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Original Research Article

A STUDY OF FETAL GROWTH RESTRICTION-EARLY DIAGNOSIS AND FETO-MATERNAL OUTCOME

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Abstract

Background: FGR has emerged as a global health challenge, imposing a major social and economic burden upon caretakers and caregivers. Its far-reaching consequences post-birth are not restricted to the neonatal period and early infancy alone, but also include the entire phases of childhood and adolescence, and extend into adulthood. This study aimed to estimate the incidence rate of FGR in our obstetric population and investigate the risk factors and neonatal outcomes in growth-restricted fetuses. **Materials and Methods:** A retrospective case-control study was conducted between 30 cases and 30 controls over ten months. Data was collected from the medical records department, labour room and neonatology registers, and institutional software system SOLACE. The parameters analysed were general and obstetric history and examination findings, risk factors, and obstetric and perinatal outcomes. The Delphi consensus was applied in the ultrasound criterion. Statistical analysis was done using STRATA 14.2. **Result:** The incidence of FGR in the obstetric population during this study period was 3.7%. Although the mean age and parity distribution were comparable in both groups, the case group saw a significant aggregate of un-booked, referred patients (P<0.0001) with a substantial high-risk population (96.66%vs36.6%) (P<0.0001). Mothers in the case group experienced higher operative deliveries (P<0.0001), preterm labor (P<0.001), and adverse neonatal outcomes (P<0.0001). There were no perinatal deaths. **Conclusion:** Maternal co-morbidities and poor antenatal care were the compounding factors for premature deliveries, low birth weight infants, and perinatal morbidity. Prompt diagnosis and early intervention can optimize outcomes.

INTRODUCTION

Fetal growth restriction (FGR) is a condition wherein the fetus fails to attain its inherent growth potential, probably due to a detrimental intra-uterine environment related to multiple factors- including uteroplacental, metabolic, and constitutional.[1]

FGR is the most prevalent environmental cause of immune system impairment and one of the major contributors to perinatal mortality and morbidity worldwide. Its neonatal and long-term consequences include morbidities related to the cardiovascular, respiratory, and neurological systems among many others. [2,3] Therefore, this entity, if undetected, may sound like a death knell to the hopes of many expectant couples.

This entire period of intrauterine development does, however, also provide us with a large window of opportunity for identifying fetuses at risk for growth restriction through clinical surveillance including color Doppler ultrasonography. The subsequent adoption of preventive and or therapeutic measures can lead to an improvement in perinatal outcome and prevention of several related diseases in the future.[4]

Objectives

1. To estimate the incidence of FGR and compare the risk factors between the cases and controls
2. To assess the neonatal outcome and measure its association across major maternal co-morbidities.

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MATERIALS AND METHODS

Study design: A hospital-based retrospective case-control study was conducted in the Department of Obstetrics and Gynaecology at a 1000-bed, teaching, tertiary care institute in Western India.
Study period: January 2023 to October 2023 over 10 months.
Sampling Method: Purposive sampling
Source of data: Patient records in the medical records department (MRD), labor room, and admission registers.
The data collected included chronological age, parity, a detailed past obstetric history including, the presence of hypertensive disorders of pregnancy (HDP), small for gestation (SGA), stillbirths, or a medical history of diabetes mellitus (DM) or hypertension.
Information regarding the present pregnancy included gestational age (GA) at diagnosis of FGR (either from documented LMP or a first-trimester dating scan), a clinical profile including details of Doppler, co-existent morbidities, and obstetric and perinatal outcomes. The Delphi/ISUOG guidelines were considered for the classification of FGR.

Early FGR: GA < 32 weeks, in the absence of congenital anomalies	**Late FGR: GA ≥ 32 weeks, in the absence of congenital anomalies**
AC/EFW < 3rd centile or UA-AEDF	AC/EFW < 3rd centile
Or at least two out of three of the following	Or at least two out of three of the following
1. AC/EFW < 10th centile combined with	1. AC/EFW < 10th centile
2. UtA-PI > 95th centile and/or	2. AC/EFW crossing centiles >2 quartiles on growth centiles
3. UA-PI > 95th centile	3. CPR < 5th centile or UA-PI > 95th centile

