Anesthetic Management of Vein of Galen Malformations

23

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Key Points

- True vein of Galen malformations are fistulous communications in the cerebral midline resulting in high-flow shunts and present as high-output cardiac failure in neonates.
- The advent of modern endovascular embolization techniques has drastically reduced mortality and improved neurological outcomes.
- Medical management of cardiac failure takes priority over definitive treatment, but emergency endovascular embolization may be carried out in a neonate with cardiac failure refractory to medical management.
- Anesthetic challenges in managing this group of children involve age-related considerations, difficult airway primarily due to increased head circumference, possible hemodynamic instability during the course of anesthesia, and requirement of postoperative ventilation.
- In neonates posted for emergency embolization, additional anesthetic considerations include difficulty in securing intravenous and invasive arterial lines, dyselectrolytemia (diuretics, digoxin, acidosis), maintenance of adequate systemic perfusion to prevent volume overload and worsening of heart failure and prevent hypoperfusion and worsening of renal injury (contrast and drug-induced).

23.1 Introduction

Vein of Galen malformations (VOGMs) are rare congenital anomalies that constitute 1% of all intracranial vascular malformations and represent 30% of vascular malformations in the pediatric population [1]. The anomaly was mentioned initially by Steinheil (1895); however, the first clinical description of a vein of Galen aneurysm was given by Jaeger et al. (1937) [2]. Variably referred to as "aneurysms of the vein of Galen," "arteriovenous aneurysms of the vein of Galen," "vein of Galen aneurysmal malformations," and "vein of Galen malformations," true VOGMs are actually persistence of the embryonic median prosencephalic vein and not the vein of Galen. The incidence of this condition in the population is 1:25,000 with a male preponderance (3:1) [3]. Left untreated, the mortality rate is almost 100% and with high morbidity in the form of cardiac failure and long-term neurodevelopmental delay [4].

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23.2 Anatomy and Embryology of VOGM

The normal vein of Galen, or the great cerebral vein, is a part of the deep venous system of the brain. It is a short (1–2 cm) thick vein located behind the splenium of the corpus callosum in the midline. It courses posteriorly where it joins the inferior sagittal sinus to form the straight sinus that ultimately drains into the confluence of sinuses (torcular Herophili) [5].

Vein of Galen arteriovenous malformations are congenital abnormal communications between arterial feeders and draining veins located in the midline in the choroidal fissure. The feeding arteries include the anterior and posterior choroidal arteries and the anterior cerebral artery, supplying the choroid plexus. Venous drainage is through the median prosencephalic vein (MPV) of Markowski, which is the embryonic precursor of the vein of Galen (Fig. 23.1) [2].

The development of cerebral vasculature can be divided into three stages, initial extra embryonal stage, extrinsic vascularization (around the neural tubes), and intrinsic vascularization, where blood vessels develop within the cerebral parenchyma. Between 6 and 11 weeks of gestation, the cortical arterial network develops from the internal carotid artery. The venous drainage of the brain at this stage is through the median prosencephalic vein, which develops in the root of the diencephalon. Except for its most distal part, regression of this vein occurs at around 11 weeks of gestation when the newly formed internal cerebral veins take over the venous drainage. The distal part of the median prosencephalic vein joins the internal cerebral vein, and the paired internal cerebral veins join to form the great vein of Galen [2].

Any abnormality in the development of cerebral vasculature during this phase between 6 and 11 weeks of gestation results in direct arteriovenous communication between the arterial network and median prosencephalic vein. The lack of a fibrous wall and its presence unsupported in the cistern of velum allows the vein to expand and balloon out as the flow increases preventing its regression [6]. The falcine sinus (connecting straight sinus and superior sagittal sinus), which usually disappears beyond the fetal stage, may continue to persist. Straight sinus may be absent.

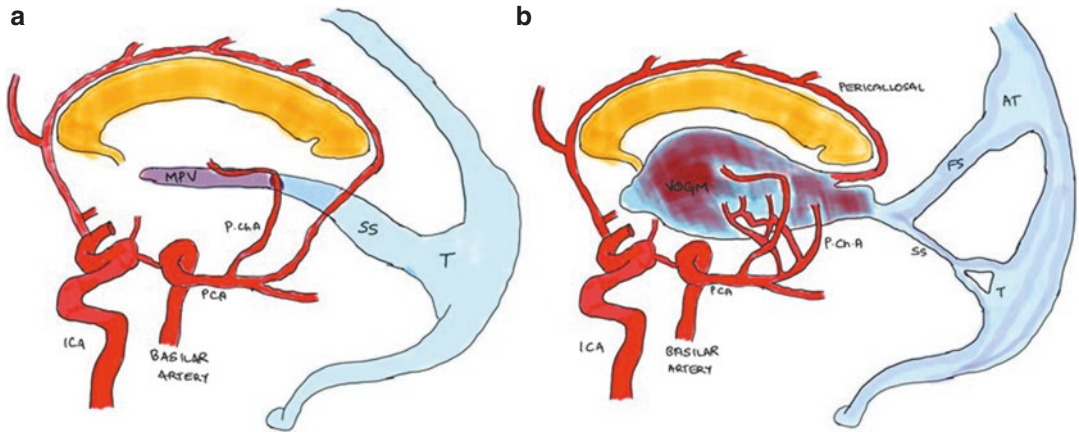

Fig. 23.1 Schematic diagram showing the development of vein of Galen aneurysmal malformation with its feeders. (**a**) The median prosencephalic vein (MPV) drains into the straight sinus (SS) and further into the torcula (T). Normal anastomosis exists between the pericallosal and the distal branches of the posterior cerebral artery (PCA). (**b**) Enlarged and malformed MPV with arterial feeders from pericallosal and posterior choroidal arteries (PChA) draining into an accessory torcula (AT) through a persistent embryonic falcine sinus (FS) and a possible stenotic straight sinus (SS)

23.3 Pathophysiology

Cardiovascular: VOGM is characterized by a low-resistance and high-flow shunt. Changes in cardiovascular physiology and blood flow patterns between fetal circulation and postnatal life determine the pathophysiology of this condition. During fetal life, the low-resistance placental circulation prevents higher shunt fraction across the VOGM. But, changes in vascular resistance in the postnatal period results in a sudden and progressive increase in blood flow through the cranial shunt. It is estimated that around 80% of cardiac output flows through the VOGM, which results in increased venous return and pulmonary overload (pulmonary hypertension), and cardiac failure. Cyanosis may result due to increased right-sided pressures and a right-to-left shunt through the ductus arteriosus and the patent foramen ovale [7].

