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Benign versus sinister aetiologies underlying basal cistern subarachnoid haemorrhage: a case series

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Abstract

Background Subarachnoid hemorrhage in the basal cisterns is usually identified on an unenhanced computed tomography scan of the head in patients presenting acutely with a characteristic sudden onset headache.

Case presentation Using imaging examples from our tertiary neurosciences center, we present six cases demonstrating a variety of causes for subarachnoid hemorrhage in the basal cisterns, ranging from benign to sinister causes. These include a venous perimesencephalic hemorrhage (35 years, female), pontine perforator aneurysm (54 years, male), vertebral artery dissection (69 years, male), cervical dural arteriovenous fistula (65 years, male), posterior fossa arteriovenous malformation (45 years, male), and vertebral artery aneurysm (78 years, female). Ethnically, all these patients were white. Specific imaging features are described and demonstrated.

Conclusion A balance between avoiding excessive investigation and overlooking what may be a mimic of a venous perimesencephalic hemorrhage is important. To refine and establish more definitive indications on when to perform computed tomography angiogram, digital subtraction angiography, delayed angiography and magnetic resonance imaging in this context requires future research to focus on large-scale prospective multicenter studies with robust data.

Keywords Perimesencephalic hemorrhage, Subarachnoid hemorrhage, Basal cisterns, Dural arteriovenous fistula, Arteriovenous malformation, Aneurysm, Vascular dissection

Introduction

A "peri-mesencephalic pattern of subarachnoid hemorrhage" refers to intracranial subarachnoid blood centered anterior to the midbrain and/or pons. This is usually identified on a nonenhanced computed tomography (NECT) head study. Patients with such distribution of hemorrhage usually present with a sudden onset thunderclap headache, characteristic of subarachnoid hemorrhage (SAH), but have a more benign clinical course compared with aneurysmal SAH. The cause of perimesencephalic subarachnoid hemorrhage (PMH) is unknown, but a venous etiology has been suggested in literature. However, there are more sinister causes of SAH that can mimic PMH. Lack of meticulous understanding of the medical history and appropriate investigations can potentially lead to misdiagnosis.

We present a case of perimesencephalic hemorrhage with presumed venous etiology followed by five cases representing these entities: namely basal artery perforator aneurysms, vertebral artery dissection, dural arteriovenous fistula, cerebellopontine angle arteriovenous malformation, and vertebral artery

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aneurysms. Our aim is to show the contrast between the clinical and imaging features of these entities and perimesencephalic hemorrhage, and to emphasise the need for further investigation when they are suspected, including conventional digital subtraction cerebral angiography and delayed magnetic resonance imaging (MRI) of the brain. Additionally, we aim to show how identifying each of these entities will significantly change management plan. To the best of the authors' knowledge, no previous article in literature has covered such a variety of case reports to serve that purpose.

Case presentation, 1—perimesencephalic hemorrhage of presumed venous origin

A 38-year-old white female patient presented with sudden-onset occipital headache at rest, neck pain and stiffness, photophobia, and vomiting. The patient had no significant comorbidities, no recent trauma, and was a non-smoker. Prior surgical history included a caesarean section and a tonsillectomy. She had no allergies other than hay fever.

A clinical examination revealed a World Federation of Neurosurgical Sciences Grading (WFNS) grade I, Glasgow coma scale (GCS) 15 (E4, V5, M6), equal and reactive pupils (3 mm) and no focal neurological or cranial nerve deficits. Vital signs were within normal limits on examination. An NECT head done at presentation revealed a Fisher's Grade 2 SAH (Fig. 1). This had typical radiological features of a benign perimesencephalic hemorrhage (PMH) with blood distribution limited to the prepontine, premedullary, and perimesencephalic cisterns. There was no intraparenchymal, intraventricular hemorrhage or acute hydrocephalus. A computed tomography angiogram (CTA) of the cerebrum did not identify an underlying cause for the bleeding. A digital subtraction angiography (DSA) performed 2 days after presentation, and subsequently repeated after another 10 days, did not demonstrate a cerebral aneurysm, intracranial arteriovenous malformation (AVM), dural arteriovenous fistula (dAVF), or vasculopathy and the dural venous sinuses were patent. A delayed MRI, including time-of-flight angiography (MRA), was performed 3 months after presentation on the basis of the recommendation of a multidisciplinary team meeting, and did not demonstrate any underlying pathology nor features of microhemorrhages, space occupying lesion, or hydrocephalus. A multidisciplinary team discussion (4 months after presentation) in the light of the delayed MRI concluded that no cause of the SAH could be identified and a venous cause was proposed.

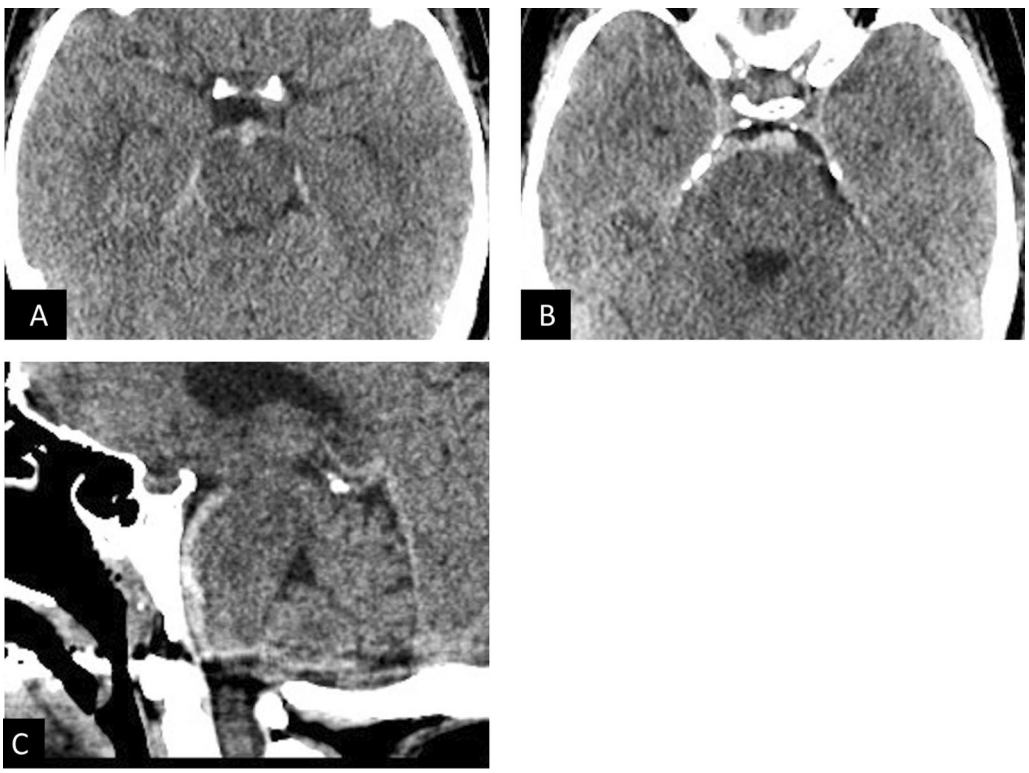


Fig. 1 Nonenhanced computed tomography head images demonstrating hyperdense hemorrhage in the perimesencephalic cisterns on an axial plane (**A**), and hyperdense hemorrhage in the prepontine cisterns on axial (**B**) and sagittal (**C**) planes

No further follow-up by imaging, after the delayed MRI, was performed, as this SAH was presumed to be venous in origin with a benign clinical course. Clinical follow-up revealed that the patient continued to complain of a persistent mild headache and some mild memory issues, and she was referred to an occupational therapist and a neuropsychologist. The patient recovered fairly well and regained functional independence with gradual improvement of headache and memory issues on clinical follow-up.

