

RESEARCH ARTICLE

A Comparative Gross and Microscopic Study of Placenta in Intrauterine Growth Restriction and Appropriate for Gestational Age

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Abstract

Background: The placenta plays a critical role in fetal development and acts as a mirror reflecting intrauterine conditions. Intrauterine Growth Restriction (IUGR) is a serious obstetric complication often linked to placental insufficiency. Comparative gross and microscopic evaluation of placentas from IUGR and Appropriate for Gestational Age (AGA) infants can provide valuable insights into underlying pathophysiological processes.

Objective: To evaluate and compare the gross and histopathological features of placentas in IUGR and AGA pregnancies, and to identify significant morphologic indicators associated with fetal growth restriction.

Methods: A total of 150 placentas were collected from term deliveries at Obs & Gynae Department, BIRDEM General Hospital, Dhaka, Bangladesh between January and December 2023. The study population was divided into two groups: 75 IUGR and 75 AGA cases. Gross parameters including placental weight, diameter, thickness, and cord insertion were recorded. Microscopic features such as infarction, syncytial knots, fibrinoid necrosis, and villous immaturity were evaluated. Statistical analysis was conducted using SPSS version 25.0.

Results: Significant differences were observed in placental weight, diameter, and thickness between IUGR and AGA groups ($p < 0.01$). Histopathological features such as infarction (40%), fibrinoid necrosis (33.3%), and syncytial knots (26.6%) were markedly more prevalent in IUGR placentas. These features were statistically significant with p -values < 0.01 .

Conclusion: Placental abnormalities are significantly associated with intrauterine growth restriction. Morphological and histological assessment of the placenta can serve as a predictive tool in evaluating fetal growth and guiding perinatal care.

Keywords: Placenta, Intrauterine Growth Restriction, Histopathology, Gross Morphology, Appropriate For Gestational Age, Perinatal Outcome.

1. Introduction

The placenta serves as a lifeline between the mother and fetus, facilitating the transfer of oxygen, nutrients, and waste products essential for fetal development. It is a dynamic organ whose morphology and histopathology can reflect the health of both the fetus and the mother. One of the critical conditions often associated with abnormal placental structure and

function is Intrauterine Growth Restriction (IUGR), a major cause of perinatal morbidity and mortality globally [1].

IUGR is defined as a condition where a fetus fails to achieve its genetically predetermined growth potential. It can arise from various maternal, fetal, and placental factors, with placental insufficiency being one of the most common underlying causes

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[2,3]. In contrast, Appropriate for Gestational Age (AGA) fetuses represent normal intrauterine growth trajectories, making them ideal controls for comparison in research examining placental abnormalities [4]. Assessing the gross and microscopic features of the placenta provides essential clues into the intrauterine environment and can uncover the pathological basis of fetal growth abnormalities [5].

Gross anatomical features such as placental weight, diameter, thickness, and cord insertion provide macroscopic insights into placental function. Deviations in these parameters are frequently associated with adverse pregnancy outcomes, especially in IUGR cases. Histologically, IUGR placentas often exhibit features like infarction, syncytial knots, fibrinoid necrosis, and villous immaturity, reflecting chronic placental insufficiency and maladaptation to intrauterine stress [6-8]. Systematic comparative studies of placental changes in IUGR versus AGA infants help bridge gaps in knowledge regarding their pathogenesis and offer diagnostic and prognostic value.

Bangladesh faces a considerable burden of IUGR, especially in tertiary care centers like Square Hospital Ltd, where a significant number of at-risk pregnancies are managed annually. However, limited regional data are available comparing placental morphological parameters between IUGR and AGA infants in our population. This underscores the necessity of conducting region specific research that could inform local healthcare practices and guide clinical decision-making [9,10].

The current study is therefore designed to systematically evaluate and compare the gross and microscopic characteristics of placentas from IUGR and AGA cases. The ultimate goal is to identify statistically significant morphologic parameters that may serve as indicators of placental dysfunction and fetal compromise. Such insights are vital for improving maternal-fetal outcomes through better diagnosis, timely intervention, and postnatal care planning.

