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SNI: Neurovascular

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Case Report

An unexpected turn: Posterior reversible encephalopathy syndrome following microsurgical resection of a brain arteriovenous malformation

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Received: 29 January 2025
Accepted: 08 April 2025
Published: 09 May 2025

DOI
10.25259/SNI_88_2025

Quick Response Code:

ABSTRACT

Background: Cerebral arteriovenous malformations (AVMs) are rare congenital vascular anomalies, often presenting with intracranial hemorrhage or seizures. Posterior reversible encephalopathy syndrome (PRES) is a distinct neurological condition characterized by vasogenic edema, primarily affecting posterior brain regions and typically associated with hypertensive crises, eclampsia, or immunosuppressive therapies. However, its occurrence following neurosurgical interventions is exceptionally rare. This case report documents the first instance of PRES following the resection of a Spetzler-Martin grade I frontal AVM, emphasizing the importance of early recognition and management of this rare complication.

Case Description: A 27-year-old woman underwent navigation-assisted resection of a right frontal AVM. The procedure was uneventful; however, in the immediate postoperative period, she experienced a generalized tonic-clonic seizure, agitation, dysconjugate gaze, and altered consciousness. Brain magnetic resonance imaging revealed diffuse high fluid-attenuated inversion recovery signal abnormalities in the brainstem, cerebellum, thalami, basal ganglia, and cerebral hemispheres, consistent with central PRES. The patient was managed with supportive care, resulting in a full clinical and radiographic recovery within 3 weeks. Follow-up imaging confirmed the resolution of PRES-related changes, and she remained seizure-free after antiseizure medication tapering.

Conclusion: This case underscores the critical importance of early neuroimaging in evaluating unexpected postoperative neurological symptoms. Recognizing central PRES and its atypical radiographic patterns enables timely diagnosis and appropriate management, avoiding unnecessary interventions. The pathophysiology likely involves postoperative endothelial dysfunction and disrupted autoregulation. This report underscores the importance of considering PRES in postoperative neurological complications and calls for further research into its mechanisms and optimal management.

Keywords: Arteriovenous malformation, Posterior reversible encephalopathy syndrome, Post-operative neurosurgical complication, Seizure

INTRODUCTION

Arteriovenous malformations (AVMs) are rare congenital vascular anomalies with direct artery-to-vein connections, posing a significant risk of intracranial hemorrhage, particularly in

young individuals.[9] Clinical presentations vary, including hemorrhage, seizures, and focal deficits, depending on lesion characteristics.[9] Cerebral angiography remains the diagnostic gold standard, guiding treatment decisions based on the Spetzler-Martin grading system.[5,8]

Posterior reversible encephalopathy syndrome (PRES), characterized by vasogenic edema predominantly affecting the posterior brain regions, is most often associated with hypertension, eclampsia, or immunosuppressive therapy but can also occur, though rarely, in postoperative settings.[4,6] Its occurrence following neurosurgical interventions is particularly rare, and the underlying pathophysiological mechanisms in this context remain poorly understood.[3] We present the first documented case of PRES following the resection of a Spetzler-Martin grade 1 right frontal AVM, emphasizing the need to consider this syndrome as a potential postoperative complication of AVM surgery.

CASE PRESENTATION

The patient is a 27-year-old right-handed woman who presented with a primary complaint of headache. Her neurological examination was unremarkable, with no observed deficits. Brain magnetic resonance imaging (MRI) revealed a Spetzler-Martin grade I right frontal AVM located along the posterior aspect of the right superior frontal sulcus, measuring 2 × 1.2 × 1.8 cm [Figures 1a and b]. Digital subtraction angiography confirmed the AVM, identifying its arterial supply from the callosomarginal branch of the right anterior cerebral artery and venous drainage through an enlarged draining vein into the superior sagittal sinus [Figure 1c]. After discussing various treatment options, including conservative management, the patient elected to undergo surgical resection of the AVM.

The AVM was resected through a navigation-assisted microsurgical approach, and the procedure was uneventful. All feeding arteries were carefully coagulated without sacrificing any major vessels. The lesion was dissected from surrounding structures, and after securing all feeders, the draining vein was clipped, coagulated, and detached.

