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Neurogenic pulmonary edema as a complication of epidural hemorrhage: A case report and review of the literature

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ABSTRACT

Pulmonary edema is accumulation of fluid in the lung interstitium and alveoli. Neurogenic pulmonary edema (NPE), a complication noted in some cases of head trauma such as subarachnoid hemorrhage (SAH) and intracerebral hemorrhage (ICH), is a life-threatening condition that requires urgent management in a timely fashion. Unfortunately, clear management protocols are currently unavailable. We present a case of a healthy young individual who presented with epidural hematoma after he had sustained a head injury. The patient then developed NPE which led to prolonged intubation and post-operative intensive care unit stay.

Keywords: Traumatic brain injury, epidural hemorrhage, neurogenic pulmonary edema, trauma

1. INTRODUCTION

Neurogenic pulmonary edema (NPE), a type of non-cardiogenic pulmonary edema, is a rare condition caused by acute nervous system injury such as seizures, traumatic brain injury and various forms of intracranial hemorrhage (Šedý et al., 2015). This rapidly developing syndrome with increased pulmonary interstitial and alveolar fluid usually occurs rapidly after the initial injury (Šedý et al., 2015). It can be classified as a type of acute respiratory distress syndrome (ARDS) and presents with abrupt onset of acute dyspnea, tachypnea and hypoxia in addition to bilateral crackles and rales on auscultation. On imaging, NPE can be appreciated as hyperdense infiltration bilaterally typical of ARDS. These common findings make NPE difficult to distinguish from other causes of ARDS; hence, it's mainly a diagnosis of exclusion (Šedý et al., 2015; Davison et al., 2012). Treatment efforts are supportive and are mainly aimed at decreasing intracranial pressure (ICP) and improving oxygenation with mechanical ventilation (MV). NPE as a complication of subarachnoid hemorrhage has a reported incidence of 2%-42.9% (Davison et al., 2012). However, there are very few previous reports of NPE as a major manifestation of an epidural hematoma (EDH); the reported

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outcomes were generally good (Hegde et al., 2017; Nakamori et al., 2018; Lee and Oluigbo, 2021).

Pathogenesis

The exact pathogenesis of NPE remains incompletely understood. However, elevated ICP is considered a key factor because it is usually associated with common neurological events (Šedý et al., 2008). Neuronal compression and ischemia following a sudden increase in ICP lead to a catecholamine surge as a result from the activityof the adrenergic system (Davison et al., 2012). The major catecholamines accounting for the pathogenesis of NPE are norepinephrine and neuropeptide Y, but the exact cascade of events remains unclear (Hegde et al., 2017). It is believed that the medulla oblongata plays a crucial role in stimulating the activity of the adrenergic system (Nakamori et al., 2018). Experimental models of bilateral lesions in medulla nuclei produced significant increase in the systemic and pulmonary pressure along with edema in the lungs. On the other hand, transection of the spinal cord at C7 and alpha-adrenergic blockade were shown to prevent the development of NPE, findings that emphasise the significant role of sympathetic activation (Davison et al., 2012). The sympathetic outflow source can be attributed to certain regions in the cerebrum. These regions include the hypothalamus and medulla, specifically the nuclei of the solitary tract, the area postrema, areas A1 and A5, and they are known collectively as the 'NPE trigger zones' (Davison et al., 2012; Ridenti, 2012; Agrawal et al., 2007; Baumann et al., 2007). Proposed theories for the progression of NPE include neuro-hemodynamic, neuro-cardiac, blast theory and pulmonary venule adrenergic hypersensitivity (PVAS) (Nakamori et al., 2018; Agrawal et al., 2007; Baumann et al., 2007).

2. CASE PRESENTATION

A medically free 28-year-old male presented to our emergency department complaining of a headache after an alleged history of falling down while horseback riding. Upon examination, he had a Glasgow Coma Scale (GCS) of 12/15 E3V4M5. He had right pupil dilatation and right-sided weakness. Imaging with computed tomography (CT) of the brain showed a right temporo-parietal EDH with 13 mm thickness (Figure 1). He was urgently transmitted to the operating theatre for an emergency craniotomy and evacuation of the EDH. The surgery was uneventful and lasted for 1.5 hours.