Secondly, the large arteriovenous shunt causes wide pulse pressure, and the resistance across the shunt may be so low that it can cause a reduced or retrograde flow in the aorta during diastole, which results in reduced coronary perfusion and increases the risk of myocardial ischemia and hence cardiac failure. Thus, cardiac failure may be multifactorial and be refractory to medical management [8]. Aortic flow reversal may also lead to renal hypoperfusion and injury.

Neurological: Immaturity of the cerebral venous system is the major pathophysiological factor in VOGM. Cerebral venous hypertension results from the large amount of blood flowing through it due to the VOGM. Structural defects like an immature deep cerebral venous system, absent sinuses including the straight sinus, and jugular bulb stenosis might result in aggravation of the cerebral venous pressure. Also, cerebrospinal fluid (CSF) reabsorption in the neonate occurs through the ventricular ependyma and brain parenchyma into the medullary veins as the arachnoid granulations do not mature until 16–18 months of age. The increased venous pressure in the brain in VOGM is reflected in the cortical and medullary veins impairing CSF reabsorption and results in hydrocephalus [9].

Together, cerebral venous hypertension and hydrocephalus lead to impaired cerebral oxygenation, edema, and subependymal atrophy. The most severe form of this cerebral atrophy is called *melting brain syndrome* [10, 11].

23.4 Clinical Manifestations

The clinical features are mainly cardiac and neurological. Age and severity of presentation may vary based on the shunt size (Table 23.1). The presentation in neonates varies from asymptom-

Table 23.1 Differences in vein of Galen malformation based on age of presentation [17–19]

	Age at diagnosis			
Parameter	Prenatal	Neonate	Infant	Older child
Clinical presentation	Incidental diagnosis	Heart failure (left-to-right shunt causing volume overload on the heart)	Macrocephaly and hydrocephalus	Headaches, developmental delay, hydrocephalus
Angiographic type		Choroidal	Mural > choroidal	Mural
Severity	Melting brain detected on fetal MRI detects poor prognosis	Most severe forms in this age group. Mortality increases with delay in diagnosis or prompt treatment of heart failure	Less severe. Heart failure controlled with medications and presenting for elective embolization	Usually mild. Developmental delay may persist
Management	Facilitate the delivery of a child at a tertiary care center equipped to manage the condition	Medical management of heart failure. Emergency embolization of feeders to reduce the shunt fraction	Elective embolization	Elective embolization

atic cardiomegaly to refractory cardiac failure. Common initial presentation in a neonate is that of the rapid deterioration of an initially unremarkable postnatal period; there is worsening of cardiac failure presenting with severe respiratory distress and usually requiring mechanical ventilation. Signs include prominent carotid pulsation and weak peripheral pulses, acrocyanosis, coarse breath sounds over both lung fields, ejection systolic murmur heard over the precordium, a continuous carotid bruit, and a soft cranial bruit (heard better over the posterior cranium) based on the shunt fraction and severity of heart failure [12, 13]. Unless promptly detected and managed, multiorgan failure may ensue. Other forms of presentation include mild cardiac failure and failure to thrive. Neurological features include seizures and macrocephaly secondary to hydrocephalus. Older children generally present with milder symptoms, which include those of venous outflow obstruction like increased head size and prominent facial veins. Initial presentation in adulthood is extremely rare but has been reported. Presenting features include headaches (SAH), focal deficits, and even incidental diagnosis [14–16].

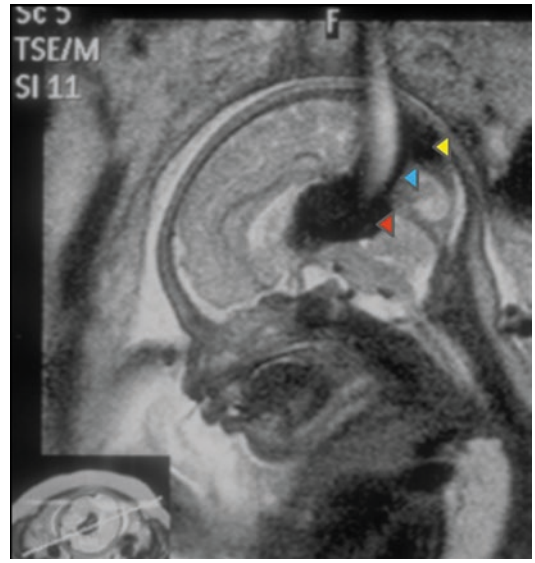

Fig. 23.2 Antenatal T2 MRI sagittal view showing a hypointense enlarged sac near the floor of the third ventricle communicating with the torcula suggestive of the vein of Galen malformation (arrowheads: red, VOGM; blue, falcine sinus; yellow, torcula)

23.5 Diagnosis

The presentation of a high-output cardiac failure in a neonate with normal cardiac anatomy on a transthoracic echocardiogram should prompt the clinician to look for a high-flow extracardiac vascular shunt in the form of a VOGM. Various investigative modalities are available for the diagnosis:

- *Prenatal ultrasound* occasionally detects the VOGM as a midline pulsatile hypoechoic lesion during the third trimester of pregnancy (28–34 weeks of gestation). Confirmation of the diagnosis is obtained with a color flow Doppler. The major significance of prenatal diagnosis is to assess the extent of the cerebral damage, to plan delivery and treatment of the lesion at a center equipped with appropriate specialties [20]. Fetal echocardiogram and antenatal MRI (Fig. 23.2) reveal the extent of cardiomegaly and neurological damage, respectively, which helps decide an appropriate further plan of management, including termination of pregnancy (abortion) [21–23].
- *Trans-fontanelle ultrasound* is done in a neonate with cardiac failure and an unremarkable thoracic echocardiogram.
- *Contrast-enhanced computed tomography (CT)/magnetic resonance imaging (MRI)* is usually the initial mode of evaluation. *MR angiography* (MRA) helps assess the type of lesion, presence of a nidus, venous drainage, and thrombosis, if any, and determine the number and the nature of arterial feeders. Imaging also reveals other features like hydrocephalus, the extent of cerebral atrophy, and calcifications and helps plan treatment appropriately (Fig. 23.3). *Susceptibility-weighted imaging (SWI)* is a recent MRI technique which allows for high-resolution imaging of the cerebrovascular architecture without the requirement of intravenous contrast administration, making it more useful in this group of patients with a higher risk

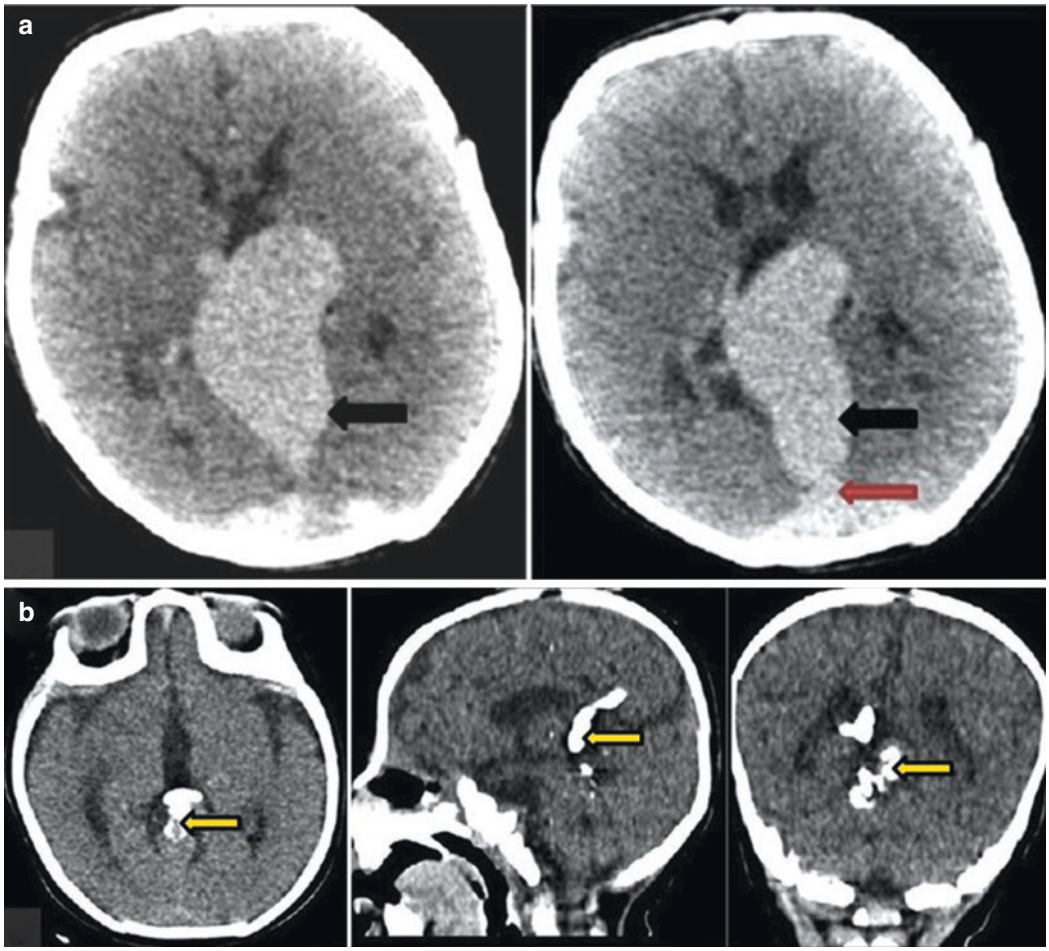

Fig. 23.3 (**a**) Non-contrast axial CT scan of the head pre-embolization showing dilated venous sac (black arrow) near the floor of the third ventricle with communication with the torcula (red arrow) and no significant evidence of brain parenchymal damage or calcifications. (**b**) Axial, sagittal, and coronal non-contrast CT showing obliteration of the venous sac post-embolization with glue cast (hyperdensity indicated by yellow arrows)

of renal injury (prematurity, multiorgan dysfunction, and multiple procedures requiring contrast agents—MRI, DSA). It also assesses the extent of cortical venous congestion, which is an indicator of cerebral parenchymal damage [24].

- *Digital subtraction angiography (DSA)* is the gold standard for the diagnosis of VOGM. It is best done at the time of embolization considering the risk of high doses of contrast dyes required for CT/MRI and DSA and the subsequent risk of renal damage and preventing the need for multiple anesthesia exposures in a neonate (Fig. 23.4).