Case presentation, 2—ruptured pontine perforator aneurysm

A 54-year-old white male patient presented owing to sudden-onset severe headache while driving. The patient was previously well with no history of recent trauma and had no allergies. A NECT head done at the time of presentation revealed a Fisher's Grade 4 SAH (Fig. 2A–C). A clinical examination revealed a WFNS grade I, GCS 15, equal and reactive pupils, no focal neurological or cranial nerve deficits, and normal power, tone, and reflexes in limbs. The patient was mildly hypertensive, but vital signs were otherwise within normal limits. The SAH was centered at the perimesencephalic and interpeduncular cisterns, extending into the prepontine and premedullary cisterns. A small volume of hemorrhage was also seen in the fourth ventricle and at the base of the Sylvian fissures. There was associated mild prominence of the temporal horns, suggestive of early hydrocephalus. An urgent CTA did not show any underlying vascular abnormality or vasospasm. A DSA at presentation also did not show an aneurysm, arteriovenous malformation or dural arteriovenous fistula.

A delayed DSA was performed 9 days postictus, revealing a right-sided 2 mm basilar artery perforator aneurysm (BAPA) (Fig. 2D–F). At 11 days post-ictus, the patient was treated with a 3.5×15 mm DERIVO (Derivo Embolisation Device, ACANDIS, Pforzheim, Germany) flow-diverting stent deployed in the basilar artery across the respective perforator vessel. Following the procedure, the patient was prescribed 90 mg of oral ticagrelor twice daily for 6 months and oral 75 mg of aspirin once daily for 2 years.

A baseline MRI/MRA performed the next day after treatment did not show any ischemic complications, and a follow-up MRI after 6 months showed resolution of the hydrocephalus. A DSA performed 9 months after the procedure showed complete occlusion of the aneurysm and a patent basilar artery (Fig. 2G). The patient complained of short-term memory issues a few days after discharge, but this has significantly improved on his follow-up visit to the neurovascular clinic and he returned to work 6 months after the procedure. He eventually recovered well without complications and the aneurysm remained completely occluded on follow-up MR angiograms performed at 6, 18, and 60 months.

Case presentation, 3—dissected vertebral artery

A 69-year-old white male patient presented with sudden-onset headache that developed at rest. The patient had no history of recent trauma and did not have any significant past medical history. Clinical examination revealed a WFNS grade I, GCS 15, equal and reactive pupils, no focal neurological or cranial nerve deficits as well as normal power, tone, and reflexes in limbs. A NECT head demonstrated a perimesencephalic hemorrhage with blood in the prepontine and interpeduncular cisterns, and prominent temporal horns (Fig. 3A–C). The CTA performed within the next 12 h showed a 2-mm right cavernous inferior cerebellar artery (ICA) aneurysm and raised suspicion of a small aneurysm at the right posterior inferior cerebellar artery (PICA). No other vascular abnormality was identified at this stage, including unremarkable appearance of the vertebral arteries (Fig. 3D). The cavernous ICA aneurysm was extradural and did not warrant any further work-up. However, a DSA was performed 8 h after the CTA to investigate the possibility of a PICA aneurysm.

The DSA revealed that the suspected abnormality was in fact a small focus of calcification (in retrospect, this was evident on the NECT head), and a separate pertinent finding of a left vertebral artery dissection involving the V3 and V4 segments (Fig. 3E, F). The right vertebral artery contrast injection demonstrated a left AICA-PICA complex with symmetrical capillary enhancement suggesting adequate bilateral blood flow to the entire

(See figure on next page.)

Fig. 2 Axial nonenhanced computed tomography head at different pontine levels demonstrate hemorrhage in the basal cisterns, medial aspect of the Sylvian fissures, and in the fourth ventricle, and mild prominence of the temporal horns of the lateral ventricles (**A**, **B**). Sagittal nonenhanced computed tomography head in the median plane demonstrates hemorrhage in the prepontine and premedullary cisterns, and the fourth ventricle (**C**). Volumetric maximum intensity projected reconstruction from a three-dimensional digital subtraction angiography demonstrates a right pontine artery perforator aneurysm (**D**). Three-dimensional spin angiography demonstrates a right pontine artery perforator aneurysm (**E**). A three-dimensional model generated from three-dimensional spin angiography demonstrates a right pontine perforator artery aneurysm (**F**). A three-dimensional model generated from three-dimensional spin angiography demonstrates complete occlusion of the right pontine perforator artery aneurysm at 9 months after treatment (**G**)

