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Delayed thalamic infarction following transvenous vein of Galen malformation embolization: Balancing complete occlusion and risk

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ABSTRACT

Background: Vein of Galen malformation embolisation achieves favourable outcomes in most patients through staged endovascular approaches. However, severe neurological complications occur in up to 30% of cases, with mechanisms of delayed ischemic events following near-complete occlusion remaining incompletely understood. This is particularly concerning in asymptomatic patients with favourable preoperative profiles, where the risk-benefit balance of pursuing complete angiographic cure versus accepting substantial flow reduction remains unclear.

Case Presentation: A 15-month-old asymptomatic boy with mural-type Vein of Galen malformation and high Bicêtre score (15 points) underwent three staged transarterial embolisations, achieving progressive flow reduction. The first two sessions successfully reduced flow by approximately 70% without complications. Given small-calibre residual arterial feeders in the third session, a combined transarterial and transvenous approach was employed, achieving near-complete occlusion. Eighteen hours post-procedure, the patient developed acute left hemiparesis. Imaging revealed right thalamic infarction with intraventricular haemorrhage. Conservative management with intensive rehabilitation resulted in complete functional recovery within one month. Follow-up angiography demonstrated persistent occlusion without recanalisation and development of superficial venous collateral drainage.

Conclusion: This case demonstrates that severe neurological complications can occur even in optimal candidates undergoing staged embolisation. The delayed thalamic infarction with hemorrhagic component following transvenous occlusion suggests acute venous hypertension as the primary mechanism, compromising deep

Keywords
vein of Galen malformation, transvenous embolization, venous hypertension; endovascular complications, thalamic infarction

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venous drainage before adequate collaterals developed. In asymptomatic patients achieving substantial flow reduction through transarterial approaches, accepting incomplete occlusion may be safer than pursuing complete angiographic cure via transvenous techniques when collateral venous pathways appear limited. Extended neurological monitoring for 48-72 hours after high-grade occlusion procedures is essential. Despite severe complications, excellent functional recovery is achievable through aggressive early rehabilitation.

Introduction

Vein of Galen aneurysmal malformation (VGAM) is a rare congenital arteriovenous malformation characterized by the presence of direct arteriovenous fistulas resulting from persistence of the embryonic median prosencephalic vein (MPV), also known as the vein of Markowski or primitive median cerebral vein [8]. The distal portion of the MPV becomes the vein of Galen [9]. This anomaly accounts for approximately 1% of all arteriovenous malformations and 30% of pediatric cases [13]. The abnormal persistence of the distal MPV and formation of anomalous connections between choroidal arterial circulation and the MPV during weeks 6-12 of gestation leads to venous hypertension, aneurysmal morphology, and a high-flow, low-resistance intracranial shunt [5,8]. Although genetic mutations in RASA1, endoglin (ENG), and ACVRL1 have been implicated, no definitive risk factors have been established [8].

Clinical presentation varies significantly by age, ranging from neonatal high-output cardiac failure—which can account for up to 80% of cardiac output [12]—and multiorgan dysfunction to later presentation with macrocephaly, communicating hydrocephalus, seizures or renal failure [5, 11]. The Bicêtre scoring system stratifies patients based on cardiac function, neurological status, and other organ involvement, guiding treatment timing and prognosis [5]. Patients with high Bicêtre scores (>12) and absence of cardiac compromise generally have favorable prognoses with appropriate management [5].

Historically, open microsurgical treatment has been associated with extremely high mortality rates, approaching 100%, prompting a paradigm shift toward endovascular management [5,6]. Staged transarterial embolization has emerged as the first-line treatment, significantly reducing mortality to approximately 15.7% [5,6]. The primary goal is safe disconnection of arteriovenous fistulas, flow reduction, and hemodynamic stabilization through gradual remodeling across multiple sessions [5]. Staged approaches allow development of collateral venous drainage pathways, reducing the risk of acute hemodynamic decompensation [5]. When residual feeders arise from small-caliber vessels such as thalamoperforators or choroidal arteries, transvenous embolization becomes necessary, though it carries considerable risk [2,5].

