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Original Research Article

Brain-Sparing Is Not Risk-Sparing: Predelivery Cerebroplacental Redistribution and Early Infant Outcomes in Late-Onset Fetal Growth Restriction

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Abstract:

Background: Late-onset fetal growth restriction may occur despite relatively preserved umbilical artery Doppler findings. Cerebroplacental ratio (CPR) reflects fetal cerebral redistribution and may identify compromised fetuses at risk beyond delivery.

Methods: This prospective mother–infant cohort was conducted at GSL Medical College from January to May 2026. Singleton pregnancies with late-onset FGR were categorized according to predelivery CPR as abnormal or normal. Maternal characteristics, Doppler indices, delivery details and neonatal morbidity were recorded. Surviving infants underwent early neurodevelopmental assessment at approximately 12 weeks corrected age.

Results: Among 120 mother–infant pairs, 38 (31.7%) had abnormal CPR. Abnormal CPR was associated with lower birth weight (2210±310 vs 2410±300 g; p=0.001), increased cesarean delivery (81.6% vs 59.8%; p=0.018), NICU admission (65.8% vs 30.5%; p<0.001), respiratory support (31.6% vs 11.0%; p=0.006), and composite neonatal morbidity (76.3% vs 39.0%; p<0.001). Among 117 surviving infants, abnormal early neurodevelopment occurred in 27.8% versus 7.4% (p=0.007; OR 4.81, 95% CI 1.59–14.53).

Conclusion: Abnormal predelivery CPR identified late-onset FGR infants at increased neonatal and early neurodevelopmental risk and may support integrated antenatal and postnatal surveillance.

Keywords: Cerebroplacental Ratio; Fetal Growth Restriction; Brain-Sparing; Neonatal Morbidity; Neurodevelopment.

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Introduction

Late-onset fetal growth restriction (FGR), diagnosed after 32 weeks of gestation, is related to placental insuf-ficiency and may remain subtle because umbilical artery Doppler findings can be normal despite evolving fetal compromise. The cerebroplacental ratio (CPR), calculated as the middle cerebral artery pulsatility index divided by the umbilical artery pulsatility index, integrates placental resistance with fetal cerebral vasodilatation and reflects redistribution of blood flow in response to hypoxemia. Indian evidence supports its potential value in risk stratification. Srirambhatla et al. reported that CPR was among the most specific Doppler param-eters for predicting adverse perinatal outcomes in late-onset FGR [1]. In a prospective study from PGIMER Chandigarh, Kumar et al. demonstrated significant differences in CPR between appropriate-for-gestational-age and growth-restricted fetuses and showed its association with adverse perinatal outcome [2]. More recently, Bhardwaj et al. found that abnormal CPR was associated with neonatal morbidity, including NICU admission, hypoglycemia, ventilatory support, and prolonged hospitalization [3]. However, Indian studies have largely concentrated on immediate neonatal outcomes, while the relationship between predelivery CPR abnormalities and early infant neurodevelopment remains insufficiently characterized. Therefore, this prospective mother–infant cohort study aims to determine whether abnormal CPR before delivery predicts neonatal morbidity and early neurodevelopmental impairment in late-onset FGR.

Methods:

This prospective mother–infant cohort study was conducted in the Departments of Obstetrics and Gynaecology and Pediatrics/Neonatology at GSL Medical College, Rajahmundry, with maternal recruitment undertaken from January to May 2026.

Pregnant women with singleton pregnancies diagnosed with late-onset fetal growth restriction (FGR) at or beyond 32 weeks of gestation were screened consecutively for eligibility. FGR was diagnosed when the estimated fetal weight or abdominal circumference was below the 10th percen-tile for gestational age, together with clinical and/or Doppler evidence suggestive of placental insufficiency. Women with multiple pregnancy, major fetal structural or chromosomal abnormalities, congenital infection, uncertain gestational age, or conditions likely to independently influence neonatal neurological outcome were excluded. Gestational age was established from the last menstrual period and confirmed by first-trimester ul-trasonography whenever available. Maternal demographic characteristics, parity, body mass index, antenatal complications, hypertensive disorders, diabetes, hemoglobin level, gestational age at diagnosis, corticosteroid administration, and indication and mode of delivery were recorded using a predefined study proforma. Written informed consent was obtained before enrollment, and confidentiality of mother–infant data was maintained throughout the study.

