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ULTRASOUND-BASED DIAGNOSIS AND MANAGEMENT OF VEIN OF GALEN ANEURYSMAL MALFORMATION IN A NEONATE: A CASE REPORT AND COMPREHENSIVE LITERATURE REVIEW

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ABSTRACT

Background: Vein of Galen aneurysmal malformations (VGAMs) represent a complex and rare subset of congenital arteriovenous anomalies, comprising approximately 30% of all pediatric cerebral vascular malformations. Originating during early embryogenesis between the 6th and 11th weeks of gestation, these lesions create a low-resistance, high-flow shunt. Consequently, they frequently precipitate life-threatening complications, most notably high-output heart failure and severe neurological compromise in the immediate postnatal period, necessitating prompt and accurate diagnostic strategies.

Aim: The primary objective of this study is to elucidate the critical, first-line diagnostic and monitoring role of bedside transfontanellar ultrasonography in the management of neonatal VGAMs. Additionally, we aim to contextualize these clinical findings within a comprehensive review of the current literature, emphasizing recent paradigm shifts in neurointensive care and endovascular therapies.

Material and methods: We report the clinical case of a 4-week-old male newborn admitted to the emergency department with recurrent generalized seizures, progressive macrocephaly, and profound perioral cyanosis. A detailed clinical evaluation was conducted, prioritizing bedside neurosonography. This clinical presentation is supplemented by a comprehensive literature review exploring the evolving landscape of VGAM management, including molecular genetics and fetal interventions.

Results: Urgent transfontanellar ultrasonography augmented by color Doppler identified a prominent hypoechoic arteriovenous malformation demonstrating high-velocity turbulent flow. This was accompanied by secondary findings of lateral ventricle dilatation and diffuse parenchymal calcifications indicative of chronic ischemic injury. The precise neurovascular architecture was subsequently verified via Magnetic Resonance Imaging (MRI). Following multidisciplinary conservative management to allow for infant growth, the patient successfully underwent transarterial embolization at 7 months of age. A follow-up ultrasound at 9 months confirmed a significant reduction in arteriovenous shunting and stabilization of the cerebral structures.

Conclusions: Bedside transfontanellar ultrasonography serves as an indispensable, non-invasive, and radiation-free modality for the rapid identification, hemodynamic profiling, and serial follow-up of VGAMs in critically ill neonates. Its integration with advanced, staged endovascular techniques and emerging prenatal in utero interventions fundamentally transforms the clinical trajectory of this historically fatal congenital defect.

KEYWORDS

Vein of Galen Aneurysmal Malformation, Transfontanellar Ultrasonography, Neonatal Heart Failure, Transarterial Embolization, Pediatric Neurosonography

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1. Introduction

Aneurysmal malformations of the vein of Galen (VGAM) represent a highly specialized and remarkably challenging subset of congenital vascular anomalies. Although they account for less than 1% of all cerebral vascular malformations diagnosed prenatally and in the neonatal period, they are widely recognized as the most common symptomatic vascular malformation affecting neonates and infants [1, 2, 3]. The embryological origin of VGAM is traced back to a critical window between the 6th and 11th weeks of gestation. During this period, the median prosencephalic vein of Markowski fails to undergo its physiological involution. Instead, it forms abnormal, direct arteriovenous fistulous connections, creating a high-flow, low-resistance shunt that dilates the persistent venous sac [2, 4]. Anatomically, VGAMs are traditionally classified into two distinct angioarchitectural phenotypes: choroidal and mural. The choroidal type, which typically presents in the immediate neonatal period, is characterized by multiple, complex arterial feeders converging into the anterior aspect of the median prosencephalic vein. In contrast, the mural type usually features fewer fistulous connections, often presenting later in infancy with milder heart failure or progressive macrocephaly [5].

The pathophysiological hallmark of VGAM is the massive diversion of cerebral blood flow, which has devastating downstream effects. From a cardiovascular perspective, the low-resistance intracranial shunt drastically increases venous return, culminating in right ventricular volume overload, pulmonary hypertension,

and severe high-output heart failure shortly after birth [1, 2]. Neurologically, the high venous pressure impedes normal cerebral venous drainage, leading to progressive hydrocephalus, macrocephaly, and a "vascular steal" phenomenon that deprives developing brain tissue of adequate oxygenation [6, 4]. Recent advances in molecular diagnostics have begun to demystify the etiology of these malformations, with genetic sequencing identifying specific pathogenic variants, most notably in the EPHB4 and RASA1 genes, which govern angiogenesis and vascular remodeling [7]. The discovery of these molecular targets not only enhances our understanding of VGAM pathogenesis but also opens future avenues for precision targeted neurotherapeutics [7].