Despite improved outcomes with staged endovascular approaches, severe neurological complications occur in up to 30% of patients [3]. Bhatia et al. reported major neurological events including extensive ischemic or hemorrhagic strokes in their multicenter series, with 50% mortality among complicated cases [3]. Wagner et al. documented a 24% complication rate, emphasizing the need for meticulous patient selection and monitoring [15]. The mechanisms underlying these complications remain incompletely understood, particularly delayed ischemic events following near-complete occlusion.

Several mechanisms have been proposed for post-embolization neurological complications: (1) acute venous hypertension following abrupt occlusion of major venous outflow routes, particularly affecting deep venous structures [3,10]; (2) inadvertent occlusion of normal perforating arteries during catheter manipulation or embolic material reflux [2]; (3) normal perfusion pressure breakthrough phenomenon in chronically hypoperfused territories [1]; and (4) procedure-related thrombosis of deep venous structures [14]. The relative contribution of each mechanism likely varies by patient characteristics, lesion anatomy, and technical approach.

A critical clinical question remains: in asymptomatic patients with favorable preoperative profiles, does the pursuit of complete angiographic cure justify the risks associated with transvenous approaches when substantial flow reduction has already been achieved? This case report describes a 15-month-old boy with mural-type VGAM, high Bicêtre score, and no cardiac compromise who developed delayed thalamic infarction following near-complete occlusion via combined transarterial and transvenous embolization. We discuss the potential mechanisms, analyze technical

considerations, and examine implications for risk-benefit assessment in similar cases.

Case Description

A 15-month-old male with age-appropriate neurodevelopment presented with macrocephaly (head circumference 54 cm, >99.9th percentile) and parental reports of frequent falls during ambulation. Developmental milestones were normal (walking at 13 months, first words at 12 months). Birth history was unremarkable (3,150g, cesarean delivery for transverse position, non-consanguineous parents aged 42 and 44 years). Past medical history was significant only for hip dysplasia.

As part of the evaluation, a plain and contrast enhanced Brain MRI [Figure 1] was performed, revealing a mural-type vein of Galen aneurysmal malformation (VGAM) with Bicêtre score of 15 points. Physical examination showed an alert, interactive child with symmetric motor function and no focal deficits. Cardiology evaluation demonstrated no heart failure, with normal echocardiography findings. The patient reported moderate headache one day prior to intervention.

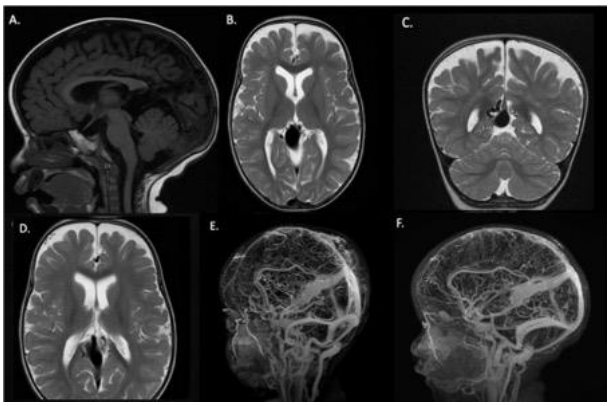

Figure 1: Preoperative brain MRI and contrast-enhanced MR angiography in venous phase. (A) Sagittal T1-weighted image shows signal void within the deep venous system suggestive of Galenic aneurysmal dilation. (B, C, D) Axial and coronal T2-weighted images demonstrate signal voids in the deep venous system consistent with VGAM. (E, F) 3D reconstructions of contrast-enhanced venous phase MR angiography showing aneurysmal dilation consistent with VGAM.

