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Original Research Article

A study of various fetal doppler indices to assess outcome in growth restricted fetuses: a comparative and cross-sectional study

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ABSTRACT

Background: Fetal growth restriction (FGR) poses significant risks of adverse perinatal outcomes. Accurate surveillance is essential to distinguish pathologically growth-restricted fetuses from constitutionally small infants and to optimize the timing of delivery.
Methods: A comparative cross-sectional observational study was conducted at Rajiv Gandhi Medical College among 118 singleton pregnancies (≥32 weeks) with diagnosed FGR. Comprehensive doppler velocimetry of the umbilical artery (UA), middle cerebral artery (MCA), and ductus venosus (DV) was performed and categorized into doppler grades 0 to 5. Primary outcomes evaluated were perinatal morbidity, neonatal mortality, APGAR scores, and birth weight.
Results: Normal doppler (grade 0) was present in 98 (83.1%) participants, while 20 (16.9%) exhibited abnormal doppler indices. Abnormalities in UA, MCA, and DV were strongly associated with adverse outcomes ($p<0.001$). Grade 0 doppler showed a high association with normal neonatal outcomes (odds ratio=24.14, $p<0.001$). Conversely, severe doppler deterioration (grade 4 and grade 5) was significantly associated with perinatal morbidity ($p=0.037$) and neonatal mortality ($p=0.011$), respectively. Infants with abnormal doppler had significantly lower gestational age at delivery (35.35±2.30 vs 37.22±0.58 weeks, $p=0.001$), lower APGAR scores at 5 minutes (6.10±3.09 vs 8.53±0.61, $p<0.001$), and reduced birth weights (1.67±0.44 kg vs 2.09±0.14 kg, $p<0.001$).
Conclusions: Fetal doppler surveillance effectively stratifies risk in growth-restricted fetuses. Abnormal doppler indices correlate strongly with hemodynamic compromise and adverse perinatal outcomes, providing critical evidence to guide timely clinical intervention prior to irreversible fetal acidemia.

Keywords: Fetal growth restriction, Doppler velocimetry, Umbilical artery, Middle cerebral artery, Ductus venosus, Perinatal outcome

INTRODUCTION

Intrauterine growth restriction (IUGR), currently termed FGR, represents one of the most significant and complex challenges in contemporary obstetrics.[1] Historically defined as an estimated fetal weight (EFW) or abdominal circumference (AC) below the 10th percentile for gestational age and sex, FGR encompasses a heterogeneous population of fetuses.[2] Epidemiological estimates indicate that the incidence of FGR ranges between 3% and 27% globally, with a disproportionately high burden in low- and middle-income countries owing to maternal nutritional deficiencies and sub-optimal antenatal healthcare access.[3,4] It is critical to clinically distinguish FGR from small for gestational age (SGA). While SGA refers strictly to statistical biometry where birth weight falls below the 10th percentile, it includes constitutionally small but otherwise healthy fetuses. In contrast, true FGR denotes a pathological state in which the fetus fails to achieve its genetically predetermined growth potential due to maternal, placental, or environmental insults.[1,2] Consequently, identifying pathological FGR within the

broader SGA population is paramount to prevent adverse outcomes.

Ultrasound biometry remains the foundational screening modality to assess fetal dimensions, yet biometry alone lacks the functional sensitivity required to differentiate physiological smallness from pathological growth restriction.[5] Pathological FGR is broadly categorized into early-onset (<32 weeks) and late-onset (≥32 weeks) patterns, or morphologically into symmetric and asymmetric FGR.[1] Asymmetric FGR, characterized by an elevated head circumference to abdominal circumference (HC:AC) ratio, typically stems from late-onset uteroplacental insufficiency, giving rise to the preferential preservation of brain growth known as the 'brain-sparing' phenomenon.[5] Symmetric FGR usually presents earlier in gestation and is often associated with early cellular hyperplasia deficits driven by intrinsic fetal anomalies, chromosomal abnormalities, or congenital infections.[1,24] Regardless of etiology, fetuses suffering from true FGR face heightened vulnerability to severe perinatal complications.

