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# Understanding Intrauterine Growth Restriction through Placental Histopathology: A Regional Perspective from Central India

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**Abstract:** ***Introduction: Intrauterine Growth Restriction (IUGR) is a major obstetric complication associated with increased perinatal morbidity and mortality. The condition arises from multifactorial causes, among which placental insufficiency plays a central role. Histopathological evaluation of the placenta can offer critical insights into the underlying mechanisms of fetal growth restriction. This study aimed to analyze the correlation between placental pathology and birth weight in IUGR cases at a tertiary care center in Central India. Materials and Methods: A cross-sectional observational study was conducted over 2.5 years involving 100 pregnant women with ultrasonographically diagnosed IUGR. Clinical data including maternal age, BMI, parity, socioeconomic status, and hypertensive disorders were collected. Antenatal Doppler indices such as umbilical artery pulsatility index (PI) and cerebroplacental ratio (CPR) were recorded. After delivery, placentas were grossly and histologically examined for lesions including infarction, chorangiosis, syncytial knotting, and fetal thrombotic vasculopathy. Statistical analysis was performed using Chi-square and Fisher's exact tests. Results: Maternal age ≤20 years and BMI <18.5 were significantly associated with low birth weight (p=0.0001 and p=0.001 respectively). Hypertensive disorders of pregnancy were present in 64% of cases and showed a strong correlation with low birth weight (p=0.0001). Placental weight <450g was significantly associated with birth weight <2.5 kg. Key histopathological lesions—placental infarction, chorangiosis, syncytial knotting, and fetal thrombosis—were significantly more frequent in low birth weight cases (p<0.05). Cumulative analysis showed that specific combinations of placental pathologies were highly predictive of adverse neonatal outcomes (p=0.0001). Conclusions: Placental pathology is a significant determinant of fetal growth outcomes in IUGR. Early identification of maternal risk factors and timely placental assessment can guide better clinical management and improve neonatal health.***

**Keywords:** Intrauterine growth restriction, placental pathology, birth weight, syncytial knotting, chorangiosis, fetal thrombosis, maternal risk factors.

## 1. Introduction

Intrauterine Growth Restriction (IUGR), also known as Fetal Growth Restriction (FGR), is a prominent obstetric concern marked by the inability of the fetus to attain its full genetic growth potential.[1] Globally, it affects an estimated 6–10% of pregnancies and is a leading contributor to perinatal illness and death. In the Indian context, where disparities in maternal nutrition, access to healthcare, and antenatal monitoring are widespread, the prevalence of IUGR is significantly elevated.[2–5] It is responsible for up to one-third of low birth weight cases, underlining its substantial influence on neonatal health. Infants affected by IUGR face a higher likelihood of stillbirth and immediate postnatal complications such as low blood sugar, difficulty maintaining body temperature, and respiratory issues. Additionally, they are more prone to long-term problems, including delays in neurological development and chronic conditions in adulthood.[6]

Fetal development is governed by a complex interplay of maternal, fetal, and placental factors. Maternal elements such as overall health, nutritional status, and conditions like hypertension, anemia, or infections can negatively influence fetal growth. On the fetal side, genetic disorders, congenital abnormalities, and intrauterine infections may also contribute. Among these, placental insufficiency is considered the most frequent and central cause of IUGR in singleton pregnancies. The placenta plays a crucial role in facilitating the exchange of oxygen, nutrients, and waste products between the mother and fetus. Any alteration in its structure, blood flow, or function can hinder fetal development. Therefore, examining placental pathology can offer key insights into the underlying causes and severity of growth restriction.[7–9]

Numerous histopathological features have been associated with IUGR, such as signs of maternal vascular underperfusion, placental infarctions, excessive syncytial knot formation, deposition of fibrin around villi, and thrombotic lesions in fetal vessels. Doppler ultrasound studies, particularly those examining the umbilical and middle cerebral arteries, often reflect these placental changes and help in identifying at-risk fetuses. However, the occurrence and type of these lesions can vary depending on maternal health conditions, regional practices, and population characteristics. It is also important to distinguish between infants who are small due to normal biological variation (constitutionally small) and those who are pathologically growth-restricted, as this distinction impacts prenatal care and postnatal management strategies.[7,8,10,11]

