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Histomorphological and morphometric analysis of placenta in complicated and uncomplicated pregnancies: A cross-sectional study

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ABSTRACT

Background: The placenta is a vital organ that reflects the intrauterine environment and plays a crucial role in fetal growth and development. Histomorphological and morphometric alterations of the placenta are frequently associated with obstetric complications such as hypertensive disorders, gestational diabetes mellitus, maternal anemia, and intrauterine growth restriction, and may influence perinatal outcomes.
Aim: To evaluate the histomorphological and morphometric parameters of the placenta in complicated and uncomplicated pregnancies and to correlate these findings with fetomaternal outcomes.
Materials and Methods: A hospital-based cross-sectional observational study was conducted over one year (April 2024–March 2025) in the Departments of Pathology and Obstetrics & Gynaecology at Rohilkhand Medical College and Hospital, Bareilly. Seventy-five freshly delivered placentas were examined, comprising 34 from uncomplicated pregnancies and 41 from complicated pregnancies. Gross morphometric parameters including placental weight, diameter, thickness, cord characteristics, and fetoplacental ratio were recorded. Histopathological examination assessed syncytial knots, fibrinoid necrosis, villous infarction, hyalinization, thrombosis, and calcification. Data were analyzed using Student's *t*-test, Chi-square test, and correlation analysis, with $p<0.05$ considered statistically significant.
Results: Complicated pregnancies demonstrated significantly lower placental weight (379.5 ± 128.6 g vs. 432.8 ± 118.3 g; $p=0.046$) and a higher frequency of increased syncytial knots (53.7% vs. 23.5%; $p=0.018$), fibrinoid necrosis (70.7% vs. 32.4%; $p=0.003$), villous infarction (58.5% vs. 23.5%; $p=0.009$), and thrombosis (53.7% vs. 29.4%; $p=0.042$). Placental weight showed a significant positive correlation with birth weight ($r=0.318$, $p=0.006$) and gestational age ($r=0.285$, $p=0.015$), while exhibiting a strong negative correlation with the fetoplacental ratio ($r=-0.925$, $p<0.001$). Villous infarction and increased syncytial knots were significantly associated with fetal distress.
Conclusion: Complicated pregnancies are associated with significant gross and microscopic placental alterations indicative of placental insufficiency. Histomorphological and morphometric evaluation of the placenta provides valuable insights into the pathophysiology of pregnancy complications and serves as an important adjunct for assessing adverse fetal outcomes and guiding future obstetric management.

Keywords: *Placenta; Histomorphology; Morphometry; Complicated pregnancy; Syncytial knots; Placental infarction; Maternal anemia; Hypertensive disorders.*

INTRODUCTION

The placenta is a transient yet vital organ that plays a central role in sustaining fetal life throughout pregnancy. Structurally complex and functionally dynamic, it represents the interface between maternal and fetal circulatory systems and performs

essential functions including nutrient transfer, gas exchange, immunological protection, and endocrine regulation. Because of these indispensable roles, placental health has a direct and profound impact on the outcome of pregnancy and fetal development.[1,2] Any deviation from normal placental growth or maturation may result in compromised fetal well-being, leading to complications such as intrauterine growth restriction (IUGR), preeclampsia, preterm birth, stillbirth, or neonatal morbidity.[3,4] Therefore, the placenta is widely regarded as the "diary of intrauterine life," providing valuable information about maternal and fetal conditions during pregnancy.[5,6]

Placental development begins with implantation and progresses through intricate phases of trophoblastic differentiation, villous branching, formation of the fetomaternal circulation, and spiral artery remodeling.[7]Normal pregnancy is characterized by efficient trophoblastic invasion and adequate uteroplacental perfusion, ensuring proper villous maturation and vascularization. Histomorphological features such as villous branching, syncytial knot formation, stromal cellularity, and vascular proliferation change progressively throughout gestation and reflect placental adaptation to increasing fetal demands.[8] Concurrently, morphometric parameters—including placental weight, diameter, thickness, and surface area—serve as quantitative indicators of placental development and functional capacity.[9.10.11]

Pregnancies are broadly classified as uncomplicated or complicated, depending on maternal and fetal factors.
In uncomplicated pregnancies, placental development is well-orchestrated to adapt optimally to fetal needs. However, in complicated pregnancies, such as those affected by preeclampsia[12,13,14], gestational diabetes mellitus (GDM)[15,16,17], maternal anemia[18], and intrauterine growth restriction (IUGR)[19] this adaptability is often compromised.[10]These conditions result in distinct histomorphological and morphometric alterations that can serve as biomarkers of disease severity and fetal compromise.

Placental examination—both gross and microscopic—is a valuable tool in understanding these pathological processes. Gross features such as placental weight, shape, cord insertion, calcifications, and infarctions provide initial clues about placental health. Detailed histomorphological assessment allows for the detection of microscopic abnormalities including villous immaturity, syncytial knot prominence, fibrinoid deposition, vascular lesions, and inflammatory changes.[20,21] Morphometric analysis adds further depth by quantitatively assessing parameters such as placental weight, placental diameter, placental thickness, feto placental ratio, placental co efficient, umbilical cord parameter, providing objective insights into placental adaptability.[22]

In low-resource settings where advanced antenatal diagnostic tools may be unavailable, histomorphological and morphometric placental assessments can serve as valuable retrospective indicators of intrauterine health, guiding the management of future pregnancies. [20]Furthermore, the placenta is increasingly being recognized as a "record of pregnancy" and a predictor of long-term health. According to the Developmental Origins of Health and Disease (DOHaD) hypothesis, structural changes in the placenta may link intrauterine exposures to future cardiovascular, metabolic, and neurodevelopmental disorders.[20]

The study done aims to provide knowledge into various diseases that are complicating the pregnancy through placental morphology. It also provides with the knowledge of the causes of fetal demise and also helps in taking precautionary measures for future pregnancies. As very few studies are available from this region assessing both histomorphological and morphometrical parameters together and hence this study is undertaken.

AIM AND OBJECTIVES

Aim:

To study the histomorphological and morphometric parameters of placenta in complicated and uncomplicated pregnancies.

Objectives:

1. To determine the histomorphological features of placenta.
2. To assess the morphometric parameters of placenta.
3. To document maternal & fetal parameters with respect to parity (primigravida/ multigravida), mode of labour (vaginal/cesarean), gestational age, complicating factors like (anemia, HTN, DM), sex of the baby, weight of the baby, maturity of the baby (pre term/ full term), visible anomalies in the baby.
4. To correlate histomorphological features and morphometric parameters with feto –maternal parameters.

MATERIAL AND METHODS

This cross-sectional observational study was conducted jointly in the Departments of Pathology and Obstetrics & Gynecology, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, over a period of one year (April 2024-March 2025). The study included freshly delivered placentas received in the Department of Pathology for detailed gross and histopathological examination. Clinical, radiological and obstetric data of the mothers was collected from departmental records and labour room case sheets after obtaining informed consent.

The sample size was determined using the standard formula for cross-sectional studies. A Total of 75 placenta cases were included in the study.[1]
These were divided into two groups:

- **Group I – Uncomplicated pregnancies**: placentae from normotensive, non-anaemic, non-diabetic mothers with no other obstetric or medical complications.
- **Group II – Complicated pregnancies**: placentae from mothers with one or more of the following complications:
    - Pregnancy induced hypertension / pre-eclampsia / eclampsia
    - Gestational diabetes mellitus
    - Maternal anaemia (as per WHO criteria)
    - Intrauterine growth restriction (IUGR)
    - Other high-risk conditions as defined by the obstetrician

The exact distribution of cases in each group was based on the number of eligible deliveries during the study period.
Relevant maternal and neonate details were recorded in a pre-designed proforma, including Maternal age, gravidity and parity, Gestational age (by LMP / USG), Presence and type of complication (PIH, GDM, anemia, IUGR, etc.), Mode of delivery, Baby's sex, birth weight

The collection and grossing of Placentae was done as per standard protocol.
All measurements were performed on fresh placentae using standardised techniques. Gross morphometric measurements was done under following headings-

Placental weight (g), Placental diameter (cm), Placental thickness (cm), Number of cotyledons, Umbilical cord Site of insertion (central, eccentric, marginal, velamentous), Fetoplacental Ratio: Calculated as: Fetal Weight (g) / Placental Weight (g) were noted. Microscopic examination was done under the following headings – Calcification, Chorangiosis, Syncytial knots, Fibrinoid necrosis, Hyalinization, and Thrombus

Statistical Analysis:
Coding, entry of the data, its clearing and compiling was done in excel sheet. The data was imported in SPSS (Statistical Package for Social Science) version 23.0 software where means and standard deviations were calculated.
Depending on the distribution and type of data, data was expressed in mean value and was analysed by t test and proportions through chi square test. A p value of <0.05 was considered statistically significant.

