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Original Research Article

Association of cranial ultra sonography findings with intrauterine growth restriction in term neonates

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ABSTRACT

Background: Fetal growth restriction (FGR) is a condition in which a fetus does not reach its full growth potential in utero. It is a significant contributor to perinatal morbidity and mortality. Cranial ultrasound (CU) is a non-invasive imaging technique used to assess brain structure and abnormalities in neonates, particularly affected by FGR or classified as small for gestational age (SGA). Aim was to determine the association between FGR and CU abnormalities (CUAs) in term neonates.
Methods: It was a single centre, hospital-based, cross-sectional comparative observational study conducted in the level IIIA neonatal intensive care unit (NICU) of Shri Shishu Bhawan Hospital for Children and Newborn, Bilaspur, Chhattisgarh. A total of 194 neonates were selected for the study. Comparative analysis between the FGR and control groups was performed using chi-square tests for categorical variables and t-tests for continuous variables.
Results: The present study observed a higher proportion of CUAs in term neonates with FGR (11.3%) compared to appropriate for gestational age (AGA) neonates 2.06% ($p>0.5$). Periventricular leukomalacia (PVL) was more commonly observed among FGR neonates (14.3%) compared to AGA neonates 6.18% ($p>0.05$). The trend suggests that FGR may predispose neonates to a higher risk of periventricular white matter damage, even at term gestation.
Conclusions: FGR has a substantial impact on neonatal brain development and increases the risk of neurodevelopmental complications. Early detection through CU screening and long-term follow-up for neurodevelopmental assessment are essential to improve outcomes in this high-risk population.

Keywords: Ultrasound sonography, Cranial ultrasound, Fetal growth restriction, Intrauterine growth restriction

INTRODUCTION

Fetal growth restriction (FGR) is a condition in which a fetus does not reach its full growth potential in utero.[1] It is a significant contributor to perinatal morbidity and mortality, affecting approximately 5-10% of pregnancies worldwide.[2] Infants born with FGR are at an increased risk of developing various complications, including metabolic disorders, respiratory distress, and neuro developmental impairments.[3] Small for gestational age (SGA) infants, who are defined as having a birth weight below the 10th percentile for their gestational age, are often used as a proxy for FGR.[4,5] However, not all SGA infants are growth-restricted, and distinguishing between the two can be challenging in clinical practice.[6]

Cranial ultrasound (CU) is a non-invasive imaging technique used to assess brain structure in neonates.[7] It is a valuable tool for identifying brain abnormalities, such as intraventricular hemorrhage (IVH), cerebral ischemia, and delayed brain growth, particularly in high-risk populations such as those affected by FGR or classified

as SGA.[8] CU is often used as a first-line imaging modality due to its accessibility, safety, and ability to provide real-time assessment of brain development and pathology.[9] The association between FGR and CUAs in term neonates remains an area of interest in neonatal medicine.[10] Previous studies suggest that FGR-affected infants may have a higher incidence of CUAs compared to those without growth restriction.[11]

This study is of considerable importance, as it addresses a critical gap in the understanding of the relationship between FGR and CUAs in term neonates. While much research has focused on the neurodevelopmental outcomes of preterm infants, the impact of FGR on the neurological health of term neonates is less well understood. By conducting this study, we aim to address the gap in the current literature by systematically evaluating the association between FGR and CU findings in term neonates. Understanding this association is critical for improving early detection of neurological abnormalities, optimizing management strategies, and ultimately improving the neurodevelopmental outcomes for neonates affected by FGR. This study will also provide valuable insights into the potential role of cerebral artery Doppler in complementing CU in the assessment of FGR neonates.

Aim

Aim was to determine the association between FGR and CUAs in term neonates.

METHODS

A single centre, hospital-based, cross-sectional comparative observational study design was employed for this research. The study was conducted in the level IIIA NICU of Shri Shishu Bhawan Hospital for Children and Newborn, Bilaspur, Chhattisgarh. The total duration of the present study was 24 months, from July 2023 to June 2025. This tertiary care hospital caters to neonates referred from nearby districts and adjacent states. The institute's ethical committee carefully scrutinized the ethical considerations, ensuring that all procedures adhered to the national and institutional guidelines for research involving neonates.

