

Neurogenic pulmonary edema after subarachnoid hemorrhage: a case report

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Case Report

Keywords: Neurogenic pulmonary edema, subarachnoid hemorrhage, aneurysmal rupture

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Abstract

Background: Neurogenic pulmonary edema is a relatively rare and severe complication of acute central neurologic injury, particularly aneurysmal subarachnoid hemorrhage. It has been rather neglected and even misdiagnosed in clinical practice due to its non-specific manifestation.

Case presentation: We describe a 44-year-old woman suffering from the life-threatening pulmonary edema with a aneurysmal subarachnoid hemorrhage. She was successfully treated with positive end-expiratory pressure and coil embolization at acute stage. The patient was discharged without neurological deficits.

Conclusions: Early endovascular intervention and proper timely management of neurogenic pulmonary edema is critical for improving

outcomes after aneurysmal subarachnoid hemorrhage.

Keywords: Neurogenic pulmonary edema, subarachnoid hemorrhage, aneurysmal rupture.

Background

Neurogenic pulmonary edema (NPE) is an acute life-threatening complication of many central nervous system injuries, particularly aneurysmal subarachnoid hemorrhage (SAH). The incidence of NPE in aneurysmal SAH ranges from 2 to 42.9%[1-3]. It usually appears within minutes to hours after brain hemorrhagic injury and has been reported to be associated with a fatal clinical outcome. In a case series of 20 patients suffering from NPE, 19 of the patients died[4]. Hence, the presence of NPE after SAH is important to recognize due to its adversely impact on clinical outcome. We now describe a case of patient developed NPE caused by aneurysmal subarachnoid hemorrhage before coil embolization.

Case Presentation

A 44-year-old woman complaining of headache and nausea after 30-minute sudden loss of consciousness. On arrival at the emergency department, her vital signs were blood pressure 110/88 mmHg, heart rate

87 beats/min, respiratory rate 22 breaths/min, and oxygen saturation 94%. Her consciousness level was E4V5M6 on the Glasgow Coma Scale (GCS) and presented as Hunt-Hess grade 2. Pink frothy sputum was found when she spitting and a few bilateral crackles and rales were revealed by physical examination. We performed a computed tomography (CT) of her brain and chest immediately, a computed tomography angiography (CTA) was followed to assess for the etiology of her arrest. A diffuse Fischer grade III subarachnoid hemorrhage (Figure 1) and acute pulmonary edema (Figure 2) were observed on the CT scans. CTA suggested a left posterior communicating artery (PComA) aneurysm (Figure 3A-C).

Less than 30 minute after the CT scans, she became tachypnea with increasing pinkish foamy sputum and her both lungs were filling with crackles and rales. She was stay alert, but dysphoric and her oxygen saturation continuous dropping to 70%. Immediately, the patient was intubated and ventilated with positive end-expiratory pressure (PEEP). Her blood pressure fell to 82/63 mmHg during the intubation and increased to 110/70 mmHg 20 minute after the fluid administration. Approximately 26 hours later, after a continuous 8 cm H₂O PEEP, her vital signs were stabilized and oxygen saturation was 99% without mechanical ventilation.

A coil embolization was successfully performed 28 hours after the onset (Figure 3D), followed by a continuous lumbar drainage for one week. Her pulmonary edema gradual improved after the endovascular treatment (Figure 4). The patient was discharged without neurological deficits 15 days after admission.

Discussion And Conclusions

It has been previously reported that NPE can be considered an exotic form of ARDS[3]. Although the exact mechanisms responsible for the development of NPE are not entirely understood, the proposed etiology involves the release of vasoactive substances and a rapid massive sympathetic discharge after a sudden increase of intracranial pressure (ICP) by severe central nervous system insult[5, 6].

The clinical presentation of neurogenic NPE are non-specific and often include dyspnea, tachypnea, tachycardia, cyanosis, pink frothy sputum, and bilateral crackles and rales on auscultation. The chest radiograph or CT scan shows diffuse bilateral hyperintensive infiltrates. It has been described that there are two forms of NPE. The early form of NPE that develops within minutes to hours following neurologic injury is most common and a delayed form that develops 12 to 24 hours after the CNS insult[7]. Clinically, the possibility of developing NPE after aneurysmal SAH correlates with increasing the severity of clinical and radiographic presentation[8]. Patients with NPE have a higher incidence of vasospasm and mortality rate compared with patients without NPE [2, 9].

