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Assessment of Oxidative Stress Biomarkers among Pregnant Women with Hypertensive Disorders in Osun State, Nigeria

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Abstract

Background: Hypertension in pregnancy, particularly pre-eclampsia and eclampsia, is a major cause of maternal and foetal morbidity and mortality in Nigeria. Oxidative stress, resulting from an imbalance between reactive oxygen species and antioxidant defences, is increasingly implicated in their pathophysiology, yet data on disease severity across Nigerian populations remain limited. This study assessed oxidative stress and antioxidant profiles among normotensive, pre-eclamptic, and eclamptic pregnant women. **Methods:** This cross-sectional study enrolled 210 pregnant women (≥20 weeks' gestation) attending antenatal clinics in Osun State, Nigeria, comprising 70 normotensive controls, 70 women with preeclampsia, and 70 with eclampsia. Blood samples were analysed using standard biochemical methods to measure malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and total antioxidant capacity (TAC). Group differences were assessed using one-way ANOVA, and Pearson correlation was used to evaluate associations with blood pressure ($p < 0.05$). **Results:** MDA levels increased progressively from normotensive pregnancy (3.87 ± 0.94 µmol/L) to pre-eclampsia (6.52 ± 1.15 µmol/L) and eclampsia (8.26 ± 1.48 µmol/L; $p < 0.001$). Conversely, SOD, CAT, GPx, and TAC declined significantly across groups, with the lowest values observed in eclampsia (all $p < 0.001$). A graded oxidative imbalance was evident from mild pre-eclampsia to eclampsia. MDA correlated negatively with SOD ($r = -0.68$), CAT ($r = -0.60$), GPx ($r = -0.63$), and TAC ($r = -0.71$), and positively with systolic ($r = 0.62$) and diastolic blood pressure ($r = 0.58$) (all $p < 0.001$). **Conclusion:** Pre-eclampsia and eclampsia are associated with a progressive increase in oxidative stress and depletion of antioxidant defences. The strong associations between oxidative markers and blood pressure support oxidative stress as a central mechanism in hypertensive disorders of pregnancy and highlight its potential value for assessing disease severity in resource-limited settings.

Keywords: Preeclampsia, Eclampsia, Oxidative stress, Antioxidant biomarkers, Hypertensive disorders.

INTRODUCTION

Preeclampsia remains one of the most significant complications of pregnancy, affecting approximately 2-5% of pregnant women globally, representing a leading cause of maternal and foetal morbidity and mortality

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worldwide (Kariori et al., 2025). The condition is characterised by new-onset hypertension and proteinuria or other evidence of organ dysfunction occurring after 20 weeks of gestation. In Nigeria, the burden is particularly substantial, with recent meta-analytic evidence revealing a pooled prevalence of 4.51% for preeclampsia and 1.39% for eclampsia among pregnant women (Kokori et al., 2024). Furthermore, maternal mortality associated with preeclampsia/eclampsia in Nigeria is alarming, with a pooled rate of 6.04%, alongside a foetal mortality rate of 16.73% (Kokori et al., 2024). These statistics underscore the urgent need to better understand the mechanisms underlying preeclampsia development in the Nigerian population to inform prevention and management strategies.

Preeclampsia is increasingly recognised as a multifactorial disorder originating from abnormal placentation and subsequent placental dysfunction (Kariori et al., 2025). Although the exact aetiology remains incompletely understood, oxidative stress plays a pivotal role in the pathophysiology of preeclampsia (Vornic et al., 2024). Oxidative stress arises from an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defence mechanisms to counteract them. In pregnancy, the placenta is a particularly vulnerable organ to oxidative stress due to the high metabolic demands required to support foetal development and nutrient transport (Sultana et al., 2023). Controlled (low) levels of free radicals are necessary for normal placental signalling and development, while excessive oxidative stress triggers abnormal placental function and immune disturbances linked to several complications, including pregnancy loss, hampered foetal growth and eclampsia (Sultana et al., 2023).

Oxidative stress is increasingly recognised as a central pathophysiological mechanism in the development and progression of preeclampsia and eclampsia (Vornic et al., 2024). Oxidative stress arises when there is an imbalance between ROS production and the capacity of endogenous antioxidant defence systems to neutralise them. The placenta is particularly susceptible to oxidative stress due to its high metabolic demands and the dynamic nature of placental vascularisation and hemodynamic changes during pregnancy (Sultana et al., 2023). In abnormal placentation, the placental tissue becomes hypoxic and ischemic, triggering excessive ROS production that exceeds the capacity of antioxidant mechanisms (Vornic et al., 2024). This oxidative imbalance has been implicated in endothelial dysfunction, systemic inflammation, and the multi-organ manifestations characteristic of preeclampsia (Sultana et al., 2023).

