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Original Research Article

Correlation of placental histopathology in fetal growth restriction with fetal outcome

Pratyusha Singh[1]*, Dilpreet Kaur Pandher[1], Poonam Goel[1], Uma Handa[2], Suksham Jain[3]

[1]Department of Obstetrics and Gynaecology, Government Medical College and Hospital, Chandigarh, India
[2]Department of Pathology, Government Medical College and Hospital, Chandigarh, India
[3]Department of Neonatology, Government Medical College and Hospital, Chandigarh, India

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***Correspondence:**
Dr. Pratyusha Singh,
E-mail: psshivangi1997@gmail.com

ABSTRACT

Background: Fetal Growth Restriction (FGR), affecting 5-10% of pregnancies worldwide, is a significant cause of perinatal morbidity and mortality. The placenta plays a central role in the pathogenesis of FGR, with various histopathological abnormalities contributing to impaired fetal growth. Understanding the relationship between placental pathology and neonatal outcomes can guide clinical management and improve future pregnancy outcomes. This study aimed to find out the specific placental histopathologies present in FGR pregnancies and correlate them with the type and severity of FGR as well as neonatal outcome.
Methods: An analytical cross-sectional study was conducted on 92 FGR pregnancies at Government Medical College, Chandigarh over 18 months. Placental samples were examined histologically, and findings were correlated with clinical data including doppler studies, birth weight, Apgar scores, NICU admission and neonatal mortality. Statistical analysis was performed using SPSS version 25.0, with significance set at $p<0.05$.
Results: Early-onset FGR was observed in 36.9% and late-onset in 63.1% of cases. Placental histopathological analysis indicated that the most common abnormalities were syncytial knots (88%), fibrinoid necrosis (85.9%), and dystrophic calcifications (52.2%). Early-onset FGR was significantly associated with doppler abnormalities such as absent or reversed end-diastolic flow (AEDF/REDF) and poorer neonatal outcomes like low birth weight, low Apgar scores, higher NICU admissions (42.9%), and increased neonatal mortality (31.8%). Placental abnormalities detected in early-onset FGR includes massive peri villous fibrin deposition (MPVFD), chorioamnionitis, and diffuse dystrophic calcification.
Conclusions: FGR is a complex condition with multifactorial etiology, often associated with multiple placental lesions. Placental abnormalities, particularly MPVFD, chorioamnionitis, diffuse dystrophic calcification are strongly associated with FGR severity and adverse neonatal outcomes. Routine placental histopathological examination in FGR cases provides valuable insights into its etiopathogenesis and optimizing fetal outcomes in subsequent pregnancies.

Keywords: Doppler abnormalities, Early-onset FGR, Fetal growth restriction, Massive peri villous fibrin deposition (MPVFD), Neonatal outcome, Perinatal morbidity, Placental histopathology

INTRODUCTION

Fetal growth restriction (FGR) is a condition where a fetus fails to achieve its full genetic growth potential. It affects approximately 5-10% of pregnancies globally. FGR is a major cause of perinatal morbidity and mortality leading to complications such as stillbirth, neonatal intensive care unit (NICU) admissions, neurodevelopmental delay and an increased risk of chronic conditions later in life including cardiovascular disease and type 2 diabetes.[1,2] FGR can be

classified into early-onset (before 32 weeks of gestation) and late-onset (after 32 weeks), with each type showing distinct clinical and pathological features.

The placenta plays a crucial role in fetal development by regulating the exchange of nutrients, gases, and waste products between the mother and fetus.[1] FGR is frequently linked to placental pathologies such as maternal vascular malperfusion (MVM), fetal vascular malperfusion (FVM), and chronic inflammatory disorders (villitis of unknown origin), MPVFD.[1,2] Identifying these histopathological abnormalities is critical in understanding the etiology of FGR and improving clinical outcomes through timely diagnosis and intervention.

This study aimed to find out the specific placental histopathologies present in FGR pregnancies and correlate them with the time of onset, severity of FGR and neonatal outcomes. We hypothesized that different placental pathologies would be more prevalent in certain types of FGR and would influence neonatal outcomes including birth weight, Apgar scores and NICU admissions.

METHODS

This was an analytical cross-sectional study conducted in the Department of Obstetrics and Gynaecology in collaboration with the Department of Pathology and Department of Neonatology at Government Medical College and Hospital, Chandigarh.

Study duration

The study was carried out from December 2022 to May 2024.