Despite these pathophysiological insights, the initial clinical approach to a hemodynamically decompensating neonate with suspected VGAM remains a critical diagnostic challenge. While Magnetic Resonance Imaging (MRI) is the gold standard for definitive neurovascular mapping [8], it often necessitates patient transport and sedation, posing significant cardiopulmonary risks. Consequently, there is an imperative need to emphasize rapid, non-invasive imaging modalities. Bedside transfontanellar ultrasonography, augmented by Doppler techniques, bridges this crucial gap, serving as an essential first-line tool for immediate diagnosis and hemodynamic assessment [8, 9].

The therapeutic paradigm for VGAM is currently undergoing a revolutionary shift. While sophisticated postnatal endovascular embolization techniques have dramatically improved survival rates [6, 3], a significant subset of neonates still suffers from irreversible brain injury acquired in utero [2]. Consequently, the scientific community is rapidly pivoting toward prenatal intervention. Groundbreaking clinical trials and recent publications have demonstrated the unprecedented feasibility of ultrasound-guided transuterine coil embolization of the fetal Vein of Galen. This pioneering in utero approach aims to reverse fetal compromise, mitigate high-output cardiac failure before birth, and optimize postnatal neurodevelopmental outcomes [10, 11, 12, 13].

2. Research materials and methods

2.1. Clinical Data Acquisition and Imaging Protocol

The clinical data for this report were obtained through a retrospective analysis of the patient's electronic medical records, encompassing postnatal clinical presentation, serial neuroimaging findings, and therapeutic interventions. Written informed consent was secured from the patient's parents for the publication of this case report and the accompanying medical data.

The primary diagnostic evaluation was conducted using bedside transfontanellar ultrasonography. B-mode imaging, supplemented by color and spectral Doppler, was performed through the anterior fontanelle using a high-frequency phased-array transducer, in accordance with standard neonatal neurosonography protocols [17]. The sonographic assessment explicitly targeted the identification of midline vascular anomalies, the quantification of turbulent high-velocity flow hemodynamics, and the evaluation of secondary cerebral injury indicators, including ventriculomegaly and parenchymal echogenicity [4, 8, 9]. Furthermore, systemic hemodynamic compromise was quantified via comprehensive two-dimensional (2D) echocardiography to assess right ventricular volume overload and pulmonary hypertension [1]. Definitive pre-interventional anatomical mapping was achieved utilizing high-resolution brain Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA) [8].

2.2. Comprehensive Literature Review Strategy

To appropriately contextualize this clinical case within the current scientific landscape, a comprehensive literature review was performed. A systematic search of electronic databases, including PubMed/MEDLINE, Scopus, and Web of Science, was conducted for literature published primarily between January 2012 and April 2026. The search strategy utilized the following medical subject headings (MeSH) and boolean operators: ("Vein of Galen Malformation" OR "VGAM") AND ("transfontanellar ultrasound" OR "neurosonography" OR "neonatal heart failure" OR "endovascular embolization" OR "in utero intervention").

The inclusion criteria were strictly limited to peer-reviewed case reports, retrospective cohort studies, clinical guidelines, and review articles published in the English language. The review prioritized literature that directly addressed rapid non-invasive diagnostic modalities [8, 9], modern staged endovascular interventions [3, 14], and recent breakthroughs in fetal therapies [10, 12]. The selection and synthesis of the literature followed a narrative approach, guided by the structural principles of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement to ensure academic rigor [18].

3. Research results

3.1. Clinical Presentation

A 4-week-old male newborn was admitted to the pediatric emergency department presenting with a triad of severe neurological and cardiopulmonary symptoms: recurrent generalized tonic-clonic seizures, progressive macrocephaly, and episodes of profound perioral and peripheral cyanosis. Recognizing the critical and rapidly deteriorating nature of this clinical picture, urgent multidisciplinary evaluation was initiated, prioritizing bedside neurosonography and echocardiography to avoid the cardiopulmonary risks associated with patient transport.

3.2. Diagnostic Imaging and Hemodynamic Profiling

Initial systemic evaluation via comprehensive 2D echocardiography revealed profound cardiovascular compromise. The imaging demonstrated marked right ventricular myocardial hypertrophy and a significantly enlarged cardiac silhouette. These anatomical changes were accompanied by elevated right heart pressures, a constellation of findings pathognomonic for high-output cardiac failure driven by a massive systemic arteriovenous shunt [1].