Inclusion Criteria:

- Singleton pregnancies delivered between 28 and 38 weeks of gestation.
- Primigravida and multigravida mothers delivering either vaginally or by caesarean section.
- Fresh, intact placentas available within 2 hours of delivery and suitable for histopathological examination.
- Mothers who provide written informed consent for participation.

Exclusion Criteria:

- Placentas from multiple (twin or higher) pregnancies.
- Mothers with non-obstetric systemic disorders (e.g., chronic renal disease, hepatic disorders, thyroid dysfunctions, autoimmune disease).
- Malformed, autolyzed, or incomplete placental specimens unsuitable for morphological study.
- Mothers who did not provide consent for participation.

OBSERVATION AND RESULTS

Table 1: Maternal and Fetal Characteristics of the Study Population (N=75)

Characteristic	Total (N=75)	Uncomplicated Pregnancies (n=34)	Complicated Pregnancies (n=41)	p-value
Maternal Age (years), Mean ± SD	25.4 ± 3.8	24.9 ± 3.5	25.8 ± 4.0	0.289
Parity, n (%)				
Primigravida	21 (28.0)	11 (32.4)	10 (24.4)	0.421
Multigravida	54 (72.0)	23 (67.6)	31 (75.6)	
Mode of Delivery, n (%)				

Vaginal	45 (60.0)	23 (67.6)	22 (53.7)	0.187
Cesarean Section	30 (40.0)	11 (32.4)	19 (46.3)	
Gestational Age, n (%)				
Pre-term (28-36 wks)	7 (9.3)	1 (2.9)	6 (14.6)	**0.032**
Full-term (≥37 wks)	68 (90.7)	33 (97.1)	35 (85.4)	
Type of Complication, n (%)				-
Anemia	20 (26.7)	-	20 (48.8)	
Hypertensive Disorders	8 (10.7)	-	8 (19.5)	
Gestational Diabetes	2 (2.7)	-	2 (4.9)	
Fetal Sex, n (%)				
Male	41 (54.7)	18 (52.9)	23 (56.1)	0.654
Female	34 (45.3)	16 (47.1)	18 (43.9)	
Birth Weight (kg), Mean ± SD	2.49 ± 0.48	2.58 ± 0.31	2.41 ± 0.58	0.132
Fetal Distress, n (%)	24 (32.0)	7 (20.6)	17 (41.5)	**0.049**

CLINICAL PARAMETERS IN UNCOMPLICATED VS COMPLICATED PREGNANCIES:

The above table illustrates that out of the total 75 study participants, the mean maternal age was 25.4 ± 3.8 years, with 24.9 ± 3.5 years in the uncomplicated group and 25.8 ± 4.0 years in the complicated group. Primigravida constituted 28% overall, including 32.4% in the uncomplicated group and 24.4% in the complicated group, while multigravidas accounted for 72% overall, comprising 67.6% in the uncomplicated and 75.6% in the complicated pregnancies. Vaginal deliveries made up 60% of all cases with 67.6% in the uncomplicated group and 53.7% in the complicated group, whereas cesarean sections accounted for 40% overall with 32.4% among uncomplicated and 46.3% among complicated cases. Pre-term births constituted 9.3% of all participants, including 2.9% in the uncomplicated and 14.6% in the complicated group, while full-term births accounted for 90.7% overall with 97.1% among uncomplicated and 85.4% among complicated pregnancies. Among all newborns, 54.7% were males and 45.3% females, with the uncomplicated group having 52.9% males and 47.1% females and the complicated group having 56.1% males and 43.9% females. The mean birth weight was 2.49 ± 0.48 kg, including 2.58 ± 0.31 kg in the uncomplicated group and 2.41 ± 0.58 kg in the complicated group, and fetal distress occurred in 32% of all cases, comprising 20.6% in the uncomplicated group and 41.5% in the complicated group.

Table 2: Gross Morphometric Parameters of the Placenta

Parameter	**Total** (N=75) Mean ± SD	**Uncomplicated** (n=34) Mean ± SD	**Complicated** (n=41) Mean ± SD	**p-value**
Placental Weight (grams)	403.7 ± 126.4	432.8 ± 118.3	379.5 ± 128.6	**0.046**
Placental Diameter (cm)	14.9 ± 2.7	15.3 ± 1.9	14.6 ± 3.2	0.255

Placental Thickness (cm)	4.1 ± 2.9	3.6 ± 2.1	4.5 ± 3.4	0.178
Feto-Placental Ratio	0.166 ± 0.053	0.154 ± 0.045	0.176 ± 0.058	0.061
Cord Length (cm)	16.5 ± 3.8	17.1 ± 3.5	16.0 ± 4.0	0.198
Number of Cotyledons	17.1 ± 1.8	17.4 ± 1.6	16.8 ± 1.9	0.134

The above table illustrates that out of the total 75 study participants, the mean placental weight was 403.7 ± 126.4 grams, with uncomplicated pregnancies showing a higher mean placental weight of 432.8 ± 118.3 grams compared to 379.5 ± 128.6 grams in complicated pregnancies. The placental diameter measured 14.9 ± 2.7 cm overall, including 15.3 ± 1.9 cm in the uncomplicated group and 14.6 ± 3.2 cm in the complicated group. Placental thickness was 4.1 ± 2.9 cm for all cases, measured as 3.6 ± 2.1 cm in uncomplicated pregnancies and 4.5 ± 3.4 cm in complicated pregnancies. Similar findings of increased placental thickness wasnoted in antenatal ultrasounds in 3rd trimester of pregnancy. The feto-placental ratio was 0.166 ± 0.053 overall, with a value of 0.154 ± 0.045 in uncomplicated pregnancies and 0.176 ± 0.058 in complicated pregnancies. The mean cord length was 16.5 ± 3.8 cm across the study population, including 17.1 ± 3.5 cm in the uncomplicated group and 16.0 ± 4.0 cm among complicated pregnancies. The number of cotyledons averaged 17.1 ± 1.8, comprising 17.4 ± 1.6 in the uncomplicated group and 16.8 ± 1.9 in the complicated group.

Placental Shape and Cord Insertion Characteristics

Out of the total 75 study participants, 64% of placentas were discoid, including 73.5% in the uncomplicated group and 56.1% in the complicated group, while bilobed placentas accounted for 14.7% overall with 5.9% in the uncomplicated group and 22% in the complicated group. Irregular placentas constituted 16% overall, comprising 14.7% among uncomplicated pregnancies and 17.1% among complicated pregnancies, whereas the category of others represented 5.3% overall, with 5.9% in the uncomplicated group and 4.9% in the complicated group. Central cord insertion was present in 38.7% of all cases, including 41.2% in the uncomplicated and 36.6% in the complicated group, while eccentric cord insertion accounted for 61.3% overall, consisting of 58.8% in the uncomplicated and 63.4% in the complicated group. The majority of placentas, 97.3%, had three cord vessels, including 97.1% in the uncomplicated group and 97.6% in the complicated group, whereas two vessel cords were present in 2.7% overall, comprising 2.9% in the uncomplicated group and 2.4% in the complicated group.

Gross Pathological Lesions Observed in the Placenta

Out of the total 75 study participants, retroplacental hematoma was observed in 10.7% of all placentas, including 5.9% in the uncomplicated group and 14.6% in the complicated group. Gross calcification was present in 25.3% of all cases, comprising 17.6% among uncomplicated pregnancies and 31.7% among complicated pregnancies. Infarction was noted in 20% overall, with 11.8% occurring in the uncomplicated group and 26.8% in the complicated group.

