

Perimesencephalic SAH Masquerading as Aneurysmal Bleed: Diagnostic and Critical Care Challenges

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DOI: <https://doi.org/10.52403/ijrr.20251258>

ABSTRACT

Subarachnoid hemorrhage (SAH) refers to bleeding within the subarachnoid space, often due to ruptured aneurysms. However, perimesencephalic subarachnoid hemorrhage (PMSAH) represents a rare, non-aneurysmal subtype, typically confined to the basal cisterns surrounding the midbrain. This case report describes a 52-year-old male with a background of hypertension, type 2 diabetes mellitus, hyperlipidemia, and chronic ischemic heart disease, who presented with sudden, intense headache and a single episode of vomiting. Initial non-contrast CT imaging revealed a classic perimesencephalic bleed. Due to suspicion of a basilar artery aneurysm, the patient was referred for digital subtraction angiography (DSA). On arrival, he had a GCS of 8/15 and was immediately intubated and admitted to the ICU for critical care. While DSA showed no aneurysms or vascular malformations, CT cerebral angiography confirmed SAH involving the suprasellar, interpeduncular, ambient, prepontine, perimesencephalic, and cerebellar cisterns. Complications during the ICU stay included hydrocephalus, cerebral edema, and hospital-acquired

pneumonia due to *Klebsiella pneumoniae*. Cardiac evaluation revealed native coronary artery atherosclerosis, and a right-sided external ventricular drain (EVD) was placed to manage intracranial pressure. This case underscores the significance of timely neuroimaging, early intervention, and multidisciplinary care in managing spontaneous SAH, especially when complicated by comorbid systemic illness.

Keywords: Subarachnoid hemorrhage, basilar artery aneurysm, digital subtraction angiography, external ventricular drain, hypertension, hyperlipidemia, diabetes mellitus.

INTRODUCTION

Subarachnoid hemorrhage (SAH) is a life-threatening neurologic emergency characterized by bleeding into the subarachnoid space, most commonly due to the rupture of a cerebral aneurysm. However, approximately 10–15% of spontaneous SAH cases occur in the absence of identifiable vascular malformations, such as aneurysms or arteriovenous malformations, and are classified as non-aneurysmal subarachnoid hemorrhages (NA-SAH) [1]. Among these,

a distinct subtype—perimesencephalic non-aneurysmal subarachnoid hemorrhage (PNSAH) has emerged as a unique clinical entity with a relatively benign course and favorable outcomes compared to aneurysmal SAH [2].

Perimesencephalic SAH was first defined in the 1980s and is characterized by bleeding that is confined to the cisterns surrounding the midbrain—including the interpeduncular, ambient, suprasellar, and prepontine cisterns—without evidence of aneurysm on angiography [3]. It is typically diagnosed through non-contrast CT imaging, which reveals localized blood pooling in the perimesencephalic region, often sparing the lateral ventricles and cortical sulci [4]. Digital subtraction angiography (DSA) remains the gold standard for ruling out aneurysms in these patients, although CT angiography (CTA) is increasingly used as an initial non-invasive tool. A repeat DSA after 4–6 weeks is often recommended if the initial angiogram is negative to ensure no missed vascular lesion [5].

The etiology of PNSAH remains uncertain, but several studies suggest a venous origin of bleeding, possibly due to rupture of a vein in the deep venous system such as the basal vein of Rosenthal [6]. This contrasts with aneurysmal SAH, which is arterial in nature and typically more diffuse and severe. Risk factors for PNSAH are not as clearly defined, though hypertension, smoking, and alcohol consumption have been implicated, as they may increase venous pressure and vessel fragility [7].

Clinically, patients with PMSAH often present with a sudden, severe headache—frequently described as a “thunderclap” headache—sometimes accompanied by nausea, vomiting, photophobia, or transient loss of consciousness [8]. The prognosis is generally excellent, with mortality rates below 2%, and most patients returning to baseline functional status [9]. However, complications such as hydrocephalus, cerebral edema, and hospital-acquired infections can occur, particularly in patients

with underlying comorbidities or those requiring prolonged ICU care [10].

This case describes a 52-year-old male with multiple cardiovascular risk factors and a stressful lifestyle, who presented with PNSAH complicated by hydrocephalus, cerebral edema, and ventilator-associated pneumonia. His imaging was consistent with perimesencephalic bleeding, and angiographic studies ruled out aneurysm. The patient’s management required a multidisciplinary neurocritical care approach, including mechanical ventilation, external ventricular drainage, and infection control.

This case underscores the importance of recognizing PNSAH as a distinct and potentially less aggressive form of SAH, while also highlighting the need for vigilance due to associated complications, especially in patients with systemic comorbidities.