In the recovery room, the patient experienced a generalized tonic-clonic (GTC) seizure and was loaded with levetiracetam. On examination, she was agitated, displayed dysconjugate eye movements, spontaneously moved all extremities, and opened her eyes without following commands. Postoperative computed tomography (CT) angiography and CT venography showed no abnormalities. She remained intubated and was transferred to the neuro-intensive care unit.

Continuous video-electroencephalogram monitoring revealed severe generalized background slowing, more pronounced over the right hemisphere, with bursts of generalized delta activity showing a bifrontal amplitude predominance. No clinical or electrographic seizures were recorded, and no epileptiform activity was observed. On postoperative day 1, she remained agitated, confused, and intermittently stuporous, with persistent dysconjugate eye movements. Brain MRI revealed an acute infarct in the left posterior pons with punctate diffusion restriction [Figure 2]. In addition, multiple ill-defined high fluid-attenuated inversion recovery (FLAIR) signal foci were noted in the inferior midbrain, extending to the left pons, cerebellar vermis, left cerebellar hemisphere, bilateral thalami, caudate nuclei, and to a lesser extent, the putamen, brainstem, left frontal lobe, and right temporal lobe [Figure 2]. The differential diagnosis included a central variant of PRES or a metabolic encephalopathy, such as hypoxic or hypoglycemic injury.

On postoperative day 2, her neurological status remained unchanged while sedated with dexmedetomidine due to

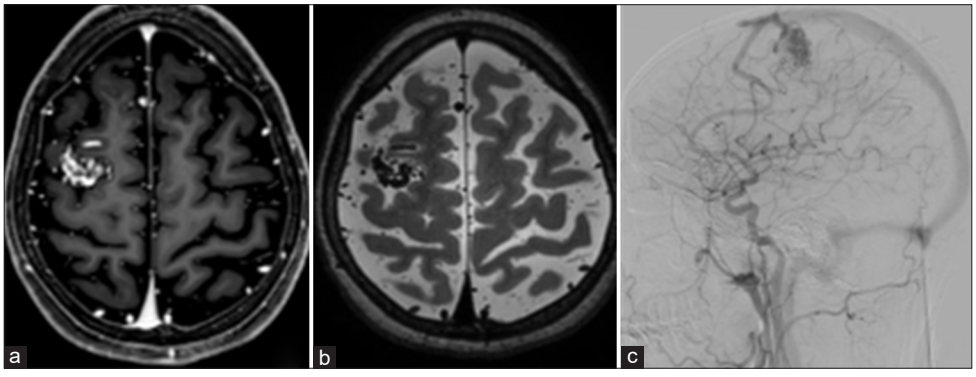

Figure 1: Preoperative brain magnetic resonance imaging, performed with axial (a) T1-weighted sequences with gadolinium injection and (b) T2-weighted sequence, revealed an arteriovenous malformation along the posterior aspect of the right superior frontal sulcus measuring 2 × 1.2 × 1.8 cm. The cerebral digital subtraction angiography in (c) lateral view showed that the arteriovenous malformation has a feeding artery originating from the callosomarginal branch of the right anterior cerebral artery and has an enlarged draining vein emptying into the superior sagittal sinus.

intermittent agitation. A repeat brain MRI showed stable edema but newly detected filling defects in the right transverse sinus [Figure 3a], suggestive of acute non-occlusive dural venous sinus thrombosis, which was subsequently confirmed by CT venography [Figure 3b]. Notably, no other venous occlusions were observed, particularly in the deep venous drainage system