Table 3: Histomorphological Features of Placental Villi and Vasculature(Microscopic)

Microscopic Feature	Total (N=75) (%)	Uncomplicated (n=34) (%)	Complicated (n=41) (%)	p-value
Syncytial Knots				
Increased (>30%)	30 (40.0)	8 (23.5)	22 (53.7)	**0.018**
Normal	45 (60.0)	26 (76.5)	19 (46.3)	
Fibrinoid Necrosis				
Present	40 (53.3)	11 (32.4)	29 (70.7)	**0.003**
Absent	35 (46.7)	23 (67.6)	12 (29.3)	
Villous Infarction				

Present	32 (42.7)	8 (23.5)	24 (58.5)	**0.009**
Absent	43 (57.3)	26 (76.5)	17 (41.5)	
Hyalinized Villi				
Present	36 (48.0)	12 (35.3)	24 (58.5)	**0.114**
Absent	39 (52.0)	22 (64.7)	17 (41.5)	
Thrombosis				
Present	32 (42.7)	10 (29.4)	22 (53.7)	**0.042**
Absent	43 (57.3)	24 (70.6)	19 (46.3)	
Dystrophic calcification				
Present	40 (53.3)	16 (47.1)	24 (58.5)	**0.451**
Absent	35 (46.7)	18 (52.9)	17 (41.5)	

The above table illustrates that out of the total 75 study participants, increased syncytial knots were present in 40% of cases, including 23.5% among uncomplicated pregnancies and 53.7% among complicated pregnancies. Fibrinoid necrosis was observed in 53.3% overall, comprising 32.4% in the uncomplicated group and 70.7% in the complicated group. Villous infarction was present in 42.7% of all placentas, with 23.5% in the uncomplicated pregnancies and 58.5% in the complicated pregnancies. Hyalinized villi were noted in 48% overall, including 35.3% in the uncomplicated group and 58.5% in the complicatedgroup.Thrombosis was present in 42.7% of placentas, including 29.4% in the uncomplicated group and 53.7% in the complicated group, while thrombosis was absent in 57.3% overall, comprising 70.6% of uncomplicated pregnancies and 46.3% of complicated pregnancies. P value was 0.042.

Dystrophic calcification was present in 53.3% of all cases, including 47.1% in the uncomplicated group and 58.5% in the complicated group, whereas calcification was absent in 46.7% overall, with 52.9% in the uncomplicated group and 41.5% in the complicated group. The P value was 0.451

Table 4: Placental Features in Hypertensive Disorders vs. Controls

Parameter	**Hypertensive Pregnancies** (n=8)	**Normotensive** Controls (n=34)	**p-value**
Placental Weight (g), Mean ± SD	387.5 ± 86.6	432.8 ± 118.3	0.315
Infarction, n (%)	**6 (75.0)**	**8 (23.5)**	**0.009**
Syncytial Knots (Increased), n (%)	**6 (75.0)**	**8 (23.5)**	**0.009**
Fibrinoid Necrosis, n (%)	7 (87.5)	11 (32.4)	**0.008**

The above table illustrates that out of the total study participants; hypertensive pregnancies had a mean placental weight of 387.5 ± 86.6 grams compared to 432.8 ± 118.3 grams in normotensive controls. Infarction was present in 75% of hypertensive placentas, whereas it was observed in 23.5% of the control group. Increased syncytial knots were noted in 75% of hypertensive cases in contrast to 23.5% in controls. Fibrinoid necrosis was present in 87.5% of hypertensive placentas compared with 32.4% in normotensive pregnancies**.**

Table5: Placental Features in Anemic Mothers vs. Controls

Parameter	**Anemic Mothers** (n=20)	**Non-Anemic Controls** (n=34)	**p-value**
Placental Weight (g), Mean ± SD	397.1 ± 150.8	432.8 ± 118.3	0.342
Placental Shape (Bilobed/Irregular), n (%)	**10 (50.0)**	**7 (20.6)**	**0.034**
Retroplacental Hematoma, n (%)	4 (20.0)	2 (5.9)	0.189

The above table illustrates that out of the total study participants; anemic mothers had a mean placental weight of 397.1 ± 150.8 grams compared to 432.8 ± 118.3 grams in non-anemic controls. Abnormal placental shape, including bilobed and irregular forms, was present in 50% of anemic mothers, whereas it was observed in 20.6% of non-anemic controls. Retroplacental hematoma occurred in 20% of anemic pregnancies and in 5.9% of non-anemic pregnancies**.**

Table 6: Correlation between Placental Weight and Fetal Parameters

Fetal Parameter	**Correlation Coefficient (r) with Placental Weight**	**p-value**
Birth Weight	0.318	**0.006**
Gestational Age	0.285	**0.015**
Feto-Placental Ratio	-0.925	**<0.001**

The above table illustrates that the correlation coefficient between placental weight and birth weight was 0.318 with a p-value of 0.006, while the correlation between placental weight and gestational age measured 0.285 with a p-value of 0.015. The correlation between placental weight and the feto-placental ratio was −0.925 with a p-value of less than 0.001.

Table 7: Association of Histological Abnormalities with Adverse Fetal Outcomes

Histological Abnormality	Fetal Distress Present (n=24) n (%)	Fetal Distress Absent (n=51) n (%)	p-value
Villous Infarction	15 (62.5)	17 (33.3)	**0.021**
Increased Syncytial Knots	14 (58.3)	16 (31.4)	**0.032**
Fibrinoid Necrosis	16 (66.7)	24 (47.1)	0.143

The above table illustrates that out of the total study participants; villous infarction was present in 62.5% of cases with fetal distress compared to 33.3% of cases without fetal distress. Increased syncytial knots were observed in 58.3% of cases with fetal distress and 31.4% of cases without fetal distress. Fibrinoid necrosis occurred in 66.7% of cases with fetal distress, whereas it was seen in 47.1% of cases without fetal distress**.**

Table 8: Placental Morphometry in Pre-term vs. Full-term Deliveries

Parameter	**Pre-term** (n=7) Mean ± SD	**Full-term** (n=68) Mean ± SD	**p-value**
Placental Weight (g)	331.4 ± 83.0	406.1 ± 126.7	**0.017**
Birth Weight (kg)	2.41 ± 0.17	2.50 ± 0.50	0.645
Feto-Placental Ratio	0.137 ± 0.031	0.167 ± 0.054	0.129
Cord Length (cm)	15.6 ± 2.1	16.5 ± 3.9	0.523
Correlation	Key Finding	Statistical Significance (p<0.05)	Clinical Implication

The above table illustrates that out of the total study participants, pre-term deliveries had a mean placental weight of 331.4 ± 83.0 grams, whereas full-term deliveries had a higher mean placental weight of 406.1 ± 126.7 grams. The mean birth weight among pre-term newborns was 2.41 ± 0.17 kg compared to 2.50 ± 0.50 kg in full-term newborns. The feto-placental ratio measured 0.137 ± 0.031 in pre-term deliveries and 0.167 ± 0.054 in full-term deliveries. The mean cord length recorded was 15.6 ± 2.1 cm in pre-term deliveries and 16.5 ± 3.9 cm in full-term deliveries.

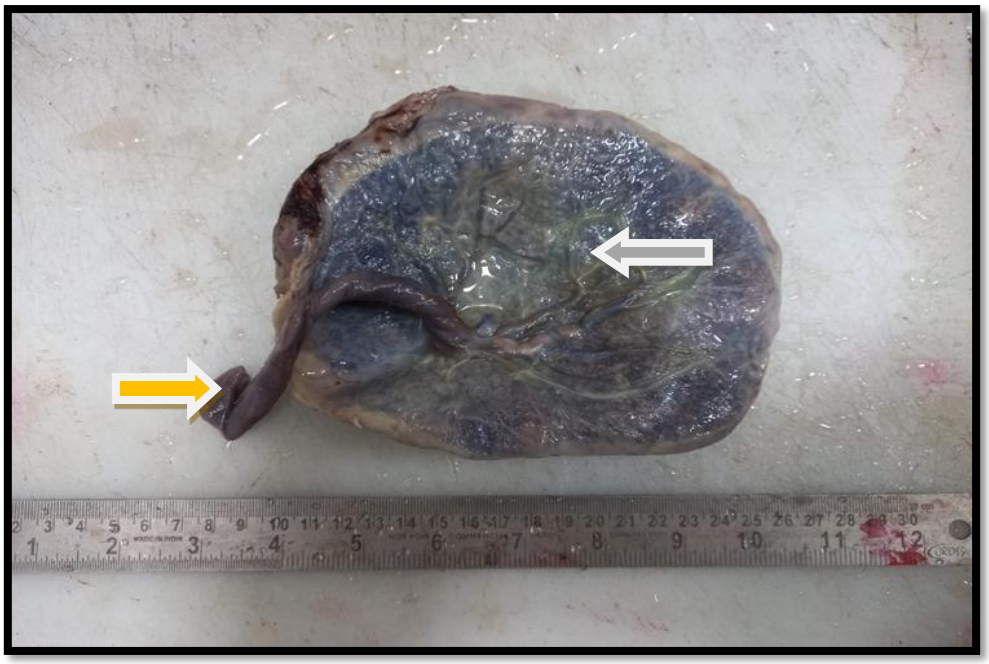

Figure 1: Gross photograph of a normal placenta having a umbilical cord showing fetal surface.

