–0.74)] compared with healthy pregnant women (both p<0.001) (Table 5 and Figure 2).Among women with GDM, no statistically significant correlations were observed between HbA1c and superoxide anion (ϱ=−0.021, p=0.911), SOD (ϱ=0.073, p=0.700), or ascorbic acid (ϱ=0.149, p=0.433). Similarly, correlations between superoxide anion and SOD (ϱ=−0.079, p=0.680), superoxide anion and ascorbic acid (ϱ=−0.024, p=0.900), and SOD and ascorbic acid (ϱ=−0.119, p=0.531) were statistically insignificant (Table 6 and Figure 3).

Table 1. Comparison of Anthropometric and Baseline Biochemical Characteristics Between Healthy Pregnant Women and Women With GDM

Parameter	Healthy pregnancy (n=30)	GDM (n=30)	p-value	Statistical test
Height (cm)	155.08 ± 5.07	155.12 ± 4.67	1.00	Independent-samples t-test
Weight (kg)	59.21 ± 6.21	69.14 ± 6.49	<0.001	Independent-samples t-test
Haemoglobin	11.45 ± 0.65	11.25 ± 0.82	0.79	Independent-samples t-test
Total protein	6.80 (6.58–7.01)	6.80 (6.62–7.00)	1.00	Mann–Whitney U test

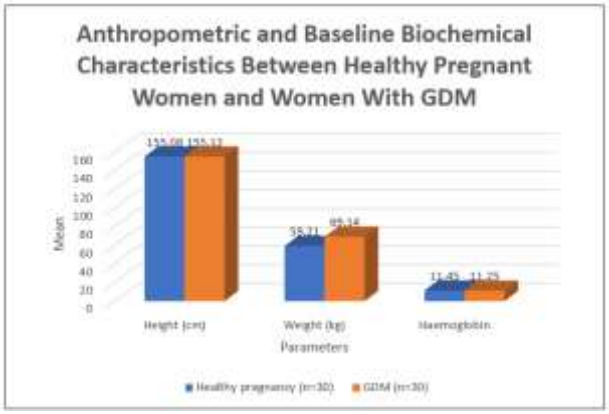


Figure 1. Comparison of Anthropometric and Baseline Biochemical Characteristics Between Healthy Pregnant Women and Women With GDM

Table 2. Comparison of Glycaemic Parameters Between Healthy Pregnant Women and Women With GDM

Parameter	Healthy pregnancy (n=30)	GDM (n=30)	p-value	Statistical test
Fasting blood glucose	81.12 ± 8.15	117.65 ± 7.43	<0.001	Independent-samples t-test
Postprandial blood glucose	108.72 ± 10.63	163.20 ± 11.03	<0.001	Independent-samples t-test
HbA1c (%)	4.81 (4.61–4.99)	6.88 (6.71–6.99)	<0.001	Mann–Whitney U test

Table 3. Comparison of Lipid Profile Between Healthy Pregnant Women and Women With GDM

Parameter	Healthy pregnancy (n=30)	GDM (n=30)	p-value	Statistical test
Total cholesterol	213.01 (210.51–218.35)	249.12 (234.84–258.40)	<0.001	Mann–Whitney U test
Triglycerides	176.65 ± 13.21	244.58 ± 17.95	<0.001	Independent-samples t-test
HDL-C	55.18 ± 5.77	41.13 ± 4.80	<0.001	Independent-samples t-test
LDL-C	119.07 ± 11.23	159.94 ± 13.24	<0.001	Independent-samples t-test
VLDL-C	35.32 ± 2.64	48.92 ± 3.59	<0.001	Independent-samples t-test
HDL/LDL ratio	0.47 ± 0.07	0.26 ± 0.04	<0.001	Independent-samples t-test

Table 4. Comparison of Oxidative Stress Marker Between Healthy Pregnant Women and Women With GDM

Parameter	Healthy pregnancy (n=30)	GDM (n=30)	p-value	Statistical test
Superoxide anion	141.38 ± 9.82	189.58 ± 10.50	<0.001	Independent-samples t-test

Table 5. Comparison of Antioxidant Status Between Healthy Pregnant Women and Women With GDM

Parameter	Healthy pregnancy (n=30)	GDM (n=30)	p-value	Statistical test
Superoxide dismutase (SOD)	5.71 (5.45–5.96)	4.20 (3.92–4.48)	<0.001	Mann–Whitney U test
Ascorbic acid	0.69 (0.65–0.74)	0.40 (0.34–0.46)	<0.001	Mann–Whitney U test

