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# Study on the Association of Mitochondrial DNA Copy Number with the Severity of Preeclampsia

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## Abstract

**Background:** Preeclampsia (PE) is a pregnancy-related multisystem disease that affects 2-8% of pregnancies globally with immense maternal and perinatal morbidity and mortality. The present study aimed to investigate the association between maternal peripheral blood mitochondrial DNA copy number (mtDNA-CN) and the severity of PE in a Bangladeshi population.

**Methods:** This case-control study enrolled 51 PE cases and 51 normotensive pregnant controls in Bangabandhu Sheikh Mujib Medical University, Dhaka. PE was identified using systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg accompanied by proteinuria and organ involvement. Blood samples were measured by a quantitative polymerase chain reaction to estimate mtDNA-CN from the ratio of ND1 and HGB genes. Categorical and continuous variables were compared using chi-square tests and unpaired t-tests, respectively.

**Results:** PE cases had statistically elevated mtDNA-CN compared to controls, with rising levels from controls (44.67±3.56) to mild PE (75.22±22.92) to severe PE (85.21±12.80) (p=0.007). The most optimal cut-off value of mtDNA-CN was determined to be >45, which had a sensitivity of 68.6% and (-specificity was 58.8% for PE detection. Females with mtDNA-CN >45 had approximately three times higher odds for PE (OR=3.1, 95% CI: 1.4-7.0, p=0.005). Overall accuracy was 63.7% (95% CI: 53.6-73.0%).

**Conclusion:** This study shows a significant correlation between increased maternal peripheral blood mtDNA-CN and PE, with values rising progressively according to the severity of the disease. These results point to mtDNA-CN as a potential biomarker for PE risk stratification and underscore the enteropathogenic role of mitochondrial dysfunctions in PE.

**Keywords:** Preeclampsia, mtDNA-CN, ND1, HGB

## Introduction

Preeclampsia (PE) is a pregnancy-specific multisystem disorder of hypertension and proteinuria, occurring in about 2-8% of all pregnancies globally [1]. PE is a major cause of maternal and perinatal morbidity and mortality, especially in the developing world [2]. It has been studied for over five decades, yet the precise pathophysiologic processes involved are still not fully elucidated, posing difficulty in early diagnosis and optimal management methods. The pathogenic mechanism of PE is suggested to be operative through abnormal placentation in early gestation, followed by placental ischemia, oxidative stress, and systemic endothelial dysfunction [3]. This pathophysiologic cascade ultimately leads to

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the development of hypertension, proteinuria, and potential multi-organ dysfunction. There have been recent studies suggesting a central role for mitochondrial dysfunction in PE pathophysiology, as these organelles are master regulators of cellular energy metabolism and oxidative stress pathways [4]. Mitochondria, the cell's energy-providing powerhouses that produce ATP via oxidative phosphorylation, possess their circular DNA (mtDNA) distinct from nuclear DNA. Mitochondrial DNA copy number (mtDNA-CN) is the number of mitochondrial genomes per cell and is a potential indicator of mitochondrial biogenesis and function [5]. mtDNA-CN alterations have been associated with a variety of pathological conditions of oxidative stress and inflammation, including cardiovascular disease and metabolic disorders [6]. The placenta is an energy-demanding organ with high energy needs to support fetal growth and development. In preeclampsia, placental oxidative stress and mitochondrial failure have been documented, suggesting potential alterations in mitochondrial dynamics [7].