Umbilical cord ( ) and fetal surface ( ) are also seen.

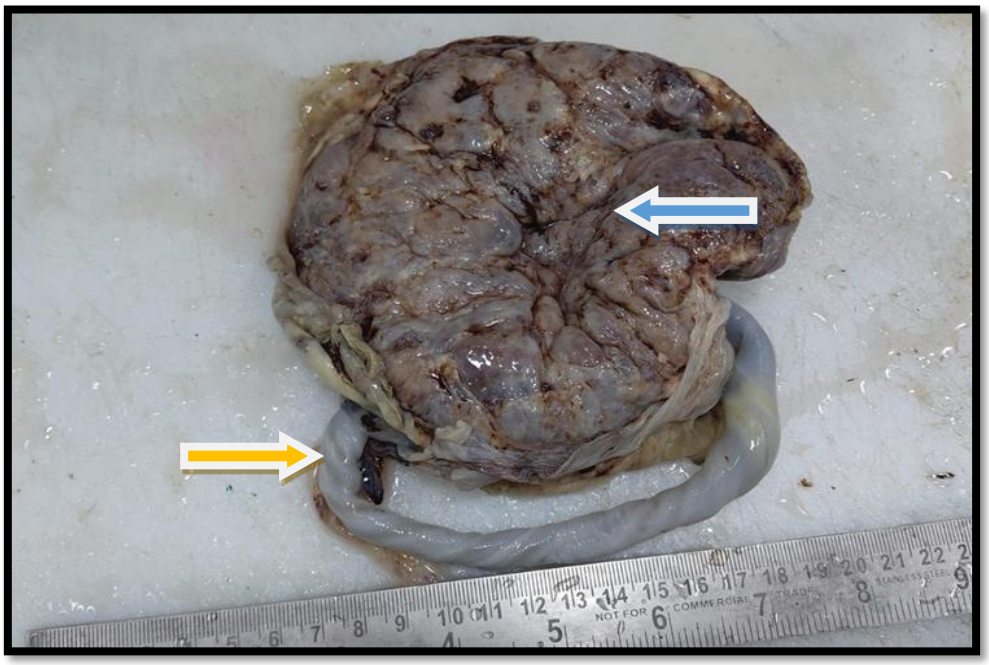

Figure 2: Gross photograph of normal placenta showing maternal surface.

Umbilical cord ( ) and maternal surface ( ).

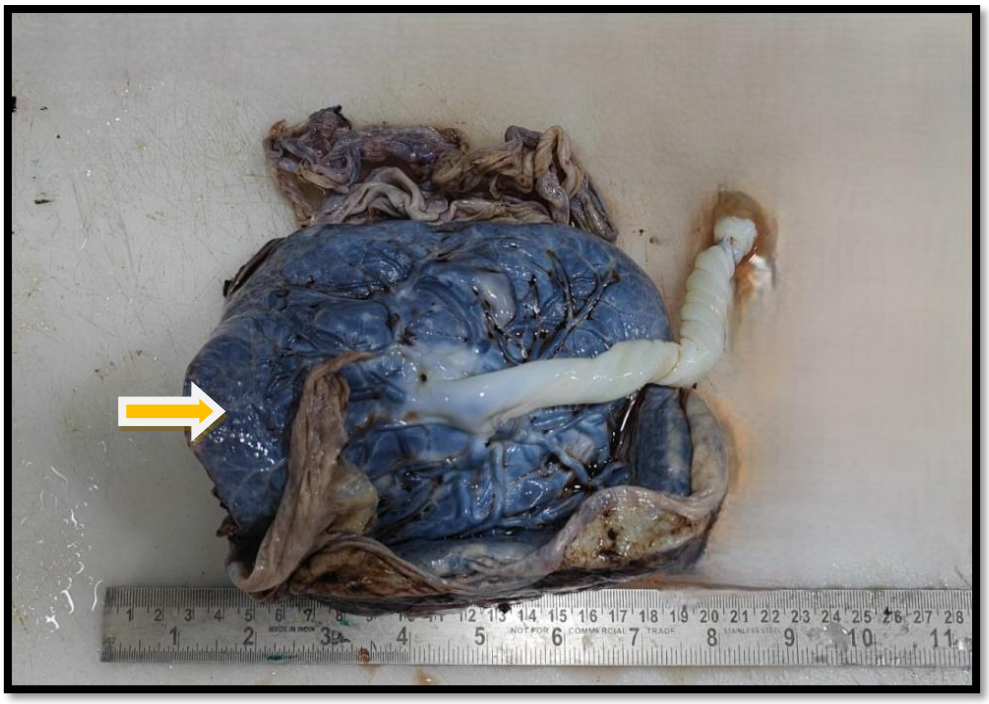

Figure 3. Gross morphology of placenta showing central attachment of umbilical cord

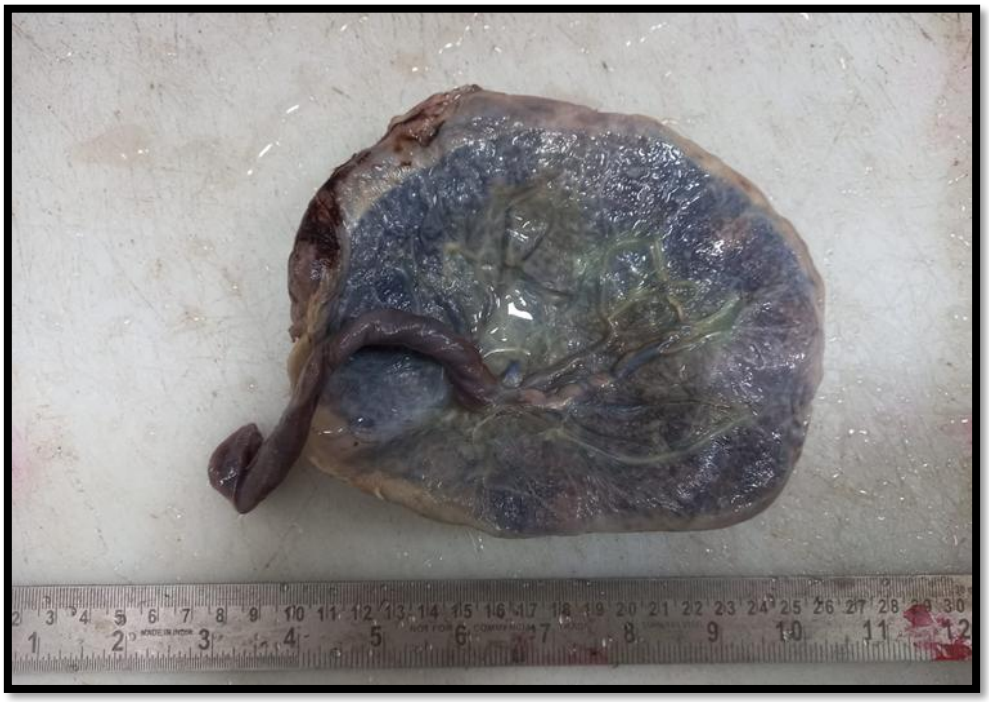

Figure 4: Gross morphology of placenta showing eccentric attachment of umbilical cord.