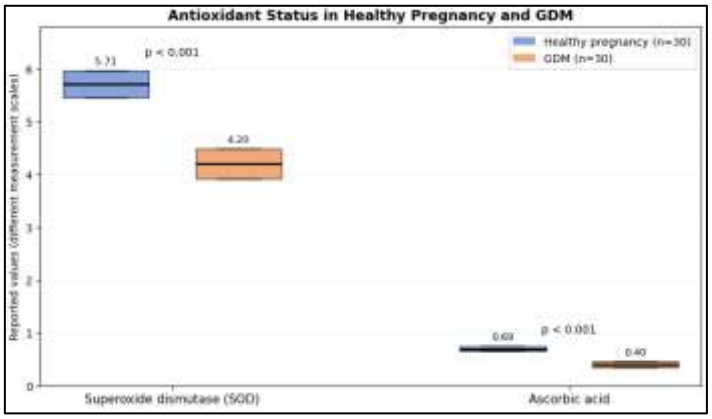


Figure 2. Comparison of Antioxidant Status Between Healthy Pregnant Women and Women With GDM

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Table 6. Correlation of Glycaemic Parameters with Oxidative Stress and Antioxidant Markers Among Women with GDM (n=30)

Parameters correlated	Spearman's ϱ	p-value
HbA1c vs superoxide anion	−0.021	0.911
HbA1c vs SOD	0.073	0.700
HbA1c vs ascorbic acid	0.149	0.433
Superoxide anion vs SOD	−0.079	0.680
Superoxide anion vs ascorbic acid	−0.024	0.900
SOD vs ascorbic acid	−0.119	0.531

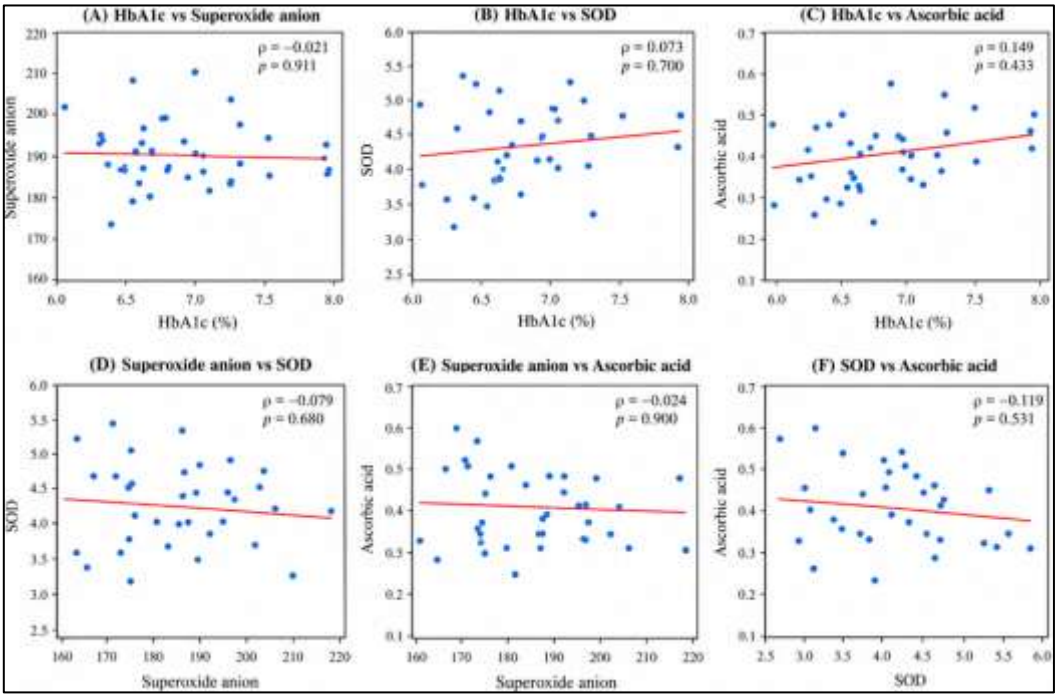


Figure 3. Correlation of Glycaemic Parameters with Oxidative Stress and Antioxidant Markers Among Women with GDM (n=30)

DISCUSSION

In the present study, mean body weight was significantly higher among women with GDM than healthy pregnant controls (69.14 ± 6.49 vs 59.21 ± 6.21 kg; p<0.001). However, height (155.12 ± 4.67 vs 155.08 ± 5.07 cm; p=1.00), haemoglobin (11.25 ± 0.82 vs 11.45 ± 0.65; p=0.79), and total protein (6.80 vs 6.80; p=1.00) were comparable. Saucedo et al. (2023) [8] described increased maternal adiposity as an important contributor to insulin resistance, inflammation, and oxidative stress in GDM, supporting the association between higher maternal weight and metabolic dysfunction observed in our study. Women with GDM demonstrated significantly higher fasting glucose (117.65 ± 7.43 vs 81.12 ± 8.15), postprandial glucose (163.20 ± 11.03 vs 108.72 ± 10.63), and median HbA1c (6.88% vs 4.81%) compared with healthy pregnant women (all p<0.001). Saucedo et al. (2023) [8] reported that maternal hyperglycaemia enhances reactive oxygen species (ROS) production through mitochondrial dysfunction and advanced glycation pathways, contributing to insulin resistance and oxidative injury. These mechanisms are consistent with the marked hyperglycaemia and increased oxidative stress observed in the present study.The present study demonstrated significantly higher total cholesterol (249.12 vs 213.01), triglycerides (244.58 ± 17.95 vs 176.65 ± 13.21), LDL-C

