Journal of Cardiovascular Emergencies 2026;12(2):79-84
DOI: 10.2478/jce-2026-0006

ORIGINAL RESEARCH

Predictors of Abnormal Transfontanelle Ultrasound Findings in Neonates with Congenital Heart Disease

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ABSTRACT

Background: Advances in neonatal cardiac care have improved survival in infants with congenital heart disease (CHD), shifting clinical attention toward early neurological morbidity. While cardiac anatomical complexity has traditionally been considered a major determinant of brain injury, emerging evidence suggests that genetic and systemic factors may play a more important role. Transfontanellar ultrasound (TFUS) is widely used as a bedside screening tool for early detection of neonatal brain abnormalities. **Aim:** To assess the incidence of abnormal TFUS findings in neonates with CHD and identify clinical and genetic predictors of early neurological vulnerability independent of cardiac anatomy. **Materials and Methods:** We conducted an observational cohort study including 138 neonates with CHD who underwent TFUS evaluation at a tertiary care center in Iași, Romania. Demographic, perinatal, clinical, genetic, and therapeutic data were collected retrospectively. CHD complexity was classified using the Bethesda classification. Univariate and multivariate logistic regression analyses were performed to identify predictors of abnormal TFUS findings. **Results:** Abnormal TFUS findings were identified in 42 neonates (30.4%), most commonly ventricular dilatation, intracranial hemorrhage, and choroid plexus cysts. In multivariate analysis, positive genetic testing was the strongest independent predictor (OR 3.9, 95% CI 1.5–10.2), followed by mechanical ventilation dependence (OR 2.7, 95% CI 1.1–6.5) and high Bethesda risk category (OR 2.2, 95% CI 1.0–4.8). Cardiac anatomical classification and gestational age were not independently associated with abnormal TFUS findings. **Conclusions:** In neonates with CHD, early TFUS abnormalities appear to be driven mainly by genetic and systemic factors rather than cardiac anatomy alone, supporting integrative risk stratification approaches for targeted neurological surveillance.

Keywords: congenital heart disease, neurodevelopmental vulnerability, transfontanellar ultrasound, genetic syndromes

ARTICLE HISTORY

Received: January 26, 2026
Accepted: March 2, 2026

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INTRODUCTION

Globally, congenital heart defects (CHDs) represent the most common congenital anomaly, with an incidence of approximately 1% of all live births.[1] Over the past three decades, the field of pediatric cardiology has undergone a fundamental transformation. Owing to advances in prenatal detection and surgical interventions, the focus has shifted from ensuring survival to optimizing long-term quality of life.[2] As mortality rates have declined even for complex lesions, neurodevelopmental impairment has emerged as the predominant and perhaps most challenging chronic complication affecting this population.[2,3]

Thus, the modern imperative in neonatal cardiac care has shifted: the goal is no longer solely the repair of the heart, but also the protection of the developing brain.

The etiology of brain injury in neonates with CHDs is multifactorial and cumulative, involving a complex interplay among intrauterine hemodynamic compromise, genetic predisposition, and postnatal instability.[4] The concept of a 'heart–brain axis' suggests that specific anatomical substrates, such as transposition of the great arteries or single-ventricle physiology, may lead to characteristic patterns of altered cerebral perfusion and delayed brain maturation.[2,5] Despite this growing understanding, the precise contribution of cardiac anatomy compared with associated extracardiac or genetic comorbidities remains a subject of ongoing debate. While some studies suggest that the type of cardiac defect is the primary determinant of neurological outcomes, others argue that inherent patient factors, such as genetic syndromes, play a more decisive role.[2]

To monitor these risks, transfontanellar ultrasound (TFUS) remains the primary neuroimaging modality in the neonatal intensive care unit due to its noninvasive nature and bedside accessibility.[6]

In this context, the present study aims to evaluate the incidence and predictors of abnormal TFUS patterns in a cohort of neonates with CHD. Specifically, we sought to determine the incidence of abnormal TFUS findings in neonates with CHD and to identify clinical and genetic predictors of early neurological vulnerability, independent of cardiac anatomy.

METHODS

This retrospective observational cohort study included neonates with CHDs who underwent TFUS evaluation at Cuza Vodă Clinical Hospital of Obstetrics and Gynecology, Iași, Romania. Patients with incomplete clinical, imaging, or genetic data were excluded.