The severity of oxidative stress is thought to correlate with disease progression and clinical severity. Studies examining oxidative stress markers such as malondialdehyde (MDA) have reported progressive increases with advancing preeclampsia severity, and further elevations in eclampsia (Vornic et al., 2024). Conversely, antioxidant enzyme activity, including superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and total antioxidant capacity (TAC), has been shown to progressively decline across the spectrum from normal pregnancy through preeclampsia to eclampsia in various population studies (Sultana et al., 2023).

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Although oxidative stress mechanisms in preeclampsia have been well characterised in populations from Europe and North America, comparative data from African populations, including Nigeria, remain limited. Evidence from sub-Saharan African studies suggests patterns of oxidative stress in preeclampsia generally consistent with findings from other regions; however, the specific expression of oxidative stress markers and antioxidant profiles in Nigerian women with preeclampsia and eclampsia has received insufficient attention (Kokori et al., 2024). Given the higher prevalence and severity of preeclampsia in Nigeria compared to developed countries, characterising the oxidative stress and antioxidant profiles across the spectrum of hypertensive pregnancy disorders in Nigerian women may provide important context-specific insights and support efforts to improve recognition and management of these conditions.

Therefore, this study aimed to assess and compare oxidative stress biomarkers (malondialdehyde) and antioxidant parameters (superoxide dismutase, catalase, glutathione peroxidase, and total antioxidant capacity) among normotensive pregnant women and those with preeclampsia and eclampsia in Osun State, Nigeria. Additionally, the study evaluated the relationship between oxidative stress markers, blood pressure parameters, and disease severity in order to enhance understanding of the role of oxidative imbalance in hypertensive disorders of pregnancy.

METHODS

Study Design

This cross-sectional study was conducted among pregnant women attending selected antenatal clinics in Osun State, Southwestern Nigeria, to assess oxidative stress biomarkers in hypertensive disorders of pregnancy. Two hundred and ten (210) participants were categorised into three groups: women with preeclampsia (n = 70), women with eclampsia (n = 70), and normotensive pregnant controls (n = 70). The diagnoses of preeclampsia and eclampsia were established using standard clinical criteria, defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg after 20 weeks of gestation with proteinuria ≥1+ on urine dipstick testing, while eclampsia was defined by the occurrence of seizures in a woman with preeclampsia (ACOG, 2020; WHO, 2023).

Study Participants

Eligible participants were pregnant women aged 18–45 years with a gestational age of at least 20 weeks who attended antenatal clinics during the study period (January to April 2025). Women receiving antioxidant supplementation were excluded to avoid potential interference with oxidative stress measurements. Additional exclusion criteria included a history of chronic hypertension, diabetes mellitus, renal or hepatic disease, as well as the use of medications known to influence oxidative balance. Eligible participants were consecutively recruited after providing informed consent.

Ethical Considerations

Ethical approval for the study was obtained from the Osun State Ministry of Health Ethical Review Board (Reference No.: OSHREC/PRS/569T/1322). Written informed consent was obtained from all participants prior to enrolment. Confidentiality of participants' information was strictly maintained, and all procedures were conducted in accordance with

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the ethical principles outlined in the Declaration of Helsinki.

Sample Collection and Laboratory Analysis

Approximately 5 mL of fasting venous blood was collected aseptically from each participant into plain sample tubes. Blood samples were centrifuged at 3000 rpm for 10 minutes to obtain serum, which was aliquoted and stored at −20°C until biochemical analysis. Serum malondialdehyde (MDA), an indicator of lipid peroxidation, was measured using the thiobarbituric acid reactive substances method described by Buege and Aust (1978). Superoxide dismutase (SOD) activity was determined using the method of Sun and Zigman (1978). Catalase (CAT) activity was assessed according to Sinha (1972), while glutathione peroxidase (GPx) activity was measured using the method described by Rotruck et al. (1973). All assays were performed according to standard laboratory protocols to ensure analytical accuracy and reproducibility.

