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Neurogenic pulmonary edema in a 13-year-old male child, following intracranial hemorrhage due to arteriovenous malformation: a case report

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ABSTRACT

Introduction: Arteriovenous malformations (AVMs) of the brain are mostly congenital vascular lesions that can present at any age. The risk of rupture and re-rupture persists until it is completely obliterated surgically. Neurogenic pulmonary edema (NPE), a rare but life-threatening event, is characterized by an acute onset of pulmonary edema subsequent to substantial central nervous system (CNS) insult.

Case presentation: We report case of 13-year-old male child who presented to our emergency room with abnormal body movement and decreased responsiveness 15 minutes prior to presentation. Past history suggested surgical intervention two years prior for right caudate hemorrhage with intraventricular extension (IVE), secondary to unknown cause, as computed tomography (CT) angiography was refused due to financial constraints. However, current imaging revealed AVM with IVE, likely a rebleed. The patient developed acute pulmonary edema, and chest radiography suggested the same. The patient was admitted and planned for decompressive craniectomy. Despite best efforts, we lost the patient on the second day of admission.

Clinical discussion: NPE due to intracranial hemorrhage is rare occurrence. The clinical presentations of NPE could be as early as first hours/minutes after injury or as late as 12–24 hours after neurological injury. Although it was identified over century ago, its sporadic and relatively unpredictable nature and lack of etiologic diagnostic and treatment modalities may be responsible for its poor recognition. If misdiagnosed or misinterpreted, it can be problematic for a successful outcome.

Conclusion: Prompt diagnosis and appropriate management of NPE are essential to maintain cardiopulmonary function and avoid fatal outcomes.

Keywords: Arteriovenous malformation, Case report, Intracranial hemorrhage, Neurogenic, Pulmonary edema

INTRODUCTION

Arteriovenous malformations (AVMs) of brains are primarily congenital vascular lesions that can appear at any age. They are comparatively uncommon in children, but they are said to have a higher rate of rupture than in adults, perhaps because most pediatric AVMs are only discovered after rupture.[1] The risk of rupture (2 to 10% per year) and re-rupture (2 to 4% per year) from an AVM persists until it is completely obliterated surgically.[1,2] The cornerstone of AVM treatment is to achieve complete angiographic obliteration with minimal neurological sequelae. Microsurgical resection remains the gold standard of treatment.[1,3]

Bleeding into the subarachnoid space due to AVM, cerebral aneurysm, and miscellaneous causes is termed subarachnoid hemorrhage (SAH).[4] AVMs occur in 1 per 100,000 children per year and are four times more common than aneurysms in patients younger than the age of 15 years.[5] Innumerable central nervous system (CNS) events, including intracranial hemorrhage (ICH), SAH, traumatic brain injury (TBI), seizure, meningitis, subdural hemorrhage (SDH), and spinal cord injury, have been associated with a syndrome called neurogenic pulmonary edema (NPE).[6] It is a relatively rare but life-threatening event, characterized by an acute onset of pulmonary edema(PE) due to a significant CNS insult.[7]

The pathophysiology is thought to be a surge of catecholamines due to hypothalamic stress caused by SAH that results in specific myocardial lesions and hydrostatic pressure injury to the pulmonary capillaries, causing PE.[8] There is sudden development of hypoxemic respiratory failure with radiographic findings of bilateral alveolar infiltrate that cannot be attributed to other causes of acute respiratory distress syndrome (ARDS).

The clinical presentations of NPE could be as early as the first hours/minutes after injury, as in our case, or a late form that could present 12–24 hours after neurological injury. There are usually non-specific signs of respiratory distress like dyspnea, tachypnea, hypoxia, pink frothy expectorations, and crepitation on auscultation. Unfortunately, there are no tools to make the specific diagnosis, and it is a diagnosis by exclusion.[4]

The incidence rate of NPE after SAH has been reported to range from 2% to 31%. However, at autopsy, the true prevalence was high, 78%.[6,9] Although NPE was identified over a century ago, it is still underappreciated, possibly because of its sporadic and relatively unpredictable nature and a lack of etiologic diagnostic and treatment modalities.[6]

Emergency physicians must have keen observation, as early recognition and immediate treatment can be lifesaving. If misdiagnosed or misinterpreted, it can be problematic for a successful outcome. There are no specific drug therapies for NPE; only treatment of the underlying CNS insult to reduce intracranial pressure (ICP) and supportive treatment with adequate ventilation to provide sufficient oxygenation are possible.[6]

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We report a case of a 13-year-old boy who presented with seizure and decreased responsiveness following a likely rebleed of AVM and subsequently developed NPE. The case highlights the importance of intense observation by the treating physician for diagnosis of NPE, as early recognition and immediate treatment is lifesaving, while delayed management has a fatal outcome.

