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Case Report

Paroxysmal sympathetic hyperactivity and cerebral salt wasting post management of arteriovenous malformation in a pediatric patient: A case report

Ahmed Hafez Mousa[1,2], Imad A. M. Abuanza[3], Sameera Hajijama[4], Yusor Al-Nuaimy[5], Arwa Jader[6], Jumana Hussain Timraz[7], Bushra Wadi Bin Saddiq[7], Mohammed Jilan Al Nahari[7], Ghaya Zeyad Alrais[8], Motaz M.B. Abudaqqa[9], Zeyad Faoor Alrais[3], Ameer Alyeldien[1]

[1]Neurosciences, [2]Department of Neurosurgery, Graduate Medical Education, Mohammed Bin Rashid University of Medicine and Health Sciences, [3]Department of Critical Care, [4]College of Medicine, Mohammed Bin Rashid University of Medicine and Health Sciences, Dubai Health, Dubai, [5]College of Medicine, Ajman University, Ajman, United Arab Emirates, [6]Department of Neurosurgery, Faculty of Medicine, Kufa University, Kufa, Iraq, [7]College of Medicine, Batterjee Medical College, Jeddah, Kingdom of Saudi Arabia, [8]College of Medicine, Dubai Medical College, [9]Department of Pediatric Neurocritical Care, HMS Al Garhoud, Dubai, United Arab Emirates.

E-mail: *Ahmed Hafez Mousa - ahmousa@dubaihealth.ae; Imad A. M. Abuanza - aboanza@hotmail.com; Sameera Hajijama - sameera.hajijama@gmail.com; Yusor Al-Nuaimy - 202110326@ajmanuni.ac.ae; Arwa Jader - arwa1999salm@gmail.com; Jumana Hussain Timraz - jomana1420@gmail.com; Bushra Wadi Bin Saddiq - bushrabinsiddiq@gmail.com; Mohammed Jilan Al Nahari - mohammedalnahari@gmail.com; Ghaya Zeyad Alrais - ghaya01alrais@gmail.com; Motaz M. B. Abudaqqa - motaz272@hotmail.com; Zeyad Faoor Alrais - zfalrais@dubaihealth.ae; Ameer Alyeldien: aalyeldien@dubaihealth.ae

***Corresponding author:**
Ahmed Hafez Mousa,
Neurosciences, Dubai Health,
Dubai, United Arab Emirates.

ahmousa@dubaihealth.ae

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ABSTRACT

Background: Spontaneous intracranial hemorrhage (ICH) is a rare presentation in healthy pediatric patients due to a myriad of conditions. Among them, arteriovenous malformations (AVMs) stand out for their potential to rupture and risk of death due to hemorrhagic strokes. A complication to consider in patients post ICH due to AVMs is the development of paroxysmal sympathetic hyperactivity (PSH) and cerebral salt wasting (CSW), as these complications further delay recovery and may lead to devastating results if left untreated.

Case Description: We report a rare case of a 13-year-old female who developed a nontraumatic intracerebral hemorrhage due to a rupture of left AVM. She was managed with a decompressive craniotomy and further stabilized with two sessions of embolization. Following surgical intervention, she developed PSH and CSW, recovering through medical management.

Conclusion: By highlighting this unique presentation in a previously healthy patient, we aim to deepen our understanding of the complexities surrounding fewer known causes of pediatric ICH, particularly in relation to nontraumatic AVMs, and to emphasize the importance of early diagnosis and intervention. Close monitoring and prompt assessment are required to prevent further complications in patients with PSH or CSW.

Keywords: Cerebral salt wasting, Emergency neurosurgery, Paroxysmal sympathetic activity, Pediatrics neurosurgery, Ruptured arteriovenous malformation

INTRODUCTION

Pediatric intracerebral hemorrhage (pICH) is critical condition that represents a significant proportion of strokes during childhood. Spontaneous pICH is due to a number of conditions. Among them, arteriovenous malformations (AVMs) stand out for their potential to rupture and lead to hemorrhagic strokes.[6,9]

AVMs are congenital vascular abnormalities that account for approximately 3% of pediatric cases of pICH. Cerebral AVMs are responsible for 35–55% of hemorrhagic strokes in children, with an incidence rate of 1.4/100,000 person-years.[21,30] The management involves a multidisciplinary approach to achieve complete angiographic obliteration while minimizing neurological sequelae. The available option's treatment includes conservative management, surgical resection, stereotactic radiosurgery, and endovascular embolization. The choice of treatment modality depends on the size, location, and hemodynamic properties of the AVM, the patient's clinical condition, and the available expertise in a multidisciplinary team.