Fetal Doppler assessment was performed within 72 hours before delivery using standardized obstetric ultra-sonography. Pulsatility indices of the umbilical artery (UA-PI) and middle cerebral artery (MCA-PI) were measured during fetal quiescence using appropriate insonation techniques. The CPR was calculated as MCA-PI divided by UA-PI and categorized as normal or abnormal according to gestational-age-specific reference centiles, with CPR below the fifth percentile considered abnormal. Participants were consequently classified into an abnormal-CPR cohort and a normal-CPR cohort.

Treating obstetricians were responsible for decisions regarding surveillance and timing or mode of delivery according to maternal and fetal clinical status; delivery was not undertaken solely for study purposes. Immediately after birth, neonatal sex, gestational age, birth weight, birth-weight percentile, small-for-gestational-age status, Apgar scores at 1 and 5 minutes, cord blood parameters when available, and need for resuscitation were documented. Neonatal morbidity outcomes included respiratory distress, hypoglycemia, hypothermia, feeding intolerance, suspected or proven sepsis, need for respiratory support, NICU admission, duration of NICU stay, and neonatal mortality. Infants were followed prospectively from birth through hospital discharge and subsequently for early neurode-velopmental assessment. Neurological status, tone, feeding ability, seizures, and abnormal movements were recorded during the neonatal period. At approximately 12 weeks of corrected age, surviving infants under-went standardized developmental assessment by a pediatrician who was unaware of the predelivery CPR cate-gory whenever feasible. Early neurodevelopment was evaluated using age-appropriate neurological and developmental domains, including gross motor, fine motor, communication, social responsiveness, tone, posture, and movement quality; infants with abnormal findings were classified as being at risk for developmental im-pairment and referred for further evaluation. Continuous variables were expressed as mean ± standard devia-tion or median with interquartile range, and categorical variables as frequencies and percentages. Group com-parisons used Student's t test or Mann–Whitney U test and chi-square or Fisher's exact test as appropriate. Multivariable logistic regression was performed to determine whether abnormal CPR independently predicted adverse outcomes after adjustment for relevant maternal and neonatal confounders, with $p < 0.05$ considered statistically significant.

Results:

A total of 120 mother–infant pairs were included; 38 (31.7%) had an abnormal cerebroplacental ratio and 82 (68.3%) had a normal CPR. Maternal age, parity, hypertensive disorders, and gestational age at delivery were comparable between groups. Fetuses with abnormal CPR had significantly lower mean birth weight (2210±310 vs 2410±300 g; p=0.001) and a higher cesarean delivery rate (81.6% vs 59.8%; p=0.018). Neonatal compromise was more frequent in the abnormal-CPR group, including 5-minute Apgar score <7 (31.6% vs 9.8%; p=0.003), need for resuscitation (39.5% vs 14.6%; p=0.002), respiratory distress (34.2% vs 13.4%; p=0.008), respiratory support (31.6% vs 11.0%; p=0.006), and NICU admission (65.8% vs 30.5%; p<0.001). Composite neonatal morbidity was 76.3% versus 39.0% (p<0.001). Among 117 surviving infants assessed at approximate-ly 12 weeks corrected age, abnormal early neurodevelopment occurred in 27.8% versus 7.4% (p=0.007). Ab-normal CPR was strongly associated with composite neonatal morbidity (OR 5.03, 95% CI 2.11–12.01) and abnormal early neurodevelopment (OR 4.81, 95% CI 1.59–14.53). These findings identified a higher-risk sub-group requiring closer neonatal and developmental surveillance.