This case report has been reported in line with the CARE criteria.[10]

Case Presentation

A 13-year-old boy presented to the ED in January 2025. The child was brought by his mother with a history of abnormal generalized body movement while having a haircut at a barber's shop 15 minutes prior to presentation, following which he had one episode of vomiting and then lost consciousness.

At presentation in the ED, the patient was responsive to pain and had involuntary intermittent abnormal flexion and extension movements of upper and lower limbs, on-and-off stiffening of the body, and clenching of the teeth. Patient vitals were recorded with a pulse rate (PR) of 76 beats per minute (bpm), blood pressure (BP) of 150/100 mm of Hg, respiratory rate (RR) of 30 per minute, and saturation (SpO_2) of 99% in room atmosphere. General random blood sugar (GRBS) was 110 mg/dl. He was categorized as triage I[11] and kept in the resuscitation bay; monitors were attached, and oxygen (O_2) was given via face mask at the rate of 6 liters per minute (L/min). Intravenous (IV) access was gained, and injectable Midazolam 2 miligrams (mg) stat was given, following which the abnormal body movement ceased. The anti-epileptic drug, injectable Levetiracetam 500 mg, was also initiated. Foley's catheterization was performed with an output of about 100 ml.

Upon inquiry with the mother, the seizure lasted for about four to five minutes with stiffening of the body, clenching of teeth, up rolling of eyes, and urinary incontinence, associated with one episode of non-projectile vomiting. Then he had loss of consciousness (LOC) for about five minutes. Thereafter, although the patient regained consciousness, he was only responsive to painful stimuli, making incomprehensible sounds, with involuntary limb movements. However, there was no history of tongue bite, frothing from the mouth, stool incontinence, altered sensorium, palpitations, diaphoresis, blurring of vision, fever, or neck rigidity. There was also no preceding history of trauma.

While digging up the past records, there was a history of bilateral frontal burr hole with external ventricular drain (EVD) insertion in the first week of February 2023 for right caudate hemorrhage with intraventricular extension and resultant obstructive hydrocephalus. Although rupture of AVM was suspected, Computed tomography (CT) angiography was not performed due to financial issues of the patient's family.

Old records also unfolded another visit to the emergency room in the last week of February, 2023, with complaints of non-projectile vomiting for four days, containing food particles, non-bilious, and not mixed with blood. Examination and investigation findings were suggestive of raised intracranial pressure (ICP) features. He was admitted for one week and was discharged after medical management. Following this event, the patient was on regular neurosurgical follow-up, and the last 22 months were quite uneventful.

In the current scenario, the patient was sedated immediately after presentation; hence, most of the neurological examinations could not be assessed at ED. Pupils, however, were unequal, 2 millimeters (mm) in the right and 3 mm in the left, with a sluggish reaction to light. The right plantar reflex was mute, and the left showed an extensor response (Babinski positive). GCS was eye-opening-1, verbal response-2, and motor response-5, summing to be 8/15. The patient was breathing spontaneously without any added sounds on chest auscultation. Cardiac and abdominal examinations were not significant.

Following symptomatic management, a non-contrast computed tomography (NCCT) head was performed, which showed acute intracerebral hematoma (63 milliliters [ml]) involving the right capsulothalamo-ganglionic region with ventricular extension, midline shift (10 mm), and ipsilateral uncal herniation with beginning hydrocephalus (Figure 1). Injectable Tranexamic acid 1 gram (gm), injectable Mannitol 20% (100 ml), and fluid NS were administered. Neurosurgical and pediatric consultations were sought, and patient relatives were counselled about the condition and need for operative management after the CT angiogram.

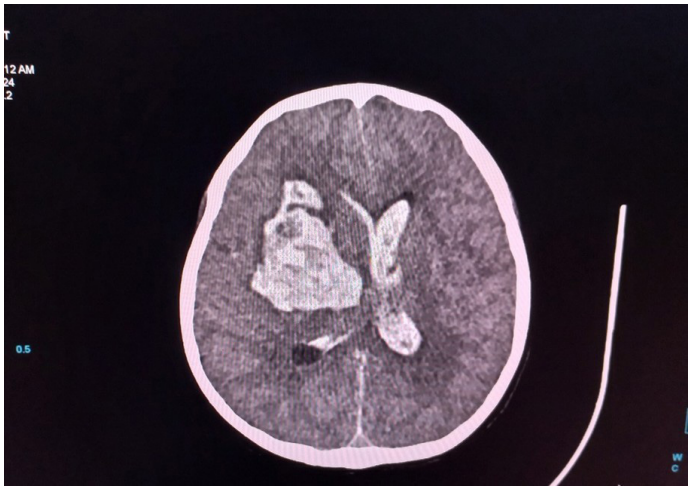

Figure 1: Acute intracerebral hematoma (63 ml) involving the right capsulothalamo-ganglionic region with ventricular extension, midline shift (10 mm) and ipsilateral uncal herniation, and beginning hydrocephalus