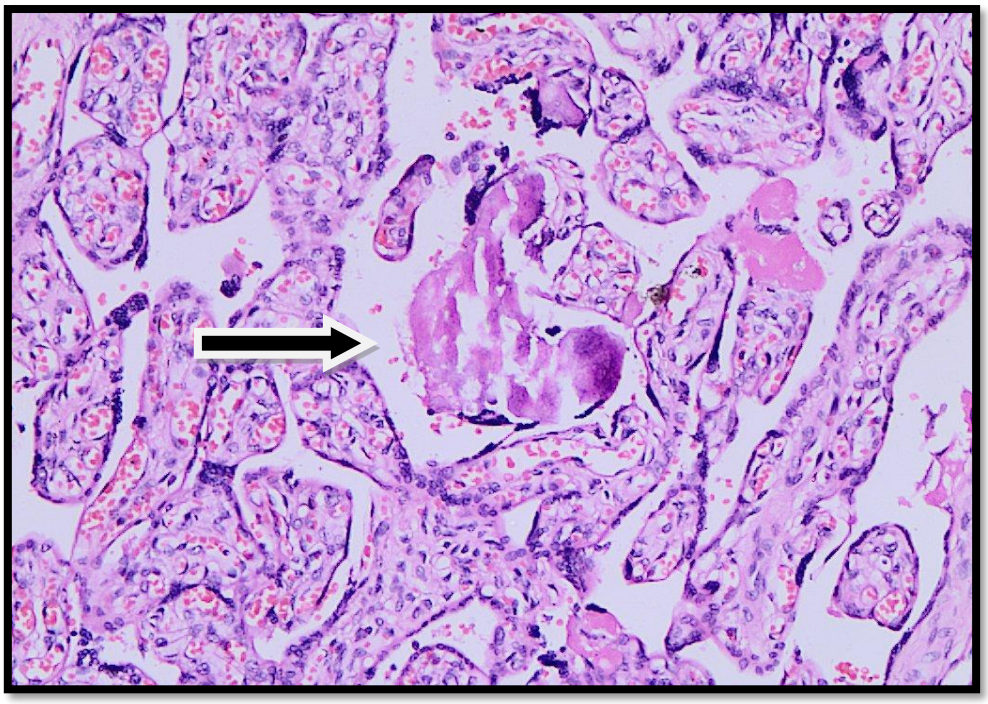

Figure 5:Calcification: Section examined shows calcification (⟶) interspersed between chorionic villi. (H & E 100 X).

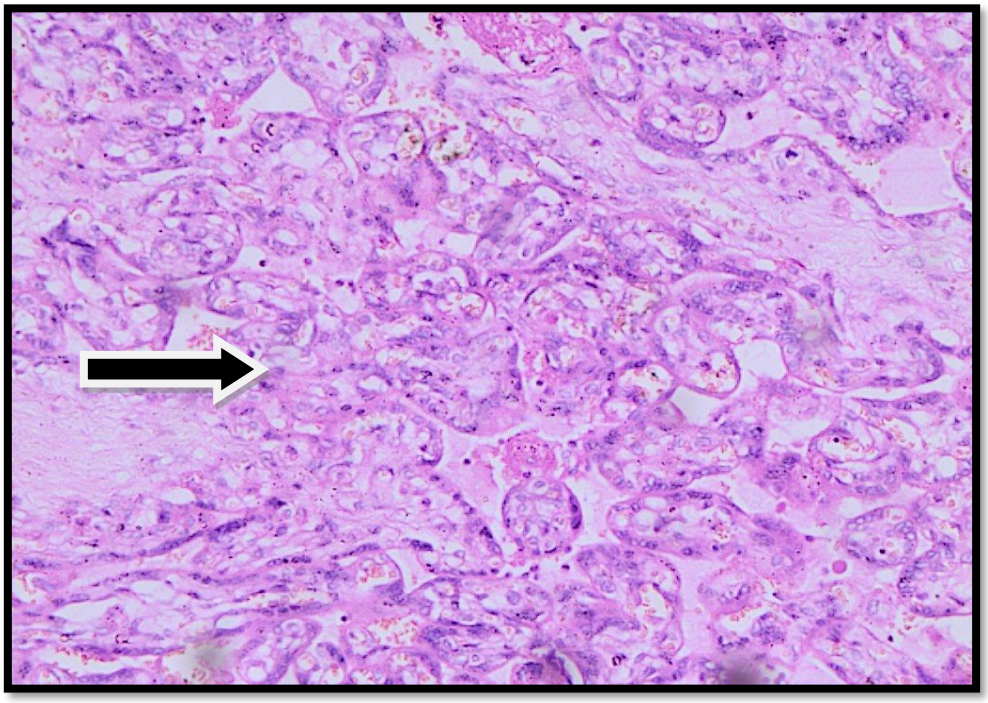

Figure 6: Decreased syncytial knots: Section examined shows immature villous maturation with decrease in syncytial knotting (⟶) -Seen in maternal diabetes.(H & E 100 X)

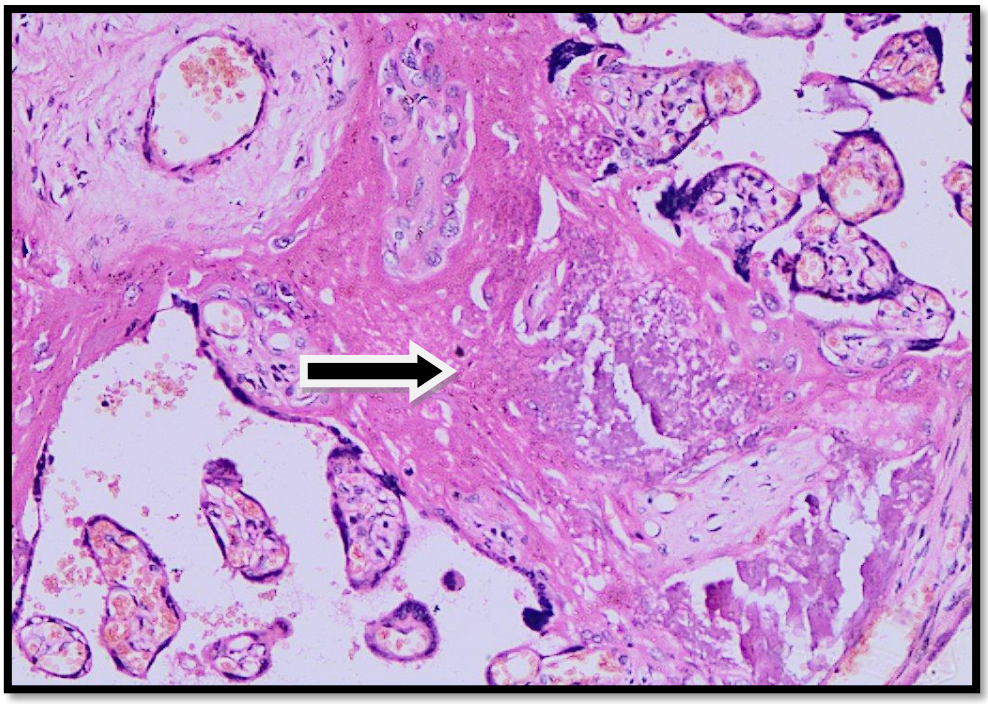

Figure7: Fibrinoid necrosis: Section examined shows extensive areas of fibrinoid necrosis of maternal vessels (⟶) with surrounding villous structures with preserved trophoblastic lining.-Seen in hypertension and pre eclampsia. (H & E 100 X).

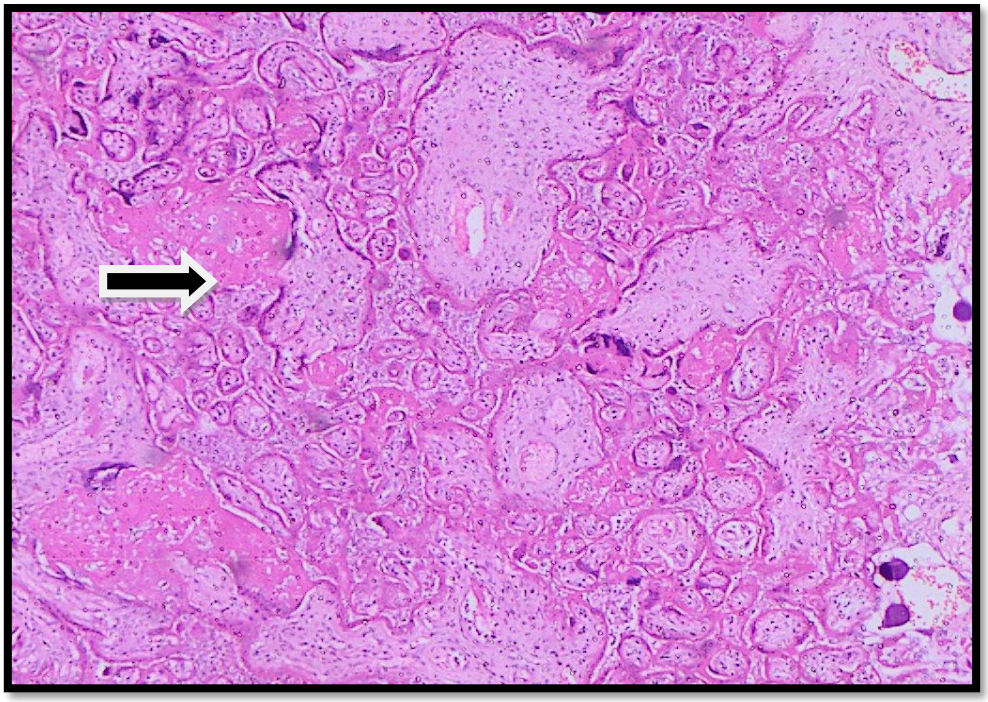

Figure 8: Hyalinization:Section examined shows extensive hyalinization(⟶) of chorionic villi. Some of which are sclerotic seen in vascular malperfusion (H&E 100X).