The key to the treatment of NPE is to control its underlying central neurologic insult in order to reduce the sympathetic discharge responsible for causing the lung injury [3]. Tracheal intubation, controlled ventilation with supplemental oxygen, and application of moderate PEEP are critical strategies to improve the pulmonary status in the acute stage[10]. In addition to these conventional treatments, it has been reported a woman with fulminant NPE following an aneurysmal SAH successfully treated by extracorporeal membrane oxygenation (ECMO) [11]. It has been reported that NPE may resolve in 48-72 hours with appropriate treatment, and patient's prognosis depends on the neurological condition[12]. In this case report, the patient was successfully treated with PEEP and early endovascular intervention, although NPE was developed rapidly and fatally after admission. Therefore, we believe that timely identification of NPE, early respiratory management and endovascular intervention at acute stage is essential to avoid poor clinical outcomes.

Abbreviations

NPE: neurogenic pulmonary edema; SAH: subarachnoid hemorrhage; GCS: Glasgow Coma Scale; CT: Glasgow Coma Scale; CTA: computed tomography angiography; PComA: posterior communicating artery; PEEP: positive end-expiratory pressure; ICP: intracranial pressure; ECMO: extracorporeal membrane oxygenation.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report.

Availability of data and material

All data generated during this study are included in this published article.

Competing interests

The authors declare no conflicts of interest in relation to this article.

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Authors' contributions

XXC collected the clinical data and drafted the manuscript. CL and JS contributed the conception of the manuscript. HJB and JS participated in the design and coordination of the study and helped revise the manuscript. XXC and MHC contributed equally to this work as co-first authors. All authors have read and approved the final version of the manuscript.

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Not applicable.

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Figures

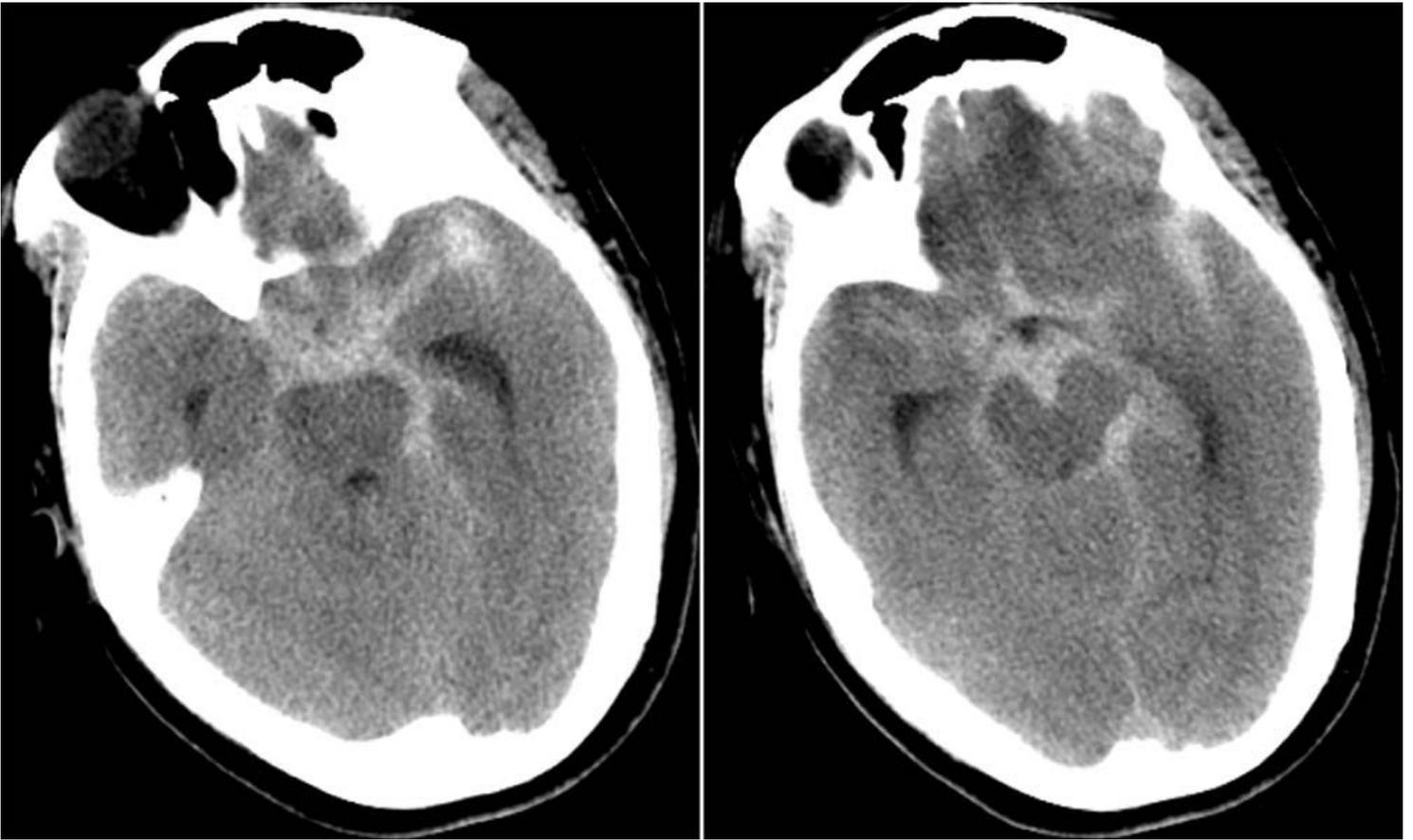


Figure 1

CT scan of brain showed diffuse subarachnoid hemorrhage.