The pathophysiological cascade of FGR is primarily driven by chronic intrauterine hypoxia, nutrient deprivation, and progressive fetal acidemia1. Placentas affected by FGR demonstrate defective spiral artery remodelling, villous vascular obliteration, and reduced syncytiotrophoblast surface area, which severely restricts oxygen and glucose transport across the syncytium.[8,10] In response to cellular hypoxia, the fetus downregulates oxidative metabolism, shifting toward anaerobic glycolysis. This metabolic alteration leads to systemic lactate accumulation and progressive metabolic acidemia, which ultimately threatens cellular integrity and organ function.[10] Clinically, growth-restricted neonates experience elevated rates of iatrogenic prematurity, intrapartum asphyxia, necrotizing enterocolitis (NEC), respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), and bronchopulmonary dysplasia.[7] Long-term epidemiological follow-up further links FGR to neurodevelopmental delays, impaired motor function, and the adult development of cardiovascular disease, metabolic syndrome, and type 2 diabetes.[8,25]

To mitigate these adverse outcomes, doppler velocimetry has emerged as an indispensable non-invasive tool for monitoring fetal hemodynamic adaptation.[13,14] Sound waves emitted from a transducer undergo frequency shifts upon reflection from circulating erythrocytes within major fetal vessels, providing detailed quantitative indices such as the pulsatility index (PI), resistance index (RI), and systolic-to-diastolic (S/D) ratio.[14] Surveillance primarily focuses on three critical cardiovascular structures: the umbilical artery (UA), middle cerebral artery (MCA), and ductus venosus (DV). The umbilical artery reflects downstream placental vascular impedance; elevated PI or absent/reversed end-diastolic velocity (AEDV/REDV) directly correlates with extensive villous arterial obliteration and elevated risks of fetal demise.[15-20] The MCA evaluates cerebral autoregulation; vasodilation within the MCA resulting in a decreased PI signals preferential blood flow to the fetal brain during early metabolic compensation.[15,25] Combining these parameters into the cerebroplacental ratio (CPR=MCA-PI / UA-PI) significantly enhances sensitivity for detecting subtle hemodynamic compromise.[23,25] Finally, the ductus venosus, a direct shunt conveying oxygenated blood to the heart, provides insight into right atrial pressure and myocardial performance. Abnormalities in the DV waveform-specifically an elevated PI or an absent/reversed 'a' wave during atrial contraction represent severe metabolic decompensation and imminent cardiac failure.[21,22]

Despite global advances in fetal surveillance, optimizing the precise timing of delivery in FGR remains a delicate clinical challenge, balancing the risk of intrauterine demise against the hazards of premature birth. The present study was undertaken to evaluate various fetal Doppler indices in established growth-restricted fetuses, correlate these hemodynamic parameters with clinical and neonatal outcomes, and provide rigorous evidence to refine antenatal decision-making.

METHODS

This comparative cross-sectional observational study was conducted in the Department of Obstetrics and Gynaecology in collaboration with the Department of Radiodiagnosis at Rajiv Gandhi Medical College and Chhatrapati Shivaji Maharaj Hospital, Kalwa, Thane, Maharashtra, India. The study was carried out over an 18-month period from January 2023 to June 2024 following formal ethical approval from the Institutional Ethics Committee (IEC). Written informed consent was obtained from all participating pregnant women prior to enrollment.

The study population comprised pregnant women attending the ANC Outpatient Department or admitted to the maternity wards of Rajiv Gandhi Medical College with clinically and biometrically diagnosed fetal growth restriction. The sample size was calculated based on an estimated national FGR prevalence of 27% in India, utilizing a standard formula for cross-sectional studies with a 95% confidence interval and a relative precision of 8%, yielding a sample size of 118 participants recruited via convenient sampling.

Eligible participants included women with singleton pregnancies having confirmed FGR with a gestational age of 32 weeks or greater, where FGR was defined as an EFW or AC below the 10th percentile for gestational age based on ultrasound biometry. Exclusion criteria were strictly enforced to prevent confounding factors; women with multifetal gestations, fetal congenital structural or chromosomal malformations, uncertain gestational dating, non-FGR pregnancies, gestations under 32 weeks, or those refusing written consent were excluded from the study. Upon enrolment, thorough clinical evaluations were

conducted for all participants, incorporating detailed sociodemographic data, obstetric history, menstrual history, accurate gestational dating based on first-trimester ultrasound, medical comorbidities, physical examination, and systemic evaluations. Fetal biometric parameters including BPD, HC, AC, FL, and AFI were systematically recorded. Comprehensive doppler velocimetry of fetal blood vessels was performed using high-resolution ultrasound machines equipped with color doppler capabilities. PI measurements were obtained from the umbilical artery, middle cerebral artery, and ductus venosus according to standardized diagnostic protocols. For the umbilical artery, spectral doppler waveforms were obtained from a free loop of the umbilical cord in the absence of fetal breathing movements. The middle cerebral artery was sampled in a transverse axial section of the fetal head at the level of the circle of Willis, maintaining an angle of incidence close to zero degrees. The ductus venosus was localized in a mid-sagittal or longitudinal section of the fetal abdomen, sampling the high-velocity turbulent jet at its inlet from the umbilical vein.

