

Volume of Parenchyma and Non-parenchyma in Human Placenta in Gestational Diabetes and Eclampsia

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ABSTRACT

Background: Eclampsia and gestational diabetes mellitus are metabolic conditions linked to pregnancy that have significant effects on the structure and function of the placenta. The goal of this study was to examine the relative and absolute volumes of the human placenta's parenchymal and non-parenchymal components in cases of eclampsia and gestational diabetes mellitus.

Materials and methods: A total of sixty placentae from the control, gestational diabetes mellitus and eclampsia groups (n = 20 each) were analysed. The point counting approach was used to estimate the volume of placental components.

Results: Without appreciable proportional alterations, placentae from gestational diabetes mellitus showed higher absolute volumes of both parenchyma and non-parenchyma. On the other hand, placentae from eclampsia displayed a proportionate rise in non-parenchymal components together with a considerable /significant decrease in absolute parenchymal volume.

Conclusion: Different patterns of placental remodelling are seen in gestational diabetes mellitus and eclampsia, with the former showing primarily adaptive modifications and the latter showing pathogenic abnormalities.

Key words: Eclampsia, Gestational diabetes mellitus, Placenta; Parenchyma; Non-parenchyma.

Introduction

The placenta is a vital organ that regulates fetal growth, metabolism and hormones during pregnancy. The placenta's structure adapts to meet fetal demands and changes in its components may indicate maternal illness processes.^{1,2}

Gestational Diabetes Mellitus (GDM) and eclampsia are severe metabolic illnesses that affect placental shape, function and neonatal outcomes. Gestational diabetes mellitus has frequently been associated with placental enlargement and increased functional tissue mass, changes that are generally interpreted as adaptive responses to altered metabolic demand.^{3,4} In contrast, hypertensive disorders of pregnancy, particularly eclampsia, are commonly linked to placental ischemia, infarction and structural compromise, resulting in reduced villous tissue and impaired uteroplacental circulation.^{5,6}

Previous morphometric investigations have reported inconsistent findings regarding the relative and absolute volumes of placental parenchymal and non-parenchymal components in these conditions. While some research found that diabetic pregnancies had increased villous mass and intervillous expansion, other studies found no structural change, especially in well-controlled gestational diabetes, which supports an adaptive rather than pathogenic interpretation.^{5,6,7,8} Similarly, studies of eclampsia and related hypertensive disorders have demonstrated varying degrees of villous loss and non-parenchymal expansion, reflecting heterogeneity in disease severity and population characteristics.^{9,10}

Although advances in placental research have emphasized refined stereological and microstructural approaches, these investigations continue to assess the same fundamental biological compartments originally evaluated through gross volumetric methods.^{11,12} Subsequent studies have confirmed that increased placental villous mass and preserved efficiency characterize gestational diabetes, whereas reductions in villous volume and ischemic injury dominate in eclampsia.^{13,14}

In this context, quantitative assessment of both proportional and absolute volumes of placental parenchymal and non-parenchymal components remains relevant, particularly in population-specific settings. The present study therefore aimed to compare the relative and absolute volumes of these placental components in gestational diabetes mellitus and eclampsia in a Bangladeshi population.

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Materials and methods

This cross-sectional study was conducted at Bangladesh Medical University (Former Bangabandhu Sheikh Mujib Medical University) Department of Anatomy. Analysis was done on 60 placenta samples from Bangladeshi mothers who underwent caesarean sections and gave delivery between 37 to 41 weeks of gestation. The study population consisted of three groups: Control (Non-diabetic, non-hypertensive) Gestational diabetes mellitus and eclampsia (n=20 each) which were determined according to World Health Organization criteria.^{18,19} Placentae were retrieved immediately after delivery, trimmed, weighed and kept at 10% formalin. The mass determination of parenchymal and non-parenchymal components was performed by a point-counting method provided by Aherne and Dunnill.¹ Parenchyma was defined as villous or intervillous space participating in maternofetal exchange, whereas based on Teasdale, non-parenchyma comprised chorionic and basal plates, massive fetal vessels, septa and pathological entities.¹⁵ Statistical analyses were based on comparisons of variance with post-hoc comparison of groups.