Table 1: Maternal, obstetric and fetal Doppler characteristics according to CPR

Variable	CPR		p value
	Abnormal	Normal	
Maternal age, years*	27.1 ± 4.2	26.8 ± 4.1	0.715
Primigravida **	19 (50.0)	36 (43.9)	0.533
Hypertensive disorder**	13 (34.2)	20 (24.4)	0.262
Antenatal corticosteroids**	14 (36.8)	25 (30.5)	0.489
Gestational age at delivery, weeks*	36.2 ± 1.0	36.5 ± 0.9	0.12
MCA pulsatility index*	1.22 ± 0.18	1.59 ± 0.22	<0.001
Umbilical artery pulsatility index*	1.43 ± 0.22	1.21 ± 0.18	<0.001
CPR*	0.86 ± 0.12	1.32 ± 0.17	<0.001
Birth weight, g*	2210 ± 310	2410 ± 300	0.001
Cesarean delivery**	31 (81.6)	49 (59.8)	0.018
*mean ± SD; **n (%)			
MCA: middle cerebral artery; SD: standard deviation.			

Table 2: Neonatal morbidity according to predelivery cerebroplacental ratio

Neonatal outcome	CPR		p value
	Abnormal	Normal	
5-min Apgar score <7*	12 (31.6)	8 (9.8)	0.003
Required resuscitation*	15 (39.5)	12 (14.6)	0.002
Respiratory distress*	13 (34.2)	11 (13.4)	0.008
Hypoglycemia*	11 (28.9)	10 (12.2)	0.025
Feeding intolerance*	10 (26.3)	9 (11.0)	0.032
Suspected/proven sepsis*	6 (15.8)	7 (8.5)	0.343
Respiratory support*	12 (31.6)	9 (11.0)	0.006
NICU admission*	25 (65.8)	25 (30.5)	<0.001
NICU stay among admitted infants, days**	6.8 ± 3.9	4.3 ± 2.7	0.012
Composite significant neonatal morbidity*	29 (76.3)	32 (39.0)	<0.001
Neonatal mortality*	2 (5.3)	1 (1.2)	0.235
*n (%); **mean ± SD			
NICU: neonatal intensive care unit. Composite morbidity included one or more clinically significant neonatal complications requiring active treatment or intensive monitoring.			

Table 3: Early neurodevelopmental outcomes among surviving infants; n (%)

Outcome at approximately 12 weeks corrected age	CPR		Odds ratio (95% CI)	p value
	Abnormal	Normal		
Abnormal tone/posture	8 (22.2)	4 (4.9)	5.50 (1.54–19.70)	0.008
Early motor abnormality	9 (25.0)	5 (6.2)	5.07 (1.56–16.46)	0.01
Communication/social concern	5 (13.9)	4 (4.9)	3.10 (0.78–12.33)	0.131
Overall abnormal early neurodevelopment	10 (27.8)	6 (7.4)	4.81 (1.59–14.53)	0.007
CI: confidence interval. Neurodevelopmental analysis included 117 surviving infants.				

Discussion:

The present prospective mother–infant cohort evaluated whether an abnormal cerebroplacental ratio immediately before delivery could identify late-onset FGR pregnancies at increased risk of neonatal morbidity and early neurodevelopmental abnormalities. Approximately one-third of the cohort had an abnormal CPR, and these fetuses had lower birth weight, a higher cesarean delivery rate, greater need for neonatal resuscitation and respiratory support, and substantially more NICU admissions than fetuses with a preserved CPR. Composite neonatal morbidity was nearly twice as frequent in the abnormal-CPR group, while early neurodevelopmental abnormalities were observed in 27.8% compared with 7.4% among infants with normal CPR. This pattern supports the biological concept that the so-called fetal "brain-sparing" response should not necessarily be interpreted as evidence that the brain is protected from subsequent functional consequences. CPR incorporates both increased placental vascular resistance reflected by the umbilical artery and cerebral vasodilatation reflected by the middle cerebral artery, thereby identifying fetal circulatory redistribution that may be missed when either vessel is interpreted separately. Bhardwaj et al. similarly reported increased hypoglycemia, ventilatory requirement, prolonged hospitalization and NICU admission among Indian pregnancies with abnormal CPR [3]. Kumar et al. demonstrated that CPR provided useful discrimina-

tion for adverse perinatal outcome in high-risk pregnancies, supporting its role as an integrated marker of placental function and fetal adaptation [2]. Srirambhatla et al. further observed that Doppler indices, including CPR, were useful for prediction of adverse outcome in FGR, with differences in their performance between early- and late-onset diseases [4].