Inclusion criteria

A total of 92 pregnant women diagnosed with fetal growth restriction (FGR) were included in the study. FGR was defined using Delphi consensus criteria as follows[2]:

1) Abdominal circumference (AC) or estimated fetal weight (EFW) <3rd centile at a gestational age (GA) >26 weeks, 2) Absence of end-diastolic flow (AEDF) in the umbilical artery at GA <32 weeks, 3) AC/EFW <10th centile combined with uterine artery pulsatility index (UtA-PI) >95th centile and/or umbilical artery pulsatility index (UA-PI) >95th centile at GA <32 weeks, 4) For GA >32 weeks, at least two of the following were required: i) AC/EFW < 10th centile, ii) AC/EFW crossing > 2 quartiles on growth centiles, iii) Cerebroplacental ratio (CPR) <5th centile or UA-PI > 95th centile.

Exclusion criteria

Pregnant women with congenital malformed fetus, suspected or diagnosed chromosomal abnormalities, multiple pregnancies, placenta previa and premature rupture of membranes. were excluded from the study.

Ethical approval and consent

The study received approval from the Institutional Ethics Committee and informed consent was taken by all participants.

Sample size calculation

A sample size of 92 FGR pregnancies was calculated using a 95% confidence level and 5% level of significance. This calculation was based on an estimated predictive accuracy of 30% for placental histopathology in forecasting adverse maternal and fetal outcomes.

Data collection

Participants' demographic and clinical data, including maternal age, gravidity, parity, gestational age at delivery and antenatal complications were recorded. In addition, neonatal outcomes such as birth weight, Apgar scores and NICU admission rates were recorded.

After delivery, placentas were collected and sent to the Department of Pathology for histological examination. Placental samples were fixed in 10% neutral buffered formalin, embedded in paraffin, and detailed histopathological examination was conducted by the pathology department. The data was correlated with neonatal outcomes.

Statistical analysis

Data were entered into a computer-based spreadsheet and analysed using SPSS software version 25.0. Quantitative variables, such as maternal age and birth weight, were summarized using means with 95% confidence intervals. Categorical variables were described using frequencies and percentages. Chi-square tests and Fisher's exact tests were used to assess the associations between placental histopathological findings and neonatal outcomes. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Maternal characteristics

The mean age of the mothers included in the study was 27.2±4.79 years, with the majority (71.7%) being between 21 and 30 years of age (Figure 1). A total of 45 participants (48.9%) were primigravida, while the remaining 47 (51.1%) were multigravida. Most FGR cases were observed in the upper class (53.3%), followed by the upper middle class (16.3%) according to the modified Kuppuswamy scale.

In terms of maternal comorbidities, it was seen that maximum mothers 23 (25%) had subclinical hypothyroidism, followed by preeclampsia without severe features in 21 (22.8%), gestational hypertension in 12

(13%), chronic hypertension was seen in 10 (10.9%), intrahepatic cholestasis of pregnancy (ICP) in 9 (9.8%), gestational and overt diabetes in 8 (8.7%) and 2 (2.2%) mothers respectively and only 1 mother had eclampsia (Table 1).

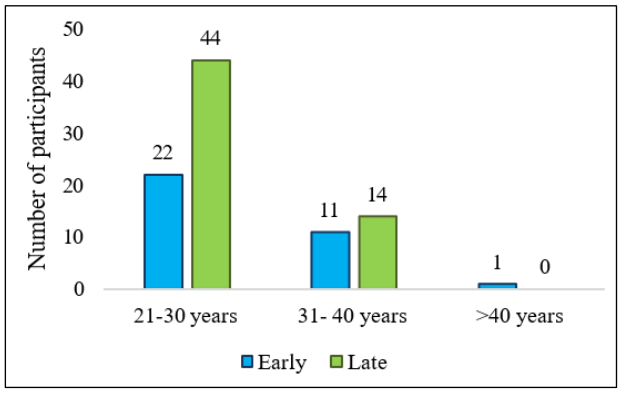

Figure 1: Age distribution among the mothers according to the onset of fetal growth retardation (FGR).

Neonatal characteristics

Out of the 92 neonates, 68.5% were delivered preterm, with 17.4% being born before 33+6 weeks of gestation (early preterm) and 51.1% born between 34 and 36 weeks (late preterm) (Figure 2). The mean birth weight of the neonates was 1,780±320 grams (Table 2).