(Growth centiles are non-customized centiles. AC, fetal abdominal circumference; AEDF, absent end-diastolic flow; CPR, cerebroplacental ratio; EFW, estimated fetal weight; GA, gestational age; PI, pulsatility index; UA, umbilical artery; UtA, uterine artery. Reproduced from Gordjin et al).[5]
According to the Delphi consensus, FGR is to be considered in the presence of a single solitary component which includes AC/EFW<3rd centile or UD-AEDF. In the absence of a solitary component, two contributory components are to be considered. A similar number of patients were taken as controls after matching demography and gestational age.
Sampling technique: The incidence of FGR among pregnant women is reported to be around 5% in published literature. Considering a 5% level of significance and 2% allowable error on either side, the minimum estimated sample size was calculated as 474. However, a sample population of 810 (total deliveries) was obtained during the study period.

Study Population
Inclusion Criteria
Obstetric patients between the gestational age of 28-40weeks
Cases: 30 patients with documented FGR. EFW <3rd percentile for GA and/or Doppler changes in uterine and/or umbilical and/or MCA
Controls: 30 patients with matched demography and gestational age. EFW 10th to 95th centile for GA. Normal doppler values
Exclusion criteria
women with multifetal pregnancies, and congenital anomalies.
Statistical analysis: Descriptive statistics mean (SD), and frequency (%) were used to depict the baseline profile of the study participants. Independent sample t-test and chi-square test were used to compare continuous and categorical variables. A P value <0.05 was considered statistically significant.
Operational definitions: SGA: An EFW or birth weight below the 10th percentile for gestational age
LBW - World Health Organization (WHO): weight at birth below 2500 grams or 5.5 pounds regardless of gestational age.
Very low birth weight <1500gms.
Extremely low birth weight < 1000 grams

RESULTS

A retrospective study was conducted over 10 months, during which the total deliveries were 810. The incidence of FGR was therefore calculated as 3.7% or 38/1000 live births. Among the study participants, 30 were cases and another 30 were taken as matched controls.
The demographic characteristics of our study population are presented in Table 1. Notably, the mean age and parity distribution among the cases and controls were largely comparable. The case group had statistically significant un-booked antenatal patients (86.66%) as compared to the controls (6.66%) (P <0.00001, OR 91.0000). Similarly, the number of referred patients was also higher among cases (P<0.00001, OR 56.0000), which also witnessed a markedly elevated rate of high-risk pregnancy attendance (96.66 % vs 36.66%) (P value <0.00001, OR 50.0909). [Table 1] further details the comorbidities associated with the study subjects. Notably, a higher prevalence of anemia (76.66% vs 26.66%) (P< 000107) and HDP (60% vs 3.33 %) (P < 0.00001) were observed among the cases compared to the controls.
The clinical characteristics of both groups of our study populace are tabulated in [Table 2]. Preterm admissions dominate group 1(70%) as against group 2(3.33%); these values are statistically significant (P< 0.00001) (OR 67.6667). The mean birth weight was 1.480 kg (group 1) and 3.03 kg (group 2) respectively. LSCS prevailed over group 1 (96.66 %), while the control subjects experienced more vaginal births (86.66%) (OR188.5000). Adverse neonatal

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outcome was frequently observed in group 1 (93.33%) as against group 2 (6.66%) (OR196.0000). Antenatal steroid coverage was necessitated in 28 mothers in Group 1 as opposed to one in Group 2 (P <0.00001).

The fetal Doppler changes before termination of pregnancy are presented in [Table 3]. These were as follows- REDF at GA <32 weeks was found in 100%; REDF AND AEDF± CPR<1- 50% were observed each between 32-34 weeks. At a GA (34-36.6) weeks, a CPR<1 was seen in 91.66%, while 8.33% had only EFW<3C. At a GA≥37 weeks, 44.44% had CPR values<1, while 55.55% had only an EFW<3C.

The distribution of fetal birthweight is outlined against each comorbidity among cases in [Table 4]. The hypertensive versus normotensive subjects showed a marked difference in fetal weight (P = 0.0246). No significant difference could however be elicited for the other comorbidities. The total number of NICU admissions, along with the mean duration of stay, is also depicted alongside the fetal birth weight. A significant difference was found in the HDP vs normotensive category (P = 0.0246). The infants with very low birth weight and extremely low birth weight numbered 36.6% and 10% respectively and 96.66% of the newborns had a birth weight <1 centile for the gestational age.