Informed consent for elective intubation was taken, following which the patient was successfully intubated with necessary drugs. Then the patient was kept on mechanical ventilation in volume control-assist control (VC AC) mode with a tidal volume of 200, positive end expiratory pressure (PEEP) of 8, 100% fraction of inspired oxygen (FiO_2), and RR of 30. The patient was clinically stable, and blood investigations were awaited as creatinine clearance was required for the CT angiogram.

After about an hour of continuous monitoring in the ED, suddenly, frothy blood-mixed secretions were visualized in the endotracheal tube, and saturation decreased to 53% and BP to 90/60 mm of Hg. Immediate ETT suctioning was done, and 35 ml of blood-mixed secretions were collected. Fluid resuscitation was initiated, but diffuse chest crepitations on auscultation suggested acute pulmonary edema, so injectable Noradrenaline was initiated as the vasopressor of choice.

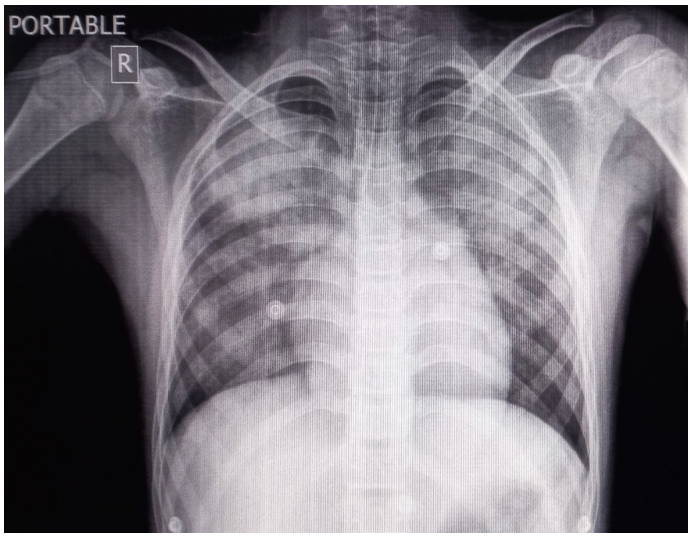

Figure 2: Portable chest X-ray with bilateral heterogenous opacities, suggestive of pulmonary edema, with ETT in situ, curved over the right bronchus

A portable chest X-ray at the ED was suggestive of pulmonary edema (Figure 2). Also, the tip of the endotracheal tube (ETT) was seen in the right bronchus; hence, it was corrected. After stabilization of vitals, the

patient was shifted for a CT angiogram, which showed AVM in the right caudate head having a feeder artery (lenticulostriate branch of the right middle cerebral artery) and draining vein (medullary vein) (Figure 3).

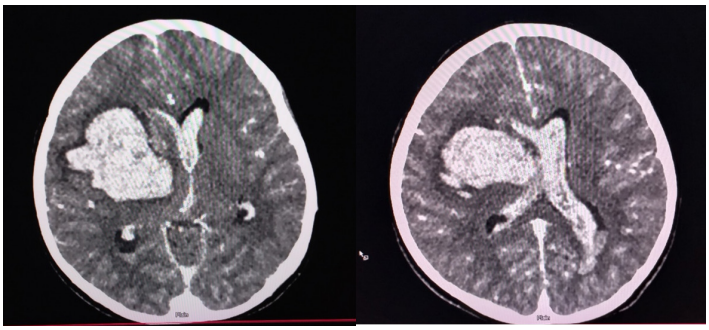

Figure 3: CT angiogram showing AVM in the right caudate head having a feeder artery (lenticulostriate branch of the right middle cerebral artery) and draining vein (medullary vein)

A chest CT was also done for pulmonary edema due to unknown etiology, which revealed diffuse ground glass opacities in bilateral lung fields with sparing of a few areas of anterior, middle, and posterior segments of the right upper lobe and the anterior and lingular segments of the left lobe, suggestive of pulmonary edema (Figure 4). The patient was then admitted to PICU with continuous monitoring by the neurosurgery and pediatric departments with a plan for emergency decompressive craniectomy. Parents were counselled about the condition of the patient, the need for operative management, the high risk of hemodynamic instability, complications, and poor prognosis.