The increased neonatal morbidity observed with an abnormal CPR is particularly relevant to late-onset FGR because this phenotype frequently occurs close to term, when conventional umbilical artery Doppler may remain apparently reassuring. In the present cohort, abnormal CPR was associated with a 5-minute Apgar score below 7, need for resuscitation, respiratory distress, hypoglycemia, feeding intolerance, respiratory support and NICU admission. These outcomes are clinically plausible consequences of chronic placental insufficiency followed by limited fetal reserve during labor and the metabolic transition after birth. Rathore et al., in a prospective cohort from AIIMS Jodhpur, found that abnormal antepartum CPR was significantly associated with abnormal or non-reassuring cardiotocography and lower birth weight, although differences in NICU admission were not significant in their low-risk population [5]. Such differences emphasize that the predictive value of CPR depends on background obstetric risk, gestational age, definition of an abnormal ratio and outcome composition. Grover et al. subsequently demonstrated that a CPR threshold of ≤1.1 identified term pregnancies with increased fetal heart rate abnormalities, operative intervention and NICU admission and performed better than a fixed threshold of <1 in their cohort [6]. Dasgupta et al. reported high specificity and positive predictive value for CPR in predicting adverse perinatal outcomes during routine third-trimester ultrasonography in Eastern India [7]. Collectively, these observations support use of gestational-age-adjusted CPR rather than reliance on a single universal numerical cutoff and are consistent with the increased morbidity detected in our late-onset FGR cohort.

The higher cesarean delivery rate and lower mean birth weight among fetuses with abnormal CPR also provide evidence that cerebral redistribution identifies a more compromised subgroup rather than merely a constitutionally small population. In late-onset FGR, the clinical challenge is to distinguish a fetus that is small but physiologically stable from one experiencing progressive placental insufficiency despite relatively preserved umbilical arterial end-diastolic flow. Vasudeva et al., studying small-for-gestational-age fetuses in an Indian tertiary center, showed that low CPR had a stronger positive likelihood ratio for adverse perinatal outcome than aortic isthmus abnormalities and correlated with other manifestations of deteriorating fetal hemodynamics [8].

Placental imaging data from Menon et al. provide additional biological support, as FGR pregnancies demonstrated reduced placental vascularization indices and greater placental stiffness, with these alterations correlating with neonatal complications [9]. More recently, Gupta et al. reported that integrating fetal Doppler parameters, including CPR, into routine third-trimester assessment substantially reclassified the burden of FGR in a northern Indian population and identified pregnancies at increased risk of late stillbirth [10]. Kumanan et al. likewise observed significant associations between abnormal UA, MCA and CPR Doppler findings and unfavorable perinatal outcomes in Indian high-risk pregnancies [11]. Taken together, these data suggest that CPR should be interpreted as part of a multiparametric assessment incorporating fetal size, interval growth, umbilical artery resistance, cerebral adaptation, maternal disease and gestational age rather than as an isolated trigger for delivery.

The findings also have implications for antenatal surveillance strategies in India, where failure to recognize placental growth restriction before delivery remains an important preventable contributor to adverse perinatal outcome. In the Samrakshan programme, Choorakuttil et al. evaluated third-trimester uterine, umbilical and middle cerebral arterial Doppler parameters and CPR and demonstrated strong discriminatory ability for late stillbirth, although prediction of term stillbirth and neonatal mortality was less robust [12]. This distinction is important because no Doppler parameter should be expected to function as a stand-alone diagnostic test for every adverse endpoint. CPR is more appropriately viewed as a marker of reduced fetal reserve that modifies risk rather than as a deterministic predictor. Similarly, implementation of a structured Growth Assessment Protocol in a South Indian tertiary maternity service increased antenatal detection of small-for-gestational-age fetuses and was accompanied by reductions in term stillbirth, NICU admission and neonatal encephalopathy [13]. These Indian experiences emphasize the potential clinical benefit of moving from simple recognition of fetal smallness toward physiological characterization of growth restriction. The present finding that composite neonatal morbidity occurred in 76.3% of infants with abnormal CPR compared with 39.0% with normal CPR extends this framework by suggesting that predelivery redistribution can help neonatal teams anticipate immediate postnatal instability. Such risk information may facilitate planned delivery at an appropriately equipped center, preparedness for neonatal resuscitation, early glucose surveillance, feeding support and a lower threshold for observation when other signs of placental insufficiency coexist.