The study included term neonates (gestational age≥37 weeks) diagnosed with intrauterine growth restriction (IUGR) or FGR based on clinical and ultrasound findings. It included neonates without FGR, classified as AGA, as the control group. Only those neonates were included whose guardians provided written informed consent for participation.

Neonates with congenital anomalies, metabolic disorders, incomplete clinical data or those who did not undergo CU, and neonates born to mothers with monochorionic diamniotic twin pregnancies or other high-risk conditions such as congenital heart disease were excluded from the study.

Sample size

The sample size for the study was calculated based on the difference between the proportions of CUAs in term neonates with and without FGR. Using a two-tailed z-test for proportions with an alpha error probability of 0.05, power of 0.8, and a 20% difference in abnormality rates between the groups (p1=0.6, p2=0.4), the required sample size for each group was determined to be 97. Therefore, a total of 194 neonates were needed for the study. All eligible participants coming to the study institute during the recruitment period, and whose guardians provided written informed consent.

Statistical analysis

The data from the paper-based data collection forms were initially entered into MS Excel and then imported into Stata software version 17.0 for analysis. The data were subjected to descriptive and inferential statistical tests. Comparative analysis between the FGR and control groups was performed using chi-square tests for categorical variables and t-tests for continuous variables. The association between FGR and CUAs was analyzed using logistic regression models to adjust for confounding variables. All the statistical and graphical analyses for this study were undertaken by Stata software version 17.0. A $p<0.05$ was considered as statistically significant.

RESULTS

As per Table 1, among neonates with FGR, 43.3% (n=42) were admitted within the first three days of life, whereas 37.1% (n=36) of the normal neonates were admitted within the same period. In the 4-7 days age group, 26.8% (n=26) of FGR neonates and 28.8% (n=28) of normal neonates were admitted. A similar trend was observed in the 8-14 days age group, where 23.7% (n=23) of FGR neonates and 21.6% (n=21) of normal neonates were admitted. However, the proportion of neonates admitted after 15 days was higher in the normal group (12.4%) compared to the FGR group (6.2%).

As per Figure 1, neonates with FGR, 73.2% (n=71) were male and 26.8% (n=26) were female. Similarly, in the normal group, 68% (n=66) were male and 32% (n=31) were female. This indicates a slightly higher proportion of male neonates in both groups, with a more pronounced male predominance in the FGR group. Additionally, A significant proportion of FGR neonates (63.9%, n=62) had a birth weight below average, whereas a larger proportion of normal neonates (81.4%, n=79) also fell into the below-average birth weight category. However, the proportion of neonates with an above-average birth weight was higher in the FGR group (36.1%) compared to the normal group (18.6%) (Figure 2). The mean birth

weight was 1860±440 grams in the FGR group and 2810±290 grams in the normal group, indicating a clear disparity in birth weight between the two groups.

Table 1: Distribution of participants based on age of admission.

Age (in days)	FGR (n=97)		Normal (n=97)	
	N	%	N	%
0-3	42	43.3	36	37.1
4-7	26	26.8	28	28.8
8-14	23	23.7	21	21.6
≥15	6	6.2	12	12.4

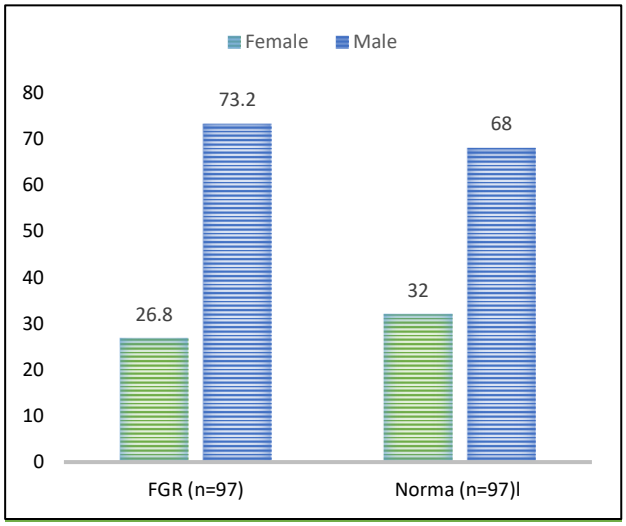

Figure 1: Distribution of participants based on gender.