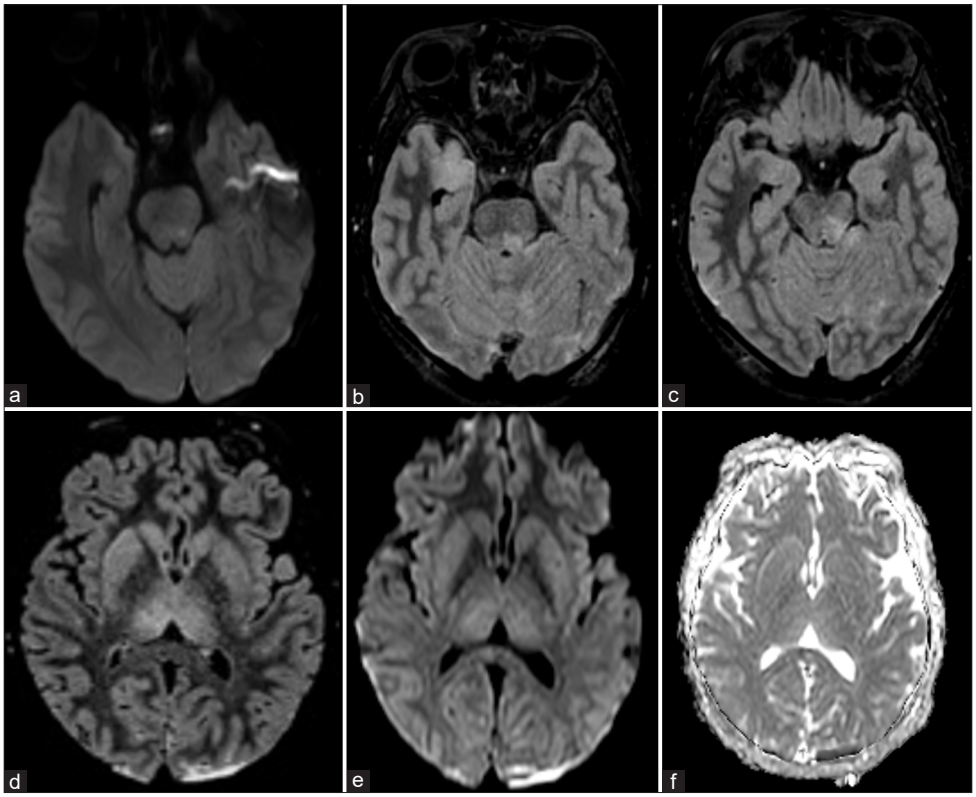

Figure 2: On postoperative day 1, brain magnetic resonance imaging in axial views showed (a) punctate diffusion restriction on diffusion-weighted imaging (DWI) involving the left posterior aspect of the pons, with a corresponding high fluid-attenuated inversion recovery (FLAIR) signal (b). In addition, axial FLAIR (b) revealed an abnormally high signal involving the right anteromedial temporal area, which was not restricting on DWI (a). (c) High FLAIR signal was also seen over the left inferior midbrain, cerebellar vermis, and left cerebellar hemisphere, as well as (d) in the bilateral thalami and caudate nuclei, (e-f) which likewise did not show any restriction on diffusion sequences.

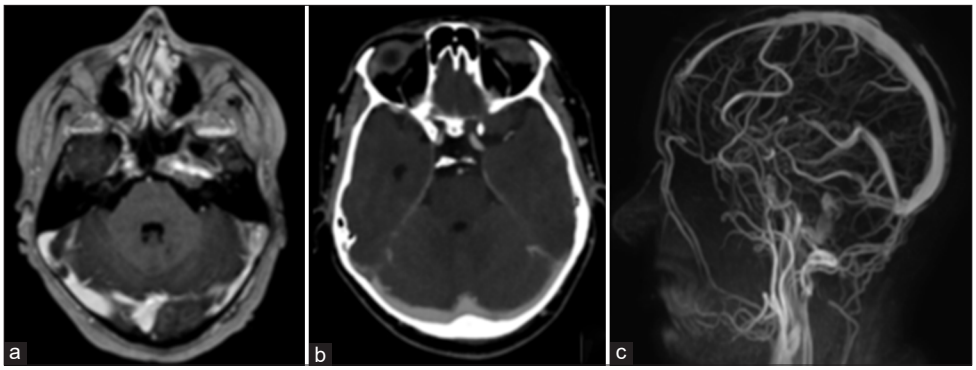

Figure 3: On postoperative day 2, brain magnetic resonance imaging with gadolinium, along with venography (a) sequences, showed an interval development of a filling defect in the right transverse sinus. (b) This was confirmed the same day with computed tomography venography of the head, in keeping with the diagnosis of acute dural venous sinus thrombosis, (c) while the remainder of the cerebral venous drainage system was patent.

[Figure 3c]. By postoperative day 3, the patient was somnolent but easily arousable, followed simple commands, and exhibited resolution of the dysconjugate eye movements. Repeat CT venography later that day demonstrated spontaneous resolution of the filling defect in the right transverse sinus, with all cerebral venous sinuses appearing patent despite the absence of anticoagulation therapy [Figure 4a]. On postoperative day 4, after significant clinical improvement, she was extubated, with full recovery of mental status and a normal neurological examination. A repeat MRI on postoperative day 8 showed substantial resolution of the previously noted high FLAIR signal in the left cerebellum and other posterior fossa regions. Magnetic resonance (MR) venography confirmed patency of the venous sinuses. The patient was discharged on postoperative day 9.