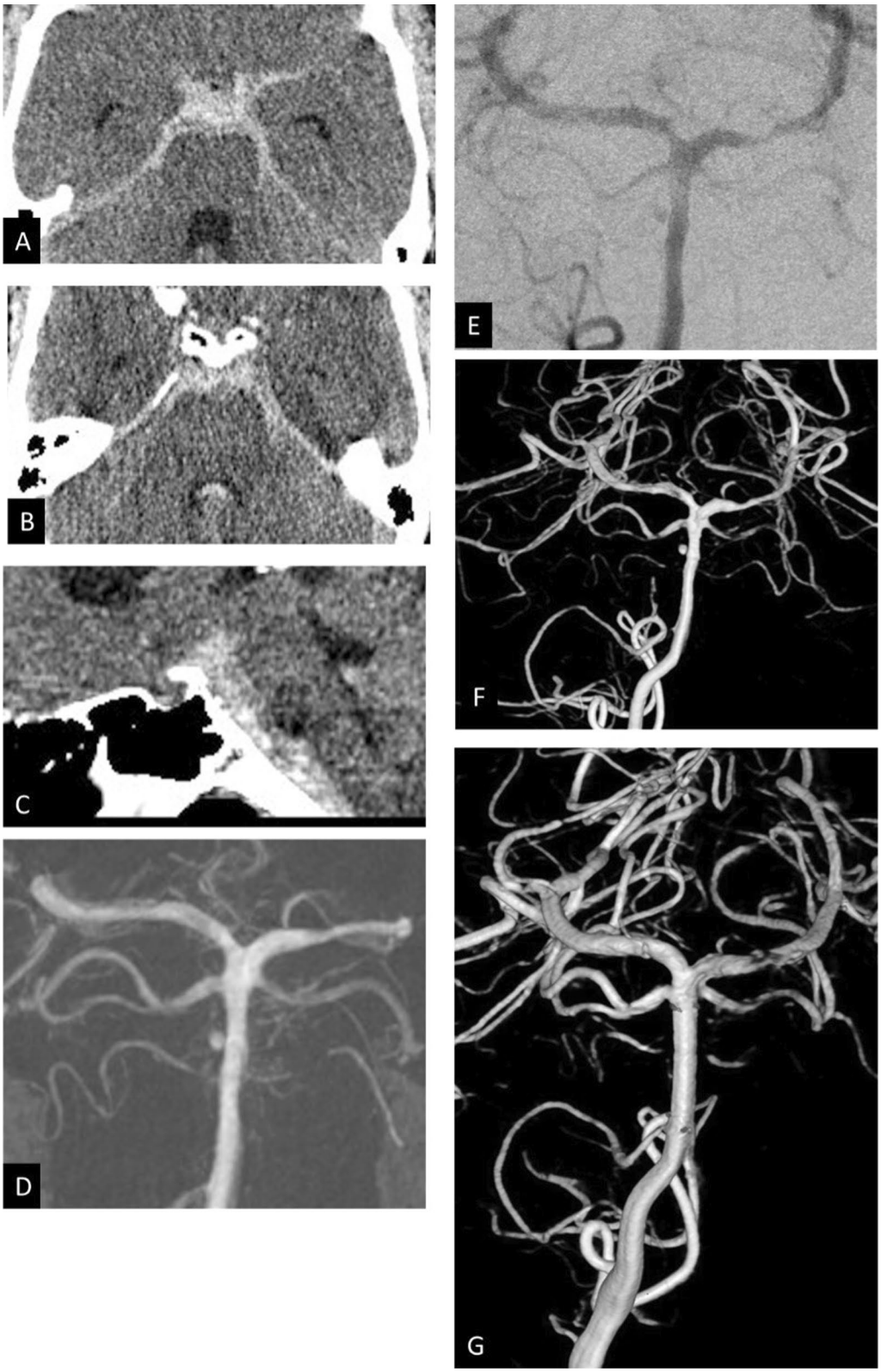


Fig. 2 (See legend on previous page.)

cerebellum (Fig. 3G, H). A MRI/MRA scan confirmed that there were no signs of cerebellar infarction but demonstrated loss of flow void in the left vertebral artery on the standard sequences and an absent left vertebral artery on time-of-flight (TOF) imaging, in keeping with dissection (Fig. 3I, J). This was treated with endovascular angioplasty. During this procedure, 10,000 U of Heparin and 1 g of Aspirin were administered intravenously. This resulted in partial recanalisation of the vessel. A postprocedural MRI/A at 3 months demonstrated remodeling of the left upper vertebral artery (Fig. 3K, L). He was seen in the neurovascular clinic 4 months after the SAH episode and at the time he had recovered well and was independent in all activities with no focal neurological deficit.

Case presentation, 4—ruptured cervical dural arteriovenous fistula

A 65-year-old white male patient became unresponsive during sexual intercourse. The patient did not have any significant past medical history. Clinical examination prior to intubation revealed WFNS grade IV, Glasgow coma scale (GCS) of 8 and fixed dilated pupils. GCS improved to 14 on the first day. He required intubation and was admitted to the intensive care unit (ICU) at a regional hospital.

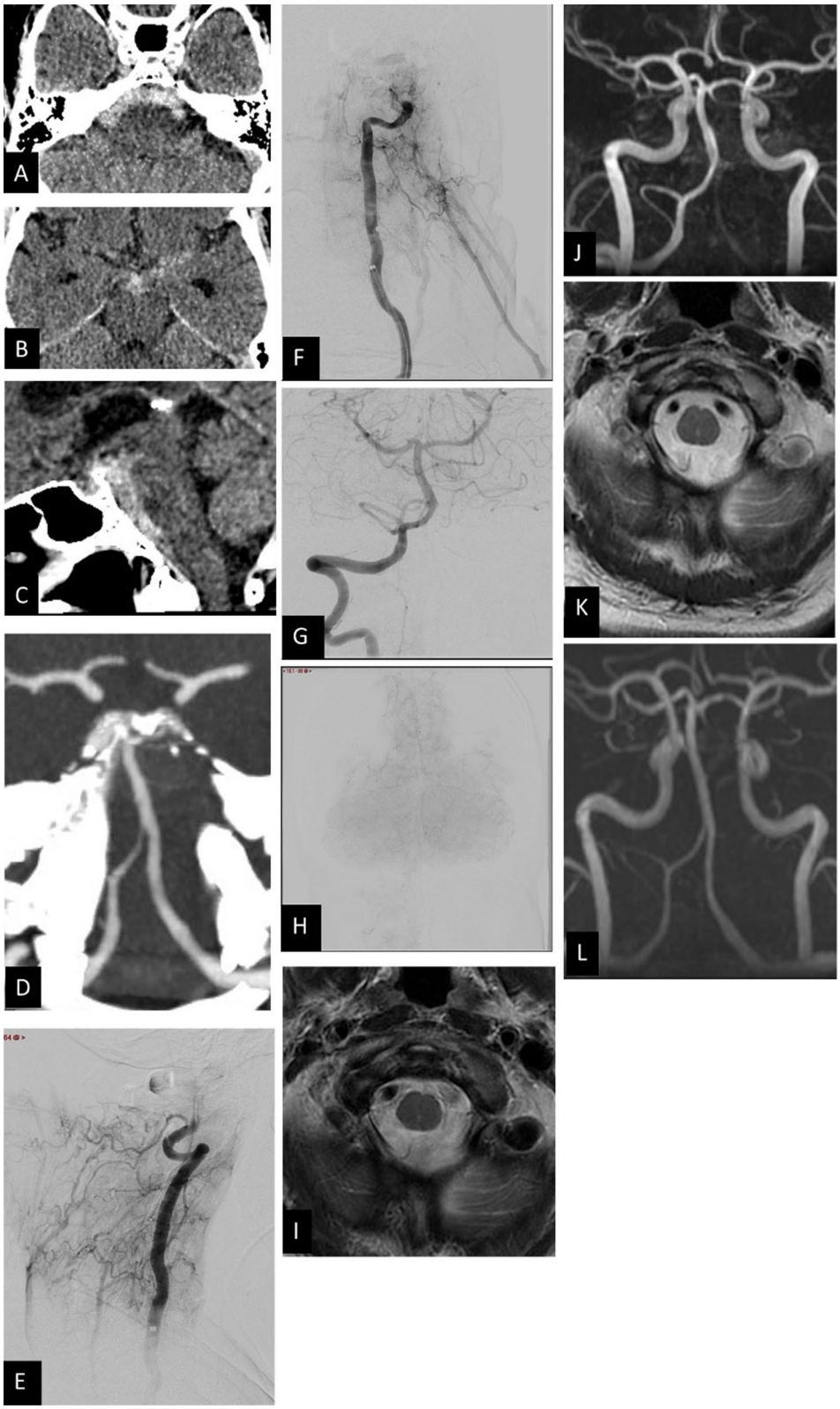
A NECT head showed a large volume of blood in the perimesencephalic region with caudal extension through the foramen magnum and around the upper cervical spinal cord (Fig. 4A–C). This also demonstrated intraventricular hemorrhage and mild hydrocephalus. The patient was transferred to our neurocenter. Neurosurgeons decided to manage the mild hydrocephalus conservatively considering the rapid neurological improvement and following improvement of ventricular dimensions on imaging studies. A CTA of the head demonstrated partially visualized abnormal tortuous vessels at the level of the foramen magnum and raised concern for a vascular malformation (Fig. 4D). A dural arteriovenous fistula (dAVF) was confirmed on DSA, which demonstrated multiple meningeal arterial feeders arising from the right vertebral artery, and associated perimedullary venous reflux (Fig. 4E). A meningeal feeder from the V3 segment harbored a 2 mm aneurysm. This feeder was catheterized with a Magic 1.5 Fr microcatheter (Balt, Montmorency, France) for endovascular embolization with injection of a liquid embolic agent. The other arterial feeders could not be catheterized. The patient had right upper and lower limb hemiparesis and right upper limb paresthesia as a consequence of the SAH episode and had a prolonged stay in the complex rehabilitation unit. On discharge, the patient had mild right upper and lower limb weakness. Follow-up imaging in 1 month demonstrated that the aneurysm had been fully occluded, but the fistula persisted owing to the other meningeal feeders. Surgical disconnection of the dural arteriovenous fistula was offered to the patient owing to risk of repeated hemorrhage from such a high-grade arteriovenous fistula (Borden III/Cognard IV). However, the patient was not keen on having surgery and opted for conservative management in view of the risks associated with complex surgery, which include hemorrhage, ischemic stroke, meningitis, pseudomeningocele, cranial nerve injury, and risk to life. His wish was respected, and he was managed conservatively. The patient was independently mobile on clinical follow-up visits and his right-sided weakness was gradually improving. The patient made good recovery on 15-month clinical review and was independent with daily activities. He was discharged from rehabilitation services.