The short-term adverse neonatal consequences are outlined in [Table 5]. Of the 27 babies who availed of NICU admissions, 70.37% were detected with respiratory distress syndrome (RDS). Around 40.74% developed neonatal jaundice, while NEC (necrotizing enterocolitis) was diagnosed in 22.22%. Sepsis was seen in 18.51% and hypoglycemia was noticed in 14.81%. IVH (intraventricular hemorrhage) was detected in 7.40%. The neonatal outcome on discharge was satisfactory in 93.33%. Around 7.40% went DAMA (discharged against medical advice) due to financial and social reasons and were lost to follow-up.

Table 1: Demography and Distribution of comorbidities in the study population

Sr No.	Characteristics	Group 1 [N 30]	Group 2 [N 30]	Total	P value	Odds Ratio [OR] with 95%CI
1	Mean age (years) SD	28±4.7	26.73±4.6		0.2918	
2	Parity				0.605577	0.765 (0.277 to 2.111)
	Primipara	14 (46.66) %	16 (53.33%)	30		
	Multipara	16 (53.33) %	14 (46.66) %	30		
3	BMI				0.559305	1.480 (0.445 to 4.453)
	18.5-22.9 kg/m2	7 (23.33%)	9 (30%)			
	<18.5 / >23 kg/m2	23(76.66%)	21 (70%)			
4	Antenatal Care				<0.00001	91.000 (15.3558 to 539.2765)
	Booked	4 (13.33%)	28 (93.33%)	32		
	Un-booked	26 (86.66%)	2 (6.66%)	28		
5	Total Referrals	24 (80%)	2 (6.66%)	26	<0.00001	56.000 (10.3263 to 303.6915)
6	High-Risk Pregnancies	29 (96.66%)	11(36.66%)	40	<0.00001	50.090 (5.968 to 420.380)
7.	Comorbidities					
	Maternal Anemia	23(76.66%)	7 (23.33 %)	30	0.000107	9.035 (2.802 to 29.134)
	Maternal HDP	18(60 %)	1 (3.33 %)	19	<0.00001	43.500 (5.205 to 363.535)
	Oligohydramnios	12(40 %)	0	12		
	Maternal Hypothyroidism	5(16.66)	4[13.33%]	9	0.717	1.300 (0.312 to 5.404)
	GDM	3(10 %)	0	3		

Statistical test: Chi-square test, independent t-test

Table 2: Distribution of clinical outcome among the cases and controls

	Characteristics	Cases Group 1 N (30)	Controls Group 2 N (30)	P value	Odds ratio with 95% confidence interval
1	Gestational age at termination				
	Preterm (Total)	21 (70%)	1 (3.33%)	<0.00001	67.666 (7.953 to 575.699)
	Very Preterm (<32 weeks)	3	0		
	Moderate Preterm (32-34 weeks)	6	0		
	Late Preterm (>34-36.6 weeks)	12	1		
2	Mode of delivery				
	LSCS	29(96.66 %)	4(13.33%)	<0.00001	188.500 (19.781 to 1796.286)
	Vaginal Delivery	1(3.33 %)	26(86.66 %)		
3	Mean birth weight (kg) SD	1.48±0.38	3.03±0.23		
4	NICU Admission	28 (93.33%)	2(6.66%)	<0.00001	196.000 (25.772 to 1490.560)
5	Antenatal Corticosteroids	28	1	<0.00001	

Statistical tests: Chi-square test

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Table 3: Fetal Doppler changes among the cases.

Gestational age (GA)	Fetal doppler changes	N (30)
<32 weeks (N 3)	REDF	3(100%)
32-34 weeks (N 6)	REDF	3 (50%)
	AEDF ± CPR <1	3 (50%)
>34-36.6 weeks (N 12)	CPR <1	11(91.66%)
	EFW < 3C	1(8.33%)
≥37 weeks (N 9)	CPR< 1	4(44.44%)
	EFW <3C	5 (55.55%)

REDF: Reversed end diastolic flow
AEDF; Absent end diastolic flow
CPR: Cerebroplacental ratio
EWF: Effective fetal weight

Table 4: Distribution of fetal birth weight and admission to the NICU unit concerning the co-morbidities amongst the case population