Based on doppler findings, fetal hemodynamic status was categorized into standardized sequential grades reflecting progressive severity: grade 0 (normal doppler velocimetry); grade 1 (increased UA PI >95th percentile without other vessel abnormalities); grade 2 (increased UA PI with decreased MCA PI and cerebroplacental ratio reversal <1.0); grade 3 (absent or reversed end-diastolic flow in UA with decreased MCA PI); grade 4 (absent or reversed end-diastolic flow in UA with increased MCA PI due to loss of cerebral autoregulation); and grade 5 (Altered or reversed 'a' wave in ductus venosus).

All participants were followed up prospectively through labor and delivery to document fetal and neonatal outcomes. Key recorded outcome measures included clinical gestational age at presentation, gestational age at delivery, mode of delivery (spontaneous/induced vaginal delivery versus lower segment cesarean section), neonatal birth weight, APGAR scores at 1 minute and 5 minutes, occurrence of intrauterine fetal death (IUD), stillbirth, perinatal morbidity (including admission to the neonatal intensive care unit (NICU), respiratory distress syndrome, necrotizing enterocolitis, hypoxic-ischemic encephalopathy, and sepsis), and neonatal death.

Statistical analysis was executed using SPSS version 21.0 (IBM Corp., Armonk, NY) and Microsoft Excel 2021. Descriptive data for continuous variables were tested for normality using the Shapiro-Wilk test; parametric continuous variables were presented as mean±standard deviation (SD), while non-parametric continuous variables were evaluated using the Mann-Whitney U test. Categorical variables were expressed as absolute frequencies and percentages. Associations between qualitative variables and Doppler gradings were analyzed using Pearson's Chi-Square test or Fisher's Exact test as appropriate, with p values less than 0.05 considered statistically significant. Strength of association was measured using Cramer's V, and risk estimation was calculated using odds ratios (OR) with 95% confidence intervals.

RESULTS

The study evaluated 118 pregnant women with established fetal growth restriction at Rajiv Gandhi Medical College. sociodemographic and parity distribution showed that 64 participants (54.2%) were multigravida, while 54 participants (45.8%) were primigravida. Out of 118 participants, doppler grading revealed normal velocimetry (grade 0) in 98 cases (83.1%), whereas 20 cases (16.9%) presented with abnormal doppler indices spanning grades 1 through 5.

Comparative statistical analysis between normal and abnormal doppler groups demonstrated significant clinical variations. The mean gestational age at doppler examination was comparable between groups (34.03±1.37 vs 33.70±1.34 weeks, p=0.292), confirming uniform baseline surveillance timing. However, clinical gestational age based on fundal height was significantly lower in the abnormal doppler group (28.90±3.28 vs 31.04±1.68 weeks, p=0.004), illustrating more severe intrauterine lag. Gestational age at delivery was significantly reduced in the abnormal doppler group (35.35±2.30 vs 37.22±0.58 weeks, p=0.001). Furthermore, birth weight was markedly reduced in neonates with abnormal doppler velocimetry (1.67±0.44 kg vs 2.09±0.14 kg, p<0.001).

Fetal doppler grading distribution among the 118 participants revealed: grade 0 (normal) in 98 (83.1%), grade 1 in 7 (5.9%), grade 2 in 4 (3.4%), grade 3 in 1 (0.8%), grade 4 in 3 (2.5%), and grade 5 in 5 (4.2%). Overall perinatal outcomes evaluated across 118 pregnancies recorded 2 intrauterine fetal deaths (1.7%) and 1 stillbirth (0.8%), all occurring in fetuses with grade 5 doppler changes. Excluding these 3 pre-delivery mortalities, live-born neonatal outcomes (n=115) demonstrated no adverse outcome in 98 neonates (83.1%), perinatal morbidity in 14 neonates (11.9%), and early neonatal death in 3 neonates (2.5%).

Statistical association analysis demonstrated that Grade 0 Doppler was highly protective, showing a significant positive association with normal neonatal outcome (pearson chi-square=39.500, p<0.001, odds ratio=24.14, Cramer's V=0.579). Conversely, presence of grade 4 doppler was significantly associated with perinatal morbidity (p=0.037, odds ratio=17.17), while grade 5 doppler exhibited a strong statistical association with neonatal mortality (p=0.011, odds ratio=13.88). Evaluating specific vascular beds individually revealed robust correlations with neonatal outcomes. As detailed in Table 4, abnormal umbilical artery doppler (grades 1-5) was associated with an adverse outcome rate of 58.9% (47.1% morbidity, 11.8% mortality) compared to only 7.1% in normal UA doppler (p<0.001). Abnormal MCA

doppler (cerebral redistribution) showed 80.0% adverse outcome rate (60.0% morbidity, 20.0% mortality, p<0.001). Abnormal ductus venosus doppler in live-born neonates resulted in a 100% adverse outcome rate (50.0% morbidity, 50.0% mortality, p<0.001), in addition to accounting for all pre-delivery deaths (IUDs and stillbirth).