Alterations in mitochondrial morphology, membrane potential, and respiratory chain activity have been described in preeclamptic placentas compared to normal pregnancy [8]. The relationship between maternal circulating mtDNA-CN and PE severity, however, remains under studied. Various biomarkers have been studied for PE prediction and diagnosis, including angiogenic factors (sFlt-1, PlGF), inflammatory markers, and placental proteins [9]. Despite these advances, there is a need for new, precise, and inexpensive biomarkers that can potentially enable early risk prediction and stratification of PE severity. Circulating mtDNA-CN has the potential to serve as a potential biomarker since it is relatively stable in peripheral blood and reflects systemic mitochondrial status [10]. Previous studies have reported conflicting results for mtDNA-CN in preeclamptic pregnancy, with some having higher levels [11] and others having lower levels [12] than in normotensive pregnancy. These differences may be due to variability in methods, timing of sample collection, and heterogeneity of PE phenotype. There have also been few studies directly investigating the correlation of mtDNA-CN with PE severity classification. Bangladesh provides a highly applicable setting for PE study as it has a high maternal mortality ratio (approximately 173 per 100,000 live births), with hypertensive disorders being a major contributor to these deaths [13]. This population might also be shaped by unique genetic, environmental, and socio-economic factors that might influence PE presentation and severity. The identification of PE biomarkers in diverse populations is required to develop globally applicable diagnostic and prognostic tools. In the present work, we seek to investigate the association between maternal peripheral blood mtDNA-CN and the severity of PE in a Bangladeshi population. By investigating the association, we hope to evaluate mtDNA-CN as a possible biomarker for risk stratification of PE and contribute to the understanding of mitochondrial dysfunction in PE pathophysiology. The findings could have the potential to direct follow-up diagnostic approaches and therapeutic interventions along mitochondrial axes in the management of PE.

## Methods

This is a case-control study that was conducted for one year in the Feto-Maternal Medicine and Obstetrics & Gynecology units of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Fifty-one preeclampsia (PE) cases and 51 normotensive pregnant controls were enrolled by using a purposive sampling method. PE patients were defined by systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, proteinuria, and organ involvement, while controls were normotensive pregnant women without proteinuria. The exclusion criteria were multiple pregnancies, chronic hypertension, renal disease, diabetes, autoimmune disease, and intrauterine fetal death.

The study aimed to evaluate maternal mitochondrial DNA copy number (mtDNA-CN) as a possible biomarker for PE. Blood samples were taken from every participant in a 3 mL tube and stored at -20°C. Samples were analyzed via a quantitative polymerase chain reaction (qPCR) method adapted for use. The mtDNA-CN was estimated from the ND1 and HGB gene ratio, and samples were all executed on a CFX Opus 96 Real-Time PCR System. Furthermore, statistical analysis of the data derived from qPCR results was performed on SPSS version 26. Categorical and continuous variables were compared by chi-square tests and unpaired t-tests, respectively. ROC curve analysis determined the potential cut-off value of mtDNA-CN for predicting the risk of PE. The p-value of less than 0.05 was taken as the criterion for statistical significance. The IRB of BSMMU approved the ethics and ensured adherence to the Helsinki Declaration. Informed consent was taken and patient confidentiality was maintained stringently. The quality control measures involved strict selection criteria, multiple data reviews, and regular supervision to maintain the integrity and reliability of data.

## Results

Table 1 denotes the comparison of socio-demographic profiles between PE cases and healthy controls. Both groups had similar age distributions with the most (49% within each group) of the participants in the 21-30 years category, although the cases were significantly older on average (30.0±4.3 compared to 28.5±4.8 years). Educational

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levels were otherwise disparate, with cases more frequently achieving higher secondary schooling (68.6%) compared with controls (45.1%). Incomes were also different, with 67.7% of the cases reporting family income of more than 25,000 BDT as opposed to 47.1% of the controls. However, group differences were not statistically significant for age, education, and income distribution (p>0.05), which suggests comparable socio-demographic features despite these differences.

Table 2 contrasts obstetric profiles between cases and controls. Primigravida (first pregnancy) distribution was near between groups: 27.5% in cases and 31.4% in controls. Most participants in both groups were in the third trimester (31-36 weeks gestation), representing 72.5% of cases and 62.7% of controls. The remaining participants were distributed across the other gestational periods, with a few percentage points less than 30 weeks and approximately 18-19% greater than 36 weeks in both groups. No differences in obstetric characteristics between PE cases and controls were found using statistical testing (p>0.05), reflecting similar gestational timing and pregnancy histories.