Recent research underscores the significance of placental analysis in clarifying the pathogenesis of IUGR and informing care in future pregnancies. Placentas from growth-restricted infants often show reduced mass, structural deviations, and indications of prolonged oxygen deprivation. Common lesions include chronic villitis, underdeveloped distal villi, and increased capillarity (chorangiosis), all suggestive of ongoing placental dysfunction. The extent and combination of these abnormalities are closely linked to the severity of fetal compromise and poor neonatal outcomes. Evaluating these patterns in a localized context enables healthcare providers to better identify modifiable risks,

anticipate complications, and deliver informed counseling.[6–9,11,12]

Although substantial research on placental pathology and IUGR exists from high-income countries, data from Central India remain sparse. This region, characterized by unique demographic, socioeconomic, and healthcare variables, presents an opportunity to investigate the association between placental findings and fetal health in a context-specific manner. Notably, there is a lack of studies correlating histological placental features directly with birth weight and maternal factors within this diverse population, highlighting the need for regionally focused investigations.

## 2. Materials and Methods

This study was a cross-sectional, observational investigation conducted in the Department of Obstetrics and Gynaecology at a tertiary care centre in Central India. The study aimed to explore the relationship between placental pathology and intrauterine growth restriction (IUGR) with reference to birth weight. The research was carried out over a period of 2.5 years and included a total of 100 participants who met the inclusion criteria.

Women who presented with diagnosed intrauterine growth restriction based on ultrasonographic criteria were enrolled in the study after obtaining informed written consent. Inclusion criteria comprised women aged above 18 years with singleton pregnancies diagnosed with IUGR via antenatal ultrasound. Exclusion criteria included mothers with known fetal congenital anomalies, multifetal gestations, and intrauterine fetal demise (IUFD), as these conditions could confound the interpretation of placental findings.

Participant recruitment was performed using purposive sampling. Each eligible participant underwent thorough clinical evaluation, and relevant maternal history including age, body mass index (BMI), parity, socioeconomic status, and any pre-existing or pregnancy-induced conditions such as hypertensive disorders or anemia was recorded. Gestational age was determined based on last menstrual period and confirmed by early ultrasonography.

All enrolled cases underwent antenatal Doppler ultrasonography. Parameters such as umbilical artery pulsatility index (PI) and cerebroplacental ratio (CPR) were documented to assess uteroplacental and fetoplacental circulation. Fetal growth was monitored using estimated fetal weight (EFW), which was plotted against standard WHO fetal growth charts. At birth, neonatal birth weight was measured and recorded along with the mode and gestational age at delivery.

Following delivery, the placenta was collected in the labor room or operating theatre and preserved in 10% formalin. Detailed gross examination of the placenta was performed to assess weight, shape, cord insertion, and any visible lesions such as infarctions or calcifications.[9,11,13] The placental tissue was then processed and sent to the Department of Pathology for histopathological evaluation.

Histopathological analysis included assessment of various lesions such as infarction, perivillous fibrin deposition, chorangiosis, fetal thrombotic vasculopathy, syncytial knotting, chorioamnionitis, and calcific foci. Each placenta was examined using standard staining techniques under light microscopy by qualified pathologists.

All collected data were compiled using a structured proforma and subsequently entered into Microsoft Excel for analysis. Statistical analysis was performed using IBM SPSS software version 24.0. Qualitative variables were expressed as proportions, while quantitative variables were presented as means with standard deviations. The association between placental pathology and birth weight, as well as other maternal variables, was analyzed using the Chi-square test or Fisher's exact test where appropriate. A p-value of less than 0.05 was considered statistically significant, while a p-value of less than 0.001 was considered highly significant.