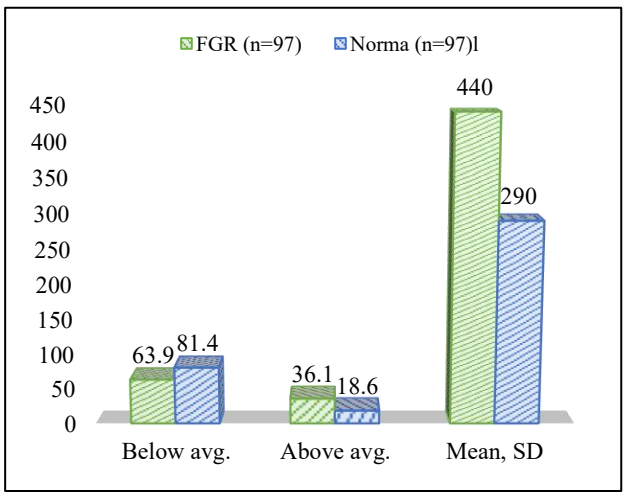

Figure 2: Distribution of participants based on birth weight.

Figure 3 shows that among neonates with FGR, 60.8% (n=58) were delivered through normal vaginal delivery (NVD), while 39.2% (n=38) were delivered via caesarean section (C-section). In contrast, a higher proportion of normal neonates (81.4%, n=79) were born through vaginal delivery, and only 18.6% (n=18) required a C-section. This indicates that FGR neonates were more likely to be delivered via C-section compared to their normal counterparts.

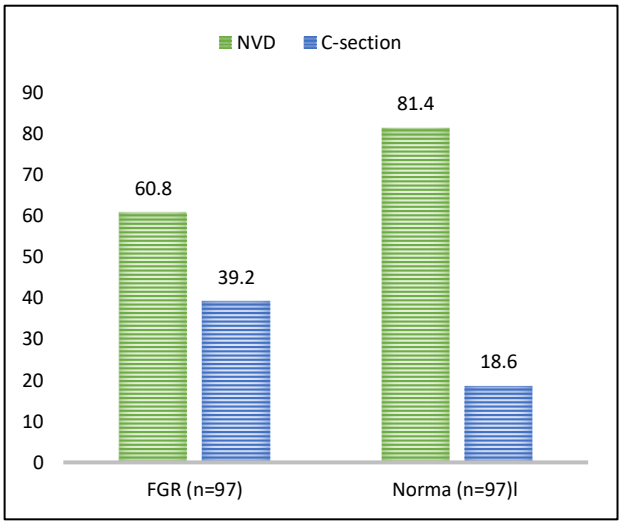

Figure 3: Distribution of participants based on type of delivery.

According to Figure 4, breathing difficulty was observed in 19.6% (n=19) of FGR neonates, compared to 14.4% (n=14) of normal neonates. Poor feeding was reported in 27.8% (n=27) of FGR neonates, whereas only 17.5% (n=17) of normal neonates exhibited this issue. Delayed cry was significantly higher among FGR neonates (9.3%, n=9) than in normal neonates (3.1%, n=3).