Surgical resection remains the gold standard for Spetzler-Martin grade 1-3 AVMs when feasible, with reported obliteration rates between 67% and 100% and associated morbidity and mortality.[13] Stereotactic radiosurgery is indicated for deep-seated or eloquent cortex AVMs, with obliteration rates around 59–69% and lower complication rates compared to surgery, though the long-term effects on the developing nervous system are not yet fully understood.[13] Endovascular embolization plays an important adjunctive role, enabling pre-surgical size reduction in large AVMs by 78% on average, and can achieve 21.2% complete obliteration of small lesions, allowing for size reduction and staged treatment, even though it is unlikely to achieve complete obliteration alone.[13]

In the context of AVM rupture, a critical complication to note post embolization is paroxysmal sympathetic hyperactivity (PSH), a clinical syndrome of an unclear mechanism thought to be caused by a disturbance in descending neuronal pathways resulting in the release of sympathetic stimulation without the counterbalanced inhibitory response.[4,35] It presents transient episodes of increased sympathetic activity, such as tachycardia, tachypnea, hypertension, diaphoresis, hyperthermia, and dystonic posturing. It is a complication often seen in comatose patients after an acquired traumatic brain injury; however, nontraumatic causes of PSH do occur.[8,17,23] The diagnosis is clinical, and measurements by Baguley *et al*.[4] use vital signs and clinical features to assess the likelihood of PSH development.

Another complication that our patient also experienced was cerebral salt wasting (CSW) syndrome, which presents as hyponatremia and extracellular fluid loss after traumatic central nervous system (CNS) injury.[20,22,31] However, nontraumatic causes such as intracranial hemorrhage (ICH) are a less likely presentation in pediatric patients.[20] The management of CSW includes sodium correction through fluid administration and sodium supplementation.

We report a 13-year-old female who developed a PSH and CSW following pICH from a nontraumatic rupture of a left AVM. This case highlights a rare presentation, and by examining this case, we aim to deepen our understanding of the complexities surrounding fewer known causes of pICH particularly in relation to nontraumatic AVMs, and to emphasize the importance of early diagnosis and intervention.

CASE PRESENTATION

A 13-year-old female was admitted to a nearby emergency department after being found unconscious with her eyes rolled up and vomit on her clothing. The patient was previously healthy and had no family history of previous neurological conditions. On arrival at the nearest hospital, her Glasgow coma scale (GCS) was 9/15, and she was sedated before being electively intubated. Stat computer tomography (CT) revealed a left ICU hemorrhage. The patient was later moved to the tertiary center as neurosurgery service was available. She was later referred to our emergency department within 6 h after her initial presentation with a GCS of 3/18 and bilateral 1 mm nonreactive pupils. She was taken for a plain brain CT scan, which revealed a left ganglionic hyperdense acute bleed measuring ~ 3.3 × 3.8 × 5.8 cm (AP × TS × CC) with perifocal edema causing mild compression of the left lateral ventricle. A midline shift to the right of about 5.5 mm [Figure 1] was noted. CT cerebral angiogram showed an Ill-defined abnormal tuft of serpiginous vasculature in the left thalamic region suggestive of AVM.

The neurosurgical team performed a decompressive craniotomy, hematoma evacuation, and right frontal intracranial pressure (ICP) monitor insertion. She remained stable throughout the process and was taken for a digital subtraction angiography shortly after occupational therapy. The results revealed a deep-seated AVM with feeders from the anterior choroidal and medial division of the left middle cerebral artery.

The interventional radiologist recommended immediate surgical management, in which the AVM's medial compartment is embolized while the lateral compartment fills through feeders from the left middle cerebral artery [Figure 2]. The patient was stable and transferred to the surgical ICU following the procedure. She received 1 unit of packed red blood cells during the procedure; as per the interventional radiologist, she needed a second session of AVM embolization the next day. During the second procedure, embolization of the feeding arteries was not possible due to the microcatheter being unable to cannulate those vessels due to them being tiny and tortuous. Thus, an angiogram was done, and the findings were reported [Figure 3].