Ethical clearance for the study was obtained from the Institutional Ethics Committee and the Board of Research Studies (BORS). The study was also registered and approved by the Maharashtra University of Health Sciences (MUHS), Nashik. All participants were informed about the purpose of the study, and their consent was obtained in the vernacular language prior to enrollment.

## 3. Results

**Table 1:** Maternal and Demographic Factors vs. Birth Weight

| Maternal Factor | Category | <2.5 kg n (%) | ≥2.5 kg n (%) | Total n (%) | p-value | Significance |
|---|---|---|---|---|---|---|
| Age (years) | ≤20 | 8 (100%) | 0 (0%) | 8 (8%) | 0.0001 | Significant |
| | 21–30 | 55 (83.3%) | 11 (16.7%) | 66 (66%) | | |
| | 31–36 | 21 (80.8%) | 5 (19.2%) | 26 (26%) | | |
| BMI | <18.5 | 12 (92.3%) | 1 (7.7%) | 13 (13%) | 0.001 | Significant |
| | 18.5–22.9 | 57 (91.9%) | 5 (8.1%) | 62 (62%) | | |
| | 23–24.9 | 11 (68.8%) | 5 (31.2%) | 16 (16%) | | |
| | ≥25 | 4 (44.4%) | 5 (55.6%) | 9 (9%) | | |
| SES | Lower Middle | 6 (60%) | 4 (40%) | 10 (10%) | 0.08 | Not Significant |
| | Upper Lower | 58 (85.3%) | 10 (14.7%) | 68 (68%) | | |
| | Lower | 20 (90.9%) | 2 (9.1%) | 22 (22%) | | |
| Parity | Primigravida | 33 (86.8%) | 5 (13.2%) | 38 (38%) | 0.54 | Not Significant |
| | Multigravida | 51 (82.3%) | 11 (17.7%) | 62 (62%) | | |
| Gestational Age | <37 weeks | 43 (100%) | 0 (0%) | 43 (43%) | – | Significant |
| | ≥37 weeks | 41 (68.3%) | 19 (31.7%) | 60 (60%) | | |

The analysis of maternal and demographic factors vs. birth weight revealed several significant associations. Maternal age showed a strong correlation with birth weight, as all mothers aged ≤20 years delivered low birth weight babies (<2.5 kg), which was statistically significant (p=0.0001). Among mothers aged 21–30 years, 83.3% had babies weighing <2.5 kg, while 80.8% of mothers aged 31–36 years also delivered low birth weight infants. Maternal BMI was another significant factor influencing birth weight (p=0.001), with 92.3% of underweight mothers (BMI <18.5) and 91.9% of mothers with a BMI of 18.5–22.9 delivering low birth weight babies. However, mothers with higher BMI (≥25) showed a higher percentage of babies with birth weight ≥2.5 kg (55.6%). Socioeconomic status (SES) did not exhibit a statistically significant association with birth weight (p=0.08), although lower socioeconomic groups had a higher proportion of low birth weight babies. Parity was also not significantly associated with birth weight (p=0.54), although primigravida mothers had a slightly higher percentage of low birth weight babies (86.8%) compared to multigravida mothers (82.3%). Gestational age showed a strong correlation, as 100% of preterm deliveries (<37 weeks) resulted in low birth weight babies, whereas 31.7% of term deliveries (≥37 weeks) had normal birth weight babies, establishing a significant relationship with birth weight.

**Table 2:** Pregnancy and Delivery Characteristics vs. Birth Weight

| Clinical Factor | Category | <2.5 kg n (%) | ≥2.5 kg n (%) | Total n (%) | p-value | Significance |
|---|---|---|---|---|---|---|
| HDP | Present | 61 (95.3%) | 3 (4.7%) | 64 (64%) | 0.0001 | Significant |
| | Absent | 23 (63.9%) | 13 (36.1%) | 36 (36%) | | |
| Significant History | Present | 31 (81.6%) | 7 (18.4%) | 38 (38%) | 0.61 | Not Significant |
| | Absent | 53 (85.5%) | 9 (14.5%) | 62 (62%) | | |
| Gestation Type | Preterm | 38 (95%) | 2 (5%) | 40 (40%) | 0.01 | Significant |
| | Term | 46 (76.7%) | 14 (23.3%) | 60 (60%) | | |
| Mode of Delivery | Vaginal | 30 (75%) | 10 (25%) | 40 (40%) | 0.04 | Significant |
| | LSCS | 54 (90%) | 6 (10%) | 60 (60%) | | |