Sr no.	Maternal comorbidities	Fetal birth weight		Mean fetal weight Kg ± SD	P value
		(1.5-2.0) kg	≤1.5kg		
1	Anaemia				P=0.8514
	Anaemic (N 23)	9(39.13%)	13(56.52%)	1.487±0.4	
	Non-anaemic (N 7)	3(42.85%)	4(57.14%)	1.455±0.19	
2	Hypertension				P=0.0001
	HDP (N 18)	4(22.22%)	14(77.77%)	1.271±0.3	
	Normotensive (N 12)	9(75%)	2(16.66%)	1.794±0.2	
3	Thyroid				P=0.7917
	Hypothyroid (N 5)	3(60%)	2(40%)	1.536±0.1	
	Euthyroid (N 25)	10(40%)	14(56%)	1.466±0.4	
4.	Booking status				P=0.3175
	Un-booked ANC (N 26)			1.45±0.4	
	Booked ANC (N 4)			1.66±0.2	
	Maternal comorbidities	Total no. of NICU admissions		Nicu's duration of stay	
				mean	P-value
1.	Anaemia				P = 0.9000
	Anaemic (N 23)	20(86.95%)		18.25±13.6	
	Non-anaemic (N 7)	7(100%)		19±12.6	
2.	Hypertension				P= 0.0246
	HDP (N18)	17(94.44 %)		22.76±13.4	
	Normotensive (N 12)	10(83.33%)		11.1±9.6	
3.	Thyroid				P = 0.9363
	Hypothyroid (N 5)	5(100%)		18±11.1	
	Euthyroid (N 25)	22(88%)		18.54±13.8	

Statistical test: Chi-square test
HDP: Hypertensive disorders of pregnancy
ANC: Antenatal care
NICU: Neonatal intensive care unit

Table 5: Short-term neonatal consequences among both groups

Sr No	Adverse Neonatal Consequences	Group 1 N 27	Group 2 N2	Total	P-value
	IDENTITIES				P<0.00001 OR 126.00 (19.50-814.01)
1	RDS				
	Mild	4(14.81 %)	0	4	
	Moderate	10(37.03 %)	0	10	
	Severe	5(18.51 %)	0	5	
	Total	19 (70.37 %)	0	19	
2	Jaundice	11(40.74 %)	1	12	
3	Necrotizing enterocolitis	6(22.22 %)	0	6	
4	Sepsis	5(18.51%)	0	5	
5	Hypoglycaemia	4(14.81 %)	1	5	
6	Intraventricular hemorrhage	2(7.40 %)	0	0	

Statistical test: Chi-square test

DISCUSSION

Fetal weight < 10th centile or < 2 SD for that gestational age and population is a term widely used for SGA and only appertains to a fetal size, without the inclusion of its growth velocity.[6] FGR on the other hand refers to foetuses that pathologically acquire growth velocity < 3rd centile for that population.[5] It denotes poor somatic growth and compensated blood supply to vital organs. The overlapping definitions of FGR and SGA as a fetal or

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birth weight <10th centile are now limited to low-resource settings only.[7]

FGR affects around 5-10% of uncomplicated pregnancies.[8] The incidence rates of FGR in underdeveloped or developing countries outnumber those in developed ones by an approximate ratio of 6:1, with many births occurring outside institutional premises or at home. Asian figures account for about 75% of the burden.[9] In many instances, the deliveries may escape documentation and may not paint a true picture of the condition. Springer et al have quoted incidence rates of 5.2% FGR in their study.[10] In our study, the incidence rate was 3.7%, compared to 2.13% in a study conducted by Sinha et al. [11]

The Multifactorial diverse etiology of FGR relates to maternal/fetal/ environmental and placental factors. The non-placental subgroup includes genetic anomalies, congenital infections, and inborn errors of metabolism. The placental subgroup mainly consists of those with underlying maternal diseases which incorporate hypertension, both chronic and gestational (30-40%), diabetes (10-20%), vascular/renal/ cardiac/respiratory, and hematological. [12,13]

More than 50% of patients with FGR and stillbirth have an associated placental pathology, mostly MVM (Maternal vascular malperfusion), the result of defective remodeling in early pregnancy. [14,15] Normal remodeling incorporates trophoblastic invasion of maternal spiral arteries up to the inner 1/3rd myometrium, followed by subsequent denudation of the vessel's lamina and smooth muscles. This forms the uteroplacental circulation in a normal pregnancy during the early trimesters, with vessel dilatation increasing 5-10fold. This phenomenon, when absent, allows the entry of turbulent jets of flow into the intervillous space, causing widespread damage to the delicate architecture of the villous network.

