Acta Scientific MEDICAL SCIENCES (ISSN: 2582-0931)

Volume 8 Issue 3 March 2024

Case Report

Neurogenic Pulmonary Edema Following Subarachnoid Hemorrhage: Case Report

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Received: October 30, 2023

Published: February 08, 2024

Abstract

Neurogenic pulmonary edema (NPO) is a rare and underdiagnosed condition that occurs within minutes to hours of central nervous system damage. The pathogenesis of NPO is not completely understood, systemic vasoconstriction, resulting from high levels of catecholamines, associated systemic inflammatory response, leads to increase capillary permeability and results in pulmonary edema. Hypoxemic respiratory failure following a catastrophic neurological event, which cannot be attributed to other causes is the main characteristic of NPO. Management is primarily based on controlling the triggering neurologic condition in order to reduce sympathetic discharge. We report the case of a female who developed hypoxemic respiratory distress following a subarachnoid hemorrhage. Clinical examination and radiological investigations led to the diagnosis of neurogenic pulmonary edema.

Keywords: Acute Pulmonary Edema; Brain Injury; Neurogenic Pulmonary Edema; Subarachnoid Hemorrhage; Sympathetic Surge

Abbreviation

NPE: Neurogenic Pulmonary Edema; SAH: Subarachnoid Hemorrhage; WFNS: World Federation of Neurologic Surgeons

Introduction

Neurogenic pulmonary edema (NPE) is characterized by the occurrence of pulmonary edema caused by acute central nervous system (CNS) insult. Although NPE was first described in 1908 [1], it has been reported in a wide range of neurological conditions, mainly subarachnoid hemorrhage, traumatic brain injury, epilepsy, meningitis and cerebral venous thrombosis. The sympathetic nervous system plays a crucial role in its pathogenesis, clinical presentation may give rise to confusion with other causes of respiratory distress [2,3]. We report a case of neurogenic pulmonary edema following subarachnoid hemorrhage.

Case Report

A 40 year old female with no comorbidities was admitted due of loss of consciousness. On admission, she had no fever, a Glasgow score of 13, no neurological deficit, blood pressure was 180/100 mmHg, a respiratory rate of 22 breaths/minute, a pulsed saturation of 82% on ambient air. Pulmonary examination revealed diffuse bilateral crackling rales.

Head Computed tomography (CT) showed spontaneous subarachnoid hemorrhage (SAH) with Fisher grade 2. The chest radiograph showed bilateral alveolar opacities and rather homogeneous airspace consolidative appearances supporting the diagnosis of pulmonary edema (Figure 2). Transthoracic echocardiography showed no cardiac failure, cerebral angiography confirm aneurysm rupture at the anterior communicating artery,

which was subsequently embolized. the Acute pulmonary edema resolved withing two days and the diagnosis of neurogenic pulmonary edema was confirmed in the absence of primary cardiac or pulmonary injury.

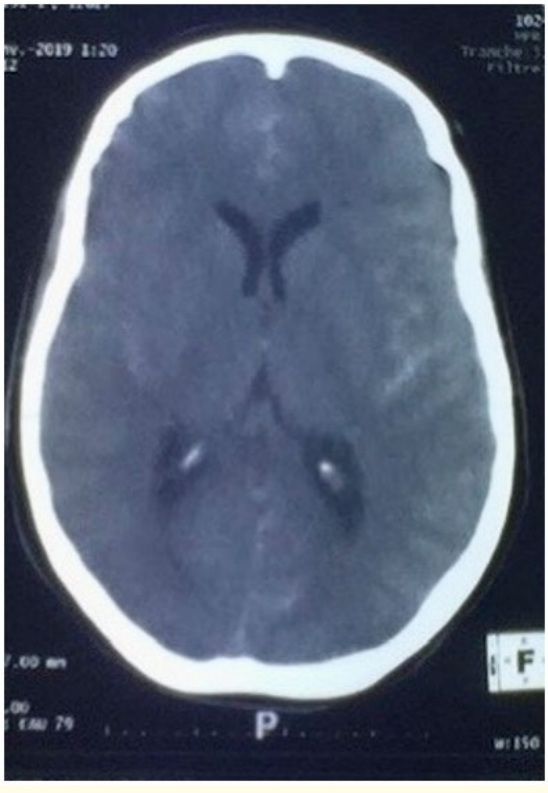

Figure 1: Head CT showing dense material in the subarachnoid space compatible with spontaneous subarachnoid hemorrhage.