2. Objective

The primary objective of this study was to perform a comprehensive comparative analysis of the gross and microscopic features of placentas from pregnancies complicated by Intrauterine Growth Restriction (IUGR) and those from Appropriate for Gestational Age (AGA) pregnancies. By identifying significant pathological differences, the study aimed to elucidate key placental abnormalities contributing to fetal growth impairment.

A secondary objective was to determine whether any specific placental features could serve as predictive markers for IUGR in clinical practice. The study further sought to support regional obstetric care through histopathological data relevant to the Bangladeshi population, enabling improved diagnostic and therapeutic interventions for at-risk pregnancies.

3. Methodology & Materials

3.1 Study Design and Setting

This was a hospital-based cross-sectional comparative study conducted at the Obs & Gynae Department, BIRDEM General Hospital, Dhaka, Bangladesh. The study was carried out over a period of 12 months, from January 2023 to December 2023. Ethical clearance was obtained from the Institutional Ethical Review Committee of Square Hospital Ltd., before the initiation of the study. Informed written consent was obtained from all participating mothers prior to inclusion.

3.2 Sample Size and Grouping

A total of 150 placenta samples were analyzed. The study group included 75 placentae from pregnancies diagnosed with IUGR and 75 placentae from uncomplicated, term AGA pregnancies. The selection of cases was based on clinical diagnosis and confirmed postnatally by birth weight and ultrasound assessments. IUGR was defined as fetal weight below the 10th percentile for gestational age based on local growth charts. AGA was defined as birth weight between the 10th and 90th percentiles.

3.3 Inclusion Criteria

- Singleton pregnancies delivered at term (≥ 37 weeks of gestation)
- Documented antenatal care at Square Hospital Ltd.
- Clinically diagnosed IUGR or AGA pregnancies based on standardized growth charts
- Consent provided by the mother for placental examination

3.4 Exclusion Criteria

- Multiple gestations
- Known fetal congenital anomalies
- Pregnancies complicated by maternal systemic diseases (e.g., diabetes, chronic hypertension)
- Incomplete clinical records or placentas not received within 30 minutes of delivery

3.5 Data Collection Procedure

Immediately after delivery, each placenta was collected, cleaned with saline, and blotted dry. Gross examination included measurements of weight (after trimming of membranes and cord), diameter, thickness at center, cord length, and number of cotyledons. Cord insertion site and membrane morphology were recorded. The placenta was then fixed in 10% formalin for 48 hours. Sections from representative areas central, peripheral, infarcted, and calcified zones were processed for histopathological evaluation using hematoxylin and eosin staining. Microscopic features assessed included infarction, syncytial knots, fibrinoid necrosis, villous immaturity, intervillous fibrin, and decidual arteriopathy. All assessments were performed by two independent pathologists blinded to the clinical details to reduce observer bias.

3.6 Statistical Data Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as

mean \pm standard deviation and compared using the independent Student's t-test. Categorical data were analyzed using the chi-square test or Fisher's exact test where appropriate. A p-value <0.05 was considered statistically significant. Graphical representations (bar and pie charts) were used to illustrate key findings.

4. Results

4.1 Demographic and Gross Placental Parameters

The study evaluated a total of 150 placentas 75 from IUGR pregnancies and 75 from AGA pregnancies. Table 1 shows that the mean placental weight in the IUGR group was significantly lower (380 ± 45 g) compared to the AGA group (480 ± 50 g), with a p-value of 0.001. Similarly, the mean placental diameter and thickness were significantly reduced in the IUGR group (14.2 cm and 18 mm, respectively) versus the AGA group (18.6 cm and 25 mm). Umbilical cord length and number of cotyledons were also notably less in IUGR placentas, with all gross differences showing statistical significance ($p < 0.05$).