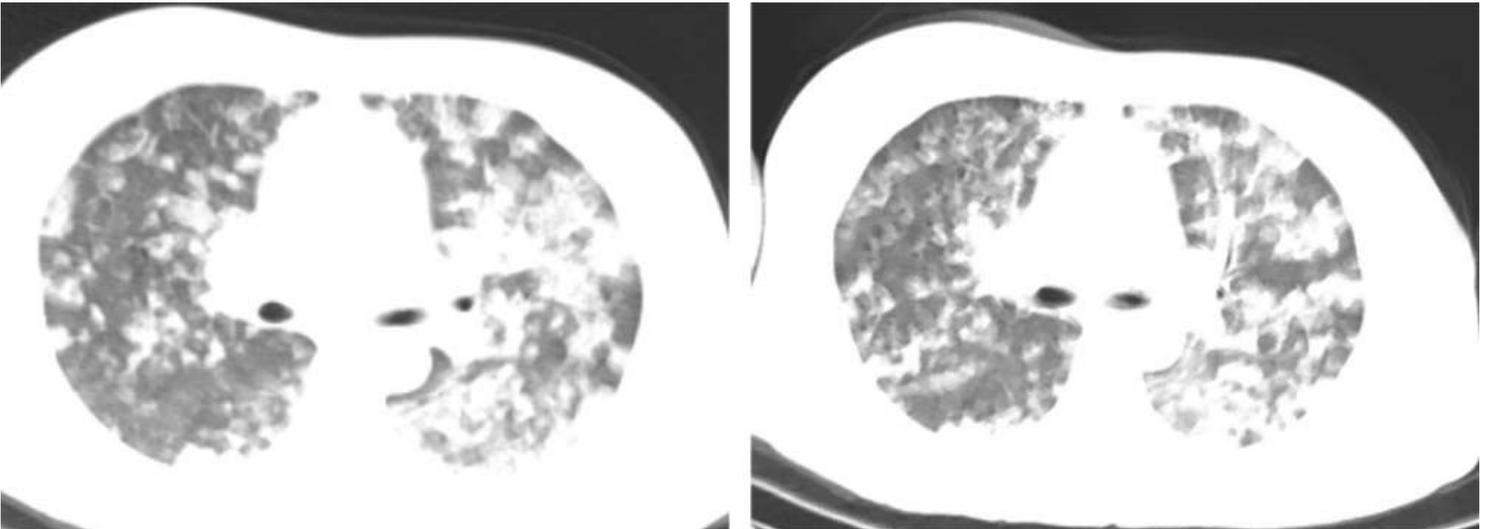


Figure 2

CT scan of chest showed diffuse bilateral hyperdense infiltrates in the lung indicating the presence of pulmonary edema.