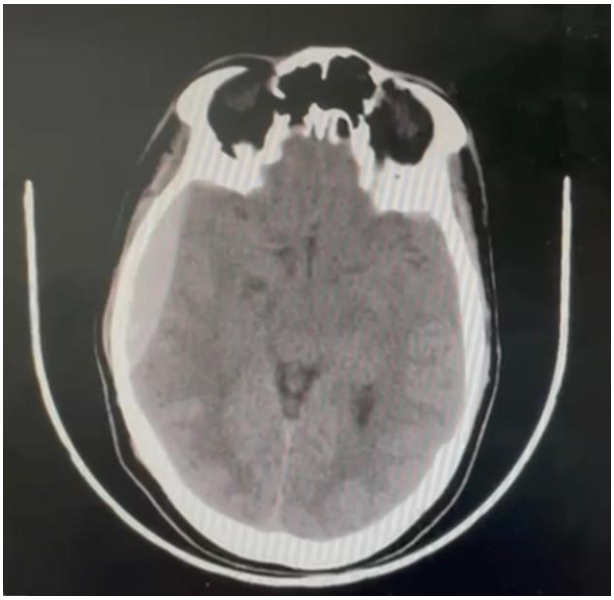
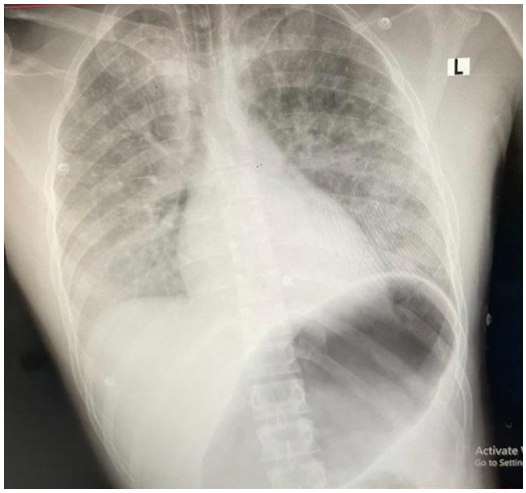


Figure 1 An axial view computed tomography scan without contrast of the brain revealing aright temporo-parietal epidural hematoma (13 mm thick).

Post-operatively, while the patient was being extubated, he went into respiratory distress, with his oxygen saturation dropping and copious amounts of clear, frothy secretions coming out of the endotracheal tube. The patient was kept on MV in the assisted control volume control (ACVC) mode on high settings (FiO2 100%, positive end expiratory pressure [PEEP 10]) and shifted to the surgical critical care unit. A bedside echocardiography was done and was normal. A chest X-ray was also done and showed non-homogenous infiltration of both lung fields, denoting pulmonary edema (Figure 2). The patient was kept on ventilatory support and started on diuretics in the form of daily intravenous furosemide 40 mg and his systolic blood pressure (SBP) was maintained at

> 100 mmHg by initially using levophed 10 ml/min. Twenty-four hours after surgery, the patient started showing signs of improvement in his oxygen saturation and his MV ACVC settings were de-escalated to FiO2 40% and PEEP 7, and the levophed dose was lowered to 5 mg/min. Serial chest X-ray showed improvement of the edema (Figure 3), so the MV ACVC settings were de-escalated further to FiO2 40% and PEEP 5, and levophed was stopped.



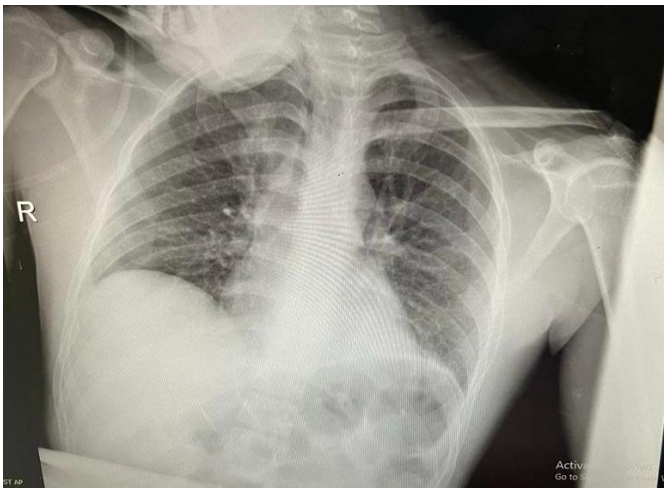


Figure 2 Chest X ray showing non-homogenous infiltration of both lung fields, denoting pulmonary edema; **Figure 3** Follow up chest X-ray showing marked improvement following pulmonary edema.

After 48 hours, the patient was extubated successfully and was shifted to the ward. He was kept under observation until his GCS improved to 15/15 and he was seen multiple times by a physiotherapist and dietician. A CT scan was done and it showed satisfactory evacuation of the EDH, with a resultant right posterior cerebral artery (PCA) territory infarction (Figure 4). The patient was discharged on phenytoin 100 mg capsules three times daily. He was seen a month later in the outpatient clinic and had resumed his normal daily activities, complaining only of a visual field defect secondary to the PCA infarction.