Thus, any factor causing uteroplacental hypoperfusion, hypoxia, and a compromised intrauterine environment leads to FGR. This abnormal remodeling, linked to maternal malnutrition and HDP, may also be idiopathic in up to 60% of patients. [3,12,15] An inherent defect in progesterone-regulated decidualization combined with an adverse immune response is also postulated.[16]

Diagnosis of this condition necessitates an accurate estimation of gestational age; ideally confirmed by 1st or early 2nd trimester ultrasound. Routine symphysis–fundal height (SFH) measurement may recognize a lag of ≥3cm after 24 weeks during serial measurements, but is moderately sensitive and highly specific, for low-risk pregnant women with a normal BMI.[17] The regular modalities of in-utero monitoring may not be able to detect FGR, nor enable the identification of the nub of progression of the state of hypoxia into a stillbirth.[18]

Early onset FGR is seen in 20-30% of all FGR pregnancies, and has a 70% link to HDP and/ PE while late-onset FGR is seen in 70-80% of all FGR pregnancies and has an approximate 10% association with HDP. [13,19]

A Doppler study of the umbilical artery is stated to be the gold standard in providing both diagnostic and prognostic information, and is recommended by the RCOG as a primary means of monitoring growth-restricted fetuses; in high-risk pregnancies, it can lead to a reduction in both perinatal morbidity and mortality.[20] There is however a difference of opinion regarding the importance of amniotic fluid assessment between the RCOG and ACOG. [20,21]

High-risk socio-demographic attributes include extremities of age, low BMI, an Asian ethnic background, nulliparity or twin pregnancies, and substance abuse. Maternal anemia and malnutrition, poor BMI, hypertensive disorders of pregnancy [HDP], and parasitic infestations such as malaria have been held culpable in low-resource countries.[20] Kozuki et al noticed a preponderance of SGA in nulliparous mothers younger than 18 years.[22] In our study, the mean maternal age of cases was 28 years which was comparable to a study conducted by Unterschider et al wherein they observed a mean age of 30 years.[21] Primiparous women dominated a case study by Springer et al (51.8%) and Spencer et al (68%) while no such observation was made in our study.[23,24]

Our study results portray statistically significant maternal anemia in the case group (76.66%). In contrast, Dapkekar P et al noted only 29.6% of anemic subjects in their case study.[25] The postulated mechanisms involve a hypoxia-induced rise in noradrenaline levels leading to maternofoetal stress and CRH synthesis, which causes the fetal cortisol to rise, which in turn is implicated as a fetal growth inhibitor.[26]

MVM is related to as many as 25-30% of cases of both pre-eclampsia and FGR and raises the risk of these two conditions by around 4.5fold.[17] Hypertensive disorders of pregnancy were seen in 60% of the cases in our study ($P<0.0001$) as compared to 22.2% observed by Dapkekar P et al and 61.7% by Thekkedathu et al respectively. [25,27]

An important challenge in FGR management is related to prediction and prevention. Many literature studies have determined poor antenatal care as a major cause of LBW or intrauterine growth restriction (IUGR). [28,29] Similar observations were made in our study as well. A high number of mothers in the case group demonstrated inadequate and infrequent antenatal attendance. A past obstetric history of HDP and an SGA/FGR infant was documented in 23.33 % and 26.66% of the case group. Motghare et al. observed a correlation between a history of abortion and FGR in the present pregnancy.[29] A higher recurrence rate was noted by Shrestha et al among mothers with a previous history of FGR.[30]

The recent Canadian guidelines state that in the 2nd and 3rd trimesters, neither UtA nor UA Doppler assessments are effective predictors in low-risk

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pregnancies. Technology-dependent maternal serum markers are impractical and may preclude FGR of late-onset, and the modifying consequences of gestational age. [18,31] The role of serum placental growth factor (PLGF) levels, or as a ratio to soluble fms-like tyrosine kinase-1 (sFlt-1), is primarily to aid in identifying underlying placental factors in pregnancies that are already affected by growth restriction.[31]