Five days after the procedure, the patient was extubated. The patient's GCS remained at 3/15, and sluggish pupils measured at 3 mm bilaterally; weaning was initiated, and the patient was sedated using intravenous fentanyl and midazolam. Thirty

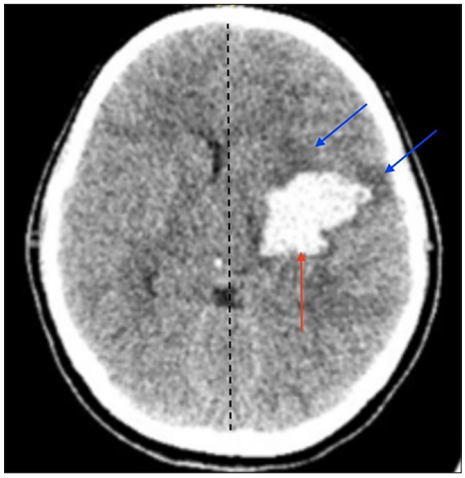

Figure 1: Computed tomography scan taken at initial presentation; axial plane revealed a left ganglionic hyperdense acute bleed (red arrow) measuring 3.3 × 3.8 × 5.8 (anteroposterior × transverse × coronal) with perifocal edema (blue arrow), with a midline shift to the right of about 5.5 cm (dotted line).

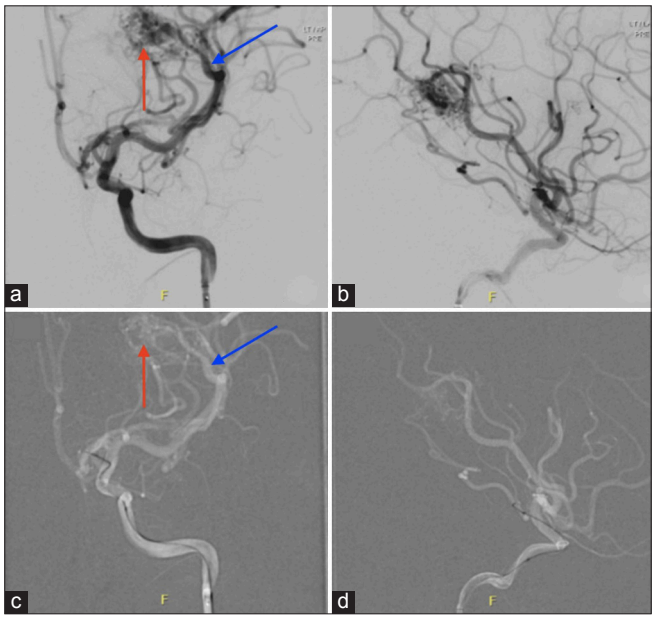

Figure 2: Initial post-selective embolization of left temporal arteriovenous malformation (AVM) (a) The partial embolization of the AVM (red arrow) with the medial component of the AVM embolized and the lateral component of the AVM filling through feeders from the middle cerebral artery (blue arrow); (b) the partial embolization of the AVM – lateral view; (c) branches of the internal carotid artery show partial embolization of the medial component of the AVM (red arrow) with the lateral component filling through feeders from the left MCA (blue arrow); and (d) branches of the internal carotid artery – lateral view.

minutes after extubating, she was placed on a nonrebreather mask, with an improved GCS to 10/15 with reassuring arterial blood gas measurements. In addition, the patient showed a right-sided weakness with both upper and lower