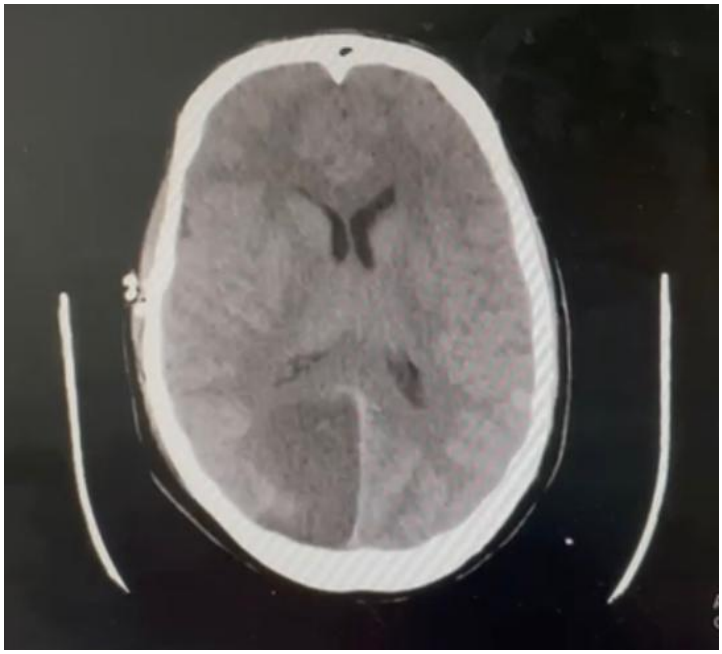


Figure 4 An axial view computed tomography scan without contrast of the brain revealing aright posterior cerebral artery infarction.

3. DISCUSSION

NPE is a life-threatening condition with high morbidity and mortality. In the literature, it has mainly been described after subarachnoid hemorrhage, with few reports of NPE after EDH (Hegde et al., 2017; Nakamori et al., 2018; Lee and Oluigbo, 2021). NPE has been classified into early and late onset (Šedý et al., 2015; Davison et al., 2012; Agrawal et al., 2007). Identifying and diagnosing NPE can be challenging, so clinical suspicion of NPE should be raised in the context of an acute insult to the brain associated with ARDS (Baumann et al., 2007).

In our case suspicion was raised because the patient developed ARDS in the context of head trauma despite having no history of cardiopulmonary disease. Our patient had a head injury associated with ARDS. Criteria for diagnosis have been suggested (Hegde et al., 2017). The cornerstone of managing NPE is to treat the causative central nervous system insult and cardiopulmonary augmentation and supportive care; there are no definitive therapeutic protocols (Šedý et al., 2015; Hegde et al., 2017). Consistent with the available data in the literature, our patient was kept on high ventilator settings post-operatively and was de-escalated as his general condition, chest X-rays and oxygen saturation improved. The treatment protocol of NPE is controversial, but the currently available management guidelines mainly recommend conservative measures in order to decrease ICP and improve oxygen saturation with MV (Davison et al., 2012).

4. CONCLUSION

NPE is considered as a type of pulmonary edema that is non-cardiac in nature. This rapidly developing syndrome of increased pulmonary interstitial and alveolar fluid usually occurs after an initial injury that elevates ICP and leads to a catecholamine surge due to the adrenergic system being activated. NPE is not a common manifestation of traumatic brain injury and it could lead to prolonged intubation, critical care unit admission and high morbidity and mortality. The outcomes of patients with NPE reported in the literature are favorable; however, as it is mainly a diagnosis of exclusion, it requires high physician awareness, especially in the context of traumatic brain injury. The cornerstone of the favorable outcomes consists of an immediate intervention to treat the underlying cause of NPE in an aim to minimize the rate of morbidity and mortality. The main management of NPE remains controversial due to the deficiency of available studies about NPE. Additional research and publications are needed to develop definitive management guidelines. A multidisciplinary approach of intensivists, anaesthetists and neurosurgeons is required to achieve good outcomes in a timely fashion.

Further Information (Conference Presentation)

This article was previously presented as an abstract at the 2022 Saudi Association of Neurological Surgery Annual Scientific conference on March 26, 2022.

Author's Contributions

Djilali Rezai: Main treating physician in the case. He was involved in writing the case presentations in the manuscript and reviewing the manuscript prior to submission.

Fahd AlAradi: Resident on call who first saw the patient and was involved in the treatment plan. He was involved in writing the case presentation.

Badr Hafiz: He was involved in collecting the data of the patient to prepare to write the case. He was also involved in writing the abstract, introduction, discussion, and conclusion.

Sara Murshid: She was involved in collecting the data of the patient to prepare to write the case. She was also involved in writing the abstract, introduction, discussion, and conclusion.

Mohammed Alamri: He was involved in writing the discussion and conclusion of the case.

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Conflicts of interest

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

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