23.6 Differential Diagnosis

The differential diagnosis of a midline hypoechoic lesion in the brain on ultrasonographic evaluation should include arachnoid cysts, porencephaly, and choroid plexus cysts. They can present with hydrocephalus, seizures, and neurodevelopmental delay, but the presence of cardiac failure effectively rules out their diagnosis. Even in the absence of cardiac manifestations, a simple demonstration of blood flow in the lesion with a color flow Doppler is sufficient [25, 26]. Further, true vein of Galen malformation has to be differentiated from a varix and an ectasia:

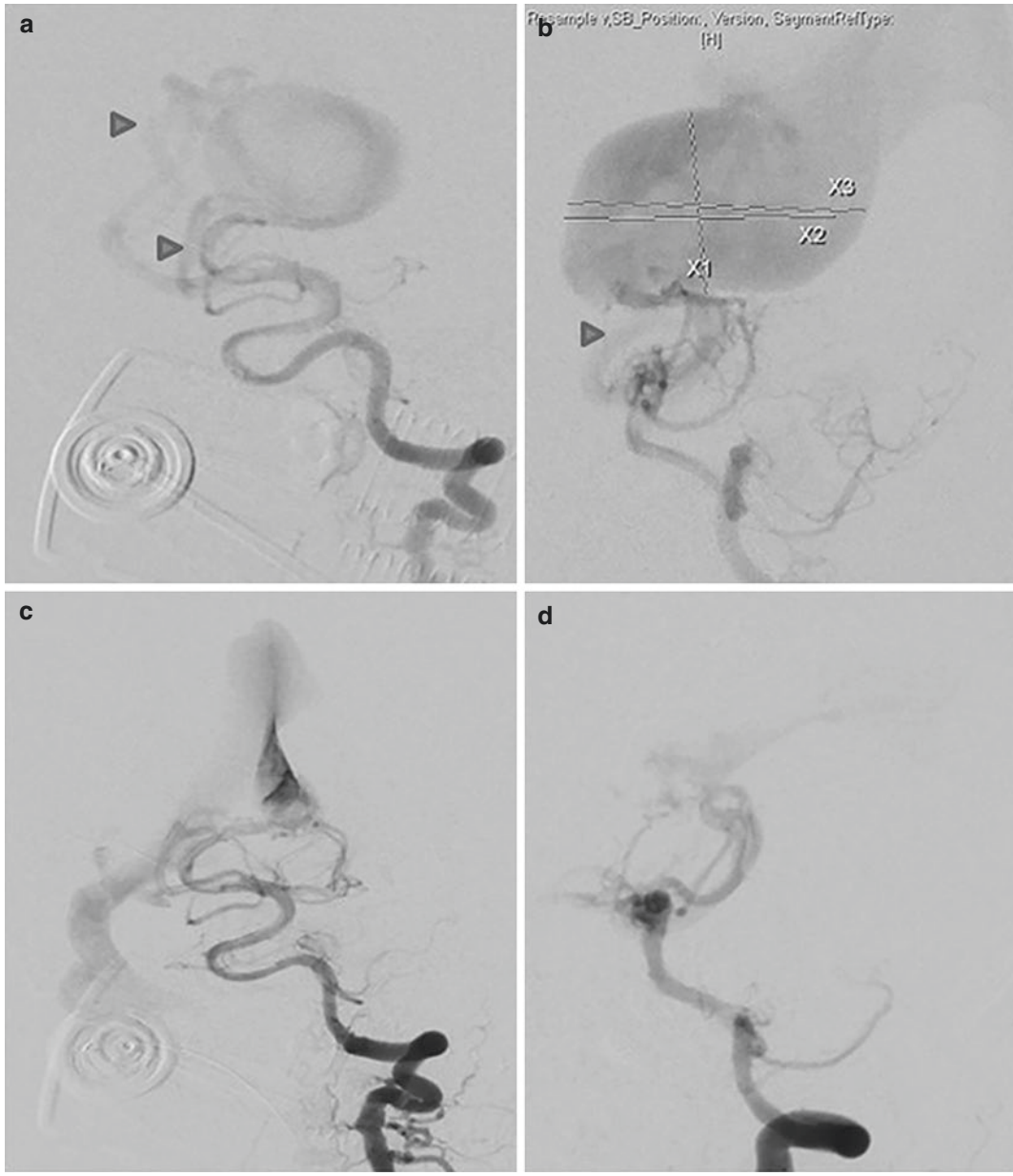

Fig. 23.4 Left vertebral angiogram: frontal (**a**) and lateral (**b**) projections showing mural-type vein of Galen malformation supplied by feeders from posterior choroidal arteries (blue arrowheads). (**c**, **d**) Post-glue embolization showing obliteration of the venous sac

- **Vein of Galen varix** represents a dilated vein of Galen without any arteriovenous shunt [17]. Neonates present with right-sided cardiac failure due to hypoxia and pulmonary hypertension. Treatment of cardiac failure generally results in the resolution of varix without the need for operative intervention [27].
- **Vein of Galen ectasia (dilatation)**: It occurs due to an outflow obstruction. The dilated vein usually drains an aneurysm in the subarachnoid space and the adjacent parenchyma. It

usually presents later in childhood with delayed psychomotor development, intracranial hemorrhages, or focal deficits depending on the degree of stenosis or thrombosis of the vein. Clinical presentation in the neonatal period is rare. Seizures and heart failure are also uncommon [28, 29].

23.7 Classification VOGM

Multiple classification systems have been proposed in the past, based on the angiographic appearance of the vessels. The most commonly used is the system proposed by Lasjaunias, which classifies the malformations into two types: choroidal and mural [30].

Choroidal type is characterized by the location of the fistula at the anterior end of the median prosencephalic vein and mural type by the fistula on the wall of the median prosencephalic vein, mostly on the inferolateral margin. The major difference between the two types is the nature of the feeding vessels. Multiple feeders in choroidal-type VOGM give the appearance of an arterial maze in contrast to the fewer feeders in mural type, which makes embolization relatively less complicated. Mural-type VOGM also presents with lesser degrees of heart failure [31].