Case presentation, 5—ruptured arteriovenous malformation at the cerebellopontine angle

A 45-year-old white male patient presented with a sudden-onset thunderclap headache, which developed at rest, associated with nausea, neck stiffness, and dizziness. The patient did not have any significant past medical history. On examination, the patient had a GCS of 15 with no focal neurological deficit. Pupils were equal and reactive and there was no cranial nerve deficit. He had normal power, tone, and reflexes in all limbs and vital signs were unremarkable. A NECT head demonstrated a SAH with blood at the perimesencephalic, interpeduncular, prepontine and premedullary cisterns, and at the foramen magnum (Fig. 5A). There was a small volume of blood extending into the basal part of the

(See figure on next page.)

Fig. 3 Nonenhanced computed tomography showing perimesencephalic hemorrhage with blood seen within the prepontine and interpeduncular cisterns on axial and sagittal images, and prominent temporal horns (**A**–**C**). Initial computed tomography angiogram revealed unremarkable vertebral arteries (**D**). Digital subtraction angiography reveals left V3 and V4 dissection (**E**, **F**). Symmetrical bilateral cerebellar capillary enhancement with right vertebral artery injections is in keeping with adequate blood flow of the entire cerebellum (**G**, **H**). A T2-weighted magnetic resonance image reveals absent flow void in the left vertebral artery (**I**). A 3D Time-of-Flight magnetic resonance angiography image reveals absent flow in the left vertebral artery (**J**). A T2-weighted magnetic resonance image image (**K**) and 3D TOF magnetic resonance angiography image (**L**) 3 months after endovascular angioplasty show restoration of the left vertebral flow void and arterial flow, respectively



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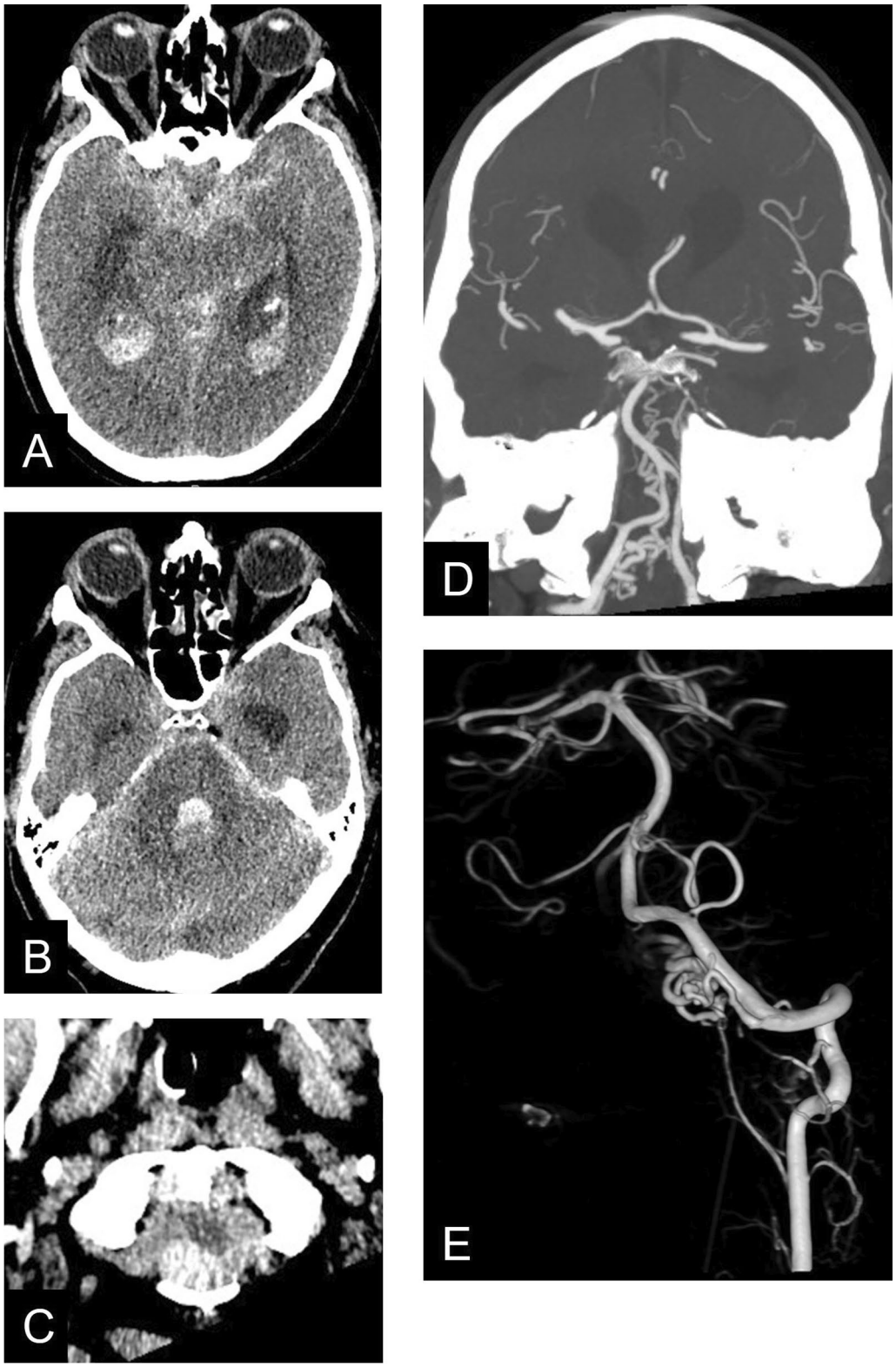