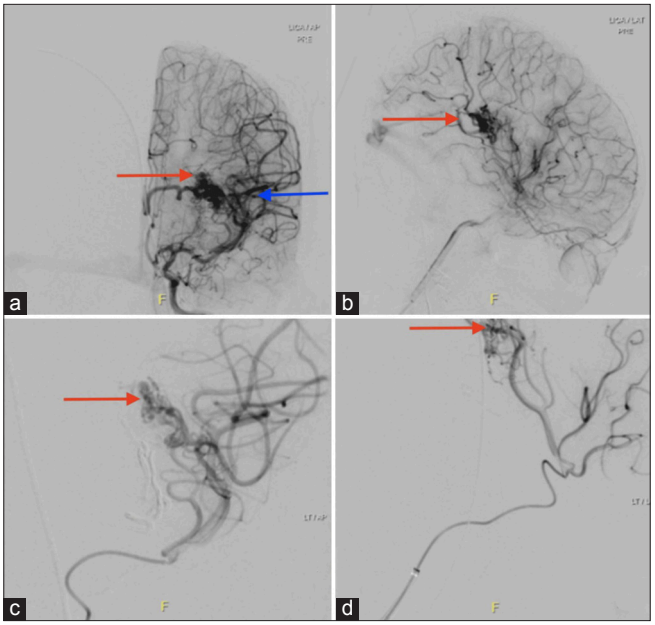

Figure 3: Angiogram of the internal carotid artery shows partial embolization of the left deep–seated cerebral arteriovenous malformation (AVM) (a) angiogram shows partial embolization of the AVM (red arrow). Embolization of the feeding arteries (blue arrow) was not possible as they could not be cannulated with the microcatheter due to being tiny and torturous; (b) partial embolization of the left AVM (red arrow) – lateral view; and (c and d) selective angiography of the internal carotid revealing left AVM (red arrow).

limbs exhibiting a power of 0/5 and an abnormal gaze in the patient's right eye. Two hours after extubating the patient, she had a drop in GCS and oxygen saturation to 49%. She was re-intubated, where she was weaned and extubated 5 days later.

Shortly after extubating, the patient began to show signs of paroxysmal sympathetic hypersensitivity [Tables 1 and 2][8] in addition to dilated sluggish pupils bilaterally at 5 mm and a raised ICP >30. She was administered lorazepam of 2mg, which lowered ICP to <20 and resolved the symptoms after 1 h. In addition, neurosurgery was consulted, which advised an urgent CT, which showed no abnormality, and pediatric neurology was consulted for an electroencephalogram, which showed no epileptiform discharges. After the recurrence of the attack to four times per day, the pediatric neurology team suspected PSH [Table 3] Carozza *et al.*,[8] and the patient was started on 150 mcg of clonidine every 6 h and 10 mg of propranolol every 8 h which led to improvement of symptoms and less frequent attacks.

In addition to PSH following extubating, the patient showed signs of CSW, which was suspected when serum sodium began to drop from 141 mmol/L to 130 mmol/L within 48 h, in addition to signs of polyuria. Random urine sodium was elevated at 179 mmol/L, elevated urine osmolality at 418 mosm/kg.H_2O and elevated plasma osmolality of 300 mosm/

Table 1: Laboratory values of a 13-year-old female patient with rupture of left arteriovenous malformation.

Lab results	Result	Normal values
WBC	16.4 10^3/uL	3.6–11.0 10^3/uL
HGB	11.5 g/dL	12.0–15.0 g/dL
Hematocrit	35.4%	36.0–46.0%
MCH	26.6 pg	27.0–32.0 pg
Neutrophil absolute	14.90 10^3/uL	2.00–7.00 10^3/uL
Lymphocytes absolute	0.90 10^3/uL	1.00–3.00 10^3/uL
Plasma osmolality		
Osmolality (Measured)		
First specimen	300 mOsm/ Kg.H_2O	275–295 mOsm/ Kg.H_2O
Second specimen	298 mOsm/ Kg.H_2O	
APPT	27.4 s	33.9–46.1 s

WBC: White Blood Cells, HGB: Hemoglobin, MCH: Mean corpuscular hemoglobin, PT: Prothrombin time, PTT: Partial thromboplastin time, APPT: Activated partial thromboplastin time

Table 2: The DLT for PSH assessment.[8]

DLT	Score
Clinical features occur simultaneously	1
Events are paroxysmal	1
Sympathetic over-reactivity to normally non-painful stimuli	0
Features persist >3 consecutive days	0
Features persist >2 weeks post-brain injury	0
Features persist despite treatment of alternative differential diagnosis	0
Medication administered to decrease sympathetic features	1
>2 episodes daily	1
Absence of parasympathetic features during episodes	1
Absence of other presumed cause of features	1
Absence of acquired brain injury	0

DLT: Diagnosis likelihood tool, PSH: Paroxysmal sympathetic hypersensitivity

kg.H_2O. She was administered IV hypertonic saline 3% at 5 mL/kg over 20 min and kept on IV normal saline 0.9% with the aim of maintaining serum sodium between 145 and 150 mmol/L. In addition, she received fludrocortisone 100 mcg in the morning and 50 mcg in the evening. Finally, strict intake and output monitoring was done to achieve a negative fluid balance, and so the patient's urine output started to improve to 2 mL/kg/h, and her sodium level was raised to 140 mmol/L. The patient was transferred from the surgical intensive care unit to the general ward after 2 weeks in stable condition. During her stay at the ward, her GCS was 15/15, and her pupils were 2 mm bilaterally with a brisk response. She also received nasogastric tube feeding and physiotherapy.