23.8 Medical Management

Treatment of VOGM consists of initial medical management and stabilization of heart failure in the neonates before proceeding with endovascular intervention. Optimal heart failure management includes reducing pulmonary hypertension, facilitation of adequate perfusion to vital organs, and prevention of multiorgan dysfunction. Beta-adrenergic agonists (dopamine, dobutamine, adrenaline) commonly used for heart failure results in tachyarrhythmias, which can further reduce diastolic coronary perfusion and potentiate the risk of myocardial ischemia [32]. The improvement in cardiac contractility expected with these drugs may not be significant due to right ventricular dilatation. The addition of arterial vasodilators such as sodium nitroprusside (SNP) 1–5 μg/kg/min, glyceryl trinitrate (GTN) 1–5 μg/kg/min, or milrinone (0.75 μg/kg/min) to low-dose dopamine (<10 μg/kg/min) produces considerable improvement in systemic perfusion and reduces metabolic acidosis. Milrinone, in particular, reduces both systemic and pulmonary vascular resistances. Hence, it favors forward flow into the systemic circulation and reduces the afterload on the failing right ventricle, respectively [33]. It also plays a significant role in preventing neurological damage by stabilizing hemodynamic parameters in the intraoperative period and preventing cerebral hyperemia in the immediate postoperative period. Once acute heart failure is resolved, maintenance therapy with a combination of digoxin and diuretics may be initiated. Although digoxin continues to be a part of the treatment for chronic heart failure, its role in offering mortality benefits has been questioned by recent trials [34, 35]. As a general rule, diuretics and varying doses of inotropic support may be initiated in mild to moderate cardiac failure without cyanosis for initial stabilization.

23.8.1 Management of Pulmonary Hypertension [36–40]

The development of pulmonary hypertension leads to severe hypoxemia, respiratory distress, and cyanosis. An increase of pulmonary artery pressures to supra-systemic levels can occur, leading to the development of a right-to-left shunt through the patent ductus arteriosus (PDA) or an arterial septal defect (ASD). Knowledge of management of such cases posted for emergency embolization is of prime importance to the neuroanesthesiologist:

1. The medications are usually continued in the perioperative period and may result in exaggerated effects of anesthetic agents.
2. Optimization of ventilatory strategies.
3. Perioperative events causing hypoxia, hypercarbia, or acidosis can further aggravate the pulmonary vascular resistance and pose difficulties in management.

Initial management of pulmonary hypertension is oxygen therapy (pulmonary vasodilator), the delivery of which depends on the severity. Usually, these neonates are intubated and mechanically ventilated with high oxygen requirements. General management includes maintenance of normothermia, normoglycemia, correction of dyselectrolytemia (neonates are prone to hypocalcemia) and adequate intravascular volume, and initiation of inotropic support.

Conventional volume-targeted mode of ventilation with low peak inspiratory pressures (PIP) and optimal positive end-expiratory pressure (PEEP) to maintain normocarbia ($PaCO_2$: 40–45 mmHg) or mild permissive hypercarbia ($PaCO_2$ up to 60 mmHg) is preferred. The target is to correct hypoxia, recruit alveoli, and also prevent ventilator-induced lung injury. Ventilator dyssynchrony increases the work of breathing, and hence the neonates should be adequately sedated, preferably with fentanyl (1 μg/kg/h), considering its hemodynamic stability.

Pharmacotherapy includes the use of inhaled nitric oxide (iNO) at a dose of 20 parts per million (ppm). iNO is a selective pulmonary vasodilator with little effect on systemic circulation. Once initiated, the effect is observed as early as 30 min, noted by an increase in PaO_2. Phosphodiesterase (PDE) inhibitors milrinone (PDE-3) and sildenafil (PDE-5) can be used as additional therapy or as alternatives to iNO. SNP and GTN can also be used as both are NO donors and cause systemic as well as pulmonary vasodilatation.

It is recommended to maintain the patency of ductus arteriosus in this condition to allow for systemic circulation (and counter for significant diastolic steal phenomenon) by using prostaglandin infusions (PGE1 or PGE2).

23.9 Definitive Treatment

The major goal of treatment of VOGM is to prevent neurodevelopmental delay that occurs as a consequence of cerebral venous hypertension. The definitive treatment for the obliteration of the VOGM is carried out once heart failure is managed initially on presentation. Treatment options available include endovascular embolization, surgery, and stereotactic radiotherapy (Table 23.2). Surgery is associated with extremely high mortality and morbidity. Radiotherapy is less effective compared to surgery and endovascular therapy.

Table 23.2 Comparison of treatment options for the vein of Galen malformation [41–44]

	Endovascular	Microsurgery	Gamma knife
Preference	Almost all treatable cases undergo embolization	Obsolete (with the advent of modern endovascular methods)	Done only in atypical presentation or secondary to failure of endovascular treatment
Success rates (clinical outcomes)	Up to 75%	10–15%	40–50%
Advantages	1. Immediate relief 2. It can be done as an emergency procedure even in patients with severe heart failure 3. Less invasive and hence less morbidity	1. Complete occlusion and immediate relief albeit in a relatively stable (some amount of neurological/cardiac compromise) patient after the failure of embolization	1. Cure rates similar to or even better than microsurgery
Disadvantages	1. Technical difficulties in the neonate 2. Complications related to embolic materials: dislodgement, hemorrhage, etc.	1. A high rate of complications: massive blood loss, cardiac arrest 2. The requirement of hypotension, deep hypothermia 3. Complications related to positioning—sitting position	1. No immediate relief 2. Done only in the simplest angioarchitecture 3. Theoretical concerns of tumors post-radiation

23.9.1 Endovascular Embolization

Endovascular embolization of the feeder vessels is a safe treatment option with superior results. It is usually performed at around 5 months of life as the procedure may be difficult in the neonatal period, where medical management of heart failure takes precedence [3, 45]. In cases of heart failure refractory to medical therapy, emergency embolization may be performed in the neonatal age group to reduce the shunt fraction and not majorly focus on complete obliteration of the malformation [9].