Fig. 4 Nonenhanced computed tomography shows a large amount of perimesencephalic and intraventricular blood, as well as blood at the foramen magnum (**A**–**C**). A computed tomography angiogram shows tortuous ectatic vessels crossing the foramen magnum, in keeping with a vascular malformation (**D**). A three-dimensional model generated from three-dimensional spin angiography demonstrates a feeder artery arising from the posterior aspect of the right V3 segment (**E**)

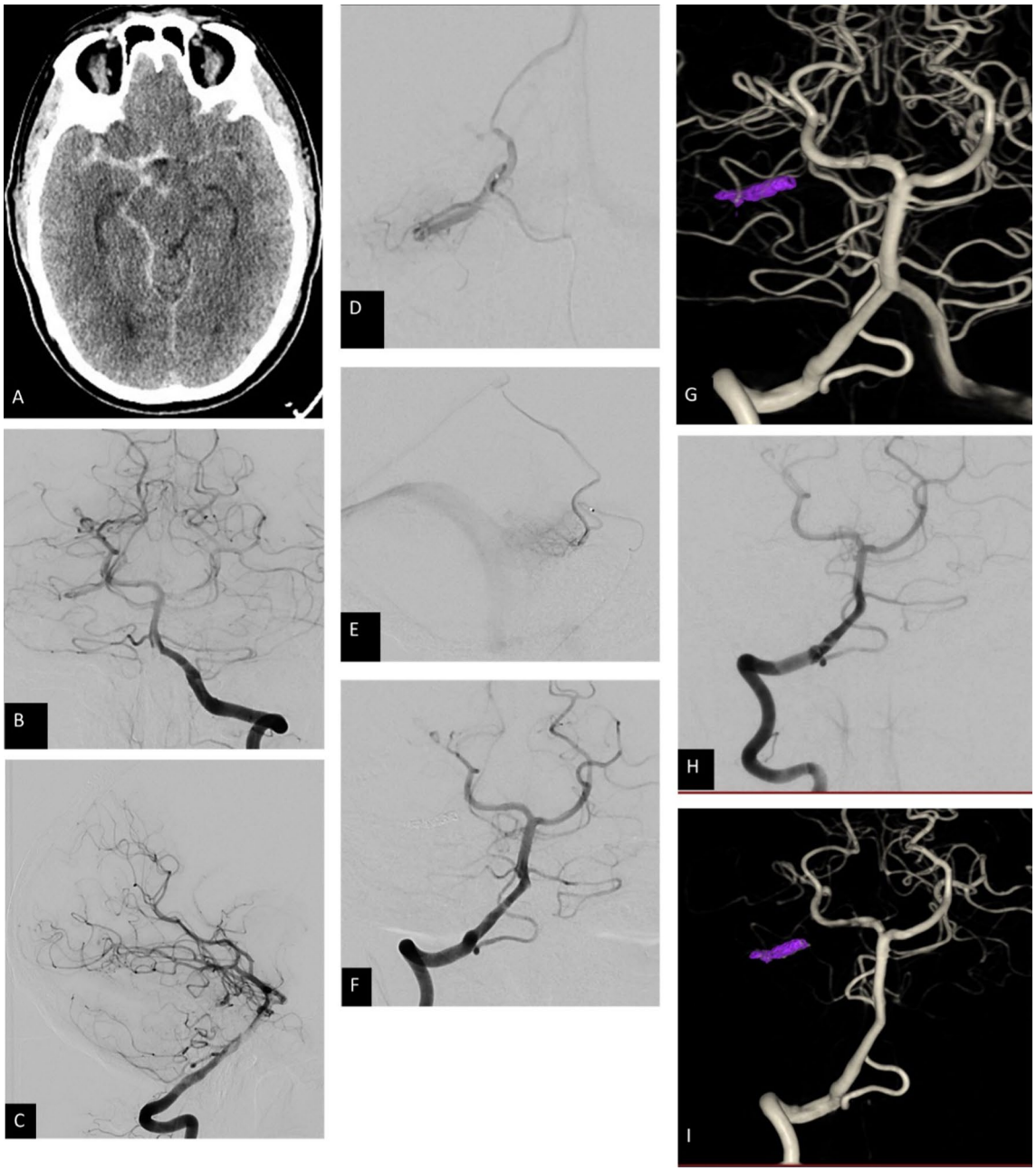

Fig. 5 Nonenhanced computed tomography shows interpeduncular and perimesencephalic blood extending into the basal parts of the Sylvian fissures, particularly on the right side (**A**). Frontal and lateral views on digital subtraction angiography demonstrate early venous shunting into right lateral perimesencephalic vein (**B**, **C**), which is better demonstrated on superselective injections with a microcatheter injection via the superior cerebellar artery (**D**, **E**). Anteroposterior view of digital subtraction angiography and three-dimensional reconstruction show no residual or recurrent arteriovenous shunting at 3 months (**F**, **G**) and 14 months (**H**, **I**)

right Sylvian fissure. There was no intraventricular or intraparenchymal hemorrhage or acute hydrocephalus. A CTA was negative for any vascular abnormality.

A DSA performed the next day demonstrated a Spetzler-Martin Grade II (SM II) arteriovenous malformation (AVM) at the right cerebellopontine (CP) angle (Fig. 5B–E). This had arterial feeders from the superior cerebellar artery (SCA) and anterior inferior cerebellar artery (AICA). Its venous drainage was to the lateral mesencephalic vein and superior petrosal sinus. The patient had worsening headache during hospital stay and significant nausea. The patient had a lumbar

puncture 4 days after admission which showed an opening CSF pressure of 25 cm H_2O which was reduced to 11 cm H_2O by draining 34 ml of cerebrospinal fluid. Endovascular embolization was carried out 2 weeks after onset, with no immediate complications. A follow-up DSA performed 3 months after treatment showed complete occlusion of the AVM (Fig. 5F, G). The patient continued to have headache after discharge, which was managed by paracetamol and codeine. The headache significantly improved on clinical follow-up reviews. The patient made a good clinical recovery, apart from fatigue and short-term memory problems (often associated with SAH) and he needed counseling because of this experience. The patient was independent in activities of daily living and financial affairs on follow-up. The patient had a second follow-up DSA 14 months after endovascular treatment which also showed no residual or recurrent arteriovenous shunting (Fig. 5H, I).