The methodological robustness of this method has been confirmed in subsequent stereological reviews.¹²

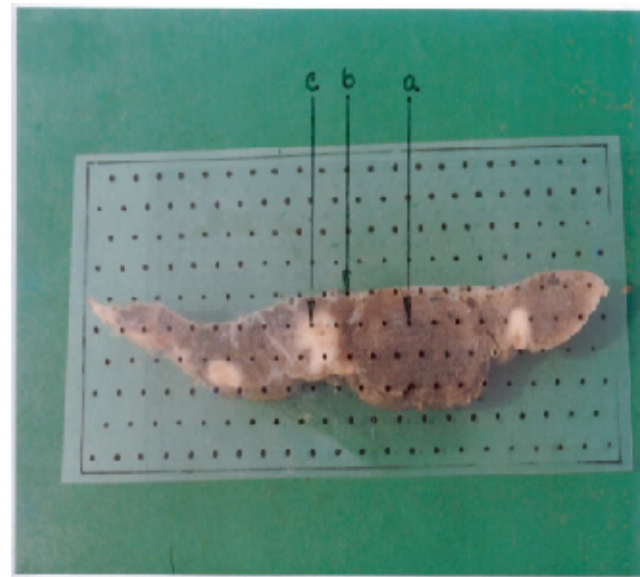
Results

A summary of the mean proportional and absolute volumes of the placental parenchymal and non-parenchymal components in the groups with eclampsia, gestational diabetes mellitus and control is provided in Table I. Placentae from Gestational Diabetes Mellitus had higher mean proportional and absolute parenchymal volumes than the Control group; however, these differences were not statistically significant. The proportional volume of non-parenchyma decreased slightly while its absolute volume increased.

The Eclampsia group, on the other hand, showed reduced proportional and absolute volumes of parenchyma along with a greater proportionate volume of non-parenchyma,



1A



1B

Figure 1A Photograph showing the internal surface of a slice of placenta. Arrowheads indicate the non-parenchymal components, while the remaining area represents the parenchyma. The red arrowheads above and below indicate the chorionic plate and basal plate, respectively.

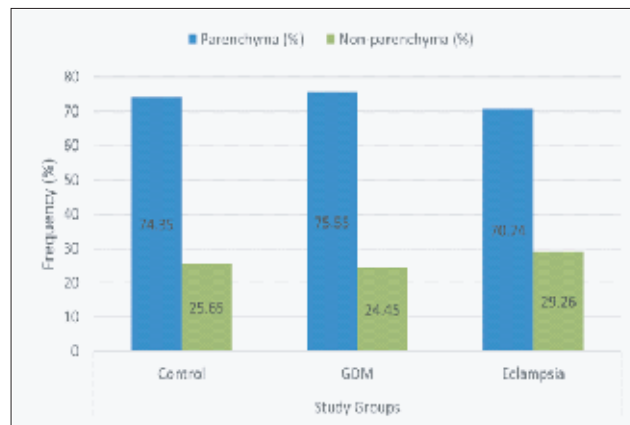
Figure 1B Photograph of a placental section overlaid with a transparent grid used for estimating the proportional volumes of different gross placental components by the point-counting method. Arrow 'a' indicates a point on the parenchyma, while arrows 'b' and 'c' indicate points on non-parenchymal components (The chorionic plate and an infarct, respectively)

which was a significant difference from the Control group in all evaluated variables. The bar diagram of proportionate volumes in Figure 2 likewise shows these disparate trends amongst groups. The Gestational Diabetes Mellitus group showed a decreased proportional amount of non-parenchymal tissue and significantly larger proportional and absolute parenchymal volumes when directly compared to the Eclampsia group.

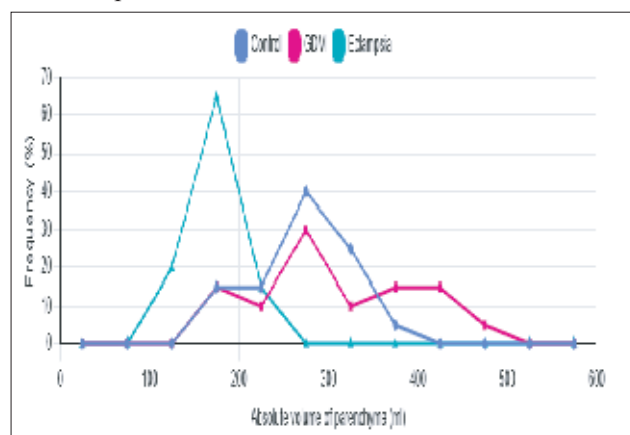
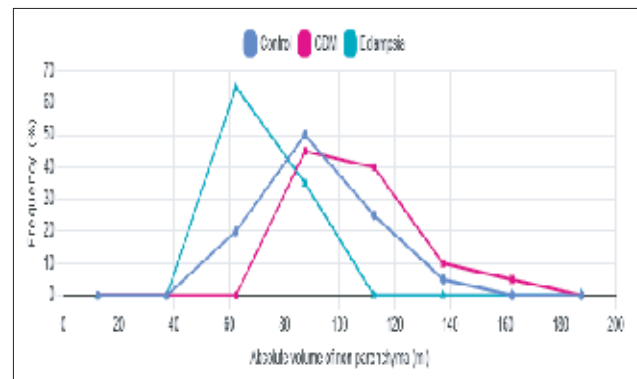
The frequency curves in Figures 3 and 4 indicate how the absolute parenchymal and non-parenchymal placental component volumes differ by study group. For Gestational Diabetes Mellitus both curves are located along larger volume ranges in comparison to the Control group. [Indicating that total absolute placental component volumes are]