(159.94 ± 13.24 vs 119.07 ± 11.23), and VLDL-C (48.92 ± 3.59 vs 35.32 ± 2.64) among women with GDM. Conversely, HDL-C was significantly lower (41.13 ± 4.80 vs 55.18 ± 5.77; all $p<0.001$). Espinoza et al. (2021) [13] described the combined effects of GDM and maternal hypercholesterolaemia on placental vascular function and fetal cardiovascular risk. Yañez and Leiva (2022) [3] highlighted the relationship between disturbed placental cholesterol transport, mitochondrial dysfunction, and oxidative stress. These findings support the metabolic and oxidative implications of the altered lipid profile observed in our study.Mean superoxide anion levels were significantly higher in women with GDM than healthy pregnant controls (189.58 ± 10.50 vs 141.38 ± 9.82; $p<0.001$), representing approximately 34.1% higher levels. Saucedo et al. (2023) [8] highlighted increased ROS production and oxidative injury in GDM due to hyperglycaemia and mitochondrial dysfunction. Similarly, Yañez and Leiva (2022) [14] described the involvement of placental mitochondrial dysfunction in oxidative stress. These findings support the elevated oxidative stress observed in our study.The present study demonstrated significantly lower median SOD [4.20 (3.92–4.48) vs 5.71 (5.45–5.96)] and ascorbic acid [0.40 (0.34–0.46) vs 0.69 (0.65–0.74)] among women with GDM compared with healthy controls (both $p<0.001$). Saucedo et al. (2023) [8] described impaired antioxidant defence and excessive ROS generation as important mechanisms underlying oxidative imbalance in GDM. Yañez and Leiva (2022) [14] further emphasized mitochondrial oxidative disturbances in placental tissues. These findings are consistent with the reduced antioxidant markers observed in our study.In the present study, HbA1c showed no significant correlation with superoxide anion ($\varrho=-0.021$, $p=0.911$), SOD ($\varrho=0.073$, $p=0.700$), or ascorbic acid ($\varrho=0.149$, $p=0.433$). Similarly, correlations between superoxide anion and SOD ($\varrho=-0.079$, $p=0.680$) and ascorbic acid ($\varrho=-0.024$, $p=0.900$) were non-significant. Saucedo et al. (2023) [8] described oxidative stress in GDM as a multifactorial process involving hyperglycaemia, inflammation, and antioxidant imbalance. Shi et al. (2005) [3] highlighted additional mitochondrial and lipid-related mechanisms.

CONCLUSION

The present study demonstrated significantly increased superoxide anion levels and reduced superoxide dismutase (SOD) and ascorbic acid concentrations among women with gestational diabetes mellitus (GDM) compared with healthy pregnant controls ($p<0.001$). These alterations were accompanied by significant hyperglycaemia and dyslipidaemia, indicating an imbalance between oxidative stress and antioxidant defence in GDM. However, no significant correlations were observed between HbA1c and oxidative stress or antioxidant markers, suggesting that additional metabolic factors may contribute to oxidative imbalance.

LIMITATIONS

The study was limited by its single-centre design and relatively small sample size (n=60), which may restrict the generalizability of the findings. Furthermore, its observational nature, lack of adjustment for potential confounding factors, and absence of longitudinal follow-up prevented assessment of causal relationships and long-term outcomes.

DECLARATION:

- **Ethics Approval and Consent to Participate:** Ethical approval was obtained from the Institutional Ethics Committee of SGT University, Gurugram. Written informed consent was obtained from all participants before data collection.
- **Consent for Publication:** Not applicable.
- **Availability of Data and Materials:** The datasets generated and analysed during the study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.
- **Competing Interests:** The authors declare no competing interests.
- **Funding:** No external funding was received for this study.
- **Authors' Contributions: S.S.:** Conceptualization, methodology, investigation, sample collection, laboratory analysis, data analysis, and manuscript drafting. S.K.B.: Research supervision, study design, methodological guidance, interpretation of findings, critical manuscript revision, and final review. Both authors reviewed and approved the final manuscript and agreed to be accountable for their contributions.
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