In PICU, a diagnosis of exclusion of NPE was made, and the child was on volume-controlled continuous mandatory ventilation (VC-CMV) with three inotrope infusions for hypotension, fluids, sedatives, analgesics, and hypertonic saline infusion to lower the ICP. The patient had an episode of seizure, which was managed with anti-epileptics. Repeated saturation fluctuation occurred but was managed subsequently. Vigorous counselling of the parents was done about the poor prognosis.

On the next day, the patient was transferred to the operation theater (OT) early in the morning as well as in the daytime for the operative procedure. Unfortunately, due to continuous desaturation, the surgery had to be postponed both times. Counselling of the parents was done again as the risks weighed more. In the PICU, the ventilatory mode was changed to pressure control-assist control (PC AC) mode with PEEP of 12 cm H_2O and FiO_2 100%, and vitals were stabilized subsequently. Throughout the day, patient vitals were fluctuating, and in the night shift, the patient became unresponsive with no cardiac pulse; therefore, pediatric advanced resuscitation was initiated. As there were no signs of return of spontaneous circulation (ROSC) even after a significant duration of resuscitation, death was declared, and patient relatives were counselled.

DISCUSSION

A 13-year-old boy presented with a seizure and decreased responsiveness following a likely rebleed of AVM and subsequently developed NPE. Following symptomatic management, intubation, neurosurgical and pediatric consultation, and about an hour of continuous monitoring in the ED, the patient developed features suggestive of acute pulmonary edema. Despite high PEEP support, initiation of multiple inotrope administrations, and substitution of fluids, the patient's cardiopulmonary condition did not stabilize, and hence definitive treatment could not be carried out for the patient.

The strength in this case management was the acute, timely management of the symptoms of the patient and the appropriate diagnostic approach and management plan. Unfortunately, there are no tools to make the specific diagnosis of NPE, and it is a diagnosis by exclusion.

Quite similar to our case, Shahid et al.[12] in June 2024, reported a case of an 11-year-old whose CT revealed right-sided frontoparietal hemorrhage with contralateral midline shift, right-sided uncal herniation, and cerebral AVM. At the hospital, she was found to have bradycardia, hypotension, a GCS of 5/15, and bilateral coarse crackles with progressively rising oxygen dependency. CXR was suggestive of pulmonary edema. An emergent right-sided decompressive craniectomy with evacuation of the clot and excision of the AVM was performed. Having excluded the more common causes, a diagnosis of NPE due to SAH was made. Contrary to our scenario, in this case, over the next few days, the patient had a gradual improvement in her symptoms, allowing her ventilation to be tapered off and later discharged.

Fontes et al.[13] reported a few NPE cases in adults since 1990. Among fourteen reports (21 cases), the most frequent underlying factor was SAH (42.9%). Symptom onset occurred <4 hours after the neurologic event in 71.4% of cases, unlike in our case (within 2 hours). One third of the patients presented with pink, frothy sputum. Chest radiography showed bilateral diffuse infiltrates in 90.5% of cases. Supportive measures included oxygen support and vasoactive drugs. Recovery was usually very rapid: 52.4% of patients recovered in <72 hours. Almost 10% of patients died of NPE.

Another case of a 14-year-old boy was reported in 2008, who presented with cardiac failure associated with pulmonary edema following ICH due to AV fistula (AVF). Emergent craniotomy with intraventricular ICP monitoring was performed before significant hypoxia and hypotension developed, potentially reducing the risk of long-term adverse neurologic consequences in the patient.[8]

An exemplary rare case of a 35-year-old man with NPE induced by ICH was reported in 2015, treated by a neurosurgical approach because of an SAH (bore hole trepanation). A critical cardiopulmonary status developed during postoperative mechanical ventilation and catecholamine administration, NPE. After use of high positive end-expiratory pressure (PEEP), adaptation of initial dobutamine doses, initiation of norepinephrine administration, and substitution of fluids, the patient's cardiopulmonary condition stabilized.[7]

AVMs of the brain are relatively rare in the pediatric population but reportedly carry a higher rate of rupture than in adults, possibly due