Statistical Analysis

Data were analysed using the Statistical Package for the Social Sciences (SPSS) version 25. Continuous variables were expressed as mean ± standard deviation. Differences among study groups were assessed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test for multiple comparisons. Pearson correlation analysis was used to evaluate relationships among oxidative stress biomarkers and clinical parameters. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics of Participants

A total of 210 participants were enrolled, comprising 70 normotensive individuals, 70 with pre-eclampsia, and 70 with eclampsia (Table 1). The mean age was 29.1 ± 4.6 years in the normotensive group, 30.4 ± 5.2 years in the pre-eclampsia group, and 31.0 ± 5.0 years in the eclampsia group ($p = 0.083$). Mean gestational age was 33.4 ± 3.5 weeks among normotensive participants, 32.6 ± 3.7 weeks in pre-eclampsia, and 31.8 ± 4.0 weeks in eclampsia ($p = 0.071$). Median parity was 2 (IQR: 1–3) in the normotensive group, 2 (IQR: 1–4) in pre-eclampsia, and 3 (IQR: 2–4) in eclampsia ($p = 0.038$). Mean body mass index increased across groups, from 25.8 ± 3.2 kg/m² in normotensive participants to 29.1 ± 4.0 kg/m² in pre-eclampsia and 31.4 ± 4.5 kg/m² in eclampsia ($p < 0.001$). Mean systolic blood pressure was 112.6 ± 7.4 mmHg, 150.5 ± 9.2 mmHg, and 166.8 ± 11.0 mmHg in the normotensive, pre-eclampsia, and eclampsia groups, respectively ($p < 0.001$). Mean diastolic blood pressure was 74.0 ± 5.8 mmHg in normotensive participants, 96.7 ± 7.9 mmHg in pre-eclampsia, and 108.3 ± 8.5 mmHg in eclampsia ($p < 0.001$).

Oxidative Stress and Antioxidant Biomarkers among Study Groups

Levels of oxidative stress and antioxidant biomarkers across study groups are presented in Table 2. Mean malondialdehyde (MDA) concentrations were 3.87 ± 0.94 µmol/L in normotensive participants, 6.52 ± 1.15 µmol/L in pre-eclampsia, and 8.26 ± 1.48 µmol/L in eclampsia ($p < 0.001$). Superoxide dismutase (SOD) levels were 12.8 ± 2.4 U/mL, 8.9 ± 1.8

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U/mL, and 6.7 ± 1.5 U/mL in the normotensive, pre-eclampsia, and eclampsia groups, respectively ($p < 0.001$). Catalase (CAT) levels were 43.5 ± 6.2 U/mL in normotensive participants, 35.1 ± 5.7 U/mL in pre-eclampsia, and 28.3 ± 4.9 U/mL in eclampsia ($p < 0.001$). Mean glutathione peroxidase (GPx) levels were 9.8 ± 1.9 U/mL, 7.2 ± 1.5 U/mL, and 5.6 ± 1.2 U/mL across the three groups, respectively ($p < 0.001$). Total antioxidant capacity (TAC) values were 1.65 ± 0.21 mmol/L in normotensive participants, 1.18 ± 0.17 mmol/L in pre-eclampsia, and 0.91 ± 0.15 mmol/L in eclampsia ($p < 0.001$).

Oxidative Biomarkers by Severity of Hypertension

Comparison of oxidative biomarkers by severity of hypertension is presented in Table 3. Among participants with mild pre-eclampsia (n = 35), mean MDA was 5.98 ± 0.89 µmol/L, SOD was 9.6 ± 1.9 U/mL, CAT was 37.8 ± 6.0 U/mL, GPx was 7.8 ± 1.4 U/mL, and TAC was 1.26 ± 0.18 mmol/L. In severe pre-eclampsia (n = 35), mean MDA was 7.06 ± 1.08 µmol/L, SOD was 8.2 ± 1.7 U/mL, CAT was 32.4 ± 5.4 U/mL, GPx was 6.6 ± 1.3 U/mL, and TAC was 1.09 ± 0.15 mmol/L. In the eclampsia group (n = 70), mean MDA was 8.26 ± 1.48 µmol/L, SOD was 6.7 ± 1.5 U/mL, CAT was 28.3 ± 4.9 U/mL, GPx was 5.6 ± 1.2 U/mL, and TAC was 0.91 ± 0.15 mmol/L.

Correlation between MDA and Antioxidant Parameters

Correlation analysis between MDA and antioxidant parameters is shown in Table 4. MDA exhibited correlations with SOD ($r = -0.68$, $p < 0.001$), CAT ($r = -0.60$, $p < 0.001$), GPx ($r = -0.63$, $p < 0.001$), and TAC ($r = -0.71$, $p < 0.001$).