Case presentation, 6—vertebral artery aneurysm (v4 segment)

A 78-year-old white female patient presented with a sudden onset severe headache and confusion. There was no history of a physical activity during onset. She had past medical history of insulin-dependent diabetes mellitus, hypertension, and hyperlipidaemia. On clinical examination, initial GCS was 9 (E2, V2, M5), which later improved to 13 on admission to our tertiary center. A NECT head revealed a SAH distributed in the prepontine, interpeduncular, cerebellopontine cisterns, and extending to the basal parts of the Sylvian fissures. There was also a small volume of blood in the third and fourth ventricles. This was complicated by acute hydrocephalus. A CTA revealed a small aneurysm at the proximal part of the V4 segment of the right vertebral artery (Fig. 6A). The hydrocephalus was initially treated by a right frontal external ventricular drain followed by a lumbar CSF drain. A DSA confirmed the presence of a 3 mm aneurysm in the V4 segment of the right vertebral artery (Fig. 6B, C). Endovascular coiling of the ruptured right V4 aneurysm was performed 1 day after presentation, and the procedure was uneventful with successful occlusion of the aneurysm with coiling (Fig. 6D, E). Following the procedure, the patient was gradually weaned off sedation. She developed vasospasm which was treated by hypertension, hypervolemia and respiratory support. She had a gradual neurological improvement during stay on the intensive care unit. The patient was then transferred back to the intensive care unit in the referring regional hospital with a GCS of 14. At the time of discharge, she had a GCS 15 with no focal neurological deficit. She was reviewed in the neurovascular clinic 3 months after discharge, which showed full recovery, and the patient was back to performing all regular activities (except driving). She had imaging follow-up by MR angiograms at 6 and 18 months after the endovascular coiling confirming complete occlusion of the treated aneurysm (Fig. 6F). Another follow-up MR angiogram is scheduled for 60 months after the endovascular procedure.

Discussion

Benign perimesencephalic hemorrhage

A distinction needs to be made between PMH as a clinical entity with a relatively benign clinical course, which has no definite identifiable underlying cause, and other causes of hemorrhage in the basal cisterns with underlying sinister pathological entities [1]. PMH as a benign entity should fit the following criteria [2–6]:

1. No identifiable underlying source for the hemorrhage on angiographic imaging (angio-negativity).
2. The epicenter of SAH must be located immediately anterior to the midbrain and/or pons. Extension of the hemorrhage to other areas is uncommon but is reported in some cases in small amounts and limited to the ambient cisterns, quadrigeminal cistern, cisterna magna, posterior aspect of the anterior interhemispheric fissure, basal (medial) portion of the Sylvian fissure, occipital horns of the lateral ventricles, and fourth ventricle.

It is estimated that the incidence of PMH is 0.5 cases per 100,000 persons-years representing about 5% of all cases of SAH [1, 2, 7, 8]. It presents in a similar way to other causes of SAH with a sudden onset headache, but exertion is known to precede the headache in some cases [9, 10]. Other features manifested, also in other causes of SAH, include nausea, vomiting, and neck pain and/or stiffness. As in the presentation of patient 1, the neurological examination is typically unremarkable in patients with PMH apart from signs of meningeal irritation [1, 2, 11].

Patient 1 did not have any radiological evidence or clinical suggestion of acute hydrocephalus as is the case with the majority of PMH presentations. In the minority of cases radiologically demonstrating acute hydrocephalus, it is rarely symptomatic [10]. The clinical picture of PMH is generally benign with a much better prognosis compared with classic aneurysmal subarachnoid hemorrhage [10–12]. However, a minority of patients do not fully recover owing to rebleeding, hydrocephalus, symptomatic vasospasm and delayed cerebral ischemia [11, 13]. Although patient 1 did not experience any acute complication of SAH and the clinical course was benign, the long-terms sequelae of