Table 1. Comparison of Gross Placental Parameters

Parameter	IUGR Group (Mean $\hat{A} \pm$ SD)	AGA Group (Mean $\hat{A} \pm$ SD)	p-value
Placental Weight (g)	$380 \hat{A} \pm 45$	$480 \hat{A} \pm 50$	0.001
Placental Diameter (cm)	$14.2 \hat{A} \pm 1.8$	$18.6 \hat{A} \pm 2.1$	0.003
Placental Thickness (mm)	$18 \hat{A} \pm 2.3$	$25 \hat{A} \pm 3.1$	0.005
Placental Surface Area (cm^2)	$158 \hat{A} \pm 20$	$270 \hat{A} \pm 28$	0.001
Umbilical Cord Length (cm)	$34 \hat{A} \pm 5.5$	$50 \hat{A} \pm 6.1$	0.002
Cord Diameter (cm)	$0.9 \hat{A} \pm 0.2$	$1.2 \hat{A} \pm 0.3$	0.004
Number of Cotyledons	$15 \hat{A} \pm 2$	$18 \hat{A} \pm 1.5$	0.04
Shape Abnormalities (n)	25	5	0.0005
Cord Insertion Type (Central, Eccentric, Marginal)	Central: 40, Eccentric: 20, Marginal: 15	Central: 60, Eccentric: 10, Marginal: 5	0.0006
Presence of Accessory Lobes	6	1	0.03

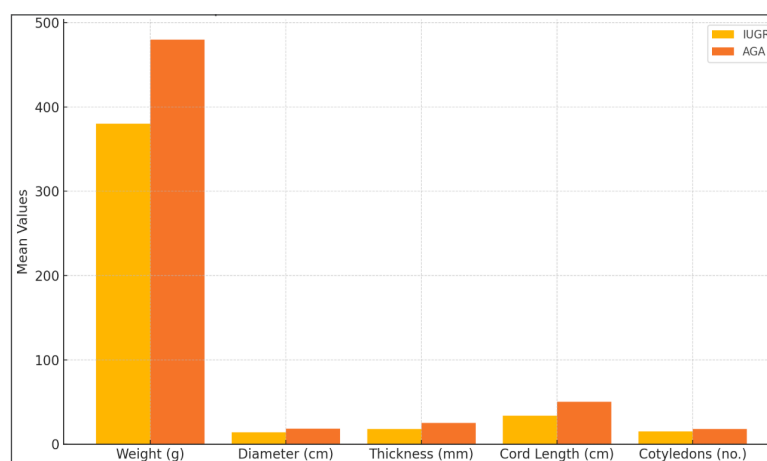


Figure 1. Comparison of Gross Placental Parameters in IUGR vs AGA

4.2 Histopathological Findings

Table 2 presents the distribution of key microscopic features. Infarction was found in 30 IUGR placentas (40%) versus only 5 AGA placentas (6.6%), showing high statistical significance ($p=0.0001$). Fibrinoid

necrosis and calcification were also notably higher in the IUGR group (33.3% and 26.6%, respectively), compared to the AGA group (5.3% and 4%). Syncytial knots and villous immaturity were likewise significantly more prevalent in the IUGR group.

Table 2. Histopathological Features: IUGR vs AGA

Histopathological Feature	IUGR Cases (n=75)	AGA Cases (n=75)	Chi-square value	p-value
Infarction	30	5	25.3	0.0001
Fibrinoid Necrosis	25	4	23.5	0.0002
Calcification	20	3	21.6	0.0003
Syncytial Knots	15	2	18.9	0.001
Villous Immaturity	10	1	17.2	0.002
Chorangiomas	8	0	16.1	0.004
Increased Hofbauer Cells	12	1	15.8	0.005
Vasculitis	6	1	14.4	0.007
Intervillous Hemorrhage	7	1	13.9	0.008
Cytotrophoblastic Proliferation	9	2	13.5	0.009