The analysis of pregnancy and delivery characteristics vs. birth weight highlighted several important associations. Hypertensive disorders of pregnancy (HDP) were significantly correlated with low birth weight (p=0.0001), with 95.3% of mothers with HDP delivering babies weighing <2.5 kg, compared to only 63.9% of mothers without HDP. Significant maternal history, which included conditions such as severe anemia, cardiac disease, or other medical comorbidities, did not show a statistically significant relationship with birth weight (p=0.61), although 81.6% of mothers with a significant history delivered low birth weight babies. Gestation type was a critical factor, with 95% of preterm births resulting in babies weighing <2.5 kg, whereas 23.3% of term deliveries had birth weights ≥2.5 kg, demonstrating a statistically significant association (p=0.01). Mode of delivery was also significantly associated with birth weight (p=0.04), where 75% of babies born through vaginal delivery and 90% of those born via lower segment cesarean section (LSCS) had low birth weight. These findings emphasize that maternal hypertensive disorders, preterm deliveries, and mode of delivery significantly influence birth weight outcomes in IUGR cases.

**Table 3:** Placental Parameters vs. Birth Weight

| Placental Factor | Category | <2.5 kg n (%) | ≥2.5 kg n (%) | Total n (%) | p-value | Significance |
|---|---|---|---|---|---|---|
| Placental Weight | <400g | 2 (100%) | 0 (0%) | 2 (2%) | – | Significant |
| | 400–450g | 61 (91%) | 6 (9%) | 67 (67%) | | |
| | >450g | 21 (67.7%) | 10 (32.3%) | 31 (31%) | | |
| Umbilical PI | <1 | 36 (42.9%) | 48 (57.1%) | 84 (84%) | 0.63 | Not Significant |
| | ≥1 | 5 (31.2%) | 11 (68.8%) | 16 (16%) | | |
| CPR | <1 | 26 (86.7%) | 4 (13.3%) | 30 (30%) | 0.63 | Not Significant |
| | >1 | 58 (82.9%) | 12 (17.1%) | 70 (70%) | | |

The analysis of placental parameters vs. birth weight revealed key findings related to placental weight, umbilical artery pulsatility index (PI), and cerebroplacental ratio (CPR). Placental weight showed a significant association with birth weight, where 100% of placentas weighing <400g were associated with low birth weight babies (<2.5 kg). Among placentas weighing 400–450g, 91% of cases resulted in low birth weight babies, whereas 67.7% of placentas weighing >450g were associated with low birth weight, while 32.3% had normal birth weight (≥2.5 kg). Although placental weight was significantly correlated with birth weight, Doppler indices such as umbilical PI and CPR did not show statistically significant associations. Umbilical PI values <1 were associated with low birth weight in 42.9% of cases and normal birth weight in 57.1% of cases, whereas PI values ≥1 were linked to low birth weight in 31.2% of cases and normal birth weight in 68.8% of cases (p=0.63, not significant). Similarly, CPR values <1 showed 86.7% association with low birth weight, whereas 13.3% had normal birth weight. In contrast, CPR values >1 showed 82.9% association with low birth weight and 17.1% with normal birth weight, which was also not statistically significant (p=0.63). These results underscore that while placental weight plays a crucial role in determining birth weight, Doppler indices such as umbilical PI and CPR may not independently predict birth weight outcomes in IUGR cases.