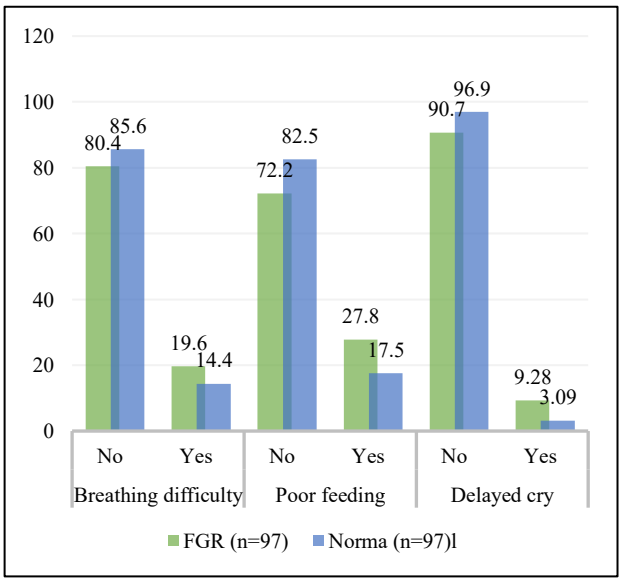

Figure 4: Distribution of participants based on presenting complaints.

Table 2 depicts the distribution of participants based on CUAs, specifically subependymal cysts and sino-venous thrombosis. Subependymal cysts were present in 12.4%

(n=12) of FGR neonates, whereas none were detected in the normal group (0%, n = 0), with a statistically significant p<0.0001. Sino-venous thrombosis was observed in 8.2% (n=8) of FGR neonates and 7.2% (n=7) of normal neonates, with a p=0.78, indicating no significant difference between the groups. These findings suggest that subependymal cysts were significantly more common in FGR neonates, whereas sino-venous thrombosis occurred at a similar rate in both groups.

Table 3 highlights the distribution of participants based on intraventricular haemorrhage (IVH). The majority of neonates in both groups did not have IVH (88.6%, n=86 in the FGR group and 96%, n=93 in the normal group). Among those with IVH, Grade I haemorrhage was observed in 2.1% (n=2) of neonates in both groups. Grade II IVH was more frequent in the FGR group (4.1%, n=4) compared to the normal group (1.03%, n=1). Similarly, Grade III IVH was present in 3.1% (n=3) of FGR neonates and 1.03% (n=1) of normal neonates. Grade IV IVH was noted only in the FGR group (2.1%, n=2), while no cases were recorded in the normal group. The p value for IVH distribution was 0.280, suggesting no statistically significant difference between the groups despite a slightly higher occurrence of severe IVH in FGR neonates.

Table 2: Distribution of participants based on subependymal cysts.

Variables	FGR, (n=97)		Normal, (n=97)		P value
Subependymal cysts	Absent	85 (87.6%)	Absent	97 (100%)	<0.001
	Present	12 (12.4%)	Present	0	
Sino venous thrombosis	Absent	89 (91.8%)	Absent	90 (92.8%)	0.78
	Present	8 (8.25%)	Present	7 (7.22%)	

Table 3: Distribution of participants based on intraventricular haemorrhage.

Variables	FGR, (n=97)		Normal, (n=97)	
	N	%	N	%
Absent	86	88.6	93	96
Grade I	2	2.06	2	2.06
Grade II	4	4.12	1	1.03
Grade III	3	3.09	1	1.03
Grade IV	2	2.06	0	0
P=0.280				

Table 4: Distribution of participants based on PVL.

Variables	FGR, (n=97)		Normal, (n=97)	
	N	%	N	%
Absent	83	85.7	91	93.84
Grade I	7	7.2	3	1.03
Grade II	3	3.09	1	1.03
Grade III	2	2.06	1	1.03
Grade IV	2	2.06	1	1.03
P=0.458				

Table 5: Distribution of participants based on CUA.