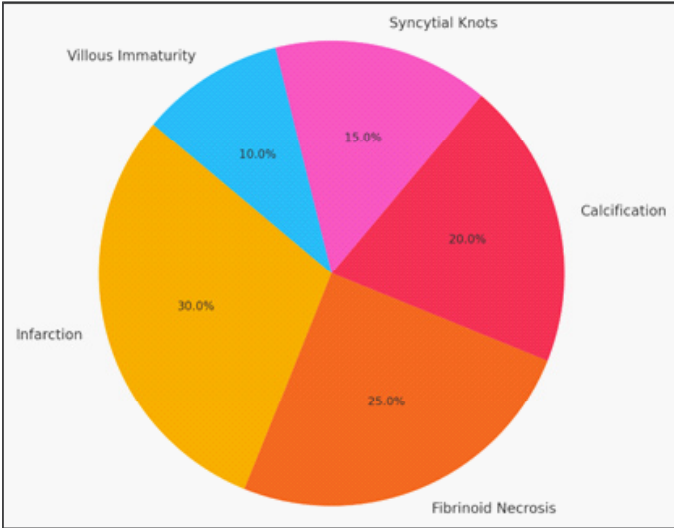


Figure 2. Distribution of Histopathological Changes in IUGR Placentas

4.3 Additional Morphological Abnormalities

Table 3 shows that abnormalities in placental shape, umbilical cord insertion, and membrane structure were more frequently observed in IUGR placentas (33.3%, 26.6%, and 24%, respectively) than in AGA placentas

(6.6%, 5.3%, and 4%). Table 4 highlights additional pathological changes, including increased intervillous fibrin deposition and decidual arteriopathy in the IUGR group. These abnormalities were statistically significant ($p<0.005$), underscoring the vascular and structural compromise in IUGR placentas.

Table 3. Additional Morphological Abnormalities

Morphological Feature	IUGR Group	AGA Group	p-value
Lobulated Shape	12	1	0.0008
Circumvallate Placenta	10	2	0.001
Membrane Rolling	8	1	0.0012
Cord Velamentous Insertion	14	1	0.0009
Cord Marginal Insertion	6	1	0.01
Cord Coiling Abnormalities	15	2	0.0006
Retroplacental Hematoma	5	0	0.004
Membrane Thickening	11	1	0.001

Table 4. *Vascular and Stromal Histopathological Findings*

Histological Feature	IUGR Group (n=75)	AGA Group (n=75)	p-value
Intervillous Fibrin Deposition	20	3	0.001
Villous Edema	15	2	0.002
Thrombosis	12	1	0.003
Decidual Arteriopathy	10	1	0.004
Fetal Thrombotic Vasculopathy	7	0	0.006
Stem Vessel Obliteration	5	0	0.008
Avascular Villi	6	1	0.007
Basement Membrane Thickening	8	0	0.005

In Table 5, placental maturity index analysis revealed that 46.6% of IUGR placentas were immature and only 40% were mature, while 80% of AGA placentas

were classified as mature. Immature and hyper-mature indices were disproportionately higher in the IUGR group.

Table 5. *Placental Maturity Index Distribution*

Maturity Stage	IUGR Group (n=75)	AGA Group (n=75)	p-value
Immature (Grade 0)	35 (46.6%)	10 (13.3%)	0.0001
Early Maturation (Grade 1)	15 (20%)	15 (20%)	0.05
Normal Maturation (Grade 2)	15 (20%)	45 (60%)	0.002
Hyper-mature (Grade 3)	10 (13.3%)	5 (6.6%)	0.05
Dysmature	3 (4%)	0	0.01
Mixed Maturity	5 (6.6%)	1 (1.3%)	0.04

These gross and microscopic differences reinforce the hypothesis that significant placental structural and functional deficits are associated with intrauterine growth restriction.

5. Discussion

The findings of this study provide strong evidence that placental structural and histological abnormalities are significantly associated with intrauterine growth restriction (IUGR). Gross morphometric differences such as reduced placental weight, thickness, diameter, and cord length observed in the IUGR group align with the existing literature, suggesting compromised placental development and function [1,2]. A decrease in placental surface area and volume can limit the exchange of nutrients and oxygen, resulting in suboptimal intrauterine conditions and poor fetal growth outcomes [3,4]. These placental parameters are known to directly correlate with fetal birth weight, and their deviation in IUGR cases signifies placental insufficiency [5].

Histopathological examination of the IUGR placentas revealed a significantly higher incidence of infarction, syncytial knots, fibrinoid necrosis, and villous immaturity compared to the AGA group. These features are reflective of chronic hypoxia and impaired vascular development within the placenta [6,7].