In our study of 92 cases, 58 (63%) were late onset FGR and 34 (37%) were early onset FGR. Early-onset FGR was more commonly associated with chronic hypertension, preeclampsia, severe preeclampsia syndromes. In our study, early-onset FGR was more significantly associated with AEDF/REDF in 77.3% compared to 22.7% cases of late-onset FGR. In our study, cerebroplacental ratio (CPR) was normal in 67% and 33% of cases showed abnormal CPR (<1). Non-stress test (NST)/cardiotocography (CTG) results showed 31.9% of mothers had non-reassuring NST/CTG, while 68.1% had reassuring results (Figure 3).

Table 1: Distribution of mothers according to comorbidities.

Comorbidity		Number	Percentage
Hypothyroidism		23	25.0
Hypertensive disorders of pregnancy	Preeclampsia with severe features	21	22.8
	Preeclampsia without severe features	6	6.5
	Gestational hypertension	12	13.0
	Chronic hypertension	10	10.9
	Chronic hypertension with superimposed preeclampsia	3	3.3
Anemia	Mild	9	9.8
	Moderate	7	7.6
	Severe	2	2.2
ICP		9	9.8
Gestational diabetes		8	8.7
Overt diabetes mellitus		2	2.2
Epilepsy		2	2.2
Renal diseases		2	2.2
Eclampsia		1	1.1
Idiopathic thrombocytopenia		1	1.1

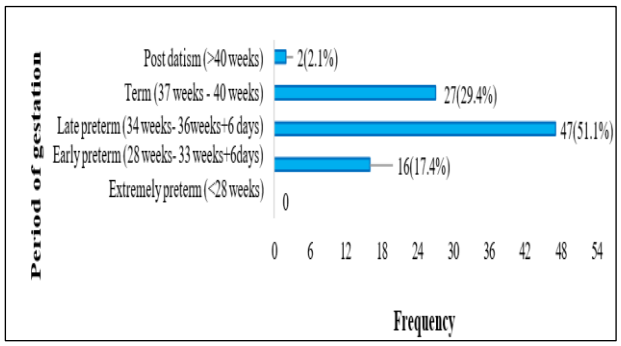

Figure 2: Distribution of the mothers according to period of gestation at the time of delivery.

Table 2: Distribution of the mothers according to birth weight of the newborn.

Birth weight in kg	Frequency (N)	Percentage (%)
>2.50	2	2.2
2.00-2.49	19	20.7
1.50-1.99	44	47.8
1.00-1.49	21	22.8
<1	6	6.5
Total	92	100

Emergency and elective Lower segment Caesarean Section (LSCS) was done in 38% and 1.1% of the mothers

respectively. Induction of labour was done in 44 cases in whom, 12 (13%) had emergency LSCS, 28 (30.4%) had normal vaginal delivery and 4 (4.3%) had instrumental vaginal delivery.

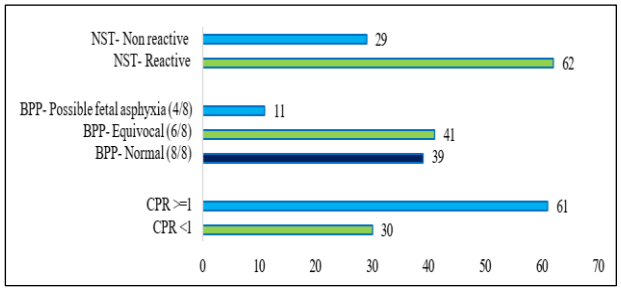

Figure 3: Distribution of the mothers according to cerebroplacental ratio and other investigation.

Twelve went into spontaneous labour, 6 (6.5%) had emergency LSCS, 4 (4.3%) had normal vaginal delivery and 2 (2.2%) had instrumental vaginal delivery. The most common indications for LSCS were non-reassuring NST/CTG (29.6%) and acute fetal distress (25%) (Table 3).

Table 3: Distribution of the mothers according to the indication of LSCS.