At her outpatient neurology follow-up on postoperative day 21, she had fully returned to baseline with no residual deficits. A follow-up brain MRI on postoperative day 76 demonstrated near-complete resolution of the previously described high FLAIR signal foci [Figure 4b,c]. She was subsequently tapered off levetiracetam without seizure recurrence.

DISCUSSION

This case highlights an unusual presentation of PRES following the surgical resection of a Spetzler Grade I AVM. While PRES is commonly associated with conditions such as hypertension, eclampsia, and immunosuppressive therapy,[4] its occurrence in the postoperative setting, particularly after neurosurgical interventions, is exceedingly rare.[3]

Our patient exhibited a GTC, agitation, dysconjugate eye movements, and altered consciousness in the immediate postoperative period. MRI findings revealed diffuse high FLAIR signal abnormalities involving the brainstem, cerebellum, thalami, and cerebral hemispheres. Given the radiographic asymmetry and involvement of deep structures, an extensive differential diagnosis was initially considered, including metabolic encephalopathy secondary to hypoglycemia or hypoxic insult and venous infarction due to deep venous thrombosis. However, MR venography and CT venography demonstrated patency of the deep venous drainage system, ruling out deep venous thrombosis as an etiology. Furthermore, diffusion-weighted imaging sequences did not show any areas of restricted diffusion, which would be expected in the case of venous infarction. Instead, the findings were consistent with vasogenic edema, characteristic of PRES. The transient non-occlusive thrombosis of the right transverse sinus identified on postoperative day 2 raised the possibility of dural venous congestion exacerbating vasogenic edema. The resolution of this defect on follow-up imaging, without the use of anticoagulation or progression of edema, further supports that deep venous congestion was not a contributing factor to the observed radiographic abnormalities. This finding ultimately corroborates the diagnosis of central PRES, an atypical presentation of the syndrome.

PRES predominantly affects the posterior parieto-occipital regions due to the limited sympathetic innervation of the posterior circulation.[2] Approximately 40% of PRES cases require intensive care due to complications such as cerebral ischemia, status epilepticus, or intracranial hemorrhage.[4] Despite these serious risks, the clinical and imaging features are typically reversible. In contrast, the central form of PRES, as seen in this case, involves the brainstem, basal ganglia, thalami, and cerebellum.[2,7] This less well-documented variant highlights the heterogeneous radiographic manifestations of PRES and underscores the need to recognize its diverse presentations.

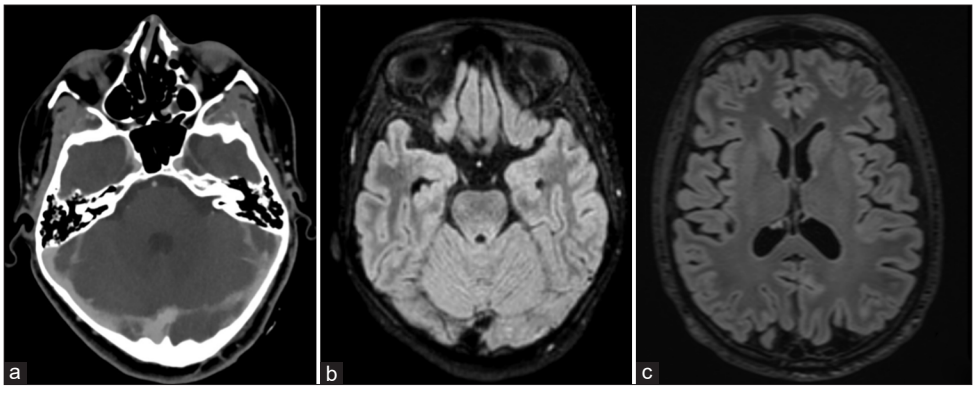

Figure 4: Computed tomography head venography (a) performed on postoperative day 3 in axial views, showed interval resolution of the filling defect in the right transverse sinus, with patency of the other dural venous sinuses. (b) Meanwhile, brain magnetic resonance imaging, performed on postoperative day 76, showed near-complete resolution of the previously described high fluid-attenuated inversion recovery signal abnormalities involving the right temporal area, midbrain, pons, and cerebellum, (c) in addition to the bilateral thalami and caudate nuclei.