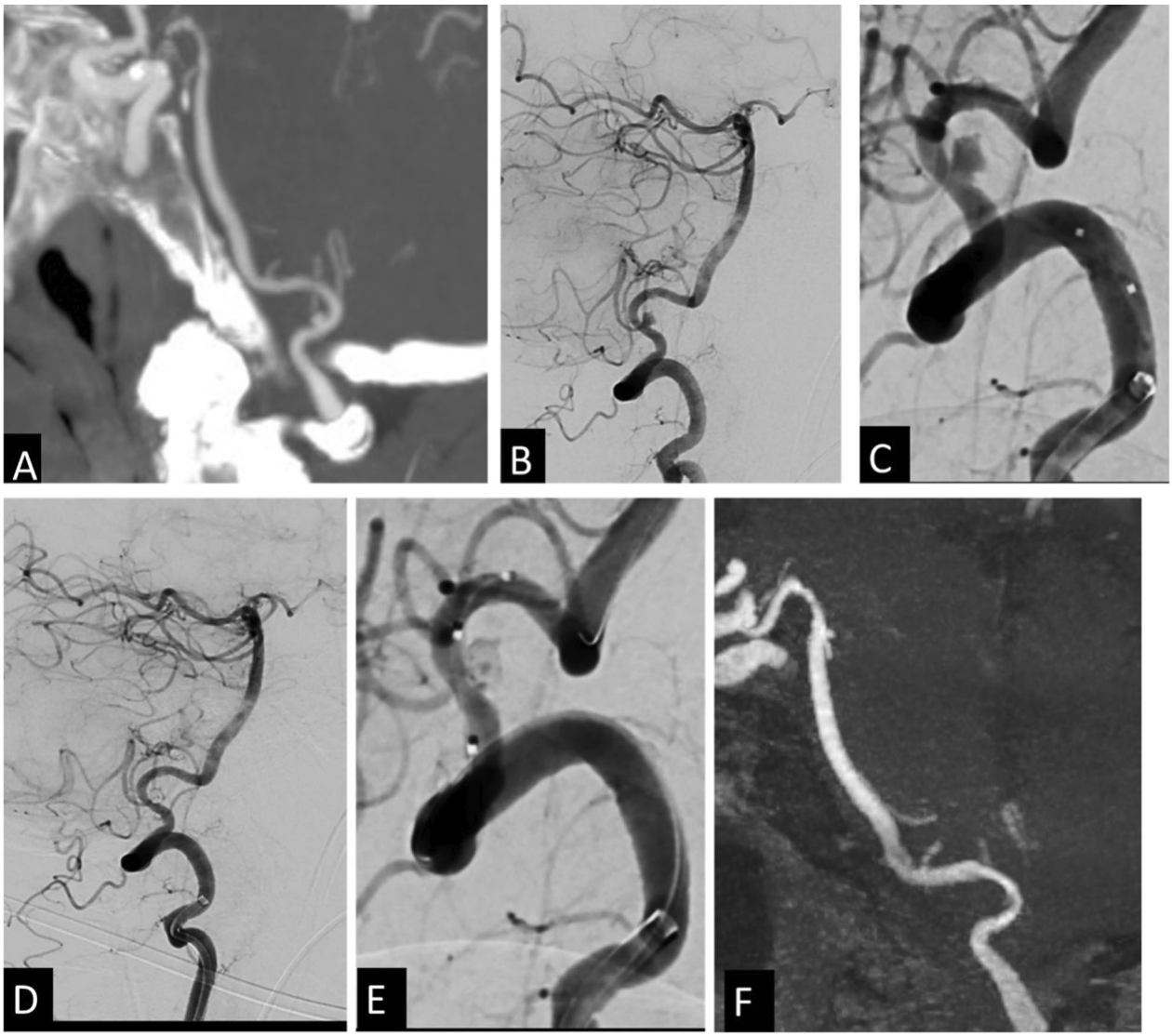

Fig. 6 A computed tomography angiogram shows a small right vertebral artery V4 segment aneurysm (**A**). A digital subtraction angiography confirms this finding (**B**, **C**). A digital subtraction angiography shows adequate occlusion of the aneurysm after coiling (**D**, **E**). Time-of-Flight magnetic resonance angiography image shows complete occlusion of the aneurysm at 18 months following endovascular coiling (**F**)

SAH, in the form of chronic headache and short-term memory issues, have implicated patient's quality of life to a degree. Similarly, some patients with PMH develop fatigue, cognitive, psychological and/or neurological impairment. Anxiety and fears related to re-experiencing SAH are also possible. Because these non-life-threatening long-term sequelae are not the primary focus in the context of acute SAH, it is important to make sure patients receive adequate clinical attention and support during long-term follow-up. A relationship between the amount of subarachnoid blood and delayed complications, such as symptomatic vasospasm or cerebral ischemia, has been suggested [2, 13–15].

Owing to the relatively benign course of PMH, it has been difficult to explore in detail the underlying pathophysiology of this entity. A limited number of surgical explorations in patients and cadaveric autopsies have not managed to demonstrate a specific cause with any certainty [16]. Radiology has been the main tool in trying to understand this entity. The dominant theory is that PMH is venous in origin, which is supported by radiological studies that investigated the pattern of venous drainage in affected patients. A primitive variant of venous drainage that directly drains into a dural sinus, rather than via the vein of Galen was seen in many patients presenting with PMH, and those patients

with unilateral venous variant presented with ipsilateral hemorrhage. Another radiological study suggesting a non-arterial source of PMH demonstrated on CT perfusion that cerebral blood flow is less compromised in these patients than in those with aneurysmal subarachnoid hemorrhage [1, 17, 18]. The dominant theory is also supported by the fact that PMH tends to have a relatively benign clinical course in most patients, and the limited distribution of blood suggests leakage of blood under low pressures [12].

While a venous etiology may be a plausible explanation for many cases, other suggested etiologies that can radiologically mimic PMH include spontaneous thrombosis of an aneurysm, a small aneurysm obscured by a large volume of SAH, vasospasm, or technical factors (e.g. imaging artifacts) [2, 3, 19]. Other cases with a hemorrhage in the perimesencephalic region may have similar radiological features to benign PMH, but also demonstrate additional features which should raise concern for an alternative and more sinister diagnosis. The case reports presented in this pictorial review highlight features that suggest benignity and other red flag features, which suggest sinister etiologies demanding more thorough investigations.

CTA tends to be the first investigation in the context of a perimesencephalic SAH. The main controversy revolves around the need for an initial and a delayed DSA, which remains the gold standard modality to diagnose vascular pathology [20]. Although rare, a DSA may have significant complications and its quality is operator dependent. Two meta-analyses by Geng *et al.* [8] and Kalra *et al.* [21] have concluded that a CTA alone is sufficient when the clinical and imaging features fit the criteria of a benign PMH, and that initial and delayed DSA should be used on a case-by-case basis. Similarly, Velthuis *et al.* [22] advised the use of CTA as an alternative to DSA to avoid risks, which outweigh the odds of finding an aneurysm exclusively on DSA. Menon [23] has suggested that DSA in the first 6 weeks may not be needed given the low risk of rebleeding and other complications.

In the context of PMH, the value of CTA can be maximized to replace DSA to a satisfactory level. For instance, adding a venous phase to cerebral CTA has the added value of assessing venous structures [2]. Additionally, having CTAs reported by specialist neuroradiologists and the practice of double reporting can increase accuracy [4]. Despite such recommendations for PMH, many centers still practice a protocol of a CTA and a single DSA. This is based on the argument that a potential miss of significant pathology presenting with atypical features may be catastrophic.

Regarding brain and cervical spine MRI, Sadigh *et al.* [3] concluded that these do not need to be included in routine follow-up of benign PMH and should be limited to selected cases with more sinister features of perimesencephalic hemorrhage. CTA tends to be preferred over MRA as it can be done simultaneously with NECT and it takes a much shorter time, making it more suitable for agitated and restless patients. The latest CT technology allows for higher resolution imaging with improved diagnostic value.

It is important to emphasize that benign radiological features alone are not sufficient to adopt a conservative approach. Patients who present with benign radiological features, but manifest serious clinical features should have a less conservative management plan in a similar manner to aneurysmal SAH [4]. In delayed presentations beyond 72 hours, the redistribution and reabsorption of the hyperdense SAH demonstrated on a NECT is likely to underestimate the original volume of SAH or even completely fail to demonstrate it. This may lead to a SAH being missed or mimicking the benign radiological features of a PMH. If a SAH is strongly suspected clinically but not evident on a NECT, a lumbar puncture should be performed to demonstrate the presence of xanthochromia and prove the presence or absence of SAH.

Basilar artery perforator aneurysm

BAPAs were first reported in 1996, and the number of reported cases in literature remains very few [24]. In most cases, these aneurysms are less than 3 mm in size and rarely exceed 7 mm [25]. Clinically, they could imitate the more commonly seen benign perimesencephalic hemorrhage in its benign course as seen in patient 2 who had an unremarkable clinical examination and a benign clinical course. This is thought to be owing to their location on low flow arteries and because they may be partially thrombosed, which also makes them difficult to detect on imaging [19].

The initial DSA for patient 2 was negative. This is usually the case with these aneurysms due to their small size and spasm associated with SAH which makes them occult on DSA and therefore likely underdiagnosed. A delayed DSA after 7 days from presentation helps to identify these aneurysms [26].

A multidisciplinary decision was made to treat the small ruptured pontine perforator aneurysm patient 2 with a flow-diverting stent. Literature describes several treatment options for such aneurysms, including stenting with a flow diverter, telescoped double stenting (e.g., LEO baby by Balt, Montmorency, France), liquid embolic agents, and in rare cases open surgical clipping [25–31]. Conservative management is also an option with some cases spontaneously resolving. The largest case series of 52 pontine perforator aneurysms suggests that the most

effective treatment with the least complications is double stenting across the basilar trunk [29].