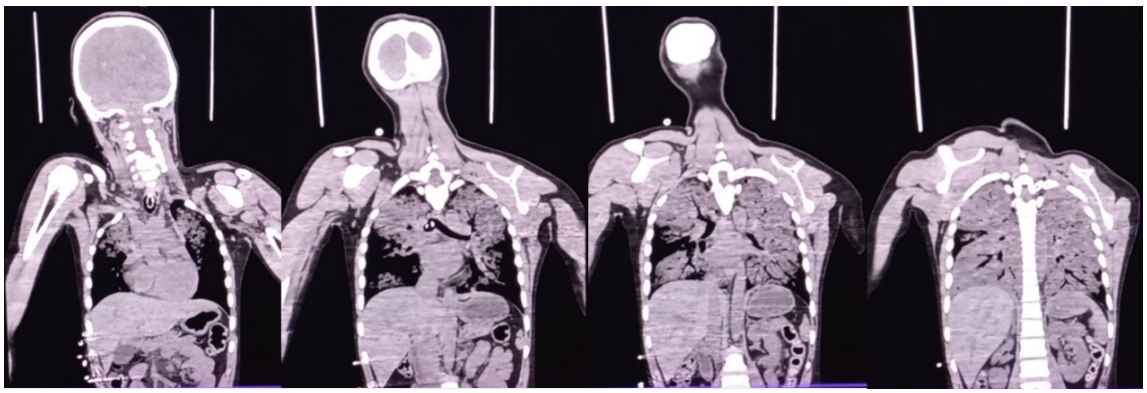

Figure 4: CT chest (anterior to posterior) revealing diffuse ground glass opacities in bilateral lung fields with sparing of few areas of anterior, middle, and posterior segments of right upper lobe and anterior and lingular segments of left lobe, suggestive of pulmonary edema

to the fact that most pediatric AVMs are only detected after rupture, as in our case scenario. AVM rupture was suspected in this patient in the first presentation. CT angiography could not be performed due to financial issues of the patient's family. Hence, the patient underwent a bilateral frontal burr hole with EVD insertion for right caudate hemorrhage with intraventricular extension with obstructive hydrocephalus of unknown cause. In the current scenario, a CT angiogram was done, which concluded the diagnosis of rupture of AVM, which could have a re-bleed. As suggested by evidence, the cornerstone of AVM treatment is to achieve complete angiographic obliteration, and microsurgical resection remains the gold standard, which could have been performed during the first admission itself if a diagnosis could have been made for AVM. Also, with correct operative management, the risk of re-rupture could have been eliminated.

NPE is rare but a lethal consequence of acute or subacute CNS insult, such as AVM rupture in this case. Sudden onset respiratory symptoms suggestive of pulmonary edema should strike a suspect of NPE in patients with a history of CNS insult. All necessary investigations to rule out other causes of acute pulmonary edema could not be performed, which could have led to a delay in diagnosis of NPE, as it is mostly a diagnosis of exclusion.

Unfortunately, there are no tools to make the specific diagnosis, and it is a diagnosis by exclusion. Essential investigations to exclude other causes of acute pulmonary edema should be executed to conclude a diagnosis of 'pure' NPE, as it is mostly a diagnosis of exclusion. Physicians should highly suspect NPE when neurologic patients suddenly become dyspneic.

It is necessary to go for aggressive treatment of patients with cardiac and/or respiratory complications following cerebral insult. Reasons are described as secondary brain injury caused by low cerebral blood flow and hypotension should be prevented, and mechanical cardiac dysfunction and pulmonary edema are highly treatable, with potentially good outcomes. Inotropic support, especially with dobutamine, can improve ventricular function. An active approach could greatly reduce the risk of long-term adverse neurologic consequences in the patient.[8] It is essentially important to provide optimal cardiopulmonary monitoring and differentiated administration of catecholamines and substitution of fluids to avoid further unfavorable consequences or even fatal outcomes.[7]

CONCLUSION

NPE is an underdiagnosed and underappreciated clinical entity despite decades of scientific experiments and case descriptions. It can occur after virtually any form of CNS insult. Emergency physicians must have a high suspicion of NPE when a patient develops sudden-onset hypoxemic respiratory failure following a catastrophic CNS event, which cannot be attributed to any other causes of ARDS, as in our case. With this keen observation, early recognition would be possible. An active approach following a definite diagnosis could greatly reduce the risk of adverse neurologic consequences in the patient and therefore could be lifesaving.

DECLARATION

Acknowledgement

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Author Contributions

PP, KHB developed concept. PP wrote the manuscript. SH, SG collected details of the case. RR, AB edited and drafted the manuscript

Conflict of interest

We have no conflicts of interest to disclose.

Ethical Approval

Not Applicable

Data Availability Statement

Not Applicable

Consent/Assent

Informed written consent was obtained from the patent's caregiver for the publication of the case report; including their photographs.

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None

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