Reason for LSCS	Frequency	Percentage
Non-reactive NST	16	29.6
Acute fetal distress	14	25.9
Poor biophysical score	8	14.8
Breech presentation	7	13.0
AEDF	4	7.4
REDF	4	7.4
MSL with poor Bishop score	3	5.6
Previous LSCS not willing for TOLAC	3	5.6
Uncontrolled hypertension	1	1.9
Anhydramnios	1	1.9
Eclampsia with poor Bishop score	1	1.9

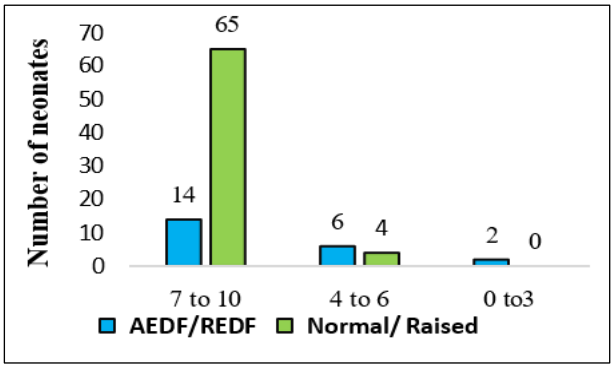

Figure 4: Association between doppler studies.

APGAR scores at 1 minute were excellent in 85.9% of babies, moderately depressed in 10.9%, and severely depressed in 3.3%. At 5 minutes, almost all babies except one had excellent APGAR scores. None of the babies with normal or raised S/D ratios had poor APGAR scores (0-3) at 1 minute.

Among those with AEDF/REDF, 9.1% had poor (0-3), 27.3% had moderate (4-6), and 63.6% had excellent (7-10) APGAR scores although infants had excellent APGAR scores at 5 minutes (Figure 4).

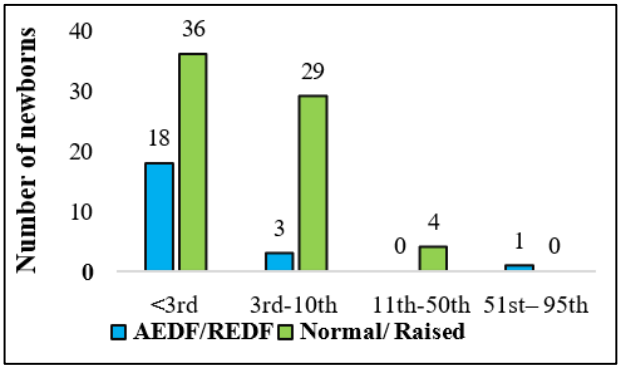

Figure 5: Association between on doppler studies and APGAR score at 1 min birth weight percentiles of the neonates.

NICU admission was required in 42% (39) and 11% (10) neonates died due to complications during NICU stay. The remaining 89% (81 babies) were discharged from the hospital in satisfactory condition. Neonates with AEDF/REDF had lower birth weights with 81% weighing below the 3rd percentile and 41% required NICU stay (Figure 5). The neonatal mortality rate in cases with AEDF/REDF was significantly higher (31.8%) compared to only 2.9% in cases with normal/raised S/D ratios. Our study showed that doppler abnormalities (AEDF/REDF) were significantly associated with poor Apgar scores, low birth weight, NICU admission and neonatal death (p-value <0.01).

Placental histopathology

The gross examination of placentas in the FGR revealed central cord insertion in most cases, while eccentric in 8 and marginal in 2 cases. In our study the most frequent histopathological finding were syncytial knots (88%), followed by fibrinoid necrosis (85.9%), diffuse dystrophic calcifications (52.2%), placental intravascular thrombi (34.8%), haemorrhagic endo vasculitis (30.4%), chorioamnionitis (27.2%), chorionic villitis (25%), MPVFD (16.3%), infarction (13%), villous fibrosis (2.2%), and avascular villi (1.1%) (Table 4).

Peri villous fibrin deposition (MVFD), diffuse dystrophic calcification and chorioamnionitis were associated with early-onset FGR and poor neonatal outcomes. On other hand histopathological features including infarction,

syncytial knots, avascular villi, villous fibrosis, chronic deciduitis, placental intravascular thrombi, chronic villitis, hemorrhagic endovasculitis, fibrinoid necrosis had no significant association with timing of onset of FGR and was seen uniformly distributed in both early and late onset FGR (Table 5). In our study there was no significant placental histopathology in low-birth-weight neonates. Neonatal deaths and intrauterine fetal demise were linked to placental abnormalities like MPVFD, thrombi, and infarction.

Table 4: Distribution of the mothers according to findings on placental histopathological examination.