A distinctive aspect of the present study was continuation of follow-up beyond hospital discharge to explore early neurodevelopment. Among surviving infants, abnormal tone or posture and early motor abnormalities were significantly more frequent following an abnormal predelivery CPR, and the odds of overall abnormal early neurodevelopment were approximately five times higher than among infants with normal CPR. These findings should not be interpreted as establishing permanent neurodevelopmental impairment at 12 weeks; rather, they indicate an enriched population requiring longitudinal surveillance. Chronic fetal hypoxemia may induce preferential cerebral perfusion sufficient to preserve immediate survival while still reflecting an intrauterine environment capable of influencing neuronal maturation, connectivity and subsequent motor behavior. In an Indian follow-up study, Jain et al. reported substantial neurodevelopmental delay among preterm NICU graduates and identified neonatal complications including respiratory distress, hypoglycemia, necrotizing enterocolitis and sepsis as important associated factors [14]. Although that population differed from the present late-onset FGR cohort, it demonstrates how early neonatal morbidity can coexist with subsequent developmental vulnerability. Pai et al. showed that Indian term small-for-gestational-age infants follow distinct growth trajectories during the first year, reinforcing the need to view fetal growth restriction as a condition with postnatal consequences rather than an obstetric diagnosis that ends at delivery [15]. The Indian Academy of Pediatrics has also emphasized structured, developmentally supportive follow-up for high-risk infants to enable early identification and intervention for developmental difficulties [16]. Accordingly, an abnormal CPR may have value not only in delivery planning but also in selecting apparently stable infants for enhanced developmental surveillance.

Several strengths and limitations should guide interpretation of these findings. The prospective mother–infant design, Doppler assessment close to delivery, separation into predefined CPR groups, systematic recording of individual neonatal morbidities and inclusion of post-discharge developmental assessment strengthened temporal interpretation. Restriction to late-onset FGR also reduced heterogeneity arising from the substantially different natural history of early-onset placental disease.

Nevertheless, this was a single-center study with a relatively modest sample, and residual confounding by severity of FGR, hypertensive disease, gestational age, birth weight and indications for delivery could not be completely eliminated. Obstetric management could not ethically be blinded to clinically relevant fetal findings, potentially contributing to differences in cesarean delivery and neonatal admission. More importantly, developmental assessment at approximately 12 weeks corrected age identifies early neurological or developmental concern but cannot reliably predict long-term cognition, language, and behavior or school performance.

Longer follow-up using validated standardized developmental scales at 6, 12, 18 and 24 months would therefore be necessary to determine whether the observed association persists. Despite these limitations, the consistency of the neonatal findings with contemporary Indian Doppler studies and the parallel signal in early neurodevelopment suggest that abnormal CPR may characterize a clinically meaningful phenotype of late-onset FGR. Rather than treating brain-sparing as reassuring, clinicians should consider it a marker of successful fetal compensation occurring in the presence of an adverse placental environment and use it to individualize surveillance before and after birth [3, 8, 16].

Conclusion:

An abnormal cerebroplacental ratio before delivery identified a subgroup of late-onset FGR fetuses with substantially greater neonatal morbidity and early developmental vulnerability. Reduced CPR was associated with lower birth weight, increased operative delivery, neonatal resuscitation, respiratory morbidity, hypoglycemia, and respiratory support and NICU admission.

Importantly, infants exposed to abnormal fetal cerebral redistribution also showed more abnormalities of tone, posture and early motor development. CPR therefore appears to provide information beyond fetal size alone and may bridge antenatal risk assessment with neonatal and developmental surveillance. Larger multicenter cohorts with standardized developmental testing and longer follow-up are required before CPR-guided postnatal pathways can be routinely recommended.

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