Table I Volumes of parenchymal and non-parenchymal components of placenta (n=60)

Variable	Control	GDM	Eclampsia	Significant Differences
Parenchyma (%)	74.35±2.63	75.55±2.41	70.74±3.90	C vs E; G vs E
Parenchyma (ml)	280.23±67.81	312.26±70.49	167.54±28.60	C vs E; G vs E
Non-parenchyma (%)	25.65±2.62	24.45±2.46	29.26±3.76	C vs E; G vs E
Non-parenchyma (ml)	95.47±19.23	100.72±23.30	69.80±13.64	C vs E; G vs E

**Figure 2** The proportional distribution of parenchyma and non-parenchyma (n=60)

Moreover, the distributions within this group are comparatively wider, suggesting greater variability based on case types. In contrast, the parenchymal and non-parenchymal frequency curves of the Eclampsia group move almost exclusively to the left and the distributions are smaller, and the dispersion of the distributions is less. These results indicate reduced inter-individual variance and smaller absolute units for both sustaining and functional placental components. The Control group is characterized by modest volume ranges and variability, sitting in a middle position for both components.

**Figure 3** Frequency curves of absolute parenchymal volume in different groups**Figure 4** Frequency curves of absolute non-parenchymal volume in different groups (n=60)

To enhance graphical clarity while maintaining the original data patterns, frequency curves were rebuilt from the original observed class wise distributions.

Discussion

This study found different patterns of placental remodelling in gestational diabetes mellitus and eclampsia, encompassing both parenchymal and non-parenchymal components. Gestational diabetes mellitus leads to increased placental parenchyma while preserving proportionate architecture, indicating an adaptive response to retain enough maternal-fetal interaction.^{3,8} Increased non-parenchymal volume without corresponding expansion suggests compensatory growth rather than severe distortion of placental anatomy. These findings are consistent with past histomorphometric research.

Diabetic pregnancies are characterized by villous proliferation, enlargement of the intervillous gap and increased villous surface area.^{4,7,16} Stereological investigations show that these structural alterations sustain placental efficiency and are a natural adaptation to changing metabolic load, not excessive or abnormal growth.^{11,12}

In this study, placentae from eclamptic pregnancies showed a considerable decrease in absolute parenchymal volume and a corresponding rise in non-parenchymal components, indicating loss of functioning villous tissue. These findings align with previous reports of placental ischemia, infarction, and stromal fibrosis linked to eclampsia and severe hypertension diseases during pregnancy.^{5,6,17} Both parenchymal and non-parenchymal frequency distributions show a concordant leftward shift and reduced dispersion, indicating that placental expansion is restricted globally rather than selectively affecting individual components.

Comparable observations have been reported in both regional and international studies, where reduced villous volume and expansion of non-parenchymal structures were attributed to uteroplacental insufficiency and maternal vascular malperfusion.^{9,10,13} More recent morphometric investigations have reinforced these conclusions, demonstrating that eclampsia-associated placental injury reflects structural loss and impaired perfusion rather than adaptive remodeling.¹⁴

Although methodological emphasis in placental morphometry has evolved toward refined stereological and microstructural analyses since the early 2000s, these approaches continue to evaluate the same biological compartments examined in gross volumetric studies. The results of this study remain physiologically coherent and compatible with subsequent peer-reviewed evidence when analysed within this modern pathophysiological framework. All of these findings lend credence to the idea that while eclampsia causes pathological placental impairment with possible negative effects on fetal outcome, gestational diabetes mellitus causes mostly adaptive placental responses.

Limitations

The results of this study may not be as broadly applicable as they could be due to the relatively very small sample size that was collected from two institutions. Without the use of supplementary micro-stereological or imaging-based approaches, volumetric assessment relied on gross stereological procedures. Furthermore, variables that might have affected placental morphology, such as the degree of glycemic control in gestational diabetes mellitus and the severity of hypertensive illness in eclampsia, could not be stratified.

Conclusion

In gestational diabetes and eclampsia, distinct placental changes are revealed by quantitative volumetric analysis. Gestational diabetes is characterized by adaptive development of placental components, whereas eclampsia has a significant loss of functional parenchyma. The examination of parenchymal and non-parenchymal volumes sheds light on placental disease and its possible negative consequences for the health of the fetus.

The validity of the initial findings is confirmed by the incorporation of new peer-reviewed morphometric studies.

Recommendations

To confirm and expand on these results, more research with larger, multicentred population is advised. The structural and functional interactions of placental tissue may be better understood by incorporating contemporary stereological and placental imaging tools. Additionally, longitudinal studies that look at placental remodelling in connection to prenatal outcome and disease severity are recommended.

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Disclosure

The authors declared no conflict of interest.

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