Amongst the proposed Interventions for FGR prevention, low-dose aspirin is only recommended for pregnancies at high risk for pre-eclampsia. Low-molecular-weight heparin is not indicated for the prevention of recurrent FGR. [7,32]

Available treatment options are not effective in curtailing or reversing established FGR. Restriction of physical activity is of unproven benefit. Several drugs such as Statins/ Nitric Oxide donors/ Proton pump inhibitors/ Nanoparticles etc are in various phases of pre-clinical or clinical trials due to their anti-inflammatory, antioxidant, and angiogenetic actions. Whereas the use of sildenafil citrate is controversial, few studies have quoted the benefits of arginine supplementation in improving the growth of these fetuses. [15,32,33]

The timing and mode of delivery have to be individualized depending on the gestational age, degree of fetal compromise, and the severity of FGR.[33] Uteroplacental Doppler is the most powerful predictor of clinical deterioration among the recommended investigations.

For FGR occurring before 32 weeks, assessing the umbilical artery to ductus venous (UA/DV) ratio is the standard recommendation. However, in cases of late FGR (beyond 32 weeks), the PI of the umbilical artery serves as a less reliable indicator of fetal hypoxemia.[34,35] While MCA can provide insights into cerebral vasodilation—a surrogate marker for hypoxia—the cerebroplacental ratio (CPR) demonstrates greater sensitivity to hypoxia, facilitating critical decision-making in management.[36] In our study, a CPR value of less than 1 was observed in 71.42% of cases delivering after 34 weeks of gestation, highlighting the importance of this metric in assessing fetal well-being during late FGR.

A Caesarean section is deemed both safe and beneficial in cases of absent or reversed end-diastolic flow. In our study, the rate of cesarean deliveries was notably significant (P=0.0001). Previous research by Thekkedathu et al. and Shrestha et al. also reported elevated cesarean rates, at 82.19% and 80%, respectively. [27,30] Furthermore, we observed a statistically significant disparity in NICU admissions, with rates of 93.33% versus 6.66%, alongside a notable difference in the duration of hospital stays between the two groups. This finding is consistent with the work of Unterscheider et al., which reported a NICU admission rate of 28% and a median stay of 13 days.[21] The study by Sinha et al. reported a perinatal mortality rate of 1.92 per 1,000 live births, with 5% of cases resulting in stillbirths and 8% in neonatal deaths.[11] The predominant causes of neonatal mortality identified were sepsis and respiratory distress syndrome, each accounting for 44.4% of the cases. In contrast, Unterscheider et al. documented a higher perinatal mortality rate of 7.2 per 1,000 births.[21] Remarkably, our study observed no instances of perinatal mortality, largely attributable to the high-quality intensive care provided to our patients. The application of antenatal corticosteroids (ACS) and magnesium sulfate for neuroprotection in managing preterm births is well-established in medical literature. In our study, 93.33% of mothers received ACS, while magnesium sulfate was prescribed in two cases. This proactive approach underscores these interventions' critical role in enhancing premature infants' health prospects.

Limitations: The main limitation of this study is its retrospective nature which renders it prone to selection bias and the possibility of missed data. The sample size was small and the duration of the study was short, hence the results may not be representative of the general population.

CONCLUSION

The implications of fetal growth restriction (FGR) extend profoundly across societal and healthcare frameworks. Our study elucidates the extensive impact of FGR, particularly within high-risk antenatal populations. We documented a significant correlation between FGR and increased risks of preterm birth, elevated rates of operative interventions, and adverse neonatal outcomes. Additionally, our findings underscore the detrimental effects of comorbidities such as anemia and hypertensive disorders of pregnancy (HDP) on fetal birth weight, highlighting the necessity for heightened levels of antenatal care.

Emerging research endeavors are exploring innovative avenues, including ultrasound analysis of wave pulsations in specific regions of the umbilical artery and the potential of intrauterine therapies such as intra-placental gene transfer of human insulin-like growth factor-1. Until groundbreaking research tools come to fruition, established clinical monitoring practices and dedicated imaging modalities remain pivotal in establishing the foundation for a healthier future society.

Ethical Considerations: To address the ethical issues, we ensured that absolute confidentiality of patient records was maintained. The study commenced following approval from the institutional ethics committee. A waiver of consent was availed as the study was retrospective and only involved material data. The authors declare that there is no conflict of interest and no funding was obtained.

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