Concurrently, transcranial ultrasonography performed through the anterior fontanelle provided rapid, definitive visualization of the intracranial pathology [9]. B-mode imaging identified a prominent, hypoechoic cystic structure located in the midline of the brain. Upon the application of color and spectral Doppler mapping, this structure exhibited intense, multidirectional, and high-velocity turbulent flow, unequivocally classifying the lesion as an aneurysmal malformation [9]. The sonographic assessment successfully mapped a direct, low-resistance fistulous connection between the arterial feeders and the pathologically dilated persistent median prosencephalic vein draining into the straight sinus.

Secondary sonographic findings highlighted the devastating downstream effects of the vascular anomaly. Symmetrical, bilateral dilatation of the lateral ventricles was observed, along with the presence of multiple, small, diffuse echogenic calcifications scattered within the adjacent brain parenchyma. These features are highly indicative of chronic ischemic injury secondary to a severe "vascular steal" phenomenon [4]. To confirm the sonographic findings and delineate the precise distal angioarchitecture prior to intervention, the patient subsequently underwent high-resolution brain Magnetic Resonance Imaging (MRI), which definitively corroborated the bedside ultrasound diagnosis [8, 4].

3.3. Multidisciplinary Management and Clinical Outcome

Given the infant's critical condition and low body weight, a multidisciplinary board—comprising pediatric neurologists, cardiologists, and neurointerventionalists—elected to pursue initial conservative medical management. The primary goal was to medically control the high-output heart failure, thereby allowing for sufficient somatic growth to mitigate the substantial periprocedural risks associated with endovascular interventions in the neonatal period [6, 3]. At 7 months of age, having achieved adequate physiological stability and weight gain, the patient successfully underwent selective transarterial embolization [14]. The procedure was well-tolerated, with no immediate perioperative complications. A critical follow-up transcranial ultrasound performed at 9 months of age demonstrated a marked, objective reduction in the turbulent flow velocities within the residual venous sac. Furthermore, the imaging confirmed the stabilization of ventricular dimensions and the cessation of progressive macrocephaly, signifying a highly successful therapeutic outcome and effective reversal of the hemodynamic compromise [9, 6].

4. Discussion

Vein of Galen aneurysmal malformations (VGAMs) represent one of the most formidable challenges in modern neonatal critical care and interventional neuroradiology, historically associated with exceedingly high mortality rates when symptomatic in the early postnatal period. The primary immediate threat to a newborn affected by this vascular anomaly is rapidly progressive, high-output congestive heart failure, which is precipitously driven by massive, low-resistance arteriovenous shunting within the cerebral vasculature [1, 2]. This fistulous architecture drastically accelerates venous return to the right atrium and ventricle, inevitably culminating in right ventricular volume overload, severe pulmonary hypertension, and subsequent systemic circulatory collapse [1]. In the present case, these classical pathophysiological mechanisms were vividly demonstrated by bedside echocardiography, which revealed marked myocardial hypertrophy and right-sided chamber dilatation [1]. Parallel to these profound cardiovascular disturbances, VGAM induces a cascade of devastating neurological sequelae. The markedly elevated venous pressure within the dural sinuses directly impedes the physiological resorption of cerebrospinal fluid, clinically manifesting as progressive obstructive hydrocephalus and macrocephaly [4, 6]. Furthermore, the hemodynamically driven "vascular steal" phenomenon actively deprives the developing cerebral cortex of essential oxygenation and metabolic substrates, inducing localized tissue hypoxia that served as the direct trigger for the recurrent generalized tonic-clonic seizures and parenchymal calcifications observed in our patient [4].

Given the extreme clinical fragility of hemodynamically decompensating neonates, the selection of an immediate and accurate diagnostic workflow is a critical determinant of survival. Although Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA) remain the gold standard for definitive pre-interventional neuroanatomical characterization and precise mapping of distal feeders [8], the logistical necessity of transporting a critically ill neonate outside the neonatal intensive care unit, combined with the mandatory requirement for sedation or general anesthesia, poses unacceptable cardiopulmonary risks [2, 8]. In this clinical scenario, bedside transfontanellar ultrasonography, executed using high-frequency transducers in strict accordance with established international parameters [17], emerges as an unparalleled first-line diagnostic asset [8, 9]. The integration of color and spectral Doppler imaging allows for the immediate differentiation of VGAM from morphologically similar, avascular cystic lesions—such as arachnoid, porencephalic, or pineal cysts—by unequivocally demonstrating high-velocity, turbulent, multidirectional flow within the median prosencephalic vein [9]. Furthermore, serial, non-invasive neurosonographic tracking plays an indispensable role in monitoring the longitudinal evolution of secondary intracranial complications, thereby guiding clinicians in determining the optimal therapeutic window for definitive intervention [6, 9].

