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Neurogenic pulmonary edema in patients with acute severe brain injury: Pathophysiology and management

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SUMMARY

In the context of critical care, this article will offer a narrative assessment of the data pertaining to theories, diagnoses, and treatments for neurogenic pulmonary edoema (NPE). Although the precise mechanisms causing NPE are still unknown, putative mechanisms for the condition include increased catecholamine release, increased vagal tone, and increased capillary permeability. This is known as the "blast injury theory" because it causes pulmonary vasoconstriction as a result. The diagnosis entails spotting pulmonary edoema symptoms in the presence of a brain damage, and the most effective treatment methods seem to be those that aim to preserve a normal physiological condition. Any acquired lesion to the brain constitutes an acute brain injury (ABI), which is a major global cause of morbidity and mortality. Twenty to thirty percent of people with ABI get lung damage. Despite being a significant complication, neurogenic pulmonary edoema (NPE) is frequently underdiagnosed.

Keywords: Prognosis; Early evolution; COVID-19; Laboratory data

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INTRODUCTION

Long-term Acute brain injury (ABI) is a common complication of trauma around the world, though less frequently occurring conditions like meningitis, stroke, status epilepticus, subdural haemorrhage (SDH), subarachnoid haemorrhage (SAH), intra-parenchymal haemorrhage (IPH), and others have also been linked to brain injury. Neurogenic Pulmonary Edema is a specific consequence of ABI that is frequently overlooked (NPE). In general, NPE is thought to occur in 20–30% of people with ABI Neurogenic pulmonary edoema develops in about 15% of individuals with either Hunt and Hess grade III-V or Fisher grade III-IV subarachnoid haemorrhage (SAH). In addition to describing the potential pathophysiologic mechanisms, this review will also discuss how to treat pulmonary problems in NPE patients.

PATIENTS AND METHODS

To A wealth of knowledge was gained from the early observations in animal experiments using noxious stimulation of the Central Nervous System (CNS) to study its effects on the cardio-pulmonary system. It was observed that lesions of the CNS produced in this way resulted in an elevation of pulmonary and systemic arterial pressures. Moreover, bilateral upper thoracic sympathetectomy or even total lung denervation did not prevent the elevation of these pressures. It was thus concluded that severe peripheral vasoconstriction induced by catecholamines released as a consequence of CNS injury results in severe systemic hypertension causing strain on the Left Ventricle (LV), similarly seen in Takotsubo cardiomyopathy .This leads to secondary LV dysfunction causing elevation of left atrial and pulmonary venous pressures, and subsequently pulmonary edema. Pulmonary edema has been seen as the sole presentation in patients with pheochromocytoma, presumably from the catecholamine surge [1-5].

Pulmonary venoconstriction: Maron MB and Dawson CA showed that in an experimental model with increased cerebrospinal fluid pressure in dogs caused catecholamine induced pulmonary venoconstriction in a denervated lobe of the lung. Indirect observations in humans using initial alveolar edema fluid to plasma protein concentration ratio in patients without heart failure or volume overload points towards a hydrostatic mechanism for the development of NPE. Smith WS and Matthay MA concluded either pulmonary venoconstriction or transient elevation in left-sided cardiovascular pressures as the contributing causes to the development of human neurogenic pulmonary edema.

CONCLUSION

In patients with ABI, NPE is a prevalent cause of lung damage and should be taken into account with other frequent causes such aspiration pneumonia, pulmonary trauma, and pulmonary embolism. NPE development is linked to worse neurologic outcomes and higher mortality. Acute CNS insults that cause catecholamine surge are the main method by which NPE is caused. Other processes include left ventricular strain, Takotsubo cardiomyopathy, shifts in blood volume from the periphery to the pulmonary circuit, and pulmonary venoconstriction. The existence of respiratory symptoms and related imaging results in the presence of a neurologic insult are necessary for the diagnosis. Euvolemia, normocapnia, and normoxemia should all be maintained as a primary goal of management measures. PEEP's impact on ICP calls for

CONFLICT OF INTEREST

The authors declare no competing interests.

All authors declare that the material has not been published elsewhere, or has not been submitted to another publisher.

DATA AVAILABILITY

Authors declare that all related data are available concerning researchers by the corresponding author's email.

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