**Table 4:** Placental Histopathology vs. Birth Weight

| Lesion Type | Status | <2.5 kg n (%) | ≥2.5 kg n (%) | Total n (%) | p-value | Significance |
|---|---|---|---|---|---|---|
| Infarction | Present | 62 (93.9%) | 4 (6.1%) | 66 (66%) | 0.0001 | Significant |
| | Absent | 22 (64.7%) | 12 (35.3%) | 34 (34%) | | |
| Chorangiosis | Present | 68 (91.9%) | 6 (8.1%) | 74 (74%) | 0.0001 | Significant |
| | Absent | 16 (61.5%) | 10 (38.5%) | 26 (26%) | | |
| Fetal Thrombosis | Present | 68 (88.3%) | 9 (11.7%) | 77 (77%) | 0.03 | Significant |
| | Absent | 16 (69.6%) | 7 (30.4%) | 23 (23%) | | |
| Syncytial Knotting | Present | 65 (95.6%) | 3 (4.4%) | 68 (68%) | 0.0001 | Significant |
| | Absent | 19 (59.4%) | 13 (40.6%) | 32 (32%) | | |
| Chorioamnionitis | Present | 27 (93.1%) | 2 (6.9%) | 29 (29%) | 0.11 | Not Significant |
| | Absent | 57 (80.3%) | 14 (19.7%) | 71 (71%) | | |
| Calcific Foci | Present | 79 (84%) | 15 (16%) | 94 (94%) | 0.96 | Not Significant |
| | Absent | 5 (83.3%) | 1 (16.7%) | 6 (6%) | | |

The analysis of placental histopathology vs. birth weight demonstrated several significant associations between placental lesions and fetal outcomes. Placental infarction was significantly correlated with low birth weight (p=0.0001), with 93.9% of cases showing infarction resulting in birth weights <2.5 kg. In contrast, 64.7% of cases without infarction also had low birth weight, while 35.3% achieved normal birth weight. Chorangiosis, observed in 74% of cases, was also significantly associated with low birth weight (p=0.0001), where 91.9% of cases with chorangiosis had low birth weight, compared to 61.5% in the absence of chorangiosis. Fetal thrombotic vasculopathy showed a statistically significant association with low birth weight (p=0.03), with 88.3% of affected cases resulting in birth weight <2.5 kg. Increased syncytial knotting, a marker of chronic placental hypoxia, was seen in 68% of cases and was strongly associated with low birth weight (p=0.0001), with 95.6% of these cases resulting in babies weighing <2.5 kg. However, chorioamnionitis and calcific foci did not show a statistically significant correlation with birth weight. Chorioamnionitis was present in 29% of cases, with 93.1% of these cases resulting in low birth weight, but the association was not significant (p=0.11). Similarly, calcific foci, present in 94% of cases, did not show a significant correlation with birth weight (p=0.96). These findings emphasize that infarction, chorangiosis, fetal thrombosis, and syncytial knotting are key histopathological markers significantly associated with low birth weight in IUGR cases.

**Table 5:** Cumulative Placental Pathologies vs. Clinical Outcomes

| Pathology Code Combination | <2.5 kg n (%) | ≥2.5 kg n (%) | Total n (%) | Notes |
|---|---|---|---|---|
| 1,2,3,5,6 | 40 (88.9%) | 5 (11.1%) | 45 (45%) | Most common pattern |
| 1,2,3,4,5,6 | 10 (100%) | 0 (0%) | 10 (10%) | All cases were low birth weight |
| 1,3,4,5,6 | 5 (100%) | 0 (0%) | 5 (5%) | All cases were low birth weight |
| 1,5,6 | 0 (0%) | 4 (100%) | 4 (4%) | All were normal birth weight |
| Others | 29 (80.6%) | 7 (19.4%) | 36 (36%) | Mixed group |
| Total | 84 (84%) | 16 (16%) | 100 (100%) | p = 0.0001 (Highly Significant) |