Given the favorable clinical profile, staged endovascular treatment was indicated. The first angiographic session identified a mural type VGMA [Figure 2] with arterial feeders from the right posteromedial choroidal artery, right pericallosal artery branches, and posterior circulation thalamoperforators draining into the prosencephalic vein. Via right femoral access with 6F sheath, Benchmark 6F guide catheter, and Echelon-10 microcatheter (Mirage 0.008" microwire), transarterial embolization of the choroidal artery was performed using Axium Prime coils (5mm × 29cm, 4mm × 32cm, 4mm × 29cm) followed by Onyx-18, achieving 30% flow reduction. The patient was extubated in the angiography suite and discharged at 48 hours without complications.

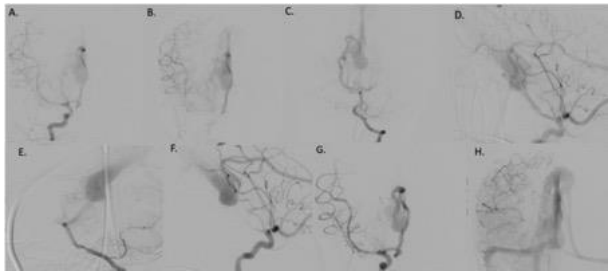

Figure 2: Diagnostic cerebral panangiography. (A–E) Anteroposterior and lateral views of right internal carotid and vertebrobasilar system injections show a high-flow VGAM with multiple feeders from anterior and posterior choroidal arteries and the splenial artery. (F–H) First endovascular embolization: embolization of choroidal feeder with coils and Onyx within the fistulous tract.

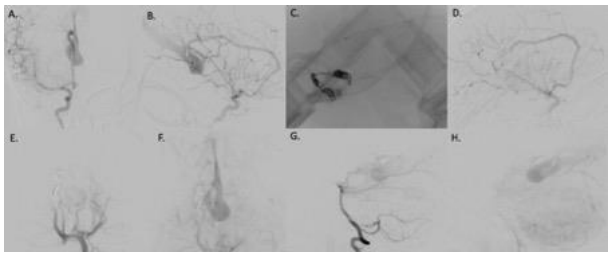

Figure 3: Second endovascular embolization. (A–H) Embolization of the high-flow splenial artery of the corpus callosum using coils and Onyx, achieving normalization of the patient's heart rate, although residual shunting persisted.

Follow-up angiography confirmed patent embolization with persistent flow through residual feeders [Figure 3]. Via left femoral access with 6F × 7cm sheath (contralateral strategy), Benchmark 6F guide catheter, and 0.035" × 260cm hydrophilic guidewire, initial Echelon-10 microcatheter navigation proved challenging due to complex vascular anatomy. Microcatheter exchange to Gama system with Hybrid 1214D and Avigo microwire successfully navigated a loop at the sac entrance.

Transarterial embolization of the right pericallosal artery was performed using progressive coil packing (Axium 12mm × 40cm, Axium Prime 4mm × 12cm, 3mm × 10cm, 2.5mm × 3cm) followed by Onyx-18, achieving cumulative 60-70% flow reduction. Postoperative course was uncomplicated with discharge at 48 hours.

After informed consent discussing risks of venous complications, near-complete occlusion was pursued. Pre-procedural angiography showed persistent flow through small thalamoperforating arteries (<1mm diameter) not amenable to safe transarterial catheterization due to vessel caliber, tortuosity, and risk to eloquent territory. Critically, detailed assessment of deep venous collateral capacity was not protocolized. Given these anatomic constraints, a transvenous approach was selected to achieve near-complete occlusion. Via femoral vein access, microcatheter navigation through the straight sinus allowed controlled coil embolization of the venous pouch, achieving >90% flow reduction. [Figure 4] Post-embolization angiography showed absence of early arterial filling with markedly delayed venous opacification and flow redirection toward superficial cortical veins. The patient was extubated and transferred to PICU in stable condition.