The decision about emergency embolization may be considered based on the Bicetre score, a 21-point scale that assesses cardiac, respiratory, neurological, hepatic, and renal functions [46, 47]. The system involvement is graded from normal (score 5) to derangements resistant to medical management such as refractory cardiac failure, permanent neurological deficit, hypoxia despite mechanical ventilation, coagulopathy with deranged liver enzymes, and anuria (score 0). A score of more than 12 indicates well-preserved major organ function, and ideal treatment in such cases would be to delay embolization until 5 months of age. A score of 8–12 indicates worsening function and is an indication for emergency embolization. A score of less than 8 indicates a poor prognosis.

23.9.1.1 Route of Embolization

Trans-arterial, transvenous, and transtorcular approaches have been described. The transtorcular route, previously believed to yield better results, has now been replaced by the arterial route, which is safer and yields better results even with embolization of limited feeders rather than complete occlusion [28, 48]. Access is usually established through the femoral artery. In neonates, the umbilical artery can also be cannulated for the procedure (up to the third day of life). It is essential to preserve this artery, especially in cases where the prenatal diagnosis has been established [49]. The transvenous route is associated with complications like hemolysis due to flow through a partially occluded feeder and disseminated intravascular coagulation (DIC) [50, 51].

23.9.1.2 Embolic Agents

Liquid acrylic agents (n-butyl cyanoacrylate) and coils are used for embolization. Liquid agents have the advantage of being able to be injected through the microcatheter into the tortuous circulation of the neonate and the infant. The advantages are a better chance of permanent occlusion and a reduced procedure time, which is crucial in these patients. Coils, used currently, are replacing the liquid agents as they can be better maneuvered to the exact location of the malformation. There is a reduced risk of distal migration with coils. Even if it occurs, coils can be retrieved easily, unlike the liquid agents [28]. Recently, Onyx glue has been used in the endovascular embolization of cerebral AVMs, including VOGM. It is a non-adhesive slow-polymerizing agent composed of a mixture of ethylene-vinyl alcohol (EVOH) in dimethyl sulfoxide (DMSO). When placed, the occlusion of flow is achieved, not by thrombosis but by the mechanical obstruction, further reducing the risk of distal migration [52]. The injection of this agent is also better controlled compared to other agents.

23.9.2 Stereotactic Radiosurgery

Different types of radiation—gamma knife, linear accelerator, and proton beam—have been used to treat cerebral arteriovenous malformations, the main mechanism being radiation-induced intimal injury. The role of stereotactic radiotherapy is currently restricted to cases with atypical presentation and those not amenable to endovascular therapy [41, 53, 54]. The disadvantages include a longer duration of treatment required, typically months or years.

23.10 Anesthetic Considerations in Vein of Galen Malformations

The anesthetic goals in VOGM are similar to other neurosurgical procedures, such as avoidance of increase in intracranial pressure (ICP), maintenance of cerebral perfusion pressure (CPP), and cerebral oxygenation. The induction and maintenance of anesthesia should be done carefully while preventing hypotension or desaturation. The anesthetic concerns for a neonate or child posted for embolization of VOGM and preparedness are mentioned in Table 23.3.

Table 23.3 Anesthetic concerns in children with the vein of Galen malformation

Anesthetic concerns	Preparedness
• Neonatal age: low weight, susceptibility to anesthetic drugs, and hypothermia • Anticipation and management of difficult airway: macrocephaly and hydrocephalus • Managing heart failure perioperatively • Fluid management and electrolyte imbalance • Considerations of anesthesia in a remote location • Considerations in interventional neuroradiology suite • Detection and management of intraoperative complications associated with the procedure	• Strict weight-based fluid calculations and administration, titration of drugs to effect, forced air warmers, warming blankets, warm intravenous fluids • Appropriate head positioning, difficult airway cart • Continue inotropic medications, maintain hemodynamic stability, and adequate depth of anesthesia, opioids, muscle relaxants • Prevention of fluid overload, maintaining adequate perfusion, use of diuretics if required, frequent monitoring of ABG for electrolytes, lactate, and blood sugar levels • Equipment availability—oxygen, suction, resuscitation, defibrillator, and monitoring. Personnel availability: reducing chances of errors [55, 56] • Radiation safety, anti-coagulation and reversal, intravenous contrast agents, and flush [57] • Bradycardia, blood loss, and hemodynamic instability. Close communication with neuroradiologist to detect and manage complications

23.10.1 Preoperative Evaluation

A complete preoperative evaluation should be carried out in a neonate/child being posted electively to embolize VOGM. This includes eliciting an appropriate history of events from the postnatal period, including heart failure and management. Records should be sought for which may reveal evidence of multiorgan dysfunction. The child may continue to be on anti-failure medications and anti-seizure prophylaxis; details of such medications should be noted. The systemic examination should be carried out to evaluate the current status of cardiac function and identify signs of heart failure if any. Neurological examination should focus on documentation of focal deficits. The presence of macrocephaly is an indicator of a potentially difficult airway and requires proper head positioning during the anesthetic induction.

Investigations to be sought include hemoglobin levels to assess for anemia and coagulation profile to rule out possible hepatic dysfunction related to cardiac failure. Failure to thrive and recurrent infections due to malnourishment may be observed in these children, resulting in anemia, altered blood counts, and hypoproteinemia. Dyselectrolytemia may be evident following nausea and vomiting associated with hydrocephalus and raised ICP and diuretic use for cardiac failure management. Digoxin, if used, warrants caution as co-administration with diuretics may cause hypokalemia and precipitate toxicity. It may also occur due to renal failure associated with multiorgan dysfunction or contrast-induced. Though not routinely recommended, a clinical suspicion or anticipation of critical events should warrant estimation of serum digoxin levels (normal therapeutic range: 0.8–2 ng/mL) [34]. Chest roentgenogram findings include cardiomegaly with pulmonary congestion (plethora), increased right

atrial and right ventricular size, widening of the superior mediastinum, and anterior displacement of the upper airway. An electrocardiogram shows right axis deviation, right atrial enlargement, right ventricular hypertrophy, severe/fulminant heart failure, or biventricular hypertrophy [18, 58]. A transthoracic echocardiogram reveals important information for the neuroanesthesiologist, like associated congenital anomalies like septal defects and coarctation of aorta, and evidence of pulmonary hypertension. Both these lesions are hypothesized to be related to the blood flow characteristics and shunt flow related to VOGM. The common findings are dilatation of cardiac chambers suggestive of volume overload, elevated pulmonary artery pressures (pulmonary hypertension), tricuspid regurgitation, PDA, or an intracardiac right-to-left shunt [52]. The presence of a sinus venosus ASD increases the chances of paradoxical embolism, especially in the case of distal migration of liquid embolizing agent; it should be carefully considered [60].

