Case Report

Neurogenic Pulmonary Edema in Traumatic Brain Injury

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Abstract

A 29-year-old male admitted with severe traumatic brain injury following a road traffic accident was sedated and ventilated uneventfully for 72 h. On the fourth posttrauma day, after stopping sedation to assess readiness for extubation, he developed sudden onset desaturation; arterial blood gas showed severe diffusion defect with very low PaO_2/FiO_2 ratio following an episode of generalized tonic–clonic seizure. The differential diagnoses and further management are discussed.

Keywords: Neurogenic pulmonary edema, seizure, severe traumatic brain injury

Introduction

In a patient with severe traumatic brain injury (TBI) complicated by immune thrombocytopenia (ITP) receiving platelet transfusions, on treatment with massive doses of steroids, identifying the cause for an acute respiratory insufficiency is challenging task even to this day. Common differential diagnoses would include transfusion-related acute lung injury (TRALI) and acute respiratory distress syndrome (ARDS). Neurogenic pulmonary edema (NPE) will usually be included in the list of differentials if the respiratory distress occurs immediately following the primary brain injury. The report stresses the importance of considering delayed onset NPE in the list of differentials when dealing with any acute respiratory decompensation that follows an acute central nervous system (CNS) event (such as seizures) occurring days after primary brain injury.

Case Report

A 29-year-old male was admitted with severe TBI sustained in a road traffic accident with a Glasgow Coma Scale score of 7/15 (E1 V2 M4) bilaterally equal and reacting pupils and no lateralizing deficits. Computed tomography brain at admission [Figure 1] showed hemorrhagic contusions in the left thalamus with intraventricular hemorrhage and acute hydrocephalus. He was planned for an emergency ventriculostomy, which was deferred as the hemogram revealed a thrombocytopenic state (platelet count 12,000/mm^3). A diagnosis of ITP was made in consultation with the hematologists as the thrombocytopenia was refractory to platelet transfusions. He was treated with high-dose steroids (dexamethasone 40 mg twice daily) following which there was an improvement in his platelet counts. Sedation and ventilation were continued uneventfully for a period of 72 h in view of the underlying brain injury following which it was stopped to assess his sensorium. He had one episode of generalized tonic–clonic seizure about 10 h following cessation of sedation and the same was terminated with a stat dose of midazolam. However, immediately following this episode, he developed sudden onset desaturation with copious pink frothy secretions from the endotracheal tube, and he required 100% oxygen to maintain an oxygen saturation of 88%–92%. The possible differential diagnoses considered were pulmonary edema, TRALI, and ARDS. Bedside two-dimensional echocardiogram showed normal cardiac contractility ruling out a cardiac etiology. He was empirically started on injection meropenem after sending routine blood, urine, and endotracheal aspirate cultures which was later discontinued once the cultures were reported sterile. TRALI was ruled out as he had not received any blood product transfusion in the preceding 24 h. Chest X-ray (CXR) revealed bilateral perihilar infiltrates [Figure 2] with a classical bat wing appearance and arterial blood gas (ABG) showed increased alveolar-arterial oxygen gradient ([A-a] O_2 = 590 mmHg) and

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a low PaO_2/FiO_2 (PF) ratio (63.5). The possibility of NPE was considered, and he was ventilated with 100% oxygen. He was also administered morphine, diuretics, and positive end-expiratory pressure (PEEP). His clinical condition gradually improved over the next 72 h as evidenced by an improved oxygenation on the ABG and clearing of the infiltrates on the CXR [Figure 3]. However, he required a tracheostomy in view of his persistent low sensorium for long-term airway protection.

Discussion

NPE is best described as rapid onset desaturation, tachypnea, and decreased PF ratio that occurs following an acute CNS injury.[1] It is often associated with aneurysmal subarachnoid hemorrhage, TBI, seizures, and stroke.[2-8] The entity often remains under diagnosed or undiagnosed in routine clinical practice.[2] Although the condition was described more than four decades ago, the exact incidence, pathophysiology, and the criteria for diagnosis remain unclear even today. The incidence of this condition ranges from 8% to 50% across reports.[3,5] NPE in TBI is reported to occur immediately following primary brain injury. Therefore, it often takes a back seat in the list of differential diagnoses of an acute respiratory insufficiency occurring later in the course of treatment. It has to be reemphasized that NPE should be considered in any rapid respiratory decompensation that follows an acute CNS injury.