Relationship between Oxidative Stress Markers and Blood Pressure

The relationships between oxidative stress markers and blood pressure parameters is presented in Table 5. MDA correlated with systolic blood pressure ($r = +0.62$) and diastolic blood pressure ($r = +0.58$), with $p < 0.001$. SOD showed correlations with systolic ($r = -0.55$) and diastolic ($r = -0.50$) blood pressure ($p < 0.001$). CAT correlated with systolic ($r = -0.49$) and diastolic ($r = -0.47$) blood pressure ($p < 0.001$). GPx correlated with systolic ($r = -0.52$) and diastolic ($r = -0.45$) blood pressure ($p < 0.001$). TAC correlated with systolic ($r = -0.57$) and diastolic ($r = -0.53$) blood pressure ($p < 0.001$).

Table 1: Demographic and Clinical Characteristics of Participants

Parameter	Normotensive (n=70)	Pre-eclampsia (n=70)	Eclampsia (n=70)	p-value
Mean age (years)	29.1 ± 4.6	30.4 ± 5.2	31.0 ± 5.0	0.083
Gestational age (weeks)	33.4 ± 3.5	32.6 ± 3.7	31.8 ± 4.0	0.071
Parity (median, IQR)	2 (1-3)	2 (1 - 4)	3 (2 - 4)	0.038*
BMI (kg/m^2)	25.8 ± 3.2	29.1 ± 4.0	31.4 ± 4.5	<0.001*
Systolic BP (mmHg)	112.6 ± 7.4	150.5 ± 9.2	166.8 ± 11.0	<0.001*
Diastolic BP (mmHg)	74.0 ± 5.8	96.7 ± 7.9	108.3 ± 8.5	<0.001*

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Table 2: Levels of Oxidative Stress and Antioxidant Biomarkers among Study Groups

Biomarker	Normotensive	Pre-eclampsia	Eclampsia	P-value
MDA (µmol/L)	3.87 ± 0.94	6.52 ± 1.15	8.26 ± 1.48	<0.001*
SOD (U/mL)	12.8 ± 2.4	8.9 ± 1.8	6.7 ± 1.5	<0.001*
CAT (U/mL)	43.5 ± 6.2	35.1 ± 5.7	28.3 ± 4.9	<0.001*
GPx (U/mL)	9.8 ± 1.9	7.2 ± 1.5	5.6 ± 1.2	<0.001*
TAC (mmol/L)	1.65 ± 0.21	1.18 ± 0.17	0.91 ± 0.15	<0.001*

Table 3: Comparison of Oxidative Biomarkers by Severity of Hypertension

Group	MDA (µmol/L)	SOD (U/mL)	CAT (U/mL)	GPx (U/mL)	TAC (mmol/L)
Mild pre-eclampsia (n=35)	5.98 ± 0.89	9.6 ± 1.9	37.8 ± 6.0	7.8 ± 1.4	1.26 ± 0.18
Severe pre- eclampsia (n=35)	7.06 ±1.08	8.2 ± 1.7	32.4 ± 5.4	6.6 ± 1.3	1.09 ± 0.15
Eclampsia (n=70)	8.26 ± 1.48	6.7 ± 1.5	28.3 ± 4.9	5.6 ± 1.2	0.91 ± 0.15

Table 4: Correlation between MDA and Antioxidant Parameters

Parameter	Correlation Coefficient (r)	p-value	Relationship
SOD	-0.68	<0.001*	Strong Negative
CAT	-060	<0.001*	Strong Negative
GPx	-0.63	<0.001*	Strong Negative
TAC	-0.71	<0.001*	Strong Negative

Table 5: Relationship between Oxidative Stress Markers and Blood Pressure Parameters

Biomarker	SBP (r)	DBP (r)	p-value
MDA	+0.62	+0.58	<0.001*
SOD	-0.55	-0.50	<0.001*
CAT	-0.49	-0.47	<0.001*
GPx	-0.52	-0.45	<0.001*
TAC	-0.57	-0.53	<0.001*

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DISCUSSION

This study demonstrates the differences in oxidative stress and antioxidant profiles across normotensive, pre-eclampsia, and eclampsia groups. Our findings indicate that the normotensive, pre-eclampsia, and eclampsia groups were comparable with respect to maternal age and gestational age, whereas parity, body mass index (BMI), systolic blood pressure, and diastolic blood pressure showed graded increases across hypertensive categories. This pattern indicates that selected baseline and clinical characteristics varied systematically with hypertensive status. At the same time, age and gestational timing were broadly similar across groups, reducing the likelihood that observed differences reflect variation in these parameters.