Variables	FGR, (n=97)		Normal, (n=97)	
	N	%	N	%
Absent	86	88.7	95	97.9
Present	11	11.3	2	2.06
P=0.10				

Table 4 illustrates the distribution of participants based on PVL. The majority of neonates in both groups did not have PVL (85.7%, n=83 in the FGR group and 93.8%, n=91 in the normal group). Among those with PVL, Grade I PVL was observed in 7.2% (n=7) of FGR neonates compared to 1.03% (n=1) in the normal group. Grade II PVL was found in 3.1% (n=3) of FGR neonates and 1.03% (n=1) of normal neonates. Similarly, grade III PVL was present in 2.1% (n=2) of FGR neonates and 1.03% (n=1) of normal neonates, while grade IV PVL was noted in 2.1% (n=2) of FGR neonates and 1.03% (n=1) of normal neonates. The p value for PVL

distribution was 0.458, indicating no statistically significant difference between the two groups.

Table 5 depicts the distribution of participants based on the overall presence of CUAs. In the FGR group, 11.3% (n=11) of neonates exhibited CUAs, whereas only 2.06% (n=2) of normal neonates had such findings. The p value for this comparison was 0.10, suggesting that while cranial abnormalities were more frequently observed in FGR neonates, difference was not statistically significant.

Table 6 highlights the distribution of participants based on intracranial parameters. The mean cerebellar vermis size was significantly smaller in FGR neonates (2.09±0.251 mm) compared to normal neonates (2.3±0.275 mm), with a p<0.0001. Similarly, the mean transverse cerebellar diameter was also reduced in FGR neonates (44.8±3.95 mm) compared to normal neonates (47.9±3.71 mm), with a p<0.0001. In Doppler studies, the mean middle cerebral artery (MCA) peak systolic velocity was 88.2±3.53 in FGR neonates and 89±3.16 in normal neonates (p=0.114), showing no significant difference. However, the MCA end-diastolic velocity was significantly lower in FGR neonates (15.7±4.82) compared to normal neonates (24.8±4.42), with a p<0.0001.

Table 6: Distribution of participants based on intracranial parameter.

Parameters	FGR, (n=97)		Normal, (n=97)		P value
	Mean	SD	Mean	SD	
Cerebellar vermis size (mm)	2.09	0.251	2.3	0.275	<0.0001
Transverse cerebellar diameter (mm)	44.8	3.95	47.9	3.71	<0.0001
MCA peak systolic velocity	88.2	3.53	89	3.16	0.114
MCA end diastolic velocity	15.7	4.82	24.8	4.42	<0.0001
MCA resistive index	0.82	0.053	0.720	0.047	<0.0001
MCA pulsatility index	1.22	0.179	1.22	0.176	0.842

Additionally, the MCA resistive index was significantly higher in FGR neonates (0.82±0.053) compared to normal neonates (0.72±0.047), with a p<0.0001. The MCA pulsatility index was similar between the groups (1.22±0.179 in FGR neonates and 1.22±0.176 in normal neonates, p=0.842), indicating no significant difference. These findings suggest that FGR neonates had smaller intracranial structures and altered cerebral blood flow patterns, particularly in end-diastolic velocity and resistive index.

DISCUSSION

The present study was conducted in the level IIIA NICU of Shri Shishu Bhawan Hospital for Children and Newborn, Bilaspur, Chhattisgarh, a tertiary care centre that receives neonatal referrals from surrounding districts and neighbouring states. The hospital setting ensured access to a wide range of neonates with diverse clinical profiles, facilitating the investigation of CUAs in term neonates affected by FGR. The study targeted full-term neonates, both with and without FGR, to evaluate the impact of intrauterine growth compromise on early neonatal brain development. A total of 194 term neonates were enrolled using a cross-sectional comparative observational design, with 97 neonates each in the FGR and AGA groups.

The present study observed a higher proportion of CUAs in term neonates with FGR (11.3%) compared to AGA neonates (2.06%), although the difference did not reach statistical significance (p=0.10). This trend suggests that FGR neonates may be more vulnerable to early brain injuries, potentially attributable to chronic intrauterine hypoxia and altered cerebral perfusion patterns associated with placental insufficiency. Even among term neonates, who are traditionally considered lower risk for cranial anomalies, the higher prevalence of CUAs in the FGR group highlights the subtle but significant impact of impaired fetal growth on early neurological development.