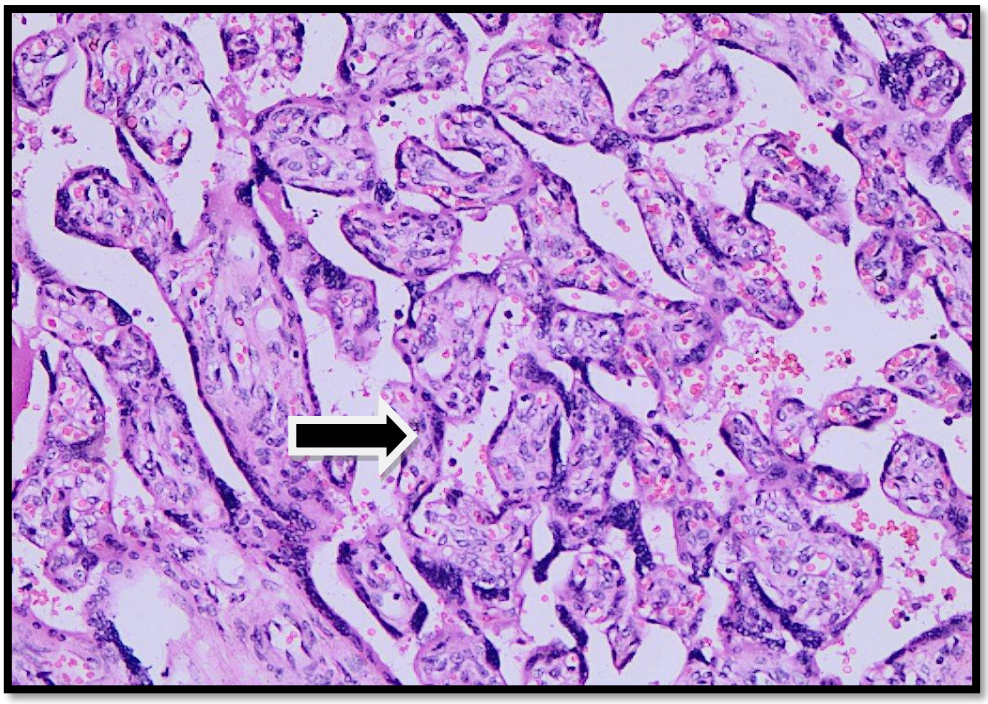

Figure9 :Increased syncytial knots: (⟶) Section examined showsaccelerated villous maturation with increased syncytial knots – seen inhypertension.(H & E 100 X).

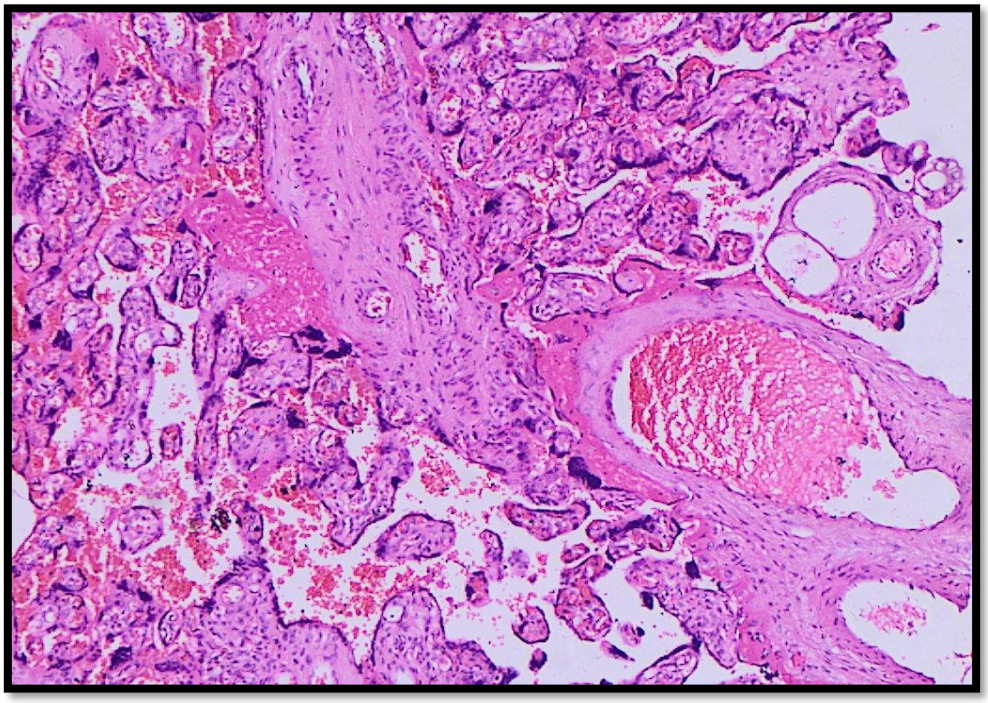

Figure10: Thrombus: Section examined shows thrombus formation (H & E 100X).

DISCUSSION

The present study aimed to analyse the histomorphological and morphometric parameters of placenta in complicated and uncomplicated pregnanciesin which 75 cases were included.The mean maternal age in our study was 25.4 ± 3.8 years, with comparable age distribution between uncomplicated (24.9 ± 3.5 years) and complicated pregnancies (25.8 ± 4.0 years), and this reflects the typical reproductive age group profile reported in Indian populations as per ICMR maternal health surveillance data (ICMR Report)[23] where the national mean age of pregnancy is documented around 24–26 years. Nelson and Crouse[16]also observed mean maternal ages of approximately 26–28 years in diabetic pregnancies. Lone *et al*[18] observed a mean maternal age of 23–25 years in anemia-affected pregnancies.

Our study reported 28% primigravida and 72% multigravida overall. This distribution mirrors the findings ofFowden*et al*[17]who described higher rates of multigravidity nearly 70% in populations at risk for placental insufficiency.

Similarly, Lelic *et al*[24] noted multigravidity in approximately 65% of anemic pregnancies, closely aligning with the 75.6% multigravida proportion among complicated pregnancies in our study.

The mode of delivery in our study showed 60% vaginal births and 40% cesarean deliveries, with higher cesarean rates among complicated pregnancies (46.3%). This pattern is consistent with WHO maternal health data (WHO report)[25] which indicates that obstetric complications increase cesarean rates to approximately 42–48%, aligning closely with the rate in our complicated cohort.

In our study, 9.3% of pregnancies were pre-term overall, but this sharply increased to 14.6% in complicated pregnancies in contrast to 2.9% in uncomplicated pregnancies. Zhou *et al*[12] demonstrated similar trends, with preeclampsia and villous maldevelopment contributing to preterm birth rates of 12–18%, comparable to ours. Mayhew[13] also described structural villous abnormalities leading to preterm birth in nearly 15% of complicated pregnancies, almost identical to our study's figure.

Fetal sex distribution was nearly equal (54.7% male and 45.3% female), which is similar to global biological norms. Birth weight, however, differed significantly, with complicated pregnancies showing lower mean birth weight (2.41 ± 0.58 kg) compared with uncomplicated pregnancies (2.58 ± 0.31 kg). Nelson and Crouse[16]reported birth weights reduced by nearly 10–18% in diabetic and hypertensive pregnancies, consistent with the 7% reduction in mean birth weight observed in our complicated cohort. Fowden*et al*[17]also described fetal growth restriction in placental dysfunction cases with birth weight reductions of 0.2–0.4 kg, again consistent with the pattern in our study.