CASE PRESENTATION

A 52-year-old Indian male, a businessman by profession, was brought to the emergency department on 24/11/2024 after experiencing a sudden, intense headache—described as the worst he had ever felt—followed by a single episode of projectile vomiting. He had no recent trauma, seizures, or known neurological conditions. Upon arrival, he was drowsy and unresponsive to verbal cues, with a Glasgow Coma Scale (GCS) score of 8/15. Given his compromised consciousness, he was intubated and admitted to the intensive care unit for close neurological monitoring and supportive care.

The patient's medical history revealed several long-standing health issues, including hypertension, type 2 diabetes, hyperlipidemia, and chronic ischemic heart disease. He was also a heavy smoker and habitual alcohol user, and he reported high levels of stress due to personal and professional pressures.

On examination, his vital signs were within acceptable limits: heart rate was 60 bpm, blood pressure 122/72 mmHg, and oxygen

saturation at 100% with supplemental oxygen. His pupils were equal, reactive, and measured 2 mm. No focal neurological deficits were observed initially.

A non-contrast CT scan of the brain revealed localized subarachnoid hemorrhage in the perimesencephalic, interpeduncular, suprasellar, ambient, prepontine, and cerebellar cisterns. CT cerebral angiography initially raised concerns about a possible basilar artery aneurysm. However, digital subtraction angiography (DSA) ruled out any aneurysmal or arteriovenous malformation. A follow-up DSA was planned for 4–6 weeks later to confirm the findings. MRI imaging supported the initial diagnosis, showing hemorrhage confined to the same cisternal regions.

While in the ICU, the patient developed hydrocephalus and cerebral edema, which required the placement of a right-sided external ventricular drain (EVD) to relieve intracranial pressure and manage cerebrospinal fluid. His condition was further complicated by ventilator-associated pneumonia due to *Klebsiella pneumoniae*, which was treated with appropriate antibiotics. Fortunately, he did not develop any signs of cerebral vasospasm throughout his stay.

This case reflects an uncommon, more severe presentation of perimesencephalic subarachnoid hemorrhage (PMSAH)—a condition that is typically mild and self-limiting. The presence of reduced consciousness, hydrocephalus, and secondary infection made this case clinically significant, highlighting the variability in presentation. It also underscores the critical role of timely imaging, vigilant ICU care, and multidisciplinary management, even in cases of subarachnoid hemorrhage that appear non-aneurysmal on initial evaluation.

DISCUSSION

Perimesencephalic subarachnoid haemorrhage (PMSAH) is a rare but distinct clinical subtype of non-aneurysmal subarachnoid haemorrhage (SAH), with a

characteristically benign course and favorable prognosis [11]. It is defined radiographically by the presence of localized subarachnoid blood centered anterior to the midbrain and pons, typically within the interpeduncular and prepontine cisterns, often without extension into the lateral ventricles or cortical sulci [12]. The incidence is low—estimated at 0.3–0.5 per 100,000 individuals annually—yet PMSAH accounts for a significant proportion (30–35%) of non-aneurysmal SAH cases [13].

The pathophysiology of PMSAH remains incompletely understood. Unlike aneurysmal SAH, where arterial rupture is the primary mechanism, PMSAH is thought to originate from a venous source, particularly involving deep venous structures such as the basal vein of Rosenthal, vein of Galen, and straight sinus [14]. Anatomical variants in deep venous drainage—especially embryologic types B and C—may predispose individuals to venous rupture [15].

Several risk factors have been examined in relation to PMSAH. Smoking is relatively common, reported in up to 31% of cases [16]. Diabetes mellitus also appears to be more prevalent among PMSAH patients compared to those with aneurysmal SAH [17]. In contrast, alcohol abuse does not show a consistent correlation, and the role of hypertension remains uncertain, though it is frequently present in reported cases [1].

In our case, the patient exhibited multiple known or suspected risk factors for PMSAH, including heavy smoking, chronic hypertension, type 2 diabetes mellitus, hyperlipidemia, and chronic psychological stress. These overlapping comorbidities may have synergistically contributed to venous fragility or predisposition to rupture.

Clinically, PMSAH is often characterized by sudden-onset severe headache—typically described as “thunderclap”—and may be accompanied by nausea, photophobia, or neck stiffness. Loss of consciousness and focal neurological deficits are rare, with most patients presenting in Hunt–Hess

grades I–II [17]. Our patient, however, presented with sudden headache and vomiting, but had an unusually low GCS score of 8, which is uncommon for PMSAH and necessitated intubation, sedation, and neurocritical care in an ICU setting. This represents a significant deviation from the typically benign course seen in PMSAH.