The analysis of **cumulative placental pathologies vs. clinical outcomes** highlighted a strong association between the presence of multiple placental lesions and birth weight outcomes, with a highly significant p-value (p=0.0001). Among the various combinations of placental pathologies, the most common pattern was the combination of pathologies 1, 2, 3, 5, and 6, observed in 45% of cases, where 88.9% of these cases resulted in birth weights <2.5 kg, while only 11.1% had birth weights ≥2.5 kg. Another combination, involving pathologies 1, 2, 3, 4, 5, and 6, was seen in 10% of cases, all of which resulted in low birth weight, demonstrating a 100% association with birth weight <2.5 kg. Similarly, the combination of 1, 3, 4, 5, and 6 was observed in 5% of cases, and all these cases also had low birth weight. Interestingly, the combination of pathologies 1, 5, and 6 was associated exclusively with normal birth weight (≥2.5 kg) in 100% of cases, though it was only observed in 4% of cases. The remaining 36% of cases exhibited a mixed pattern of pathologies, where 80.6% resulted in low birth weight and 19.4% had normal birth weight. Overall, 84% of the total cases had birth weight <2.5 kg, while only 16% had birth weight ≥2.5 kg. These findings underscore that the cumulative burden of multiple placental pathologies significantly contributes to adverse fetal outcomes, with certain combinations of lesions showing a higher predictive value for low birth weight in IUGR cases.

## 4. Discussion

Intrauterine growth restriction (IUGR) remains a significant challenge in obstetric practice due to its multifactorial etiology and association with adverse perinatal outcomes.[2] This study analyzed 100 cases of IUGR to assess the correlation between placental pathology and birth weight at a tertiary healthcare center in Central India. The findings reaffirmed and extended results from several previous investigations conducted in different settings, offering valuable insights into the pathophysiology of IUGR.

Maternal age and body mass index (BMI) demonstrated a statistically significant association with birth weight in the present study. A higher incidence of IUGR was noted in mothers aged ≤20 years, a finding consistent with the study by Fikree et al.[14] (1994), who reported that young maternal age and short interpregnancy intervals were independent risk factors for fetal growth restriction. Maternal undernutrition, reflected by a BMI <18.5, was also associated with a higher proportion of low birth weight babies, aligning with findings by Rondo et al.[15] (1997), who demonstrated a strong link

between maternal malnutrition and impaired fetal growth in a Brazilian cohort. These results emphasize the critical role of maternal nutritional and demographic factors in determining fetal growth potential.

Maternal comorbidities, particularly hypertensive disorders of pregnancy (HDP), were significantly associated with IUGR in this study, with 95.3% of women with HDP delivering babies weighing less than 2.5 kg. This finding aligns with the results of Guo et al.[16] (2023), who reported a higher incidence of maternal vascular malperfusion and infarction in hypertensive pregnancies, resulting in compromised placental function and fetal growth restriction. Similarly, Thekkedathu et al.[17] (2015) observed that preeclampsia and chronic hypertension significantly contributed to IUGR by inducing placental vascular abnormalities. Additionally, preterm delivery was more common among IUGR cases in this study, a finding consistent with Zeitlin et al.[18] (2000), who demonstrated that growth-restricted fetuses often undergo spontaneous or medically indicated preterm delivery due to compromised uteroplacental circulation.

Placental parameters provided further insights into the pathophysiology of IUGR. Low placental weight (<450g) was significantly correlated with low birth weight, reflecting reduced functional reserve and nutrient exchange capacity. These findings are consistent with Khajuria et al.[19] (2019), who reported that placentas from IUGR pregnancies were significantly smaller and had a higher incidence of infarction and villous abnormalities compared to normal pregnancies. Mukhopadhyay et al. [20](2021) also noted that reduced placental weight and size were significantly associated with fetal compromise in growth-restricted pregnancies. Interestingly, although abnormal Doppler indices such as elevated umbilical artery pulsatility index (PI) and altered cerebroplacental ratio (CPR) were observed in some cases, they did not demonstrate a statistically significant correlation with birth weight in this study. This contrasts with findings by Veerbeek et al.[21] (2014), who reported a high correlation between abnormal Doppler findings and adverse fetal outcomes in IUGR pregnancies. The lack of significant association in the present study may be attributed to variability in gestational age at Doppler evaluation or transient compensatory mechanisms.