Table 1: The demographic characteristics, clinical parameters, gestational ages, delivery modes, APGAR scores, and birth weights of the study cohort stratified by doppler normality.

Clinical parameter	Overall cohort (n=118)	Normal doppler (n=98)	Abnormal doppler (n=20)
Parity (multigravida/primigravida)	64 (54.2%)/54 (45.8%)	52 (53.1%)/46 (46.9%)	12 (60.0%)/8 (40.0%)
Gestational age at doppler (weeks)	33.97±1.36	34.03±1.37	33.70±1.34
Clinical gestational age (weeks)	30.68±2.18	31.04±1.68	28.90±3.28
Gestational age at delivery (weeks)	36.91±1.28	37.22±0.58	35.35±2.30
Mode of delivery (vaginal/LSCS)	89 (75.4%)/29 (24.6%)	81 (82.7%)/17 (17.3%)	8 (40.0%)/12 (60.0%)
Neonatal birth weight (kg)	2.02±0.27	2.09±0.14	1.67±0.44

Table 2: The clinical comparison and statistical significance across continuous surveillance variables.

Variables	Normal doppler (mean±SD)	Abnormal doppler (mean±SD)	Statistical test	P value
Gestational age at doppler (wks)	34.03±1.37	33.70±1.34	Mann-Whitney U	0.292
Clinical gestational age (wks)	31.04±1.68	28.90±3.28	Mann-Whitney U	0.004*
Gestational age at delivery (wks)	37.22±0.58	35.35±2.30	Mann-Whitney U	0.001*
APGAR score at 1 minute	8.54±0.71	6.05±3.30	Mann-Whitney U	0.001*
APGAR score at 5 minutes	8.53±0.61	6.10±3.09	Mann-Whitney U	<0.001*

*Statistically significant.

Table 3: The detailed breakdown of perinatal outcomes according to specific doppler severity grades.

Outcome category	Grade 0	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5	Total
Intrauterine death	0	0	0	0	0	2	2
Stillbirth	0	0	0	0	0	1	1
Normal neonatal outcome	91	5	2	0	0	0	98
Perinatal morbidity	6	2	2	1	2	1	14
Neonatal death	1	0	0	0	1	1	3
Total participants	98	7	4	1	3	5	118

Table 4: Outcomes based on umbilical artery, middle cerebral artery, and ductus venosus doppler findings.

Vessel examined	Doppler finding	Normal outcome	Perinatal morbidity	Neonatal death	Chi-Square (P value)
Umbilical artery (UA)	Normal (n=98)	91 (92.9%)	6 (6.1%)	1 (1.0%)	30.893 (<0.001*)
	Abnormal (n=17)	7 (41.2%)	8 (47.1%)	2 (11.8%)	
Middle cerebral artery	Normal (n=105)	96 (91.4%)	8 (7.6%)	1 (1.0%)	38.743 (<0.001*)
	Abnormal (n=10)	2 (20.0%)	6 (60.0%)	2 (20.0%)	
Ductus venosus (DV)	Normal (n=113)	98 (86.7%)	13 (11.5%)	2 (1.8%)	21.650 (<0.001*)
	Abnormal (n=2)	0 (0.0%)	1 (50.0%)	1 (50.0%)	

*Statistically significant.

DISCUSSION

The primary objective of antenatal care in fetal growth restriction is the timely identification of fetal hemodynamic compromise to prevent intrauterine demise and severe acidemic injury while avoiding unnecessary premature delivery.[1,6] The present study conducted at Rajiv Gandhi Medical College demonstrates that fetal Doppler velocimetry serves as a powerful diagnostic and prognostic tool, directly reflecting the continuum of fetal adaptation to placental dysfunction. The findings demonstrate a strong progressive correlation between worsening doppler severity grades and adverse neonatal outcomes, consistent with established physiological principles of fetoplacental circulation.[8,12]