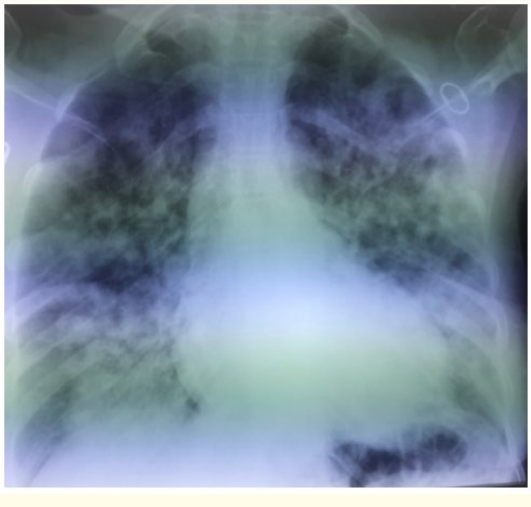

Figure 2: Chest radiograph showing bilateral alveolar opacities and rather homogeneous airspace consolidative appearances which supported the pulmonary edema.

Discussion

The pathogenesis of NPE is not fully understood. An increase in intracranial pressure leads to sympathetic overflow, resulting on systemic vasoconstriction and the mobilization of blood volume to the pulmonary circulation. This leads to increase in hydrostatic pressure and pulmonary capillary permeability [3,4]. Another mechanism involves a systemic inflammatory response secondary to endothelial damage, which increases capillary permeability and contributes to pulmonary edema [2].

Incidence of NPE is difficult to estimate, most publications refer to simple clinical cases. The main cause is subarachnoid hemorrhage (SAH), the incidence of NPE has been estimated to be up to 78% in cases of death from ruptured aneurysms. Poor prognosis is primarily due to the initial bleeding severity rather than respiratory failure [5]. Other causes reported was encephalitis particularly caused by enterovirus, traumatic brain injury, and epilepsy [6,7].

Diagnosis of NPE is often retrospective, based on the occurrence of acute pulmonary edema in the context of central nervous system insult and in the absence of other obvious causes [8], it has frequently been identified post mortem in patients who died suddenly without an apparent cause [8,9]. Diagnostic criteria have been proposed, which include the presence of acute pulmonary edema, bilateral radiological opacities, rapid resolution of opacities, acute brain injury responsible for increased intracranial pressure, absence of elevated left ventricular pressures, and the absence of another obvious cause responsible for respiratory distress [10,11]. The diagnosis of NPE is typically established by exclusion, with the main differential diagnosis including aspiration pneumonitis, community-acquired pneumonia, negative-pressure pulmonary edema, left ventricular failure and pulmonary contusion [12].

In our case, the clinical and radiological findings were consistent with neurogenic pulmonary edema. The patent airway makes the diagnosis of negative pressure pulmonary edema unlikely. Absence of left ventricular failure on transthoracic echocardiography rules out a cardiac origin. The rapid respiratory improvement after stabilizing the cerebral aneurysm without the use of diuretics along with bilateral pulmonary involvement, make an infectious origin unlikely. Furthermore, the absence of any history of traumatism rules out the hypothesis of pulmonary contusions.

Few studies have focused on identifying a specific treatment for NPE. Alpha-blocking agents have been used successfully

in a few documented cases [9], but their role in NPE requires further clarification through larger studies. Apart from providing ventilatory support, the treatment of NPE is primarily focused on managing the underlying neurological condition. This approach aim to reduce intracranial pressure thereby halting the sympathetic surge responsible for pulmonary edema. In our case, measures to control intracranial hypertension, maintain of optimal cerebral perfusion, prevent of vasospasm and, notably manage the ruptured cerebral aneurysm improved NPE [5,11].

Patients with NPE have a higher mortality rate, which ranges from 60 to 100%. The prognosis is generally poor, depending essentially on the severity of the initial brain injury rather than the presence of NPE [9]. In our case, the patient had a low grade of SAH (WFNS: II and Fisher 2) and the neurological and respiratory improvement was rapid after stabilizing the intracranial bleeding.

Conclusion

Despite several decades of scientific hindsight. Identifying and managing neurogenic pulmonary edema remains a challenge, the occurrence of an hypoxemic respiratory failure following severe neurological injury without evidence of any other systemic cause remains its main feature. We reported a case of an NPE following a low-grade subarachnoid hemorrhage that improved after stabilizing the underlying neurological condition.

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