23.10.2 Induction

The major anesthetic concerns of induction are hemodynamic responses associated with the use of pharmacological agents and those of laryngoscopy and intubation. Both inhalational and intravenous agents can be used for induction while attempting to maintain hemodynamic parameters within baseline levels. Opioids like fentanyl (1–2 μg/kg) to prevent hemodynamic responses and non-depolarizing muscle relaxants to facilitate smooth endotracheal intubation are routinely used. Also of equal concern is the relatively short apnea time and possible hypoxia in this age group.

There may be difficulty securing IV lines in neonates posted for the emergency procedure due to the peripheral vasoconstriction secondary to heart failure and its pharmacological management. In such cases, it is prudent to opt for central venous cannulation, preferably under ultrasound guidance.

23.10.3 Monitoring

Apart from the American Society of Anesthesiologists (ASA) standard monitors, namely, peripheral oxygen saturation (SpO_2), non-invasive blood pressure, ECG, capnography ($EtCO_2$), and temperature, all the patients undergoing embolization should have invasive arterial blood pressure monitoring. Central venous cannulation might pose challenges due to a pulsatile venous column due to the high-flow shunt and a bright red appearance of blood due to a higher oxygen content secondary to the lower oxygen extraction in the brain [61]. Temperature monitoring, warm IV fluids, and forced-air warming blankets are essential for preventing hypothermia and its associated complications. Urine output monitoring is an indirect estimator of volume status and should be continued in the post-procedural period as there is a risk of contrast-induced renal injury. Furthermore, the child may be on diuretics, and the possible use of osmotic agents warrants urinary catheterization and monitoring in the periprocedural period.

23.10.4 Concerns During the Procedure

During the procedure, anesthesia is maintained with a balanced technique consisting of inhalational agents, opioids, and a non-depolarizing neuromuscular blocker to maintain immobility.

23.10.4.1 Fluid Management

Fluid management is challenging due to multiple factors. Little evidence is available regarding the optimal fluid therapy during the procedure, especially in neonates with VOGM and heart failure. Various concerns are discussed below:

1. Neonates do not tolerate fluid overload in cardiac failure. The volume of contrast dye and arterial flush used during the procedure are major contributors to fluid overload. Although the literature suggests the use of up to 10 mL/kg

body weight, it is advisable to restrict the usage of contrast to a maximum of 5 mL/kg (preferably iso-osmotic) to reduce the risk of volume overload and contrast-induced nephropathy [62–64]. At the same time, pre-procedural dehydration due to fasting status and diuretics might result in hypotension and possible cardiovascular collapse due to depressant effects of anesthetic agents; blood loss due to any cause may further complicate hypotension.

2. Determining the volume of maintenance fluids using the standard Holliday and Segar method in these children does not account for the fact that the method devised was intended for use in children assuming normal urine output and energy expenditure. Energy expenditure in mechanically ventilated children reduces due to various reasons: sedation, humidification of inspired air resulting in reduced insensible water loss, and increased ADH secretion. Ultimately, the fluid requirement is reduced by approximately one-third [65, 66].
3. Choice of fluid in the perioperative period is also a matter of concern and debate over several years considering the metabolic requirements of glucose in neonatal age group, concerns of sodium handling by the immature renal system, and tonicity of fluid administered influencing cerebral edema in a susceptible population [66, 67].
4. During the procedure, the neonate usually receives fluid and infusions of inotropic medications and anesthetic agents (opioids and muscle relaxants) that add to the total volume of fluids administered.
5. Monitoring intravascular volume status through central venous pressure (CVP) is unreliable in such neonates.
6. Urine output monitoring is hindered by factors like osmotic diuresis induced by contrast media, creating a false impression of adequate volume status, while it may also precipitate renal failure.
7. Other indicators of fluid responsiveness, both static (including CVP) and dynamic, do not apply to the pediatric population, whereas no such study is available for the neonatal age group. The reason cited for this includes a more compliant thoracic wall and arterial tree, unlike adults. Studies indicate that only a few parameters are better indicators of fluid responsiveness in the pediatric population undergoing mechanical ventilation, which include plethysmography variability index (PVI) and pulse pressure variation (PPV). However, these (findings) are small studies, and the values are confounded by vasoactive medications or hypothermia causing peripheral vasoconstriction, both common to neonates with CCF undergoing emergency embolization [68–70].

With the available evidence, it may be reasonable to consider isotonic crystalloid (0.9% normal saline) as the maintenance fluid of choice and restriction of the volume administered to two-thirds of the total calculated requirement. It is essential to monitor the total volume of contrast and flush injected during the entire procedure. At the authors' institute, the maximum volume of contrast agent is restricted to 5 mL/kg and used in a diluted form (with normal saline). It might be prudent to monitor and maintain the hourly urine output at 0.5–1 mL/kg/h. This can be facilitated by a continuous infusion of diuretic (furosemide 0.1–0.4 mg/kg/h) and titrating the dose of inotropic agents to maintain the arterial blood pressure at preoperative levels or that appropriate for age. Arterial blood gas (ABG) analysis may be performed at regular intervals to monitor for electrolyte levels (sodium, base deficit), blood glucose levels (supplement if levels below 70 mg/dL), serum lactate levels (an indicator of peripheral perfusion; fluid bolus if levels are high), PaO_2 (hypoxia may be aggravated by fluid overload), and $PaCO_2$. Available dynamic indices of fluid responsiveness may be used according to institutional practice.

