

CASE REPORT

An early presentation of neurogenic pulmonary edema in acute subarachnoid hemorrhage

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Abstract

Subarachnoid hemorrhage (SAH) is a common occurrence among patients with altered mental status who arrive at the emergency department (ED), particularly for emergency physicians. However, the combination of neurogenic pulmonary edema (NPE) followed by SAH is generally considered a rare condition in the ED setting. Chest radiographs in these patients often reveal lung infiltrations, which are frequently misinterpreted by many emergency physicians as resulting from chest compression during cardiopulmonary resuscitation or aspiration due to altered mental status. This case report highlights a patient with SAH who developed NPE, with the aim to raise awareness among emergency department physicians about NPE and provide comprehensive insights regarding its management, as well as clinical, prognostic and diagnostic information.

Keywords

Subarachnoid hemorrhage; Neurogenic pulmonary edema; Emergency department

1. Introduction

Although subarachnoid hemorrhage (SAH) has a relatively low global incidence of 7.9 cases per 1 million, emergency physicians often encounter it frequently in patients with altered mental status. Conversely, emergency physicians commonly perceive neurogenic pulmonary edema (NPE) as a rare condition. However, despite its classification as a rare syndrome, the incidence of NPE in patients with traumatic brain injury (TBI) has been estimated to be as high as 20%. Furthermore, the severity of the injury seems to be correlated with an increased likelihood of developing NPE [1]. Multiple studies have provided insights into the prevalence of clinically manifest NPE in patients with SAH. One study found a prevalence of 31% in clinically diagnosed cases, while the true prevalence based on autopsy findings was reported to be as high as 78%. Another study corroborated these findings, reporting a prevalence of 31% during patient care and an even higher prevalence of 71% when assessed postmortem [2, 3]. In addition, several studies have reported varying prevalence rates of NPE in patients with SAH. One study documented a prevalence of 1.7% among 3521 patients admitted within three days of acute SAH from 1980–1983 [4], another study found a higher prevalence of 23% in 457 patients admitted within 7 days of acute SAH from 1987–1989 [5], a separate study reported a prevalence of 14% among 580 patients admitted between 1996 and 2002, although the time from acute SAH to admission was not specified [6], and more recently, a study involving 350 patients who underwent microsurgical or endovascular repair between 2014 and 2017 reported a prevalence of 4.6% [7]. These findings

suggest that NPE is most commonly observed between 3 and 7 days after the onset of acute SAH [8–10]. It is important to note that NPE can present in two distinct clinical forms. The first is the late form, which typically manifests 12–24 hours or several days after the initial neurological injury. The second is the early form, which develops within the first few hours or minutes following the onset of symptoms [8–10]. Although the causes of NPE can vary and include TBI such as epidural hemorrhage, traumatic subarachnoid hemorrhage or traumatic subdural hemorrhage, non-traumatic factors such as epilepsy, infarction or tumor may also contribute to the development of NPE [11].

In the emergency department, it is important to identify patients with early presentation NPE. Therefore, healthcare providers should remain vigilant for respiratory symptoms or warning signs in all patients with an acute neurological process. This is particularly crucial for individuals with SAH who require urgent management of their neurological condition. Unfortunately, respiratory symptoms can often be overlooked or mistakenly attributed to other causes. Notably, plain chest radiographs in patients with altered mental status frequently show lung infiltration, which has been reported to be present in 66.35% of patients with out-of-hospital cardiac arrest in a study [12]. Consequently, many emergency physicians may misattribute NPE to chest compression during cardiopulmonary resuscitation or aspiration resulting from altered mental status. Therefore, maintaining awareness of the potential for NPE and considering it in the differential diagnosis is essential for accurate management and appropriate care.

Prompt recognition and management of NPE are crucial due

to their significant impact on patient outcomes.

2. Case report

A 27-year-old female with no previous medical history presented to the emergency department (ED) in a comatose state. Upon arrival, her initial Glasgow Coma Scale (GCS) score was 3, and there was no evidence of physical trauma. The patient lacked a bilateral pupillary reflex, and both pupils were fixed at a size of 6 mm. According to her boyfriend, she was last observed in a normal state 3 and a half hours before the emergency medical service was contacted. Her boyfriend found her gasping for breath at that time and immediately dialed 911. When the emergency medical service arrived, the patient was unconscious and foaming from the mouth. Her boyfriend reported that she had not complained of a headache or suicidal ideation.