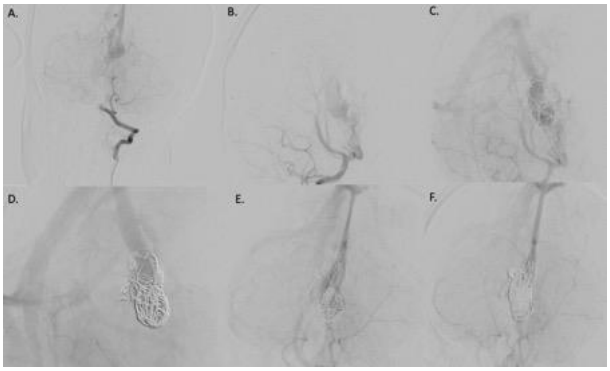

Figure 4: Third endovascular embolization. (A–F) Combined arterial and venous approach with complete occlusion of the aneurysmal sac and remaining arterial feeders.

Postoperative Complication

Initial examination showed intact consciousness (GCS 15/15) and symmetric motor strength (5/5). At 18 hours post-procedure, the patient developed acute irritability and asymmetric movements. Examination revealed new left hemiparesis (upper extremity 3/5, lower extremity 4/5) with preserved consciousness. Emergency CT demonstrated right thalamic infarction with ipsilateral intraventricular hemorrhage originating from subependymal vessels, without hydrocephalus, mass effect, or herniation. [Figure 5] Neurosurgical evaluation determined no surgical intervention was indicated.

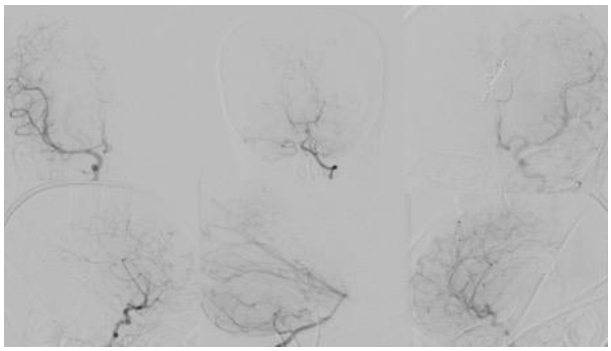

Figure 5: Non-contrast cranial CT 18 hours post-embolization. Right thalamic hypodensity and supratentorial intraventricular hemorrhage.

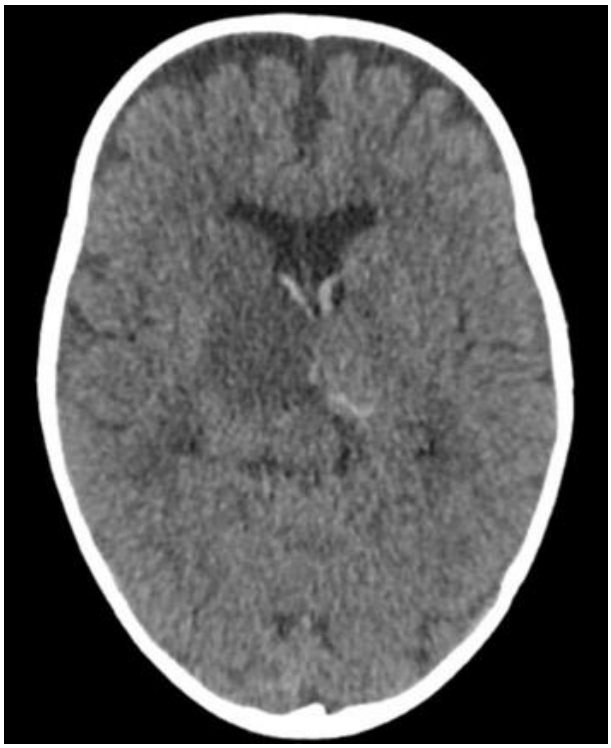

Figure 6: Postoperative control panangiography. Absence of early venous filling; normal arterial and venous return with redirection into the superficial venous system.

The patient remained hemodynamically stable without progression of the motor deficit. Conservative management was initiated including close neurological monitoring, maintenance of

adequate cerebral perfusion pressure, and initiation of physical and occupational therapy. Follow-up imaging at 48 and 72 hours showed no worsening of ischemia, no increase in intraventricular bleeding, and no signs of acute hydrocephalus.