Table 3: The paroxysmal sympathetic hyperactivity assessment tools.

PSH assessment tool	Score	Severity
CFS score	0	Nil
	1–6	Mild
	7–12	Moderate
	≥13	Severe
PSH-AM score (CFS score+DLT score)	<8	Unlikely
	8–16	Possible
	≥17	Probable
Our patient's PSH-AM score (CFS score+DLT score)	22	Probable

DLT: Diagnosis likelihood tool, PSH: Paroxysmal sympathetic hypersensitivity, PSH-AM: Paroxysmal sympathetic hypersensitivity-assessment measure, CFS: Clinical features scale

The PSH-assessment measure (PSH-AM) tool has two components: the clinical features scale (CFS) that evaluates and scores six key features associated with PSH, and the diagnosis likelihood tool (DLT) that observes the patterns of such features to provide further evidence to diagnose PSH. The combined PSH-AM score utilizes both the CFS and DLT scores to estimate the probability of PSH occurring.[8]

DISCUSSION

AVMs within the pediatric population present a unique set of challenges due to their rarity, interesting presentation, and immediate care that they entail. The case of our 13-year-old patient, who was presented with a non-traumatic left intracerebral hemorrhage (ICH) secondary to an underlying AVM, sheds light on the acute and serious nature of these anomalies in children. This patient developed a sudden onset of symptoms, including unconsciousness, eye deviation, and vomiting, all of which are typical of acute pICH. She was managed with decompressive craniotomy followed by two sessions of embolization. Despite these interventions, she later developed PSH and CSW, further complicating her case. Although it is well established in the literature that traumatic brain injury may result in electrolyte imbalances, namely, that posterior pituitary damage leading to CSW syndrome or syndrome of inappropriate antidiuretic hormone (SIADH),[1] further studies are required to fully understand the correlation between traumatic brain injury, autonomic dysfunction, and sodium imbalance.[22] There is also a documented incidence of elevated catecholamine release and autonomic dysfunction in patients after traumatic brain injury.[14,22] However, the display of both complications is uncommon in pediatric AVM cases,

making this case rare. This case report sheds light on the vitality of adopting a multidisciplinary management approach for pediatric AVM cases.

PSH management post non-traumatic ICH in children

Prompt diagnosis and intervention are vital if PSH is suspected, as patients who develop PSH experience longer mechanical ventilation, longer hospital stays, and higher illness severity scores.[2,8] Neurocritical supportive care plays a key role in ameliorating PSH episodes, as it has been shown that lower autonomic function indices are associated with poorer outcomes in patients with AVM rupture.[8] Supportive measures involve reducing stimulation, especially those that trigger symptoms, managing hyperthermia in PSH using medications, and eliminating unnecessary pressure support, as hyperventilation serves as a trigger. If PSH develops, the management consists of abortive therapy and preventative therapy.[5]

Baclofen is used as a first-line agent as it manages hypertonicity diazepam and are second and third line, respectively. Other abortive drugs include morphine, dantrolene, clonidine, bromocriptine, and propranolol. Certain drugs may be used as preventative therapy, which includes sedatives such as oral beta blockers, clonidine, and gabapentin.[3,7,8] Allodynic hyperresponsiveness is pathognomonic for PSH due to increased neuronal excitation, producing an exaggerated response, so pain control is necessary.[8]

Gabapentin is effective for controlling allodynia in PSH. Pharmacotherapy must be tapered slowly to prevent complications such as dehydration, contractures. A close monitoring of vital signs and hydration status is important to prevent possible triggers of the condition. Responses to drugs vary between patients. Expert consultation is required for refractory cases of PSH, which is noted when 2 drugs do not help symptoms or if there is no improvement after 48 h, whichever comes first.[8] After the acute onset of PSH, adjustment of medications may be warranted.[3,7]

CSW management post non-traumatic ICH in children

A complication that our patient also endured was CSW syndrome. CSW is a type of hypovolemic hyponatremia usually within the first 48 h of initial CNS injury or post-operative treatment, including pICH. Early signs of hyponatremia may include headache, nausea, weakness, and confusion. Severe hyponatremia can lead to medical emergencies such as seizures, apnea, arrhythmias, and coma.[19] A thorough investigation should be made as the right diagnosis is vital before treatment, and treating CSW and SIADH differs using serum and urine sodium level, plasma and urine osmolarity, and creatinine with urea level can help aid in diagnosis.[29]