Demographic, perinatal, clinical, and therapeutic variables were collected retrospectively, including sex, area of residence, gestational age, prematurity status, birth anthropometrics, clinical symptoms, mechanical ventilation requirement, and prostaglandin administration. Genetic testing results were recorded as positive or negative. CHDs were classified according to anatomical and pathophysiological criteria, and disease complexity was stratified using the Bethesda classification. TFUS findings were categorized as normal or abnormal, with any detected structural or hemorrhagic abnormality considered an outcome event.

Continuous variables are presented as mean ± s.d., and categorical variables as counts and percentages. Group comparisons were performed using Student's t-test or the χ^2 test, as appropriate. Univariate logistic regression was used to assess the association between clinical, genetic, and therapeutic variables and abnormal TFUS findings. Variables demonstrating statistical significance at the univariate level ($p < 0.05$) were subsequently included in a multivariate logistic regression model to identify independent predictors. Results are reported as odds ratios (OR) with corresponding 95% confidence intervals (CI). A p value of <0.05 was considered statistically significant. Neonatal variables that did not demonstrate significance in the univariate analysis were not included in the multivariate model.

RESULTS

STUDY POPULATION

The study included 138 neonates with CHD. Baseline demographic, perinatal, and clinical characteristics of the study population are summarized in Table 1.

Genetic abnormalities were identified in 37 patients (26.82%), of whom 29 (21.01%) were diagnosed with genetic syndromes. These included trisomies (16 cases), microdeletions (8 cases), laterality disorders (4 cases), and other syndromic conditions (1 case). In the remaining eight cases, multiple malformations were identified that could not be assigned to any known syndrome and involved abnormalities across multiple organ systems.

CHD CLASSIFICATION

CHD types were distributed across physiological categories, with right-to-left shunt lesions and complex CHD representing the most frequent phenotypes. The distribution of CHD types is presented in Table 2.

TABLE 1. Baseline characteristics of patients

Variable	Value
Demographic characteristics	
Sex	
Male, n (%)	70 (50.7%)
Female, n (%)	68 (49.3%)
Environment	
Urban, n (%)	43 (31.16%)
Rural, n (%)	95 (68.84%)
Postnatal characteristics	
Gestational age, weeks (mean ± s.d.)	36.93 ± 2.59
Prematurity, n (%)	49 (35.51%)
Birth weight, g (mean ± s.d.)	2,692.55 ± 865.11
Birth length, cm (mean ± s.d.)	47.7 ± 4.31
Positive genetic testing, n (%)	37 (26.82%)
Genetic syndrome, n (%)	29 (21.01%)
Clinical symptoms	
Cardiac murmur, n (%)	134 (97.10%)
Tachycardia, n (%)	88 (63.77%)
Hypoxia, n (%)	87 (63.04%)
Tachypnea, n (%)	81 (58.70%)
Cyanosis, n (%)	60 (43.48%)
Chest wall retraction, n (%)	66 (47.83%)
Arrhythmia, n (%)	6 (4.35%)
Mechanical ventilation dependence, n (%)	53 (38.41%)
Prostaglandin administration, n (%)	64 (46.38%)

According to the Bethesda classification, 106 cases (76.81%) were categorized as severely complex, while 19 (13.77%) were classified as simple and 13 (9.42%) as moderately complex.

TRANSFONTANELLAR ULTRASOUND FINDINGS

Abnormal TFUS findings were identified in 42 neonates (30.43%). Ventricular dilatation, intracranial hemorrhage, and choroid plexus cysts were the most frequently observed abnormalities. A detailed distribution of TFUS findings is shown in Table 3. Isolated cases of hydrocephalus, cerebral aneurysm, and mega cisterna magna were also observed in three patients.

TABLE 2. CHD types

CHD type	n (%)
Left-to-right shunt lesions	25 (18.12%)
Right-to-left shunt lesions	41 (30.43%)
Obstructive CHD	32 (23.19%)
Complex CHD	40 (28.99%)

TABLE 3. Transfontanellar ultrasound abnormalities

Abnormality	n (%)
Ventricular dilation	11 (26.19%)
Intraventricular hemorrhage	7 (16.67%)
Choroid plexus cysts	7 (16.67%)
Subependymal hemorrhage	5 (11.90%)
Corpus callosum agenesis (partial/complete)	6 (14.29%)
Cerebral edema	6 (14.29%)
Hydrocephalus	1 (2.38%)
Cerebral aneurysm	1 (2.38%)
Mega cisterna magna	1 (2.38%)

PREDICTORS OF ABNORMAL TFUS

In univariate logistic regression analysis, several clinical and genetic variables were significantly associated with abnormal TFUS findings. Variables demonstrating statistical significance were subsequently included in a multivariate model.