Findings on histopathological examination	Frequency	Percentage
Syncytial knots	81	88.0
Fibrinoid necrosis	79	85.9
Diffuse dystrophic calcifications	48	52.2
Placental intravascular thrombi	32	34.8
Haemorrhagic endo vasculitis	28	30.4
Chorioamnionitis	25	27.2
Chorionic villitis	23	25.0
Massive peri villous fibrin deposition (MPFD)	15	16.3
Infarction	12	13.0
Chronic deciduitis	8	8.7
Villous fibrosis	2	2.2
Avascular villi	1	1.1
Villous edema	0	0.0
Chorioangioma	0	0.0

Table 5: Association between onset of FGR and findings on placental histopathological examination.

	FGR onset, N (%)		P value
	Early	Late	
Syncytial knots			
Present	31 (91.2)	50 (86.2)	0.74
Absent	3 (8.8)	8 (13.8)	
Fibrinoid necrosis			
Present	30 (88.2)	49 (84.5)	0.76
Absent	4 (11.8)	9 (15.5)	
Diffuse dystrophic calcification			
Present	23 (67.6)	25 (43.1)	0.02
Absent	11 (32.4)	33 (56.9)	
Intravascular thrombi			
Present	13 (38.2)	19 (32.8)	0.59
Absent	21 (61.8)	39 (67.2)	
Hemorrhagic endovasculitis			
Present	13 (38.2)	15 (25.9)	0.21
Absent	21 (61.8)	43 (74.1)	
Chorioamnionitis			
Present	14 (41.2)	11 (19.0)	0.02
Absent	20 (58.8)	47 (81.0)	
Chronic villitis			
Present	10 (29.4)	13 (22.4)	0.45
Absent	24 (70.6)	45 (77.6)	
Massive peri villous fibrin deposition (MPVFD)			
Present	11 (32.4)	4 (6.9)	<0.01
Absent	23 (67.6)	54 (93.1)	
Infarction			
Present	7 (20.6)	5 (8.6)	0.12
Absent	27 (79.4)	53 (91.4)	
Chronic deciduitis			
Present	1 (2.9)	7 (12.1)	0.25

Continued.

	FGR onset, N (%)		P value
	Early	Late	
Absent	33 (97.1)	51 (87.9)	
Villous fibrosis			
Present	1 (2.9)	1 (1.7)	1.00
Absent	33 (97.1)	57 (98.3)	
Avascular villi			
Present	1 (2.9)	0 (0.0)	0.37
Absent	33 (97.1)	58 (100)	

*Fisher's exact test

DISCUSSION

In the present study, the mean maternal age was 27±4.7 years. There was no significant correlation found between maternal age and the timing of onset of FGR. Similarly, Vişan et al and Günyel et al found no significant age differences between FGR and uncomplicated pregnancies.[3,4]

Most FGR cases in this study were seen in the upper class (53.3%), contrasting with other studies like Blumenshine et al in 2010 that found FGR more common in lower socioeconomic groups.[5] The higher prevalence of comorbidities such as diabetes, hypertension, and obesity among individuals in higher socioeconomic groups may help explain this observed difference. In our study FGR was more common in nulliparous cases (64%). This aligns with Shinde et al.'s findings, where FGR was 36% in nulliparous women compared to 16% multiparous women.[6]

In our study, subclinical hypothyroidism was the most common comorbidity (25%) followed by preeclampsia (22.8%) and gestational hypertension (13%). Other conditions included chronic hypertension (10.9%), intrahepatic cholestasis (9.8%) and diabetes. Vishwa et al found anaemia (88%) and pregnancy-induced hypertension (44%) as major causes of FGR.[7]

Early-onset FGR in our study was linked to chronic hypertension, preeclampsia and doppler abnormalities, often requiring early termination and leading to higher neonatal morbidities. In our study 7 out of 11 neonatal mortalities were seen in cases of hypertension and preeclampsia. Although no significant association between timing of onset of FGR and preeclampsia was found. In contrary, studies by M. Kovo et al and Diane et al found that hypertensive women, especially those not on antihypertensives, face higher risks of poor neonatal outcomes.[8,9] In Kovo et al study, the recurrence rate of FGR was 33%. In contrast, our study found a lower recurrence rate of 7.7% in multigravida cases.[8]

Our study found that doppler abnormalities such as AEDF/REDF were strongly associated with early-onset FGR, poor neonatal outcomes, low birth weights, higher NICU admissions and increased neonatal deaths (31.8%). Abnormal cerebroplacental ratio (CPR <1) was also linked to poor outcomes. Non-reassuring NST/CTG occurred in 31.9% of cases and was similarly associated with adverse neonatal outcomes. These findings align with studies by Rashmi et al, Shmueli et al, and Istvan et al, which highlighted the role of doppler and CTG abnormalities in predicting adverse perinatal outcomes.[10-12]