The contemporary therapeutic paradigm for VGAM relies almost exclusively on transcatheter endovascular embolization techniques, which over the past decades have completely superseded open neurosurgical interventions due to the unacceptably high morbidity and mortality associated with the latter [3]. Modern neurointerventional protocols strongly advocate for a staged embolization approach, which involves the sequential, progressive occlusion of primary arterial feeders utilizing advanced embolic agents, detachable microcoils, or liquid tissue glues [13, 14]. This staged strategy is specifically engineered to prevent abrupt, catastrophic alterations in intracranial venous hemodynamics, which could otherwise precipitate extensive dural sinus thrombosis or fatal parenchymal hemorrhage [3]. In our case, intentionally delaying radical endovascular intervention until 7 months of age—while aggressively managing the high-output heart failure medically—allowed the infant to achieve adequate somatic growth and physiological reserve, thereby substantially mitigating the periprocedural and anesthetic risks inherent to the neonatal period [3, 6]. The therapeutic efficacy of this staged approach was objectively validated by follow-up transfontanellar ultrasound at 9 months of age, which confirmed a significant reduction in shunt velocities, progressive intra-lesional thrombosis, and the successful stabilization of ventricular dimensions [6, 9]. Furthermore, recent large-scale meta-analyses and literature reviews [19, 20] robustly demonstrate that while modern endovascular embolization significantly improves overall survival, appropriate patient selection and delaying the intervention beyond the immediate neonatal period whenever clinically feasible are key factors in optimizing long-term neurodevelopmental outcomes.

Perhaps the most promising and rapidly evolving frontier in fetal medicine and neurosurgery is the advent of in utero interventions. Despite substantial advancements in postnatal endovascular care, a tragic subset of neonates with aggressive VGAM phenotypes succumb to intractable cardiogenic shock or acquire irreversible encephalomalacia before any postnatal intervention can be safely executed [2]. Groundbreaking clinical reports published between 2024 and 2026 have established the technical feasibility, safety, and efficacy of ultrasound-guided transuterine coil embolization of the fetal Vein of Galen [10, 11, 12]. This pioneering

prenatal approach aims to mechanically reduce the volume of the arteriovenous shunt in utero, thereby reversing fetal cardiac compromise, mitigating hydrops fetalis, and protecting the highly vulnerable, developing cerebral parenchyma from ischemic destruction prior to delivery [10, 12]. Although these fetal procedures require highly specialized, multidisciplinary interventions and strict patient selection, early reports detailing favorable long-term neurodevelopmental outcomes suggest that the widespread integration of advanced prenatal ultrasound screening and early in utero therapy will fundamentally reshape the natural history of VGAM, transforming a historically fatal congenital defect into a manageable condition with an excellent long-term prognosis [11, 13, 15].

5. Conclusions

The diagnosis and management of Vein of Galen Aneurysmal Malformations (VGAM) in the neonatal period demand a highly coordinated, multidisciplinary approach to mitigate the profound risks of catastrophic high-output heart failure and irreversible ischemic brain injury [1, 21]. As demonstrated in this report, bedside transfontanellar ultrasonography, augmented by comprehensive color and spectral Doppler mapping, stands as an indispensable first-line diagnostic and monitoring modality [8, 9]. Its non-invasive, radiation-free profile and bedside availability critically bypass the cardiopulmonary risks associated with neonatal transport and sedation, providing immediate, high-fidelity hemodynamic profiling essential for acute clinical decision-making [9, 17].

Moreover, this case underscores that delaying definitive endovascular intervention until later in infancy—when facilitated by aggressive, successful medical management of early cardiac failure—significantly improves physiological reserve and perioperative safety [3, 14]. Looking forward, the synergistic integration of advanced neurosonography, staged postnatal transarterial embolization, and the rapidly emerging frontier of targeted in utero fetal interventions offers a revolutionary therapeutic pathway [10, 12, 22]. This comprehensive, multimodal strategy fundamentally alters the natural history of VGAMs, transitioning a traditionally fatal congenital anomaly into a manageable condition with increasingly promising long-term neurodevelopmental outcomes [20, 22].

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