In our study, placental weight was significantly lower in complicated pregnancies (379.5 ± 128.6 g) compared with uncomplicated pregnancies (432.8 ± 118.3 g), with a p-value of 0.046.Lone*et al* [18]demonstrated that anemic mothers had placental weights reduced by 40–80 g compared with controls, similar to the 53 g reduction seen in our data.

Regarding placental diameter, our study found mean values of 14.9 ± 2.7 cm, with slight reduction in complicated pregnancies (14.6 cm vs. 15.3 cm). Mayhew[13] has described that placental diameter reduces by approximately 0.8–1.2 cm in placentas with villous pathology, consistent with the 0.7 cm reduction seen in our complicated group.

Placental thickness in our study showed non-significant differences 3.6 ± 2.1 cm in uncomplicated vs 4.5 ± 3.4 cm in complicated pregnancies. Teasdale[26]described similar variability in thickness, noting that abnormal placentas can show either thinning or localized thickening due to infarcts or fibrinoid necrosis. This variability mirrors the high SD values in our thickness measurements.

The feto-placental ratio (FPR) in our study was 0.154 in uncomplicated pregnancies and 0.176 in complicated pregnancies. An increased FPR typically indicates fetal disproportion or placental insufficiency. Fowden*et al*[17]described increased FPR in placentas affected by metabolic stress, where FPR values increased by nearly 10–15%, similar to the 14% elevation seen in our complicated pregnancies.

Cord length in our study averaged 16.5 ± 3.8 cm, with shorter cords observed in complicated pregnancies (16.0 cm vs. 17.1 cm).

Our study observed discoid placentas in 64% of cases, bilobed placentas in 14.7%, and irregular forms in16%cases.Uncomplicated pregnancies were more likely to show discoid placenta 73.5%whereas complicated pregnancies showed higher bilobed frequency22% cases Burton *et al*[27]described bilobed placentas in nearly 12–15% of high-risk pregnancies, similar to the 14.7% seen overall in our study.

Cord insertion patterns in our study showed central insertion in 38.7% of cases and eccentric insertion in 61.3%. Eccentric insertion was more common in complicated pregnancies 63.4% aligning with the work of Kingdom & Kaufmann [19] who described eccentric insertion in 55–65% of placentas with impaired villous vascular remodeling.

The finding of 97.3% three-vessel cords in our study is consistent with normal population data, and only 2.7% two-vessel cords were identified. Nelson & Crouse[16]reported a similar 2–3% frequency of two-vessel cords in complicated pregnancies.

In our study, retroplacental hematoma was found in 10.7% of cases, more common in complicated pregnancies 14.6% compared with uncomplicated pregnancies 5.9%.Teasdale[26]described similar hematoma rates of 12–18% in preeclamptic placentas.

Calcification was present in 25.3% of our samples, higher in complicated pregnancies (31.7%). Lone *et al*[18]reported calcification rates of 22–28% in anemia-affected placentas, similar to our complicated group.

Gross infarctions were observed in 20% of placentas overall, with 26.8% in complicated pregnancies compared with 11.8% in uncomplicated cases. This matches findings from Kingdom&Kaufmann[19]who observed infarcts in 25–35% of placentas with vascular malperfusion. Teasdale[26] reported infarct rates of 18–28% in preeclampsia.

Syncytial knot prominence was significantly higher in complicated pregnancies (53.7%) compared with uncomplicated pregnancies (23.5%). Mayhew[13]reported increased syncytial knots in nearly 50% of placental insufficiency cases.

Fibrinoid necrosis was present in 70.7% of complicated pregnancies in our study versus 32.4% in uncomplicated pregnancies.Lone *et al*[18]reported fibrinoid necrosis in 55–65% of anemic mothers, while a studydocumented it in 68–72% of diabetic pregnancies, both comparable to our findings.

Villous infarction was present in 58.5% of complicated pregnancies in our study compared with 23.5% in uncomplicated pregnancies. Kingdom & Kaufmann [19] described villous infarctions in 50–65% of placentas with vascular malperfusion, consistent with our 58.5%.

Hyalinized villi were present in 58.5% of complicated pregnancies vs. 35.3% of uncomplicated pregnancies. A studynoted hyalinization in 45–60% of placentas from complicated pregnancies, consistent with the pattern seen in our study.[15,18]

The microscopic vascular alterations in our study demonstrated that thrombosis was present in 42.7% of all placentas, with a significantly higher proportion in complicated pregnancies 53.7% compared with uncomplicated pregnancies 29.4% with a p-value of 0.042.This pattern of increased thrombotic lesions in complicated pregnancies is supported by classical work by Teasdale *et al*[26] who reported thrombosis in nearly 50–60% of preeclamptic placentas, demonstrating comparable vascular pathology to the 53.7% seen in our complicated cohort. AdditionallyHeazell[20] observed thrombosis in 44–52% of placentas associated with fetal compromise and stillbirth, reflecting a strong correlation between compromised uteroplacental perfusion and the presence of vascular thrombi, which is consistent with our finding that thrombosis doubled in complicated pregnancies.[16,20]

Dystrophic calcification was present in 53.3% of our study samples, with slightly higher frequency in complicated pregnancies (58.5%) compared with uncomplicated pregnancies (47.1%). Lone *et al*[18] described calcification in 50–60% of placentas from anemic mothers, nearly identical to our 53.3% total and 58.5% complicated category. Fowden*et al*[17]documented abnormal mineral deposition in 45–55% of placentas with metabolic stress, and showed similar patterns, demonstrating that calcific changes are common across many forms of placental insufficiency. These parallels confirm that our observation of higher vascular pathology in complicated pregnancies is consistent with established literature describing compromised perfusion, oxidative stress, and villous remodeling as key contributors to thrombotic and calcific lesions.[13] In our study, placental weight did not differ significantly between primigravida (401.2 ± 119.0 g) and multigravida mothers (404.5 ± 129.5 g), with a p-value of 0.919, indicating parity did not substantially influence placental mass. Other studies also showed similar results.[13,18]

Placental thickness also showed no significant difference (4.3 cm in primigravida vs. 4.0 cm in multigravida). concluded that parity does not independently alter placental thickness unless confounded by comorbid conditions.[16]Jirkovská*et al*[15] also demonstrated similar thickness patterns across parity groups in diabetic pregnancies. Cord length showed a slight increase in multigravidas (16.8 cm) compared with primigravidas (15.4 cm), though statistically non-significant. This resembles observations by Fowden et al [17]who found cord length to be more influenced by fetal activity and intrauterine environment than by parity.

Our feto-placental ratio (0.171 in primigravida vs. 0.163 in multigravida) demonstrates only minimal variation, consistent with the physiological stability noted that FPR varies primarily with fetal growth restriction rather than parity. Overall, these findings align with the consistent consensus that parity does not significantly alter morphometric placental indices.[19] There was no significant difference in placental weight between vaginal deliveries (399.2 ± 130.8 g) and cesarean deliveries (409.9 ± 121.1 g), with a p-value of 0.716.A study similarly reported that mode of delivery has negligible influence on placental weight, with variations primarily dependent on pathological conditions rather than delivery type as also shown by Jirkovská*et al*[15,18]

Placental thickness was higher in cesarean deliveries (4.4 cm vs. 3.9 cm), though non-significant. This mild increase aligns with findings by Heazell[20] who associated emergency cesarean due to fetal distress with slightly thicker, edematous placentas. The feto-placental ratio (0.166 in both groups) was identical in our study, highlighting that mode of delivery does not influence placental efficiency. Birth weight was slightly higher in cesarean deliveries (2.58 ± 0.69 kg vs. 2.43 ± 0.27 kg), reflecting a pattern similar toMayhew[13] and Fowden[16]who observed higher birth weights in planned cesarean deliveries, often due to better-controlled pregnancies[13,.16]

In our study, male babies showed slightly lower placental weight (393.6 ± 134.8 g) than female babies (415.4 ± 116.0 g), though non-significant. This contrasts somewhat with findings of Fowden*et al*[17] who reported that male fetuses typically have marginally heavier placentas by 10–20 g; however, the variability seen in our population aligns with observations in

one study where it was demonstrated that there was no consistent sexual dimorphism in placental weight among complicated pregnancies.Nelson[16]similarly found inconsistent sex differences in placental weight in anemia-affected pregnancies.[18]

Birth weight was nearly identical between sexes in our study 2.50 kg for males vs. 2.47 kg for females mirroring the global findings reported in WHO data [25] which indicates minimal sex differences in low-risk South Asian populations.