Across sub-Saharan Africa and Asia, prior evidence shows strong concordance with BMI as a consistent correlate of hypertensive disorders of pregnancy, with overweight and obesity more frequently reported among women with pre-eclampsia and eclampsia (Meazaw et al., 2020; Hounkpatin et al., 2021; Shao et al., 2017). In contrast, findings on parity remain mixed. While syntheses from a meta-analysis emphasise primiparity as a risk factor (Meazaw et al., 2020), other studies from similar population reported higher parity or multigravidity among women with more severe disease, aligning with the higher median parity observed in the eclampsia group in this study (Jikamo et al., 2023; Li et al., 2019; Shi et al., 2021). Studies in Chinese and other Asian populations indicate that multiparity or higher parity may be inversely associated with risk in certain cohorts or show weaker nulliparity effects compared to Western settings, potentially aligning with our higher parity in more severe cases (Shi et al., 2021; Li et al., 2019). Maternal age and gestational age show less consistent regional differentiation, with several reports indicating weak or absent associations, broadly consistent with their comparability across groups here (Meazaw et al., 2020; Shi et al., 2021). This mixed parity evidence may reflect variations in study populations, referral biases in severe cases, or distinctions between early- and late-onset disease. Age and gestational age alignments reduce confounding concerns in our comparisons.

A graded pattern was observed across hypertensive severity for oxidative stress and antioxidant markers. MDA levels increased with severity (from pre-eclampsia to eclampsia), whereas SOD, CAT, GPx, and TAC showed significant reductions. The progressive oxidative imbalance across disease severity likely reflects the cascade of placental dysfunction. In mild preeclampsia, initial compensatory upregulation of antioxidant enzymes may partially offset ROS overproduction. However, in severe preeclampsia and eclampsia, the overwhelming oxidative burden exceeds these compensatory mechanisms, resulting in enzyme depletion and accumulation of lipid peroxidation products. This exhaustion of antioxidant defences is consistent with the stepwise reduction in SOD, CAT, and GPx observed here.

Similar severity-associated patterns have been reported across sub-Saharan African and Asian populations, where higher MDA levels and lower antioxidant indices are more frequently observed in severe pre-eclampsia and eclampsia compared with milder disease or normotensive pregnancy (Adu-Bonsaffoh et al., 2016; Onovughakpo-Sakpa et al., 2021; Taravati et al., 2018; Sharma et al., 2006; Afrose et al., 2022). The marked elevation of MDA and significant correlation with antioxidant enzymes in eclampsia suggests these markers could potentially serve as indicators of critical disease progression. The graded pattern across all groups indicates oxidative stress markers

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reflect disease severity in a dose-dependent manner, potentially useful for clinical decision-making regarding escalation of care or timing of delivery.

In the Nigeria, where early antenatal care uptake is limited and many women present late in pregnancy with established disease, oxidative stress markers measured at presentation may reflect accumulated damage rather than early placental dysfunction. This differs from developed nations where early biomarker detection enables intervention. Our findings could suggest examining whether early detection interventions are feasible and could improve outcomes in this high-burden population in future studies.

Although individual antioxidant enzymes show variability across studies, the predominant evidence supports a consistent pattern of higher oxidative stress marker levels and lower antioxidant measures with increasing disease severity (de Araújo et al., 2023). Studies highlight increased lipid peroxidation products like MDA and diminished enzymatic antioxidants in preeclamptic pregnancies, aligning with our opposing stepwise patterns across mild, severe, and eclamptic categories (Afrose et al., 2022). While direct comparisons of graded severity are less frequently reported, the predominant evidence supports intensified oxidative stress in more severe hypertensive disorders, with limited divergence primarily in compensatory antioxidant responses that do not fully offset the damage. These findings are consistent with a progressive pattern of oxidative imbalance across hypertensive disorder severity, similar to that observed in our study.