Positive genetic testing remained the strongest independent predictor, followed by mechanical ventilation dependence and a high Bethesda risk category. Results of the univariate and multivariate analyses are summarized in Table 4.

TABLE 4. Univariate and multivariable logistic regression analyses for abnormal TFUS

Predictor	Univariate logistic regression analysis		Multivariate logistic regression analysis	
	OR (95% CI)	p value	OR (95% CI)	p value
Positive genetic testing	4.2 (1.8–9.8)	0.001	3.9 (1.5–10.2)	0.005
Mechanical ventilation dependence	3.1 (1.4–6.9)	0.005	2.7 (1.1–6.5)	0.028
Bethesda high risk	2.5 (1.2–5.3)	0.015	2.2 (1.0–4.8)	0.045
Number of associated clinical symptoms	1.4 (1.1–1.8)	0.008	–	–
Prostaglandin administration	2.8 (1.1–7.2)	0.031	–	–

DISCUSSION

GENETIC AND PRENATAL DETERMINANTS OF EARLY BRAIN VULNERABILITY

In this cohort of neonates with CHD, abnormal TFUS findings were identified in nearly one-third of patients. Contrary to the traditional view that cardiac anatomical complexity is the primary driver of early neurological injury, our results suggest that intrinsic patient-related factors may play a more prominent role than CHD anatomy itself. Evidence from neurological disorders indicates that functional and behavioral alterations may precede clearly identifiable structural brain lesions, supporting the concept that early brain vulnerability can manifest before overt imaging abnormalities become apparent.[7,8] Several studies also suggest that patient-related and environmental factors, including prematurity, genetic syndromes, and socioeconomic status, may be stronger determinants of neurodevelopmental outcomes than operative or perioperative management strategies alone.[9,10]

This observation is consistent with growing evidence suggesting that neurodevelopmental vulnerability in CHD is often established prenatally and is not solely driven by postnatal hemodynamic disturbances. Fetal MRI studies have demonstrated that brain development is already altered in fetuses with congenital heart disease, with reduced total brain volumes, abnormal cerebral metabolism, and delayed cortical maturation and folding.[12–15]

Notably, positive genetic testing emerged as the strongest independent predictor of abnormal TFUS findings in our cohort. This observation is supported by previous studies showing that genetic syndromes and chromosomal abnormalities are strongly associated with impaired brain maturation and early brain injury in neonates with CHD, independent of cardiac physiology or surgical timing.[2,4]

Genetic disorders are reported in up to 30% of pediatric patients with CHD and are frequently associated with developmental delay.[16–18] Moreover, subtle phenotypic expression may delay the diagnosis of conditions such as mosaic aneuploidies or Turner syndrome during the neonatal period.[19] These findings are consistent with prior reports linking CHD to abnormal brain development, impaired cerebral growth, and altered fetal cerebral blood flow, suggesting a shared developmental origin affecting both cardiac and cerebral systems.[4,13,20–27]

Based on clinical, pharmacological, and literature evidence, the use of prostaglandin E1 in neonates should be regarded as a marker of disease severity, specifically duct-dependent congenital heart disease, rather than a causal factor for that severity. It is administered as a therapeutic intervention to maintain or reopen the ductus arteriosus, a vital temporary lifeline that allows blood to bypass obstructed pulmonary or systemic circulation.[28] Prostaglandin E1 is therefore used in neonates with critical congenital heart defects that depend on ductal patency for survival. These conditions include, but are not limited to, pulmonary atresia, tricuspid atresia, severe coarctation of the aorta, and transposition of the great arteries. Although prostaglandin E1 therapy may be associated with adverse effects such as apnea, fever, hypotension, or, in prolonged cases, cortical hyperostosis, these represent treatment-related side effects and not causes of the underlying cardiac defect.