Table 3 illustrates a gradual increase in mitochondrial DNA copy number (mtDNA-CN) based on preeclampsia severity. The control group had the lowest mean mtDNA-CN (44.67±3.56), while mild preeclampsia (75.22±22.92) was in the middle, and severe preeclampsia cases had the highest (85.21±12.80). Statistical comparison with ANOVA confirmed that these were significant differences (p=0.007), which demonstrates a potential biological correlation between mitochondrial dysfunction and preeclampsia pathophysiology. This result supports the hypothesis that increased mtDNA copy number may be a marker of severity in preeclampsia and may reflect oxidative stress and mitochondrial damage in the condition.

Table 4 provides the information compulsory to pick the best cut-off for clinical application by comparing the diagnostic performance characteristics of mtDNA-CN at multiple possible threshold values. For each potential cut-off, the table provides the resultant sensitivity (ratio of true preeclampsia cases correctly classified), specificity (ratio of true non-preeclampsia cases correctly classified), and Youden Index (sensitivity + specificity - 1), which is an index of the overall discriminatory ability at that cut-off. There is an increasing trade-off between sensitivity and specificity as the cut-off increases. For lower thresholds (e.g., >20), sensitivity is extremely high (0.96) but so is the false-positive rate (0.14), so virtually all instances of preeclampsia would be detected but with an overwhelming number of false positives. Increasing the threshold reduces sensitivity but increases specificity. With the Youden Index, which considers these two opposing factors, an optimal cut-off value of >45 was determined with sensitivity of 0.69 and specificity of 0.61, with the highest Youden Index of 0.29.

Table 5 assigns a quantitative value to the strength of the relationship between raised mtDNA-CN and preeclampsia with the generally accepted optimum cut-off value of >45. The values illustrate that more than two-thirds of women with preeclampsia (68.6%) had values above this cut-

**Table 1:** Socio-demographic characteristics of participants

| Characteristics | Case N (%) | Control N (%) | p-value |
|---|---|---|---|
| **Age (years)** | | | |
| ≤20 | 3 (5.9%) | 5 (9.8%) | 0.744[a] |
| 21-30 | 25 (49%) | 25 (49%) | |
| >30 | 23 (45.1%) | 31 (41.2%) | |
| Mean±SD | 30.0±4.3 | 28.5±4.8 | |
| **Educational Qualification** | | | |
| Illiterate | 4 (7.8%) | 3 (5.9%) | 0.063[a] |
| Primary | 6 (11.8%) | 8 (15.7%) | |
| SSC | 6 (11.8%) | 17 (33.3%) | |
| HSC | 35 (68.6%) | 23 (45.1%) | |
| **Monthly family income (BDT)** | | | |
| <10,000 | 2 (3.9%) | 3 (5.9%) | 0.281[a] |
| 10,000-25,000 | 17 (33.3%) | 24 (47.1%) | |
| >25,000 | 32 (67.7%) | 24 (47.1%) | |
| **Total** | **51 (100%)** | **51 (100%)** | |

[a]p-value obtained from chi-square test

**Table 2:** Obstetrics characteristics of participants

| Characteristics | Case N (%) | Control N (%) | p-value |
|---|---|---|---|
| **Gravida** | | | |
| Primigravida | 14 (27.5%) | 16 (31.4) | 0.664[a] |
| Multigravida | 37 (72.5%) | 35 (68.6) | |
| **Gestational age (weeks)** | | | |
| 20-25 | 0 (0%) | 1 (2%) | 0.333[a] |
| 26-30 | 4 (7.8%) | 9 (17.6%) | |
| 31-36 | 37 (72.5%) | 32 (62.7%) | |
| >36 | 10 (19.6%) | 9 (17.6%) | |
| **Total** | **51 (100%)** | **51 (100%)** | |

[a]p-value obtained from chi-square test

**Table 3:** Comparison of Mitochondrial DNA copy number between severity of PE and control

| Mild preeclampsia | Severe preeclampsia | Control | P value |
|---|---|---|---|
| **Mean ± SE** | **Mean ± SE** | **Mean ± SE** | |
| 75.22±22.92 | 85.21±12.80 | 44.67±3.56 | 0.007[c] |

[c]p-value obtained from ANOVA F-test

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point, compared to only 41.2% of controls. The proportion difference was statistically significant (p=0.005), providing conclusive proof of association. The odds ratio (OR) of 3.1, with a 95% confidence interval of 1.4-7.0, offers a quantitation of this association, and from it, one can observe that women with mtDNA-CN >45 had approximately three times higher odds of getting preeclampsia than with less than these values. The reason the lower limit of the confidence interval is well outside 1.0 is to ensure that the association is statistically significant.