Similar findings were reported by Cruz-Martinez et al in a prospective study on 180 neonates born between 28 and 34 weeks, where 40% of IUGR neonates demonstrated CUAs compared to 12.2% of controls (p<0.001).[12] Their study further emphasised that fetal Doppler parameters, especially middle cerebral artery vasodilation and retrograde flow in the aortic isthmus, were stronger predictors of CUAs than gestational age at birth. Roufaeil et al performed a meta-analysis including 168,136 infants and found that FGR/SGA neonates had an almost twofold increased risk of any CUA compared to AGA neonates (RR=1.96; 95% CI: 1.26-3.04), supporting the findings of the present study despite differences in study design and population.[13]

In the present study, PVL, a marker of white matter injury, was more commonly observed among FGR neonates (14.3%) compared to AGA neonates (6.18%). Although this difference was not statistically significant (p=0.458), the trend suggests that FGR may predispose neonates to a higher risk of periventricular white matter damage, even at term gestation. Grade I PVL was the most frequent form in both groups, but higher-grade lesions (II-IV) were also slightly more common among FGR neonates. These findings are clinically relevant, as

PVL is associated with long-term neurodevelopmental impairments, including cerebral palsy, cognitive delay, and visual-motor dysfunction.

Comparable observations were reported in the systematic review and meta-analysis by Roufaeil et al which assessed CUAs in SGA and FGR neonates born over 32 weeks. Their analysis found an increased risk of white matter injuries, including PVL, in growth-restricted infants, although the evidence quality was low due to methodological heterogeneity.[9] Similarly, Khazardoost using MRI in term FGR neonates, identified significant microstructural white matter abnormalities, which were not always evident on conventional CU but suggest an underlying vulnerability to PVL-like pathology.[14]

In the present study, IVH was more frequently observed in the FGR group (11.34%) compared to the normal group (4.12%), though the difference was not statistically significant (p=0.280). Notably, the FGR group showed a higher prevalence of severe haemorrhages-grade III (3.09%) and grade IV (2.06%)-whereas no grade IV cases were found among normal neonates. This pattern suggests that neonates affected by FGR may be more susceptible to cerebral vascular fragility and haemodynamic instability, even when born at term, thereby increasing the risk of the clinically significant IVH.

These findings align with those of Roufaeil et al whose meta-analysis involving 167,060 infants showed that FGR/SGA neonates had a significantly increased risk of IVH compared to AGA infants (RR=2.40; 95% CI: 2.03-2.84).[9] Though most existing literature has focused on preterm populations, several studies have drawn attention to the vulnerability of term FGR neonates. For example, Cruz-Martinez et al found that 40% of IUGR neonates born between 28 and 34 weeks developed CUAs, and IVH was one of the most frequently encountered abnormalities.[8] Within their cohort, middle cerebral artery vasodilation and retrograde flow in the aortic isthmus were strong predictors of the haemorrhagic lesions.

The MCA resistive index (RI) was significantly elevated in the FGR group (0.82 vs. 0.72; p<0.0001), suggesting reduced cerebral perfusion efficiency. This finding mirrors the results of Acharya et al who demonstrated that higher MCA-RI values were associated with impaired neurodevelopment in growth-restricted infants.[15] The elevated RI in FGR neonates may reflect cerebrovascular constriction due to prolonged intrauterine hypoxaemia. In contrast, MCA pulsatility index (PI) did not differ significantly between the two groups (p=0.842), suggesting that while RI is sensitive to diastolic flow changes, PI may not consistently reflect subtle haemodynamic alterations in term FGR neonates.

CONCLUSION

This study demonstrates that FGR neonates exhibit a higher prevalence of CUAs, smaller cerebellar structures, and altered cerebral blood flow compared to normal neonates. These findings suggest that FGR has a substantial impact on neonatal brain development and increases the risk of neurodevelopmental complications. Early detection through CU screening and long-term follow-up for neurodevelopmental assessments are essential to improve outcomes in this high-risk population.

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