23.10.4.2 Other Concerns

During the procedure, hypotension may be requested before injection of the embolizing agent in order to reduce the high flow across the shunt

that can cause migration of the embolizing agent. The child with heart failure may not tolerate it and, hence, be avoided. Currently, detachable coils are deployed at the distal end of the feeding arteries to reduce the flow at the required. This avoids the need for induced hypotension [71].

The general consensus in emergency embolization is that the goal should be to achieve acceptable levels of improvement in heart failure. This can be achieved with partial embolization, which should be done in an acceptable duration (2 h). By limiting the procedure and the time is taken, certain complications like hypothermia and contrast-induced toxicity can be prevented. Also, the redistribution of cerebral blood flow after embolization can be limited to levels that can be handled by the immature cerebral vasculature [72].

23.10.5 Complications

Intra-procedural complications include bradycardia during or immediately after embolization attributed to a transient increase in ICP. Strict vigilance is required during this step, and pharmacological intervention is rarely required. Communication between the neuroanesthesiologist and the interventional neuroradiologist is essential. Other procedure-related complications include rupture of the thin-walled fistula or the feeding vessels from an iatrogenic perforation or a microcatheter rupture resulting in hemorrhagic complications [71]. Commonly encountered preventable complications include hypothermia in the interventional neuroradiology suite and fluid overload. Repeated femoral artery puncture may lead to occlusion and lower limb ischemia. It can be prevented by imaging-guided cannulation at the beginning of the procedure, and proper care, including compression and hemostasis after the procedure is over [73].

Major procedural complications are expected with larger shunts. The immature cerebral vasculature at neonatal age groups results in poor handling of the increased blood volumes that occur immediately after the procedure. Venous hypertension secondary to embolization may result in intracranial hemorrhage and perfusion pressure breakthrough. It can be prevented by doing a staged embolization and maintaining normotension postoperatively [74]. A less common but dreaded complication is distal migration of the embolizing agent resulting in pulmonary embolism. In the presence of a right-to-left cardiac shunt, a paradoxical embolism can occur too.

23.11 Post-Procedure Management

Mechanical ventilation may be required in the post-procedural period to maintain hemodynamic and respiratory stability until improvement is noted in the clinical condition [75]. Indicators of response to emergency embolization in neonates include improvement in signs of heart failure evidenced by a reduction in the requirement of inotropic support echocardiographic findings of reduced pulmonary artery pressure and closure of the right-to-left shunt (PDA) [59]. These changes may be seen in the first few hours to few days after emergency embolization. Maintaining the lower limb in a neutral position for 6 h (if femoral artery access was obtained), frequent neurological assessment and monitoring of distal pulses at regular intervals all form a part of post-procedural care [73]. In this period, the role of neuroanesthesiologist involves, and not restricted to, maintenance of adequate sedation, monitoring for clinical improvement, and weaning from mechanical ventilation.

Development of new focal neurological deficits, seizures, worsening of hydrocephalus, cerebral hyperperfusion, cerebral edema, venous infarcts, and precipitation of congestive cardiac failure are all possible in the immediate post-procedural phase [61]. Maintenance of hemodynamic stability is essential to prevent possible post-procedure complications, including cerebral venous thrombosis and hemorrhagic complications akin to perfusion pressure breakthrough.

23.12 Brain Melting Phenomenon and Hydrocephalus

The brain melting phenomenon refers to the most severe form of neurological injury in VOGM. It is characterized by progressive and extensive cerebral atrophy, white matter calcifications, hydrocephalus, and severe cognitive dysfunction that occurs secondary to cerebral venous hypertension. It may also occur after CSF diversion procedures for hydrocephalus in VOGM [23, 45]. In utero diagnosis of this condition is one of the indications for termination of pregnancy.

Hydrocephalus in VOGM is described as hydrodynamic (hydrocephalus without raised ICP) with defective CSF absorption due to immature arachnoid granulations and the increased venous sinus pressure [76]. Primary treatment of the lesion usually leads to gradual resolution of hydrocephalus. Rarely, hydrocephalus results due to obstruction of the aqueduct of Sylvius by the lesion as well. In such conditions, the child may require a CSF diversion procedure (ventriculoperitoneal shunt or endoscopic third ventriculostomy). Such procedures are usually done after primary embolization and are associated with a significant risk of hemorrhage due to the venous engorgement [77, 78]. Placement of ventricular shunts before embolization reverses the pressure gradient between the ventricles and brain parenchyma, resulting in aggravation of cerebral atrophy and white matter calcification [45, 46].

23.13 Prognosis

One of the major prognostic factors in VOGM is the age of presentation: earlier onset indicates a higher severity of the disease and probable severe brain damage in utero, decreasing the possibility of good neurological recovery [28, 79]. Near-normal neurodevelopmental outcomes have been described in children with a later age of presentation consistent with lesser degrees of cerebral damage and lower shunt flows. Treatment strategies also influence the outcome. Surgery carries the highest risk of mortality with large amounts of blood loss and poor neurological outcomes in survivors. Although considered safer, mortality rates of nearly 40% have been reported after the endovascular interventions [79, 80]. According to a recent meta-analysis, significant risk factors indicating poor outcomes include prenatal diagnosis, neonatal cardiac failure, and low neonatal Bicetre score [80].

23.14 Conclusion

Management of VOGM poses multiple periprocedural challenges and requires a multidisciplinary approach consisting of neonatologists, neurologists, pediatric cardiologists, interventional neuroradiologists, and neuroanesthesiologists. Adequate pre-procedural planning and vigilant procedural monitoring help prevent and prompt management of complications, thereby preventing neurodevelopmental dysfunctions.

Conflict of Interest None to declare.

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