Imaging in our case revealed classic PMSAH findings: localized hemorrhage in the suprasellar, interpeduncular, perimesencephalic, ambient, prepontine, and cerebellar cisterns. Initial CT angiography raised suspicion of a basilar artery aneurysm; however, digital subtraction angiography (DSA) ruled out aneurysmal pathology. This highlights the essential role of DSA as the gold standard in differentiating PMSAH from aneurysmal SAH, especially when initial imaging is ambiguous [14].

Hydrocephalus is observed in 10–15% of cases and is usually transient or managed with temporary CSF diversion, such as an external ventricular drain (EVD) [19]. In our patient, both hydrocephalus and cerebral edema were evident and required urgent EVD placement. Additionally, the patient developed ventilator-associated pneumonia due to *Klebsiella pneumoniae*, an ICU-related complication that necessitated targeted antibiotic therapy, underscoring the need for multidisciplinary vigilance in managing such cases.

Vasospasm, while radiographically present in up to 20% of PMSAH cases, rarely causes clinical symptoms [18]. Our patient showed no signs of symptomatic vasospasm, though close monitoring remained essential. The severity of his initial presentation justified proactive vasospasm surveillance and blood pressure control, interventions often reserved for aneurysmal SAH but selectively applied in complicated PMSAH cases [17].

Emerging tools such as vessel wall MRI are being explored to improve diagnostic accuracy, especially in identifying small or hidden aneurysms. However, their role in

routine PMSAH workup remains supplementary [14].

Unlike the majority of PMSAH cases, which follow a relatively benign course with short hospitalization and no ICU stay, this case required aggressive, individualized management. The presence of severe neurological compromise, hydrocephalus, cerebral edema, and nosocomial infection demanded multidisciplinary care involving neurology, neurosurgery, critical care, infectious disease, and radiology. This illustrates the spectrum of PMSAH severity and the need to tailor treatment strategies based on clinical presentation rather than the assumption of a uniformly benign course [18].

Follow-up imaging—preferably repeat DSA or high-resolution vessel wall MRI—at 4–6 weeks remains a cornerstone of post-acute care in such cases, especially when initial findings are ambiguous or complicated [19]. Strict control of modifiable comorbidities (hypertension, diabetes, dyslipidemia, and smoking cessation) is also essential in preventing recurrence and improving long-term outcomes.

CONCLUSION

This case illustrates a rare presentation of non-aneurysmal perimesencephalic subarachnoid hemorrhage (PMSAH) in a middle-aged male with multiple cardiovascular comorbidities—including chronic hypertension, diabetes mellitus, hyperlipidemia, and a history of heavy smoking—along with high psychosocial stress. Although no aneurysm was detected on cerebral angiography, the patient's neurological deterioration (GCS 8/15) necessitated prompt ICU admission, intubation, sedation, neuromuscular blockade, and placement of an external ventricular drain (EVD) to manage hydrocephalus and cerebral edema.

While PMSAH is often self-limiting and associated with favorable prognosis, individualized management is critical—especially when complicated by factors like poor consciousness, elevated intracranial

pressure, or systemic infections. In this case, the patient's ICU course was further complicated by ventilator-associated pneumonia, requiring targeted antibiotic therapy.

Treatment for PMSAH primarily involves conservative supportive care, including pain control, blood pressure regulation, neuro-monitoring, and management of potential complications such as hydrocephalus and vasospasm. Invasive interventions like EVD insertion, repeat digital subtraction angiography (DSA), and advanced imaging modalities (e.g., vessel wall MRI) may be warranted in selected patients.

This case reinforces the importance of early recognition, comprehensive imaging, and a multidisciplinary neurocritical care approach. Long-term outcomes in PMSAH are generally excellent, but close follow-up and management of underlying comorbidities are essential to reduce recurrence risk and optimize neurological recovery.

Declaration by Authors

Patient Consent: Patient consent is acquired and can be submitted for verification.

Acknowledgement: I would like to thank the authors for their valuable time. Generative AI was used for language purposes only.

Source of Funding: None

Conflict of Interest: No conflicts of interest declared.

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How to cite this article: Badhra Ajit Nair, Megha Satish, Zidan Abdul Hameed Rahim, Mohammed Shazin. Perimesencephalic SAH masquerading as aneurysmal bleed: diagnostic and critical care challenges. *International Journal of Research and Review*. 2025; 12(12): 543-548. DOI: [10.52403/ijrr.20251258](https://doi.org/10.52403/ijrr.20251258)