Arterial dissection

A dissection of the vertebrobasilar system can also cause hemorrhage in the basal cisterns. The history of patient 3 was negative for major or minor trauma and the cause of the left vertebral dissection was unknown. Additionally, the exact mechanism of how a dissection leads to SAH is not entirely clear. However, one possible explanation is extension of dissection across the arterial wall causing rupture of adventitia or the formation of a thin-walled pseudoaneurysm which ruptures later [32–34]. Another suggested explanation is the development of a dissecting aneurysm, which ruptures causing subarachnoid hemorrhage [35]. The internal elastic lamina is important for integrity of the arterial wall and its disruption is thought to play an important role in this process [36, 37].

Patient 3 had a similar acute clinical presentation to a benign perimesencephalic hemorrhage as well as a similar pattern of SAH on NECT. Although we believe the patient would have had a cerebral angiogram anyway in this clinical scenario, perhaps the misinterpretation of a calcification close to the right PICA as an aneurysm has played a positive role in reinforcing the decision for further investigation despite the benign clinical presentation and the very similar NECT appearance to benign perimesencephalic hemorrhage. This again emphasizes the necessity of considering these rare alternative diagnoses even in cases with clinical and radiological features suggestive of a benign perimesencephalic hemorrhage. In our center, we have a low threshold for performing DSA for such cases and further studies are needed to refine indications for further radiological investigation including an initial and delayed DSA.

Vascular malformations

Patient 4 is the first in this series to present with a high-grade SAH and deteriorated consciousness which necessitated further investigation beyond initial CT studies. This case shows how meticulous review of initial CTA by a neuroradiologist is important for finding clues (sometimes subtle) for a serious underlying pathology. While the lower grades of dAVF (Borden I/Cognard I–IIa) typically have a benign clinical course and many of these patients are asymptomatic, the presence of cortical venous drainage (CVD) in the higher grades (Borden II–III/Cognard types IIb–V) seems to account for the higher risk of hemorrhage and mortality rate [38–40]. The accuracy of these systems can be improved by further classification of dAVFs with CVD into symptomatic and asymptomatic types [38, 41].

Patient 5 was relatively young and presented with nonspecific clinical and initial radiological features that are very similar to benign perimesencephalic hemorrhage. However, he went on to develop manifestations of increased intracranial pressure during hospital stay. Further investigation by a DSA revealed a CP angle arteriovenous malformation. Cerebral arteriovenous malformations (AVMs) are estimated to cause 2% of cases of hemorrhagic stroke. Although they represent a relatively rare cause of intracranial hemorrhage, they are especially important as they tend to affect relatively younger patients, and as there is an increased rate of detection with the increased utilization of cross-sectional brain imaging [42, 43]. The estimated annual risk of hemorrhage within a previously unruptured cerebral AVM ranges from 2 to 4%, which increases in case of ruptured cerebral AVMs [43–46]. A meta-analysis by Gross *et al*. in 2013 showed an annual risk of hemorrhage of 4.5% in already ruptured brain AVMs compared with 2.2% in unruptured AVMs [47].

Posterior circulation aneurysm

Although patient 6 presented with serious clinical manifestations in the form of confusion, the SAH pattern on the plain CT study was similar to benign perimesencephalic hemorrhage involving the basal cisterns. However, the presence of an intraventricular component was an important differentiating feature on imaging. It is estimated that aneurysms of the posterior circulation are responsible for nearly 4% of cases presenting with a perimesencephalic pattern of SAH [1, 48]. In addition to vertebrobasilar aneurysms, other possible locations of posterior circulation aneurysms include the PCA, SCA, and PICA [11, 22, 48, 49]. It is good practice to closely assess the CTA in the posterior fossa and pay attention to these reported locations of posterior fossa aneurysms [48].

It is well-known that the risk of rupture of posterior circulation aneurysms is significantly higher than that of anterior circulation aneurysms [50–56]. Clarke *et al*. [57] have estimated the annual bleed rate of posterior circulation aneurysms to be 1.8% compared with 0.49% with anterior circulation aneurysms. The reasons why bleed rates differ are not fully understood. However, factors such as differences in hemodynamics between anterior and posterior circulations based on differences in configurations and geometries of arteries are thought to play a role. The extent of perianeurysmal spaces allowing for aneurysmal growth in the posterior circulation might also be involved [55]. Some studies have also shown a larger dome size [53, 55] and parent artery [53] in cases of posterior circulation aneurysms which again might be related to the higher risk of rupture.

Thanks to advances in endovascular techniques in treatment of intracranial aneurysms in general, and posterior circulation aneurysms in particular, trends have shifted from microsurgical treatment to the minimally invasive endovascular coiling, reducing mortality and morbidity, including the risk of epilepsy [58]. This was influenced by two studies: the International Study of Unruptured Intracranial Aneurysm (ISUIA) and the International Subarachnoid Aneurysm Trial (ISAT) [50, 59]. Microsurgical treatment is particularly more difficult for posterior circulation aneurysms owing to several factors, such as challenging surgical exposure, related brainstem perforators, absence of an aneurysm neck, and closeness of critical structures, such as the brainstem, eloquent areas of the brain, and cranial nerves. Furthermore, SAH and the associated edema and adhesions add to the complexity of the situation [51, 58, 60–62].

Conclusion

There are several causes of a basal-cistern-predominant pattern of SAH, which could radiologically and often clinically mimic the more benign perimesencephalic hemorrhage in the basal cisterns. It is important to consider these differentials whenever faced with an initial diagnosis of a benign perimesencephalic hemorrhage and to have a low threshold for further radiological investigation including DSA (initial and delayed) and delayed MRI studies where appropriate. Meticulous interpretation of initial radiological studies by a neuroradiologist and paying attention to clinical and radiological clues are important to avoid misdiagnosis as benign perimesencephalic hemorrhage, particularly outside tertiary neurocenters. Radiological red flags include caudal, cranial, or peripheral extension of the perimesencephalic SAH, extensive intraventricular hemorrhage, or intraparenchymal hemorrhage. Sinister clinical features warrant further investigation by DSA and/or MRI despite benign perimesencephalic pattern on plain CT. To refine and establish more definitive indications on when to perform delayed imaging in these patients requires future research to focus on large-scale prospective multicenter studies with robust data.

Abbreviations

AICA	Anterior inferior cerebellar artery
AVM	Arteriovenous malformation
BAPA	Basilar artery perforator aneurysm
CP angle	Cerebellopontine angle
CTA	CT angiogram
CVD	Cortical venous drainage
dAVF	Dural arteriovenous fistula
DSA	Digital subtraction angiography
EVD	External ventricular drain
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
NECT	Non-enhanced CT study
PICA	Posterior inferior cerebellar artery
PMH	Perimesencephalic hemorrhage
SAH	Subarachnoid hemorrhage
SCA	Superior cerebellar artery

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Declarations

Ethics approval and consent to participate

The data is completely anonymized and therefore the requirement for individual consent was waived as per institutional policy.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

No competing interests to declare.

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