The overall management includes an IV bolus of 3% sodium chloride (2–5 mL/kg) over 20 min. If hyponatremia persists despite bolus dosing, start a continuous infusion at a rate of 0.08–0.25 mmol/kg/h. For adequate ICP control, serum sodium levels should be maintained at 145–155 mmol/L. Older patients will require strict fluid restriction. It is important to frequently monitor serum sodium levels to ensure the rise in serum sodium does not exceed 10 mmol/L in the first 24 h and 18 mmol/L in the first 48 h.[18,29,31] Using a dose error reduction software pump can help reduce the risks associated with sodium overcorrection.

Fludrocortisone has been shown to help and treat children with CWS. Isolated case reports have said that mineralocorticoids can be an effective additive, although the resolution of CSW with steroids has been shown to have a variable time course. Mannitol 20% has also been used to lower high ICP;[29] however, its effect can be brief. Regarding our patient, we started IV hypertonic saline 3% at 5 mL/kg over 20 min. We also used normal saline 0.9% to keep her sodium level between 145 and 150 mmol/L. We monitored intake and output to achieve a negative fluid balance of −100 mL in 24 h. Fludrocortisone started at 100 mcg in the morning and 50 mcg in the evening. Urine output improved to 2 mL/kg/h, and her sodium level rose to 140 mmol/L.

Modalities of AVM management

Management of brain AVMs (bAVMs) in the pediatric population involves various strategies that weigh the benefits and risks of intervention, particularly given the higher frequency of a hemorrhagic presentation in children compared to adults.[16] Treatment options are varied but fall under one of three main categories: microsurgical resection, stereotactic radiosurgery, and endovascular embolization.[12] The goal of AVM management is the total obliteration of the AVM to mitigate the risk of rupturing post treatment. Accomplishing this goal depends on the size of the AVM, its hemodynamic properties, patient stability, and the therapeutic modality chosen.[25]

Although endovascular embolization is most used adjunctively with surgery or radiosurgery, it can also be used as a stand-alone therapeutic modality. Embolization is unlikely to completely remove the AVM's, yet staged embolization remains a cornerstone in treating large AVMs. In a study including 1246 patients with bAVMs, total obliteration was reported to be 5%.[15] The rate of total obliteration using embolization alone varies drastically with the embolic agent used, with angiographic cure reported in several small case series. A study reported a total occlusion rate of 20% when using cyanoacrylate-based liquid embolic agents[36]. Other studies involving EVOH reported success rates of 51%, with some cases of bAVMs with simple angiographic features achieving up to 96% total

occlusion.[12,27,28,34] The complication rate post embolization could be as high as 26%.[33] However, almost none of the patients suffered from permanent neurological deficits or expired, with all reported complications resolving over time.

In acute settings of ruptured AVMs, partial or targeted embolization is crucial for management.[10,24] When the AVM has recently ruptured and the bleeding source is identifiable, partial embolization plays a role in stabilizing the patient before subsequent definitive management. Moreover, even though complete embolization is preferable, partial embolization can also be a therapeutic option for AVMs with complex and difficult anatomy.[11] Once a targeted endovascular approach is selected, it remains imperative to form a consequent plan to target the residual AVM.

Earlier studies and case series suggested that conservative management and observation are suitable approaches for non-emergent pediatric cases.[32] However, this has since been largely rejected except in select cases where further therapy would be ineffective or when the risk-benefit ratio heavily favors conservative management. Even in cases with high-grade or deep-seated AVMs, where interventions may carry significant risk, it is still advisable to pursue aggressive management as opposed to observation.[11,26] Despite the limited evidence regarding the ideal management of AVMs in the pediatric population, a multidisciplinary approach is generally considered to yield the best clinical outcomes.

CONCLUSION

We report a rare case of PSH and CSW syndrome following a non-traumatic ICH due to a rupture of left arteriovenous malformation in a previously healthy 13-year-old patient. By documenting and highlighting the presentation and subsequent complications, we aim to highlight the initial surgical modalities of AVM management and further emphasize the importance of prompt assessment to prevent complications in pediatric patients with ICH.

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