Significant associations were also observed between oxidative stress markers and blood pressure parameters. MDA showed positive correlations with both systolic and diastolic blood pressure, whereas SOD, CAT, GPx, and TAC exhibited negative correlations with these measures. The strong positive correlations between MDA and both systolic and diastolic blood pressure, coupled with inverse correlations between antioxidant parameters and blood pressure, suggest oxidative stress as a mechanistic link between placental dysfunction and maternal hypertension. Oxidative stress impairs nitric oxide availability and endothelial function, promoting vasoconstriction and hypertension. The magnitude of these correlations provides evidence supporting oxidative stress as a critical node in the pathophysiology of hypertensive pregnancy disorders. The consistency of these associations across disease severity groups suggests oxidative imbalance may reflect a common underlying mechanism driving the hypertensive phenotype.

Comparable associations have been reported in studies from sub-Saharan Africa and Asia, where lipid peroxidation markers were higher among women with elevated blood pressure, while antioxidant parameters are lower (Sharma et al., 2006; Adu-Bonsaffoh et al., 2016; Akinloye et al., 2021; Taravati et al., 2018). While the strength and specificity of associations for individual antioxidants vary across studies, the overall pattern of opposing associations between oxidative stress markers and antioxidant indices in relation to blood pressure is broadly consistent. These concordances indicate consistent associations between oxidative stress markers and blood pressure measures in preeclampsia, consistent with our significant associations.

Inverse correlations were further observed between MDA and all assessed antioxidant parameters. Oxidative stress-induced endothelial dysfunction is a hallmark of the maternal syndrome in preeclampsia. The progressive elevation in MDA and decline in

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antioxidant capacity observed here likely underlie the increasing endothelial injury reflected in progressively worse blood pressure parameters. This oxidative imbalance may complement other biomarkers of placental dysfunction (such as antiangiogenic factors) in characterizing preeclampsia severity.

The progressive depletion of antioxidant enzymes with increasing disease severity suggests that endogenous antioxidant capacity becomes insufficient to manage the ROS burden. While SOD and CAT may be upregulated initially, the overwhelming and sustained oxidative stress in severe disease and eclampsia likely exhausts these systems through multiple mechanisms: (1) direct enzyme inhibition by oxidative products, (2) substrate depletion, and (3) impaired cofactor availability. The particularly marked reduction in GPx activity in eclampsia may reflect a critical loss of glutathione reserves, a key antioxidant cofactor. This progressive failure of compensatory mechanisms underscores the severity of oxidative imbalance in eclampsia.

Regional studies from Africa and Asia similarly report higher MDA levels alongside lower enzymatic and total antioxidant measures in pre-eclampsia and eclampsia (Adu-Bonsaffoh et al., 2016; Onovughakpo-Sakpa et al., 2021; Taravati et al., 2018; Afrose et al., 2022). Although some reports describe variability or isolated compensatory changes in specific antioxidants, the prevailing evidence supports an overall inverse relationship between lipid peroxidation markers and antioxidant parameters across populations.

Implications for Disease Management and Research

Our study population represents a setting where many women present to healthcare facilities with established rather than early-stage disease, reflecting limited access to early antenatal screening and monitoring. Oxidative stress markers like MDA and antioxidant enzymes, if validated for clinical utility, could potentially be measured using relatively simple and less expensive laboratory methods compared to more sophisticated biomarkers. However, standardization of measurement techniques and establishment of Nigerian-specific reference ranges would be necessary before clinical implementation.

These findings raise the question of whether antioxidant therapies could be therapeutically beneficial in preeclampsia, particularly in eclampsia. Some animal and in vitro studies suggest that mitochondria-targeted antioxidants may improve outcomes (Yang et al., 2021), though clinical translation remains challenging. In the Nigerian context, simpler interventions such as selenium or vitamin supplementation might be feasible, though their efficacy in correcting oxidative imbalance once eclampsia has developed remains unclear. Future prospective studies measuring oxidative stress markers in early pregnancy and their trajectory toward eclampsia could identify windows for intervention.

Study Limitations

The cross-sectional nature of our study makes it observational, and thus, it can not establish causality. The directionality between oxidative stress and eclampsia severity remains inferential. The study focused on classical oxidative biomarkers but did not evaluate placental/angiogenic markers such as sFlt-1 and PlGF. This restricts the clinical translation of the findings.

Conclusions

This study demonstrates a progressive oxidative imbalance across the spectrum of hypertensive pregnancy disorders, with malondialdehyde levels increasing 2.1-fold from normotensive to

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eclampsia, while antioxidant capacity declines. The significant antioxidant depletion in eclampsia further suggests that antioxidant mechanisms become exhausted in severe disease, implicating oxidative stress as a mechanistic target for future therapeutic investigation.

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