Table 6 represents an overall appraisal of the diagnostic precision of mtDNA-CN with a cut-off of >45 as established. This 68.6% sensitivity (95% CI 54.1%-80.9%) implies that this cut-off would recognize roughly two for every three preeclampsia women but miss roughly a third (false negatives). With the specificity being 58.8% (95% CI 44.2%-72.4%), this would imply that roughly 60% of non-preeclampsia women would correctly be diagnosed negative and roughly 40% of them would mistakenly be reported positive. The positive predictive value (PPV) of 62.5% (95% CI 53.3%-70.8%) is that 63% of women with mtDNA-CN >45 would have preeclampsia. The negative predictive value (NPV) of 65.2% (95% CI 54.1%-74.9%) is that among women with mtDNA-CN ≤45, 65% would not have preeclampsia. The overall accuracy of 63.7% (95% CI 53.6%-73.0%) is the proportion of all women (both with and without preeclampsia) who would be correctly classified by using this threshold.

**Table 4:** Sensitivity and specificity of maternal mitochondrial DNA copy number in different cut-off values

| Cut-off value | Sensitivity | Specificity | Youden Index (Sensitivity+Specificity-1) |
|---|---|---|---|
| >20 | 0.96 | 0.14 | 0.1 |
| >25 | 0.94 | 0.24 | 0.18 |
| >30 | 0.92 | 0.31 | 0.24 |
| >35 | 0.84 | 0.39 | 0.24 |
| >40 | 0.77 | 0.49 | 0.26 |
| **>45** | **0.69** | **0.61** | **0.29** |
| >50 | 0.59 | 0.65 | 0.24 |
| >55 | 0.45 | 0.8 | 0.26 |
| >60 | 0.37 | 0.82 | 0.2 |
| >65 | 0.33 | 0.86 | 0.2 |
| >70 | 0.33 | 0.9 | 0.24 |

**Table 5:** Association between maternal mitochondrial DNA copy number and preeclampsia (n=102)

| Mitochondrial DNA copy number | Case N (%) | Control N (%) | P-value | OR (95% CI) |
|---|---|---|---|---|
| >45 | 35 (68.6%) | 21 (41.2%) | 0.005[a] | |
| ≤45 | 16 (31.4%) | 30 (58.8%) | | |
| **Total** | **51 (100%)** | **51 (100%)** | | |

[a]p-value obtained from chi-square test

**Table 6:** Diagnostic accuracy of serum mtDNA-CN level to identify preeclampsia

| Statistics | Value | 95% CI |
|---|---|---|
| Sensitivity | 35/51=68.6% | 54.1% to 80.9% |
| Specificity | 30/51=58.8% | 44.2% to 72.4% |
| PPV | 35/56=62.5% | 53.3% to 70.8% |
| NPV | 30/46=65.2% | 54.1% to 74.9% |
| Accuracy | (35+30)/102=63.7% | 53.6 to 73.0% |