Upon arrival at the ED, her initial vital signs were: blood pressure, 109/70 mmHg; heart rate, 123 beats per minute; oxygen saturation, 75% on room air; and body temperature, 36.1 °C. We then performed endotracheal intubation immediately to secure airway patency. Arterial blood gas analysis performed immediately after intubation at a fraction of inspired oxygen (FiO_2) of 1.0 yielded the following results: pH of 7.33, the partial pressure of carbon dioxide (pCO_2) of 41.0 mmHg, the partial pressure of oxygen (pO_2) of 138 mmHg, and bicarbonate (HCO_3) level of 21.7 mmol/L. Chest plain radiograph demonstrated diffuse bilateral infiltrations (Fig. 1A). Initial laboratory tests revealed a lactate level of 6.5 mmol/L, a d-dimer level exceeding 20 $\mu\text{g/mL}$, a white blood cell count (WBC) of 9190/uL, a platelet count of 223,000/ μL , an activated partial thromboplastin time (aPTT) of 34.7 seconds, a prothrombin time international normalized ratio (PT INR) of 0.99, and a blood sugar level of 209 mg/dL. Other laboratory findings, including a urine drug screening test, were within normal ranges. A chest radiograph showed diffuse bilateral infiltrations and an electrocardiogram showed sinus tachycardia with a rate of 123 beats per minute. Initial laboratory findings did not reveal any evidence of heart failure or chronic kidney disease, as the patient's blood urea nitrogen (BUN)/creatinine (Cr) levels were 11.7/0.84 mg/dL, the B-type natriuretic peptide (BNP) test showed a value of 14.4 pg/mL, the CK-MB (creatinine kinase-MB) level was 1.87 ng/mL, the troponin T level was 0.025 ng/mL, and her electrocardiogram showed a normal sinus rhythm with a heart rate of 86 beats per minute.

A brain computed tomography (CT) scan revealed SAH, which was further confirmed through an angiogram showing rupture of an A-commissural aneurysm (Fig. 2). Lung setting view from the bottom of the brain CT angiogram revealed significant pulmonary edema compared to the chest plain radiograph (Fig. 1B). Unfortunately, the patient's condition deteriorated, leading to neurogenic shock and eventually, cardiac arrest. Despite resuscitation efforts, the patient's condition did not improve, and she passed away three hours after arriving at the emergency department.

3. Discussion

The exact pathophysiology of NPE remains incompletely understood; however, numerous studies indicate that a temporary central sympathetic discharge is likely the main trigger for the subsequent pulmonary pathology [10, 13–17]. An increase in intracranial pressure resulting from a brain injury activates the hypothalamus and medulla oblongata, which are linked to the sympathetic nervous system, leading to increased sympathetic activity, resulting in systemic and pulmonary vasoconstriction [14, 15].

Furthermore, the permeabilization of capillaries is another contributing factor to the development of pulmonary edema in NPE. This permeability is induced by the release of inflammatory mediators like cytokines and histamines, which are triggered by the brain injury, thereby increasing blood flow to the pulmonary circulation and manifesting as pulmonary edema [13, 15, 18–20].

As described earlier, NPE is present in 72% of SAH patients and can be difficult to diagnose when it is still in its mild stages [2, 3]. The predominant symptoms commonly experienced include dyspnea, while mild hemoptysis may occur in severe cases. Chest radiography typically reveals a normal heart size and bilateral alveolar opacity [11]. The severity of NPE is directly proportional to the extent of hemorrhage. A diagnosis of NPE primarily relies on clinical evaluation and involves the exclusion of other potential causes of pulmonary edema, such as aspiration pneumonia, heart failure, acute kidney failure or fluid overload. Imaging techniques such as bedside ultrasound, plain chest radiography and CT scans can provide valuable assistance in the diagnostic process [21].

The treatment for NPE primarily focuses on addressing the root neurological cause, such as coil embolization or surgery in the case of SAH, while concurrently providing respiratory support. Ventilator care is essential to NPE management in the emergency department, aiming to maintain sufficient oxygenation and ventilation while preventing further lung injury. Positive End-Expiratory Pressure (PEEP) is frequently used to reopen collapsed alveoli and enhance oxygenation. However, it's critical to use PEEP cautiously since high levels can cause barotrauma and worsen lung injury. In severe situations, extracorporeal membrane oxygenation (ECMO) may be required to support gas exchange and provide time for lung recovery. Close monitoring of fluid balance is also essential, as over-administration of fluids can aggravate pulmonary edema and increase the likelihood of complications like pneumonia and acute respiratory distress syndrome (ARDS) [18, 20, 22, 23].