Cases of PRES following neurosurgical procedures, though rare, have been reported, primarily in the context of intracranial tumor resections and spinal surgeries.[3] The literature includes no prior reports of PRES following AVM resections, making this, to our knowledge, the first documented case. A recent review identified 47 cases of PRES as a postoperative complication, with 21 of these occurring after cranial surgeries, predominantly for brain tumors. The mean onset of symptoms post-surgery was 4.7 days, though many patients experienced symptoms on the day of surgery.[3] Seizures were the most common clinical manifestation, occurring in 45% of cases as the sole symptom.[3] Our patient's presentation included a more severe and prolonged course of agitation and obtundation, resolving over approximately 72 h. Reported recovery times for PRES symptoms span from 1 day to 18 months, underscoring the variability of this syndrome's clinical trajectory.[3]

The pathogenesis of PRES remains incompletely understood, particularly in normotensive patients like the one described in this case. The syndrome is believed to arise from cerebrovascular autoregulation failure and endothelial dysfunction, resulting in vasogenic edema. In the perioperative context, several factors may exacerbate endothelial injury, including systemic inflammation, anesthetic agents, and perioperative stress. Preoperative hypoperfusion in AVM lesions, due to significant shunting, is corrected after resection, potentially leading to reperfusion of previously hypoperfused areas, while distant regions may experience relative hypoperfusion. While these processes are more commonly associated with larger AVMs, smaller lesions can also be involved, as seen in this case. Historically, two complementary theories have been proposed to explain hemodynamic disturbances following AVM resections: the normal perfusion pressure breakthrough (NPPB) theory and the occlusive hyperemia theory. The NPPB theory, introduced by Spetzler *et al*. in 1978 [10], suggests that loss of autoregulatory capacity in chronically hypoperfused brain regions leads to hyperperfusion after resection, causing vasogenic edema or hemorrhage. The occlusive hyperemia theory, proposed by Al-Rodhan *et al*. in 1993 [1], postulates that the sudden restoration of blood flow to previously shunted brain tissue after resection causes occlusion of small venules due to a mismatch between arterial and venous inflow resulting in microvascular congestion and edema.

However, neither theory fully accounts for the neuroimaging findings in our patient, particularly the central form of PRES observed in regions such as the caudate nuclei, thalami, brainstem, and cerebellum, which are distant from the site of resection. This suggests that the pathophysiology of PRES, in our case, may involve more diffuse disturbances. We propose that postoperative stress, endothelial dysfunction, and microvascular injury from the surgical resection may have led to a systemic disruption of cerebral autoregulation. In addition, alterations in blood-brain barrier integrity and neurovascular coupling could have contributed to the development of vasogenic edema in regions remote from the resection site.

CONCLUSION

This case underscores the importance of early neuroimaging in patients with unexpected postoperative neurological deterioration. Recognizing atypical radiographic patterns of central PRES facilitates timely diagnosis and prevents unnecessary interventions. Supportive care focused on optimizing systemic and cerebral hemodynamics remains the cornerstone of management, as evidenced by the reversibility of symptoms and imaging findings in this patient.

This case broadens the differential diagnosis of postoperative complications in neurosurgical patients and highlights the potential for PRES as a rare but reversible condition. The findings emphasize the importance of understanding the hemodynamic changes associated with AVM resections, even in small lesions, and the need for vigilance in identifying PRES in patients with postoperative neurological decline. Further research is warranted to elucidate the underlying mechanisms of PRES and guide management strategies, particularly in the context of neurosurgical procedures.

Ethical approval: The Institutional Review Board approval is not required.
Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.
Financial support and sponsorship: Nil.
Conflicts of interest: There are no conflicts of interest
Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript, and no images were manipulated using AI.

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How to cite this article: El Halabi T, El Houshiemy M, Kawtharani S, Ali B, Ftouni L, Darwish H, *et al*. An unexpected turn: Posterior reversible encephalopathy syndrome following microsurgical resection of a brain arteriovenous malformation. Surg Neurol Int. 2025;16:176. doi: 10.25259/SNI_88_2025