The exact pathophysiology of this condition remains unclear although any mechanisms have been proposed. The most accepted theory is "The Blast theory" described by Theodore and Robin in 1976 which considers an intense vasoconstriction secondary to a massive surge of sympathetic amines that occurs following an acute CNS injury such as a seizure or increased intracranial pressure to be the cause for the fluid accumulation in the lungs due to altered hydrostatic pressure gradient across the alveolar capillary membrane and increased pulmonary vascular protein permeability.[9-11] Other theories include neurocardiac pulmonary edema, neurohemodynamic NPE, and pulmonary venule adrenergic hypersensitivity. These theories stress the role on catecholamines in the causation of NPE acting directly on the pulmonary vasculature or indirectly by causing a myocardial dysfunction.[12]

Two distinct forms of NPE have been described in literature. The more common early form which is characterized by an abrupt onset respiratory distress immediately following a CNS injury and a less common delayed form, in which the onset of respiratory distress is delayed by 12–24 h.[12] Appropriate management of this condition lies in the early recognition and timely institution of mechanical ventilation with PEEP, morphine for pulmonary vasodilatation, and guarded use of diuretics, especially in a setting of TBI.

Conclusion

NPE, an underdiagnosed entity, should be considered in any patient with a rapid desaturation and a low PF ratio that follows an acute CNS injury such as a seizure or increased intracranial pressure. When presenting late after a severe TBI in a patient on mechanical ventilation, infectious and cardiac causes cause a considerable amount of confusion in diagnosing this entity.

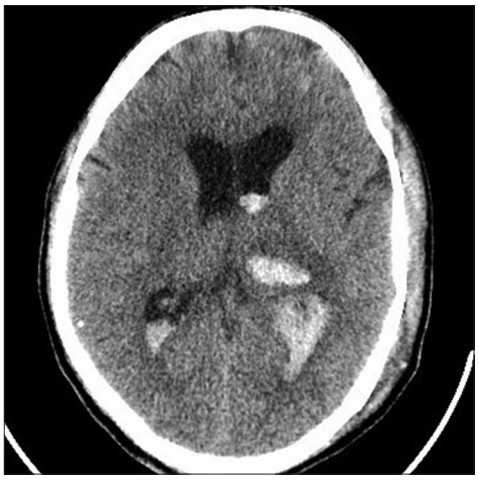

Figure 1: Computed tomography brain at admission showing thalamic and intraventricular bleed with hydrocephalus

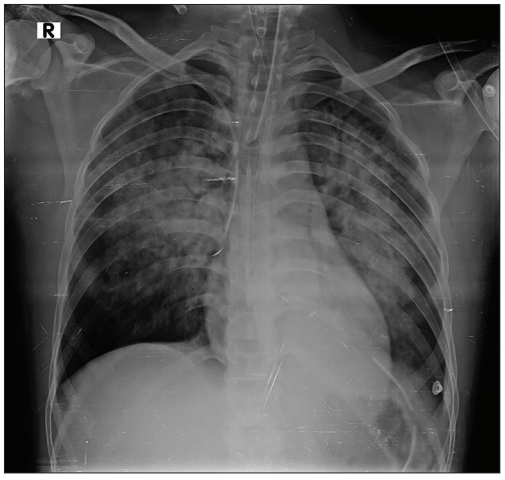

Figure 2: Pulmonary edema with bilateral perihilar infiltrates and a normal cardiac shadow

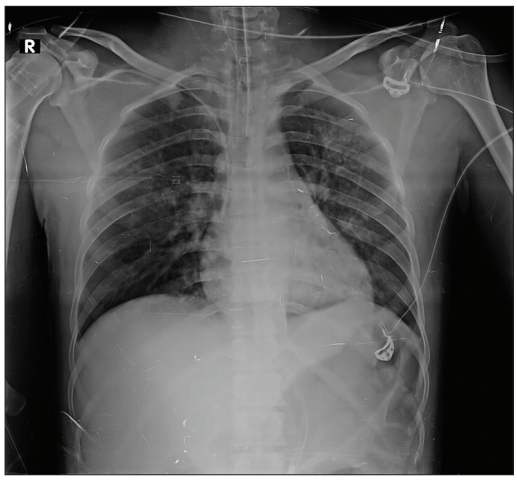

Figure 3: Resolved pulmonary edema after a period of 72 h

Early diagnosis and institution of appropriate therapy may improve patient outcome.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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