In the emergency department, there are no definitive guidelines for hemodynamic support. Both hypovolemia and volume overload need careful monitoring, and appropriate fluid resuscitation should be administered accordingly. Additionally, the optimal vasopressor for neurological conditions leading to NPE remains uncertain. In patients with traumatic brain injury, phenylephrine, norepinephrine and vasopressin are commonly utilized vasopressors. However, the administration of dobutamine or milrinone is often suggested in the case of neurogenic stress cardiomyopathy [23].

Collectively, it is highly important for emergency physicians to be cognizant of NPE and its potential occurrence in patients

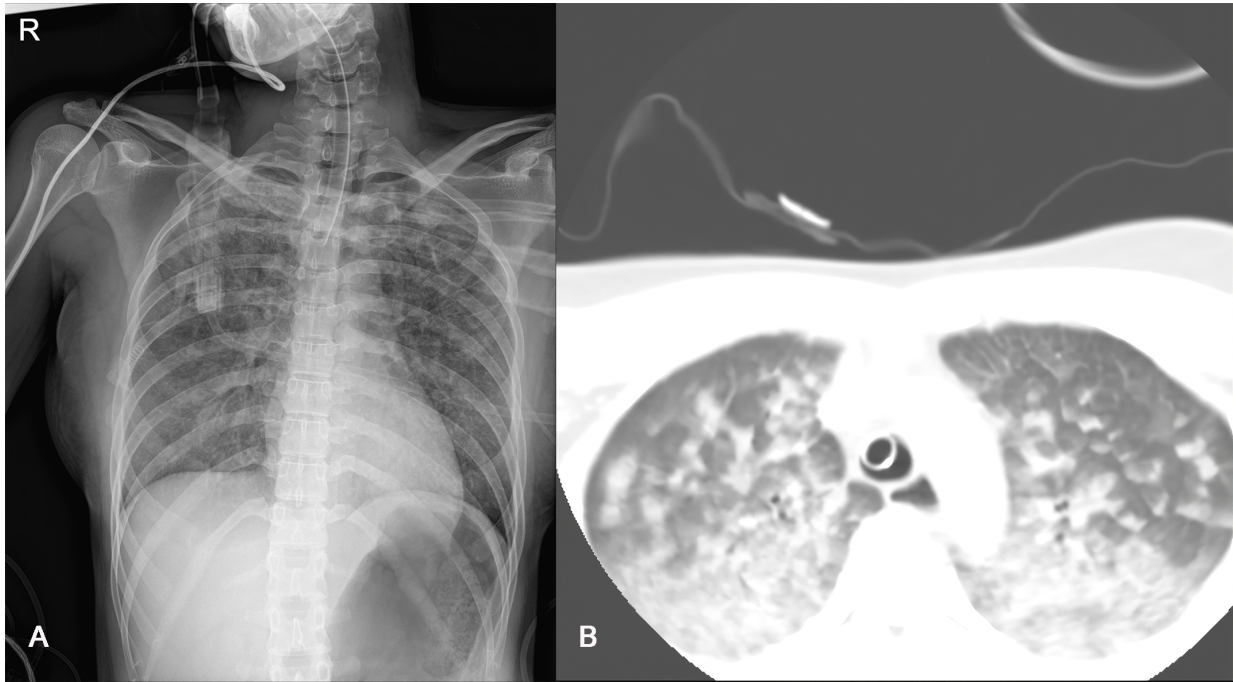


FIGURE 1. Radiologic results of neurogenic pulmonary edema. (A) Patient's chest radiography demonstrating diffuse bilateral infiltrations. (B) Lung setting view from the bottom of the brain CT angiogram revealed significant pulmonary edema compared to the chest plain radiograph. It is important to note that our institution's protocol for performing brain CT angiograms involves acquiring images from the aortic root level.



FIGURE 2. Axial slice of patient's brain CT scan demonstrating the presence of subarachnoid hemorrhage.

with SAH, given that it affects 72% of these patients. Adequate treatment and management strategies should be promptly administered to ensure optimal patient outcomes.

AVAILABILITY OF DATA AND MATERIALS

The data are contained within this article.

AUTHOR CONTRIBUTIONS

JP, HC and SL—designed the research study. HK, HO and SL—performed the research. HK, HO and SL—analyzed the data. HK and SL—wrote the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This case report was approved by the Institutional Review Board (IRB) of Korea University Ansan Hospital (IRB No. 2023AS0080) and waived the requirement for informed consent.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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