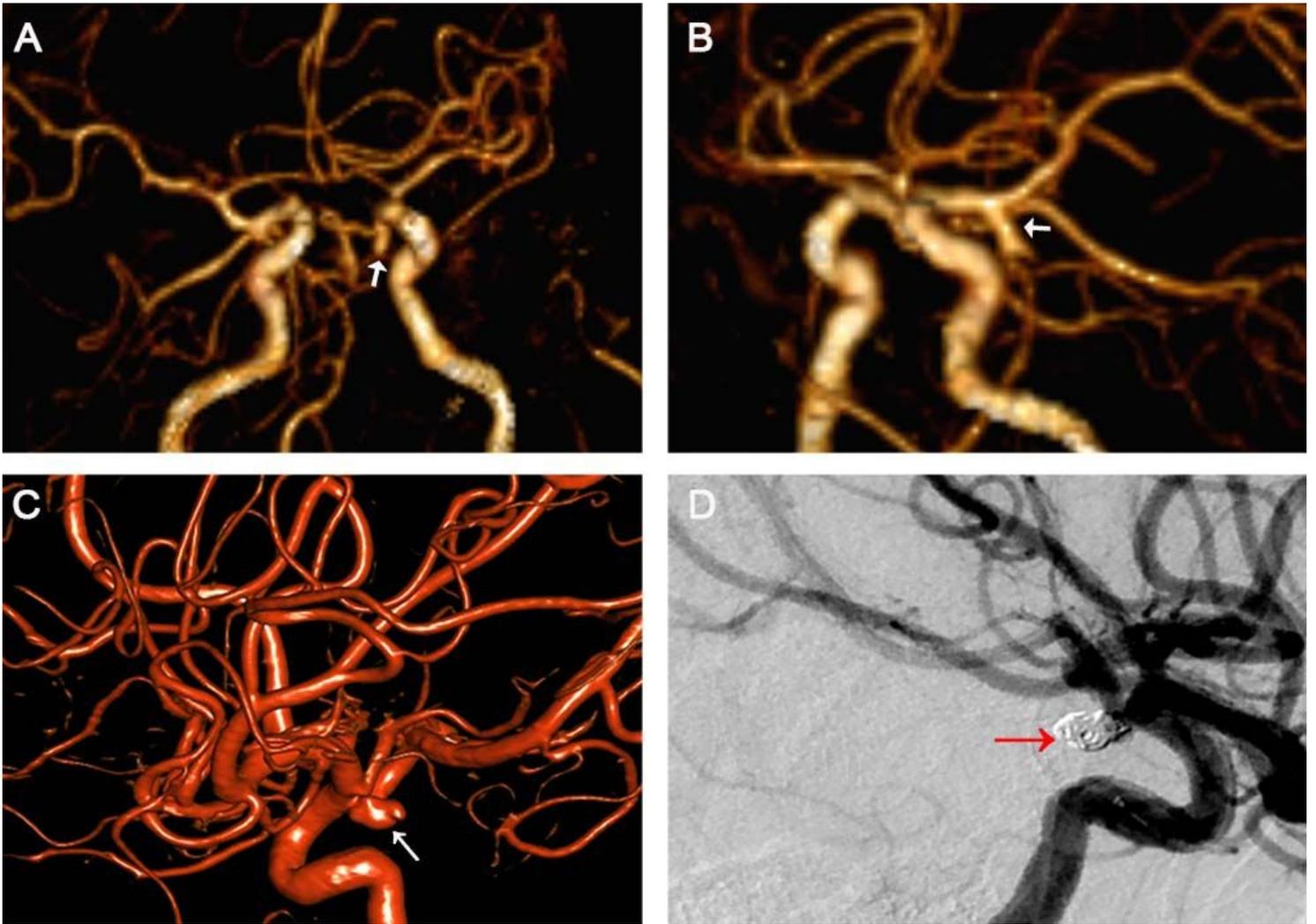


Figure 3

Cerebral angiography confirmed an aneurysm of left posterior communicating artery (A-C). The aneurysm was completely embolized by detachable coils (D).

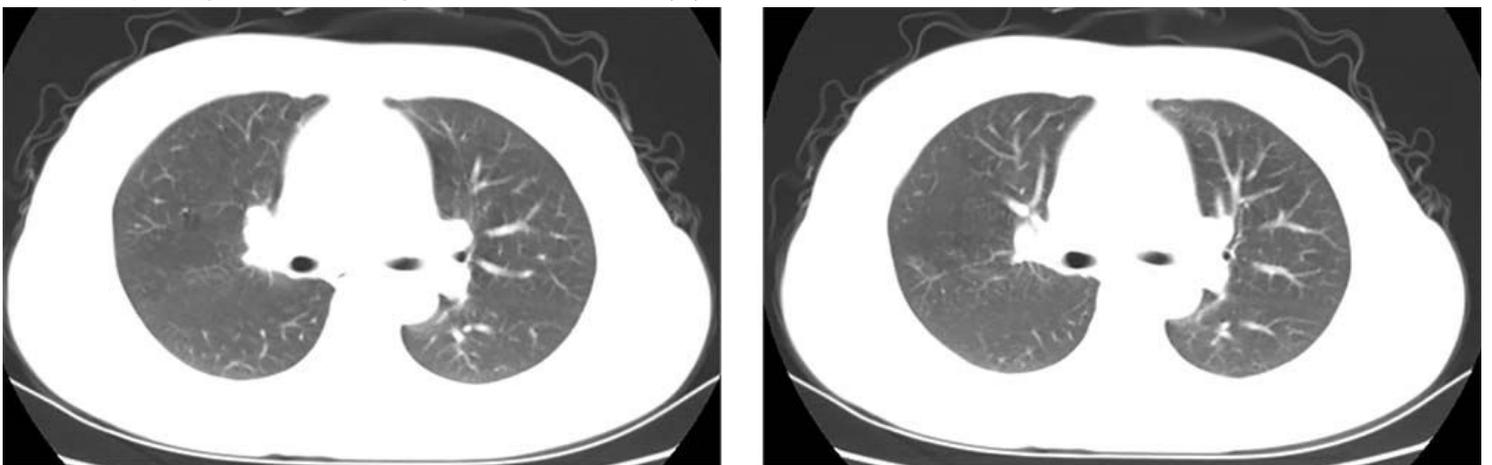


Figure 4

The respiratory condition of the patient was significantly improved 10 days after the endovascular treatment.

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