## Discussion

This study demonstrated a significant association of elevated maternal peripheral blood mitochondrial DNA copy number (mtDNA-CN) with preeclampsia (PE) and mtDNA-CN correlating with PE severity in a positive manner. Application of >45 as a cut-off value for mtDNA-CN generated moderate diagnostic sensitivity (63.7%) with 68.6% sensitivity and 58.8% specificity and can potentially serve as a biomarker for PE risk stratification. Our finding of higher mtDNA-CN in PE cases compared to normotensive controls is in line with previous research by Qiu et al. [19], which reported elevated mtDNA-CN in placental tissue from preeclamptic pregnancies. This elevation can be viewed as a compensatory response to oxidative stress and mitochondrial damage characteristic of PE pathophysiology [17]. The current dose-response relationship between mtDNA-CN and PE severity further adds to this biological likelihood and suggests worsening mitochondrial dysfunction in accordance with clinical severity. The pathophysiologic explanation for elevated mtDNA-CN in PE is most likely the result of placental ischemia-induced oxidative stress, a central mechanism in PE pathogenesis [14]. During an episode of oxidative stress, mitochondrial damage evokes compensatory biogenesis to maintain cellular energy homeostasis [15]. The mechanism of elevated mtDNA-CN results from the cells' efforts to maintain mitochondrial function despite ongoing damage. McCarthy and Kenny [15] have already established mitochondrial dysfunction as a central component of PE pathophysiology, making systemic endothelial dysfunction by virtue of increased generation of reactive oxygen species and release of pro-inflammatory cytokines. Though our findings are contrary to some investigations which have shown decreased mtDNA-CN in PE [20], such variation most likely is ascribed to technical differences, sampling time, and heterogeneity in the population. Worth noting, however, is the fact that our finding of increased mtDNA-CN is paralleled by

other pathologies with similar definitions involving systemic inflammation and oxidative stress, i.e., cardiovascular disease and metabolic derangement [16]. This similarity argues for the fact that mtDNA-CN alterations constitute a common response to oxidative stress in different diseases.

The diagnostic performance of mtDNA-CN at our established cut-off value (>45) represented moderate utility as a potential biomarker. The odds ratio of 3.1 (95% CI: 1.4-7.0) suggests that women with mtDNA-CN above this value have three-fold greater odds of PE. However, the sensitivity (68.6%) and specificity (58.8%) suggest that mtDNA-CN alone may be insufficient for absolute diagnosis. Alternatively, mtDNA-CN may also have potential as a component of a panel of multi-markers besides established biomarkers such as angiogenic factors (sFlt-1, PlGF) [18] towards enhancing prediction and risk stratification for PE. Socio-demographic concordance between our control and case groups strengthens the validity of our findings by minimizing potential confounding. Both groups also had comparable distributions of age, education, income, gravidity, and gestational age, which suggests differences observed in mtDNA-CN are less likely to be due to demographics than PE pathophysiology. Our study offers novel data from a Bangladeshi cohort, in which PE is a leading reason for maternal mortality [21]. This ethnic and geographical context is particularly valuable since the majority of the literature on mtDNA-CN in PE thus far has been within Western or East Asian groups. The generalizability of our findings to this South Asian context supports the potential global utility of mtDNA-CN as a marker for PE in various populations. From a translational perspective, our findings suggest mitochondrial dysfunction as a potential target for therapy in the treatment of PE. Mitochondria-targeted antioxidants and mitochondrial biogenesis-promoting agents could be novel therapeutic strategies worthy of investigation in preclinical and clinical studies. In addition, serial measurement of maternal mtDNA-CN during pregnancy could provide valuable insights into PE development and response to treatment.

## Limitations of the Study

There are several limitations to the study including the relatively small sample size that may constrain statistical power for subgroup analyses, the single-center case-control design that limits causal inference and generalizability, and peripheral blood measurement of mtDNA-CN rather than placental tissue which may not reflect localized mitochondrial dysfunction at the maternal-fetal interface.

## Conclusion

This study demonstrates a robust correlation of elevated maternal mitochondrial DNA copy number with preeclampsia and a stepwise increase with disease severity. At a clinically applicable cut-off of >45, mtDNA-CN demonstrated moderate diagnostic accuracy, heralding its potential as a biomarker for preeclampsia risk stratification. The findings position mitochondrial dysfunction at the center of preeclampsia pathophysiology and offer the potential for the development of mitochondria-targeted therapeutic interventions. Additional research must focus on longitudinal assessment of mtDNA-CN changes throughout pregnancy and validation in diverse populations to further its clinical applications.

## Recommendations

Subsequent studies should include longitudinal multicenter cohorts with greater numbers and serial measurements of mtDNA-CN during pregnancy, as well as the exploration of therapeutic interventions targeted at mitochondrial function for the prevention and treatment of PE.

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