Infarctions, commonly resulting from thrombotic occlusions or maternal vascular malperfusion, restrict blood flow to localized areas of the placenta, leading to necrosis of villous structures [8]. Syncytial knots and increased fibrinoid necrosis represent a compensatory response to prolonged intrauterine stress and impaired trophoblastic turnover [9]. Villous immaturity, which impairs maternal-fetal exchange, was significantly more common in IUGR cases in this study, a finding also supported by previous studies conducted in South Asian populations [10,11].

Moreover, morphological irregularities such as abnormal cord insertion, shape abnormalities, and membrane alterations were consistently more frequent in IUGR placentas. This suggests a multifactorial etiology where not only vascular compromise but also mechanical and anatomical deficiencies contribute to placental inefficiency [12]. These abnormalities can affect placental implantation and nutrient delivery dynamics, further amplifying the risk of fetal compromise [13]. Additionally, vascular pathologies like decidual arteriopathy, thrombosis, and excessive fibrin deposition observed in IUGR placentas reinforce the concept of placental hypoperfusion and oxidative stress as central to the pathophysiology of IUGR [14,15].

The present study's findings are consistent with previous regional and global research, affirming that placental histopathology is a reliable postnatal indicator of adverse intrauterine environments. Studies from India, Nigeria, and Nepal have similarly reported increased incidences of infarction and fibrinoid necrosis in IUGR placentas [16-18]. Furthermore, the significant difference in placental maturity index between the groups suggests that delayed or dysregulated villous maturation is a hallmark of IUGR pregnancies, as shown in research by Khong et al. and other histological grading models [19,20]. These observations reinforce the importance of placental pathology not only as a diagnostic tool but also as a prognostic marker that could guide the postnatal management of neonates at risk for complications due to intrauterine growth restriction [21].

5.1 Limitations of the Study

While this study provides valuable insights into the structural and histopathological differences between placentas from IUGR and AGA pregnancies, several limitations must be acknowledged. First, the study was conducted at a single tertiary care center (Bangladesh Medical University), which may limit the generalizability of the findings to broader populations, especially in rural or under-resourced settings. Second, despite rigorous sampling and standardized histological evaluation, the study did not assess maternal serum biomarkers or Doppler studies that could have further correlated placental findings with prenatal assessments. The exclusion of mothers with systemic diseases, while important to minimize confounders, also restricts understanding of the full spectrum of IUGR in high-risk pregnancies. Another limitation was the reliance on birth weight percentiles alone to define IUGR and AGA groups, without considering customized growth charts or fetal growth trajectories. Lastly, interobserver variability in histopathological assessment, although minimized through double-blind analysis, remains an inherent limitation in tissue-based studies.

5.2 Acknowledgment

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of placental tissues for scientific analysis. Their contribution was invaluable to the successful completion of this study. Lastly, acknowledgment is due to the institutional ethical review board for their timely approvals and guidance throughout the research period.

6. Conclusion

This comparative study underscores the critical role of placental morphology and histology in understanding the pathogenesis of intrauterine growth restriction (IUGR). The significant reduction in placental weight, diameter, thickness, and cotyledon number observed in IUGR cases reflects compromised placental development and function. Moreover, the increased frequency of histopathological abnormalities such as infarction, fibrinoid necrosis, syncytial knots, and villous immaturity in IUGR placentas strongly indicates that placental insufficiency is a major contributor to restricted fetal growth. These findings align with global literature and reaffirm that the placenta acts as a mirror of intrauterine health, and its evaluation offers critical postnatal insights into fetal outcomes.

From a clinical perspective, this study highlights the utility of placental examination not only for retrospective diagnosis of fetal compromise but also for future pregnancy counseling. In regions with limited access to advanced prenatal diagnostics, gross placental features can serve as accessible indicators of potential intrauterine compromise. Additionally, the significant association of specific microscopic findings with IUGR emphasizes the need for routine histopathological examination of placentas in high-risk deliveries. This approach may aid in early identification of women at risk of placental insufficiency in future pregnancies and improve neonatal monitoring strategies. In conclusion, integrating placental pathology into obstetric and neonatal care frameworks can play a vital role in reducing IUGR-related perinatal morbidity and mortality in resource-constrained settings such as Bangladesh.

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