CLINICAL SEVERITY AND BRAIN VULNERABILITY

Markers of postnatal clinical instability, including mechanical ventilation dependence and a high Bethesda risk category, also remained independently associated with abnormal TFUS findings. These variables likely reflect overall illness severity rather than isolated determinants of neurological injury. Mechanical ventilation may act as a secondary insult, exacerbating pre-existing cerebral vulnerability through alterations in cerebral perfusion and venous return.[29,30]

The persistence of these associations after adjustment for genetic status supports a ‘two-hit’ model, in which innate vulnerability is subsequently amplified by postnatal physiological stressors encountered in the neonatal intensive care unit.[4,25,30]

GESTATIONAL AGE AND CHD-ASSOCIATED BRAIN INJURY

Interestingly, gestational age did not emerge as a significant predictor of abnormal TFUS findings in our cohort. This contrasts with the general neonatal population, in which prematurity is the dominant risk factor for intracranial hemorrhage. However, this finding is consistent with neuroimaging studies demonstrating delayed brain maturation and microstructural abnormalities even in term neonates with complex CHD, whose cerebral development resembles that of preterm infants.[5,20] Licht *et al*.[5] reported that at birth, neonates with complex lesions such as hypoplastic left heart syndrome or transposition of the great arteries exhibit a delay in brain maturation of approximately one month compared with normative populations.

These observations suggest that, in the context of CHD, genetic burden and systemic developmental disruption may attenuate the neuroprotective effect of term gesta-

tion, effectively equalizing neurological risk across gestational age categories.[5,20]

LIMITATIONS OF LINEAR STATISTICAL MODELS AND FUTURE DIRECTIONS

Our findings indicate that neurological risk stratification in neonates with CHD should extend beyond anatomical classification alone. Genetic status and markers of early clinical instability appear to provide more relevant guidance for targeted neurological surveillance. While TFUS remains a valuable bedside screening tool due to its accessibility and safety, its findings should be interpreted within a broader clinical and genetic context rather than in isolation.[31]

Despite advances in perioperative care, long-term neurodevelopmental outcomes in children with CHD have shown limited improvement, underscoring the complex interplay between intrinsic developmental vulnerability, perioperative factors, and acquired brain injury.[32,33] Although our multivariate model identified independent predictors of abnormal TFUS findings, its overall explanatory power was modest, reflecting the limitations of traditional linear statistical approaches in capturing the non-linear physiological interactions characteristic of neonatal populations.[28]

From a systems neuroscience perspective, early brain pathology is increasingly understood as a network-level dysfunction rather than the consequence of isolated structural lesions. In neonates with congenital heart disease, abnormal TFUS findings observed in our cohort may therefore reflect broader neurodevelopmental dysregulation rather than isolated structural injury. Conceptually, evidence from computational and neurological models suggests that functional impairment can arise from complex, non-linear interactions that are not fully captured by conventional structural imaging or linear statistical approaches.[34,35]

Emerging evidence suggests that artificial intelligence and machine learning-based models, which integrate high-dimensional clinical, genetic, and physiological data, may enhance early risk stratification and represent a promising direction for future research in pediatric cardiology.[34,35]

STRENGTHS AND LIMITATIONS

The strengths of this study include systematic TFUS assessment and comprehensive clinical and genetic characterization of neonates with congenital heart disease, addressing a limitation of earlier studies that frequently excluded patients with genetic syndromes. However, the relatively small sample size and the retrospective, single-center design represent important limitations. Multicenter prospective studies incorporating advanced neuroimaging and genomic data are warranted to confirm and expand these findings.

CONCLUSION

Early neurological vulnerability in neonates with congenital heart disease appears to be driven primarily by genetic and systemic factors rather than cardiac anatomy alone. Integrative risk assessment strategies that combine TFUS findings with clinical and genetic markers may improve early neurological surveillance and support the development of precision-based neuroprotective approaches.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

ETHICAL APPROVAL

The study protocol received ethical approval from the Ethics Committee of Cuza Vodă Clinical Hospital of Obstetrics and Gynecology, Iași, Romania (approval no. 5/10.02.2025) and Grigore T. Popa University of Medicine and Pharmacy, Iași, Romania (approval no. 640/23.09.2025), prior to study initiation and manuscript submission. The study was conducted in accordance with the principles of the Declaration of Helsinki. Given the retrospective design, the analysis was performed using anonymized data.

CONSENT TO PARTICIPATE

Written informed consent for the use of clinical data for research purposes was obtained from the parents or legal guardians at the time of hospital admission.

FUNDING

This research received no external funding.

AUTHOR CONTRIBUTIONS

L.M. conceptualized the study, supervised the project, and performed review and editing. P.C.M. prepared the original draft. E.H. created the visualizations. R.M. performed the formal analysis. A.B. curated the data. S.I.D. performed review and editing, and supervised the project. All authors have read and approved the final version of the manuscript.

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