In our study cohort of 118 growth-restricted fetuses, 83.1% exhibited normal doppler velocimetry (grade 0), whereas 16.9% presented with abnormal doppler indices. Infants with normal doppler velocimetry demonstrated favorable outcomes, with 92.9% experiencing no adverse neonatal events (odds ratio=24.14, p<0.001). This finding aligns with observations by Seyam et al and Ghosh et al who reported that growth-restricted fetuses with normal umbilical artery impedance have a benign clinical course, allowing safe expectant management until term.[16,17] Conversely, fetuses with abnormal doppler parameters in our cohort experienced significantly higher rates of prematurity (mean delivery at 35.35±2.30 vs 37.22±0.58 weeks, p=0.001), reduced birth weight (1.67±0.44 kg vs 2.09±0.14 kg, p<0.001), lower APGAR scores at 5 minutes (6.10±3.09 vs 8.53±0.61, p<0.001), and elevated rates of cesarean delivery (60.0% vs 17.3%). These findings corroborate the results of Nanthakomon et al and Jang et al confirming that doppler abnormalities reflect underlying hypoxia that impairs fetal functional reserve during labor.[19,20]

Evaluating specific blood vessels highlights the sequential pathophysiological progression of FGR. The umbilical artery provides a direct measure of downstream villous vascular resistance. In the study, abnormal UA doppler was present in 17 cases (14.8%) and was significantly associated with adverse neonatal outcomes (47.1% morbidity, 11.8% mortality, p<0.001). As documented by Baschat et al and Morris et al elevated UA pulsatility index occurs when over 50% of placental tertiary villous arterioles are obliterated, progressing to absent or reversed end-diastolic flow when obliteration exceeds 70%.[8,11,22] The observed morbidity in severe UA abnormalities agrees with findings by Ozyuncu et al who demonstrated that absent/reversed UA diastolic flow dramatically increases the odds of perinatal death, RDS, and NICU admission.[27]

Cerebral vascular autoregulation during fetal hypoxia manifests as vasodilatation of the middle cerebral artery, reducing MCA PI and reversing the cerebroplacental ratio (CPR<1.0). In the cohort, 10 participants (8.7%) exhibited abnormal MCA doppler, which was strongly associated with perinatal morbidity (60.0%) and mortality (20.0%, p<0.001).

This brain-sparing adaptation reflects early hemodynamic compensation. However, as noted by Stampalija et al and Johnson et al prolonged cerebral redistribution is not entirely benign it indicates chronic hypoxemia and correlates with an increased risk of adverse neurodevelopmental outcomes and intrapartum distress.[28,29]

The ductus venosus represents the final cardiovascular threshold before complete metabolic decompensation. In our study, abnormal DV flow velocimetry (grade 5, altered or reversed 'a' wave) was observed in 5 cases. Notably, DV abnormalities accounted for all 2 intrauterine fetal deaths and 1 stillbirth in the cohort, while live-born infants with abnormal DV exhibited 50% morbidity and 50% mortality (p<0.001). This critical finding reflects right atrial hypertension and cardiac failure secondary to severe acidemia, fully supporting the conclusions of Baschat et al, Turan et al and Senat et al. Alterations in the DV 'a' wave signify profound acidemia and impending demise, establishing DV Doppler as the ultimate parameter for timing delivery in severe early-onset FGR.[8,18,28,30]

The study has several limitations that should be acknowledged. First, the sample size of 118 participants, while statistically sufficient for primary outcome correlation, contained relatively small numbers in the advanced doppler sub-grades (grades 3 to 5), limiting sub-group comparative power. Second, the study was conducted at a single tertiary medical center, which may select for a higher proportion of severe cases compared to general community settings. Third, long-term neurodevelopmental and pediatric follow-up of neonates beyond the immediate perinatal period was not performed. Future multi-center prospective studies incorporating long-term neurodevelopmental tracking and routine uterine artery doppler integration are recommended to further refine surveillance protocols.

CONCLUSION

This study confirms that comprehensive fetal doppler velocimetry of the umbilical artery, middle cerebral artery, and ductus venosus provides exceptional diagnostic and prognostic surveillance in pregnancies complicated by fetal growth restriction. Normal doppler velocimetry strongly predicts favorable neonatal outcomes, allowing safe continuation of pregnancy, whereas sequential doppler deterioration culminating in cerebral redistribution and ductus venosus 'a' wave reversal correlates significantly with severe prematurity, low birth weight, neonatal morbidity, and perinatal mortality. By accurately mapping the spectrum of fetal hemodynamic adaptation and identifying severe distress prior to the onset of irreversible acidemia, multi-vessel doppler assessment advances clinical understanding and provides an essential, evidence-based framework to optimize delivery timing

and improve perinatal survival in growth-restricted fetuses.

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