Over the subsequent three weeks, the patient achieved progressive improvement in motor function with intensive guided physical therapy and rehabilitation. At discharge, motor strength had improved to 4+/5 in the upper limb and 5/5 in the lower limb. At one-month follow-up, the patient demonstrated complete functional recovery with age-appropriate motor skills and no residual deficits. Follow-up cerebral CT and panangiography [Figures 6-7] showed expected post-embolization findings with no signs of recanalization and development of collateral venous drainage through the superficial venous system. The patient continues to do well with ongoing clinical and imaging surveillance at current date.

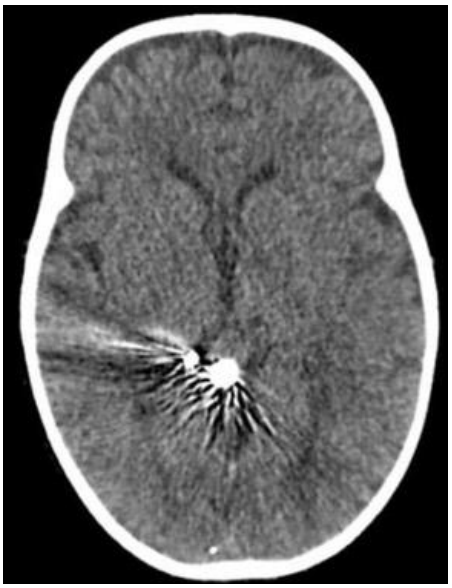

Figure 7: Non-contrast cranial CT one month postoperatively. Normal cortico-subcortical differentiation and metallic artifact from embolization material.

Discussion

This case demonstrates that severe neurological complications can occur following staged VGAM embolization even in patients with optimal preoperative profiles—specifically, asymptomatic presentation, high Bicêtre score (15 points), absence of cardiac compromise, and appropriate neurodevelopment. The development of thalamic infarction with intraventricular hemorrhage 18 hours after achieving near-complete occlusion via transvenous approach highlights a critical clinical dilemma: in favorable-risk patients, does pursuit of complete angiographic cure justify the risks associated with aggressive final-stage embolization?

Multiple pathophysiological mechanisms may have contributed to the observed complication, though acute venous hypertension appears most likely given the clinical and radiographic features of our case.

Acute Venous Hypertension as Primary Mechanism

The thalamic location of infarction, presence of intraventricular hemorrhage, delayed onset after transvenous occlusion, and involvement of deep brain structures all support venous congestion as the predominant etiology. In mural-type VGAM, the deep venous system (thalamostriate veins, internal cerebral veins) drains primarily through the prosencephalic vein. Although staged embolization typically allows gradual adaptation of venous drainage pathways, abrupt near-complete occlusion in the final session—particularly via transvenous coil packing—can acutely compromise residual deep venous outflow before adequate collateral superficial venous drainage develops. The resulting pressure surge impairs capillary perfusion, induces venous congestion, and triggers deep venous ischemia [3,10]. This mechanism explains both the ischemic and hemorrhagic components observed in our patient.

Alternative Contributing Mechanisms

Inadvertent occlusion of thalamoperforating or choroidal branches arteries during catheter manipulation or embolic material reflux represents a second potential mechanism [2]. However, isolated arterial occlusion would not fully explain the intraventricular hemorrhage component. Normal perfusion pressure breakthrough phenomenon, described in arteriovenous malformation literature, theoretically could contribute to hemorrhagic complications following abrupt flow reduction [1], though this mechanism typically manifests during or immediately after the procedure rather than 18 hours later. Finally, procedure-related thrombosis of deep venous structures remains a recognized risk

[14], potentially contributing to the mixed ischemic-hemorrhagic presentation.