The histopathological analysis of placentas in this study revealed key patterns that correlated with poor fetal outcomes. Placental infarction was observed in 66% of cases and was significantly associated with low birth weight ($p<0.0001$), mirroring the findings of Salafia et al.[22] (1992), who demonstrated that placental infarcts were present in 63% of IUGR cases and were associated with chronic placental insufficiency. Similarly, So-Young Park et al.[23] (2002) identified placental infarcts, villous fibrosis, and increased syncytial knotting as consistent histopathological features in IUGR placentas. Chorangiosis was observed in 74% of IUGR cases in the current study, reflecting a compensatory increase in vascular density in response to chronic hypoxia. This finding parallels the work of Vafaei et al.[24] (2021), who reported a higher prevalence of chorangiosis in placentas from pregnancies complicated by IUGR, often associated with maternal conditions such as preeclampsia and gestational diabetes. Increased syncytial knotting, indicative of chronic hypoxia and placental aging, was found in 95.6% of cases, aligning with the results of İskender-Mazman et al.[25] (2014), who identified increased syncytial knots as a hallmark of IUGR placentas.

Fetal thrombotic vasculopathy was detected in 77% of cases in this study, corroborating the observations of Nigam et al.[26] (2014), who reported a higher incidence of fetal thrombotic vasculopathy and obstructive vasculopathy in growth-restricted placentas. Although chorioamnionitis and calcific foci were present in some cases, they did not demonstrate a statistically significant correlation with birth weight, consistent with findings by William Mifsud et al.[27] (2014), who noted that these changes were more commonly incidental or related to advancing gestational age rather than IUGR. An important observation in this study was the significant association between the cumulative number of placental pathologies and both low birth weight and hypertensive disorders. This finding aligns with the study by Spinillo et al.[28] (2019), who demonstrated that the presence of multiple placental lesions, particularly massive perivillous fibrin deposition and infarctions, increased the likelihood of adverse neonatal outcomes in IUGR pregnancies.

Despite the robust findings, this study has certain limitations. The sample size was relatively small (n=100), and the study was conducted at a single tertiary care center, which may limit the generalizability of the results. Furthermore, a control group of normal placentas was not included, which would have allowed for a more comprehensive comparison of histopathological features between normal and IUGR placentas. Genetic and environmental factors that may influence fetal growth could not be evaluated in this study. Additionally, inter-observer variability in histopathological interpretation was not assessed, which may introduce potential biases in pathological findings. Future studies with larger sample sizes, inclusion of control groups, and multicentric data can provide more robust conclusions and help refine clinical management strategies for IUGR.

## 5. Conclusions

This study highlights the significant influence of maternal, clinical, and placental factors on birth weight outcomes in intrauterine growth restriction (IUGR) cases. Younger maternal age, low body mass index (BMI), and hypertensive disorders of pregnancy (HDP) were associated with a higher risk of low birth weight, emphasizing the impact of maternal health on fetal growth. Preterm deliveries further compounded this risk by introducing the dual challenge of growth restriction and prematurity.

Placental weight showed a strong correlation with birth weight, reflecting its critical role in nutrient and oxygen exchange. However, Doppler indices such as umbilical artery pulsatility index (PI) and cerebroplacental ratio (CPR) did not independently predict birth weight outcomes. Placental histopathology revealed that lesions such as infarction, chorangiosis, fetal thrombotic vasculopathy, and increased syncytial knotting were significantly associated with low birth weight.

The cumulative presence of multiple placental pathologies further increased the likelihood of adverse birth weight outcomes, with certain combinations of lesions showing a stronger correlation. These findings underscore the importance of identifying maternal risk factors and conducting detailed placental examinations to better understand the pathophysiology of IUGR and improve perinatal outcomes.

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