Hypertensive pregnancies in our study exhibited marked placental pathology. Placental weight in the hypertensive group was lower (387.5 ± 86.6 g) compared with normotensive controls (432.8 ± 118.3 g), though the p-value (0.315) was not statistically significant. Lean *et al*[28]also demonstrated reduced placental mass in hypertensive disorders, with weights 40–70 g lower than controls, comparable to the nearly 45 g reduction in our study.

Villous infarction was significantly higher75% in hypertensive pregnancies compared with 23.5% in controls (p = 0.009)This mirrors the classical findings of who reported infarction rates of 60–75% in hypertensive pregnancies, nearly identical to our 75%.[19] Similarly, Jirkovská*et al*[15]described infarction in 58–72% of hypertensive diabetic patients, and described infarction in nearly 70% of severe preeclampsia.[97]

Syncytial knots were markedly increased in hypertensives (75% vs. 23.5%, p = 0.009). This aligns with findings of Zhou *et al*[12] who linked impaired trophoblast invasion with increased syncytial knotting ranging from 65–80%.

Fibrinoid necrosis was drastically more frequent in hypertensive pregnancies (87.5% vs. 32.4%, p = 0.008). Teasdale et al[26]reported fibrinoid necrosis in 80–90% of hypertensive placentas, matching our 87.5% almost exactly. Lone et al[18]also reported necrosis rates of around 60–70% in anemia, but significantly higher (up to 85%) in combined hypertensive states, showing strong comparability to our findings.

It was concluded that hypertensive pregnancies exhibit the most severe histopathological alterations, strongly consistent with global literature linking maternal hypertension to pronounced placental malperfusion, infarction, necrosis, and accelerated villous maturation.

In our study, placental weight was slightly lower in anemic mothers (397.1 ± 150.8 g) compared with non-anemic controls (432.8 ± 118.3 g), although this difference did not reach statistical significance (p = 0.342). This trend closely resembles findings reported by Lone et al[18]who demonstrated a reduction of nearly 30–60 g in placental weight among anemic mothers, reflecting impaired oxygen transport and maternal under-nutrition that limit placental growth.[18]Similar reductions were documented by Lelic *et al*[24] who observed placental weights approximately 50 g lower in anemic pregnancies, paralleling the modest decrease seen in our studysupported that impaired maternal oxygenation decreases placental metabolic capacity and suppresses placental growth, which helps explain the weight reduction in our anemic cohort despite lack of statistical significance.

Abnormal placental shape was markedly more frequent in anemic mothers, with 50% showing bilobed or irregular placentas compared with 20.6% in controls (p = 0.034). This strong association is well aligned with the findings of Lelic *et al*[24] who reported abnormal placental morphology in approximately 40–50% of anemic pregnancies, nearly identical to our 50% abnormal shape frequency. Mayhew [13]explained these structural abnormalities as consequences of villous remodeling and stromal fibrosis associated with chronic maternal anemia.

Retroplacental hematoma was seen in 20% of anemic mothers compared with 5.9% of controls in our study. Although the p-value (0.189) was non-significant, the trend is meaningful and mirrors findings of other studies.Lone *et al*[18]similarly described hematoma prevalence of 12–18% in maternal anemia.

In our study, placental weight showed a moderate positive correlation with birth weight (r = 0.318, p = 0.006), indicating that heavier placentas support better fetal growth. This observation aligns with the classical metabolic principle described by Fowden*et al*[17]who emphasized that placental mass is directly proportional to nutrient transfer capacity, demonstrating correlations of around r = 0.30–0.40 between placental and fetal weights.

Gestational age showed a positive correlation with placental weight (r = 0.285, p = 0.015).A studyreported nearly identical associations (r = 0.28–0.32), demonstrating that placental growth continues progressively with advancing gestation.[13]Lelic *et al*[24] also found gestational age–placental weight correlation between 0.20 and 0.30, while noted similar trends in anemic pregnancies.

A strong negative correlation was observed between placental weight and feto-placental ratio (r = –0.925, p < 0.001), one of the most physiologically meaningful findings in this study. This aligns with explanations by Kingdom et al[19]who described that lower placental weight or inadequate growth forces an increase in FPR due to fetal-maternal disproportion. Such an extremely strong negative correlation is consistent with severe placental imbalance and highlights placental functional inadequacies, especially in pregnancies complicated by vascular or metabolic disorders.[8,19]

The present study found that villous infarction was significantly associated with fetal distress, occurring in 62.5% of distressed fetuses compared with 33.3% of non-distressed fetuses ($p = 0.021$). This strong association closely mirrors observations of Teasdale[26] who documented that 55–70% of fetuses experiencing distress or growth restriction exhibited infarction.

Increased syncytial knots were found in 58.3% of fetal distress cases compared with 31.4% when distress was absent ($p = 0.032$). Jirkovská*et al*[15]and Teasdale[26]emphasized syncytial knots as markers of chronic insufficiency contributing to fetal compromise.

Fibrinoid necrosis was more frequent in the fetal distress group (66.7%) compared with the no-distress group (47.1%), though the association was not statistically significant ($p = 0.143$). Nevertheless, the trend aligns withKingdom etal[19]who reported necrosis in 60–70% of placentas with fetal hypoxia.Lone *et al*[18]described similar associations, strengthening the interpretation that necrosis contributes to impaired oxygen exchange.

The present study demonstrated significantly lower placental weight in pre-term births (331.4 ± 83.0 g) compared with full-term births (406.1 ± 126.7 g), with a p-value of 0.017. This finding aligns strongly with the observations of Mayhew[13]who reported that reduced placental weight is a critical determinant and predictor of preterm delivery, with reductions often exceeding 60–80 g, comparable to the 75 g reduction in our dataset. A studyalso highlighted that impaired villous development and surface area reduction significantly predispose to preterm birth.

Birth weight did not differ significantly between preterm (2.41 kg) and full-term infants 2.50 kg though the expected difference was small. Nelson and Crouse[16] similarly observed that in some populations, late-preterm infants may have near-overlapping birth weight ranges with term infants, particularly in the presence of maternal metabolic disorders. Feto-placental ratio was lower in preterm deliveries (0.137) than full-term 0.167 supporting the principle described byFowden*et al*[17]that suboptimal placental development precedes fetal growth limitation.

Cord length was marginally shorter in preterm births (15.6 cm) compared with term births (16.5 cm)a studydescribed similar findings, attributing reduced cord length to restricted fetal movements or prolonged fetal hypoxia.[20]

The integrated findings of our study demonstrate that complicated pregnancies consistently showed lower placental weight, increased infarcts, excessive syncytial knots, and higher fibrinoid necrosis, all with significant associations ($p < 0.05$). These findings agree closely with classical descriptions by Teasdale[26]who emphasized that vascular malperfusion profoundly alters placental structure[8,13]. The positive correlation between placental weight and birth weight ($r = 0.318$) seen in our study is consistent with global data from Fowden*et al* [17]and WHO[25] reinforcing that a heavier placenta supports better fetal growth. The strong association between hypertension and placental histopathology—specifically infarction and syncytial knots.[12].[19,20] The relationship between histological abnormalities and fetal distress, including increased infarcts and syncytial knots, closely mirrored findings by a study preterm delivery was significantly associated with reduced placental weight.

CONCLUSION

In this extensive analysis of all parameters we concluded that complicated pregnancies consistently had lower placental weight, higher gross lesions, more severe histopathological abnormalities including syncytial knots, fibrinoid necrosis, infarction, vascular thrombosis, and calcification, all of which adversely affected fetal outcomes and significantly contributed to increased rates of fetal distress and preterm delivery. The strong correlations between placental weight, fetal birth weight, gestational age, and feto-placental ratio underline the centrality of placental health in determining fetal well-being, the study conclusively establishes that placental morphology and histopathology serve as powerful markers of fetomaternal health, with measurable and statistically significant associations with pregnancycomplications, supporting the critical need for placental evaluation as an integral part of maternal-fetal medicine.

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