FGR alone is not an indication for caesarean section. However, primary caesarean section may be considered in severe FGR cases where successful vaginal delivery with optimal neonatal outcome is unlikely. On analysing our study, the most common indication for caesarean section were non-reassuring NST/CTG and acute fetal distress (29.6% and 25% respectively). A study done by Rashmi et al it was found that reduced AFI and abnormal doppler had a high rate of operative delivery.[10] In a study Sudhakara et al, 64% of FGR had caesarean section which similar to results seen in our study (i.e. 57%).[13]

Our study provides significant insights into the placental histopathological findings associated with fetal growth restriction and their impact on neonatal outcomes. In our study, the most common placental histopathological findings were syncytial knots (88%), fibrinoid necrosis (85.9%), and diffuse calcifications (52.2%). Syncytial knots, markers of placental ischemia, were more frequent compared to other studies done by Visan et al (75%) and Shinde et al (48.6%).[3,6] MPVFD (16.3%), villous fibrosis (2.2%) and avascular villi (1.1%) was seen in lesser cases when compared to studies done by Visan et al (75%) and Shinde et al (48.6%).[3,6] Similar findings were reported in studies by Novac et al and Mardi et al but their direct role in causing FGR remains uncertain.[14,15]

In our study, early-onset FGR was significantly associated with placental pathologies such as MPVFD, chorioamnionitis, and diffuse dystrophic calcification, which were linked to poor neonatal outcomes. This aligns with Istvan et al.'s findings on early-onset FGR.[12] Other histopathological features like infarction, syncytial knots, avascular villi, and fibrinoid necrosis were found in both early and late-onset FGR. Regarding birth weight, no significant association was found with placental histopathology in our study, contrasting with Erum et al.'s findings that massive peri villous fibrin deposition and decreased vascularity were linked to lower birth weight and poor neonatal outcomes.[16]

In our study, placental lesions like infarction and MPVFD were more prevalent in hypertensive mothers. In a 2023

study by Daniela et al, placental lesions like retroplacental haemorrhage, villous maturation, infarctions and calcium associated fibrin deposition were more found in FGR with preeclampsia compared to normotensive pregnancies suggesting maternal malperfusion.[17] Similarly, Kovo et al found that FGR due to preeclampsia was associated with more maternal placental vascular lesions and worse neonatal outcomes.[8]

In a study by Raymond et al, non-infectious chronic villitis of unknown etiology is seen in obese mothers and carries a tendency of recurrence in a more severe form in subsequent pregnancies; though uncertain but treatment with low dose aspirin is advocated in such cases.[15] In our study, non-infectious chronic villitis of unknown origin was not seen to be associated with obese women.

In our study, intrauterine fetal demise and neonatal death were associated with placental pathologies like MPVFD, intravascular thrombi, infarction and chorioamnionitis. Similar findings were reported by Gunyel et al, Silver et al and Spinillo et al highlighting the link between FGR, stillbirth and placental insufficiency.[4,18,19]

One of the strengths of this study is the detailed histopathological examination of placental samples, which allowed for a comprehensive analysis of the different types of placental pathology associated with FGR. The cross-sectional design enabled us to capture a wide range of FGR cases providing a robust dataset for analysis. However, the study is not without limitations. The small sample size may limit the generalizability of our findings and the cross-sectional design prevents drawing conclusions about causality. Additionally, while we were able to identify associations between placental pathology and neonatal outcomes, further longitudinal studies are needed to explore the long-term effects of these findings on childhood development.

CONCLUSION

Histopathological evaluation can provide valuable insights into the etiology of FGR and help clinicians in improving the outcome of subsequent pregnancies with counselling and necessary treatment. The findings from our study underscore the importance of placental examination in cases of FGR. Four major types of placental histopathologies are maternal vascular malperfusion (decidual arteriopathy, agglutinated villi, increased syncytial knots, intervillous fibrin deposition and villous infarcts), Fetal vascular malperfusion, Villitis of unknown etiology, massive peri villous fibrinoid deposition (MPVFD) all commands screening for thrombophilia and managed with appropriate treatment for better neonatal outcomes in subsequent pregnancy.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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