Our complication rate aligns with published series. Bhatia et al. reported major neurological complications in 30.3% of 76 patients undergoing VGAM embolization, with ischemic stroke representing 63% of these events and 50% mortality among complicated cases [3]. Wagner et al. documented 24% complication rates [15]. Savage et al.'s meta-analysis found similar thrombosis rates in single-stage (27%) versus staged (24%) approaches [14].

However, our case differs in a critical aspect: the patient had a favorable preoperative profile rarely described in complication reports. Most published series include predominantly symptomatic neonates with cardiac compromise and lower Bicêtre scores. Our patient's high Bicêtre score, absence of symptoms, and normal cardiac function suggested low baseline risk, making the severe complication particularly instructive regarding inherent procedural risks even in ideal candidates.

Clinical Implications and Technical Considerations

This case raises several important clinical considerations. When substantial flow reduction (60-70%) has been achieved through transarterial approaches and the patient remains asymptomatic without cardiac compromise, clinicians face a critical decision point regarding pursuit of complete angiographic cure. While achieving >90% occlusion via transvenous approaches may offer theoretical benefits, it introduces immediate risks of venous disruption and thrombosis. In retrospect, accepting 70% occlusion in our asymptomatic patient may have been the safer strategy, particularly given the favorable preoperative profile.

Pre-procedural evaluation should include detailed analysis of alternative deep and superficial venous drainage routes. Future cases might benefit from dedicated venous phase acquisitions with delayed imaging to map available drainage pathways before pursuing near-complete occlusion. This becomes particularly critical when considering transvenous approaches, which—while effectively addressing small-caliber arterial feeders inaccessible to transarterial catheterization—may more abruptly compromise deep venous drainage compared to gradual arterial occlusion. In asymptomatic patients with favorable baseline characteristics, this risk warrants especially careful consideration.

The delayed presentation of our patient's complication at 18 hours post-procedure, beyond typical immediate monitoring windows, has important implications for postoperative care protocols. We recommend extended neurological monitoring (minimum 48-72 hours) after procedures achieving >80% occlusion, with a low threshold for obtaining imaging if subtle neurological changes occur. Finally, despite the severity of the thalamic infarction, our patient achieved complete functional recovery within one month, highlighting the remarkable neuroplastic capacity of the developing brain. This underscores the importance of initiating aggressive physical and occupational therapy as soon as the patient is medically stable.

As a single case report, generalization is limited. The absence of intraoperative hemodynamic monitoring prevents definitive confirmation of the proposed mechanism. Pre-session 3 venous phase imaging was not specifically protocolized to assess collateral pathways. Follow-up duration (one month) is relatively brief for assessing long-term neurodevelopmental outcomes.

Conclusions

Endovascular embolization has transformed the vein of Galen malformation outcomes, reducing mortality from near 100% to approximately 16%. However, this case demonstrates that severe neurological complications remain a significant risk even in patients with optimal preoperative profiles. The delayed thalamic infarction with intraventricular hemorrhage following transvenous near-complete occlusion likely resulted from acute venous hypertension before adequate collateral pathways developed. Extended neurological monitoring for 48-72 hours after high-grade occlusion procedures is essential, and pre-procedural assessment should include detailed evaluation of collateral venous pathways.

In favorable-risk patients achieving substantial flow reduction through transarterial approaches, accepting incomplete occlusion may be safer than pursuing complete angiographic cure via transvenous techniques when collateral venous drainage appears limited. Despite severe complications, excellent functional recovery is achievable through early aggressive rehabilitation,

reflecting the remarkable neuroplastic capacity of the developing pediatric brain.

Abbreviations

ACVRL1: Activin A receptor-like type 1
AVM: Arteriovenous malformation
BNP: Brain natriuretic peptide
CT: Computed tomography
ENG: Endoglin
MPV: Median prosencephalic vein
MRI: Magnetic resonance imaging
RASA1: RAS p21 protein activator 1
VGAM: Vein of Galen aneurysmal malformation
PICU: Pediatrics Intensive Care Unit
CHF: Congestive heart failure
IVH = intraventricular hemorrhage

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