

A case with neurogenic pulmonary edema

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Abstract

Fibrolamellar hepatocellular carcinoma (FL-HCC) is an uncommon histological subtype of Hepatocellular carcinoma, which seldom invades the gall-bladder (GB). Here we report a case of FL-HCC invading the GB, that resembled gall bladder carcinoma invading the liver on both US and CT. A central calcification and/or central stellate scar that are clue to the diagnosis of FL-HCC were absent.

Introduction

Neurogenic pulmonary edema (NPE) is a relatively rare and underdiagnosed clinical entity which is an increase in pulmonary interstitial and alveolar fluid. NPE develops within a few hours after a neurologic insult including spinal cord injury, subarachnoid hemorrhage (SAH), traumatic brain injury (TBI), intracranial hemorrhage, status epilepticus, meningitis, and subdural hemorrhage, have been associated with this syndrome [1-6]. We aimed to present a patient who developed NPE following subarachnoid haemorrhage.

Case Presentation

A 33-year-old man with no history of any significant past medical illness, admitted to the emergency room sudden onset fainting and loss of consciousness. CT scan of the head revealed the massive subarachnoid blood which consistent with subarachnoid haemorrhage (Figure 1A and 1B).

Shortly after admission, respiratory failure was developed with tachypnea and tachycardia. He was intubated and hemorrhagic pulmonary fluid was aspirated from endotracheal tube. Chest roentgenography showed the nonspecific, bilateral, rather

homogeneous airspace consolidative appearances with an apical predominance which supported the non-cardiogenic pulmonary edema (Figure 2).

Thoracic CT scans demonstrated the bilaterally diffuse alveolar edema which consistent with neurogenic pulmonary edema (Figures 3 and 4).

The pulmonary edema recovered after 24 hours of IV fluid and furosemid treatment (Figure 5).

Discussion

NPE is a potential early contributor to the pulmonary dysfunction. It develops within a few hours and caused by a myriad of CNS insults that raises Intracranial Pressure(ICP). Prevailing view is that a rapid elevation of ICP leads to neuronal compression, ischemia or damage; followed by an autonomic response. Sympathetic nervous system activation leads to release of catecholamines that results in cardiopulmonary dysfunction. The cellular mechanisms that cause capillary leakage are also not well understood. Modifications in neurovegetative pathways are probably the cause of sudden, significant increases in microvascular pressure in the lungs, particularly in the pulmonary venules. This leads to reduced venous outflow, which in turn causes pulmonary capillary and arterial hypertension [7]. Sudden onset of respiratory distress is a chief feature of NPE. Due to lack of treatment modalities, management of NPE consist of treating the underlying neurologic condition. Neurogenic pulmonary edema is described clinically as

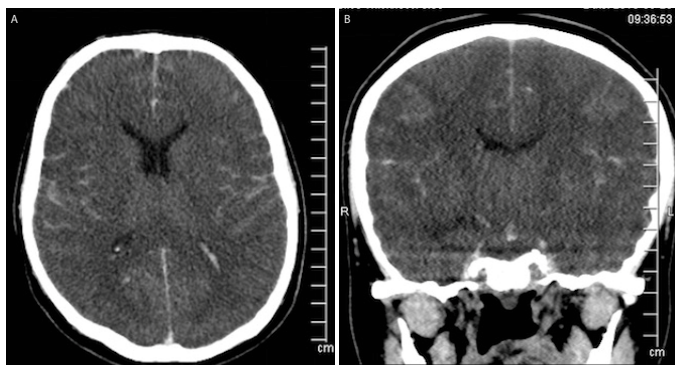


Figure 1. CT scan of the head showing the massive subarachnoid blood which consistent with subarachnoid haemorrhage

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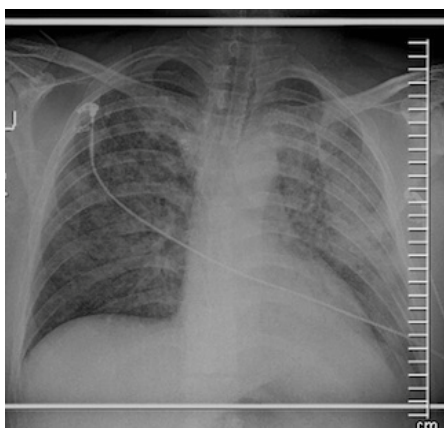


Figure 2. Chest roentgenography showing the nonspecific, bilateral, rather homogeneous airspace consolidative appearances with an apical predominance which supported the non-cardiogenic pulmonary edema

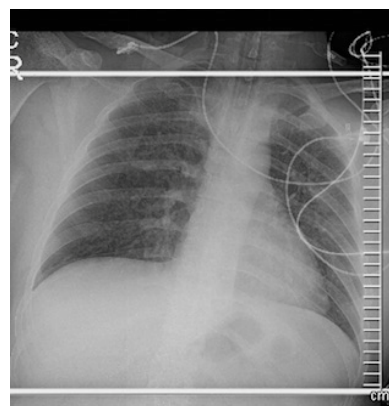


Figure 5. Chest radiography showing the recovered pulmonary edema after the treatment

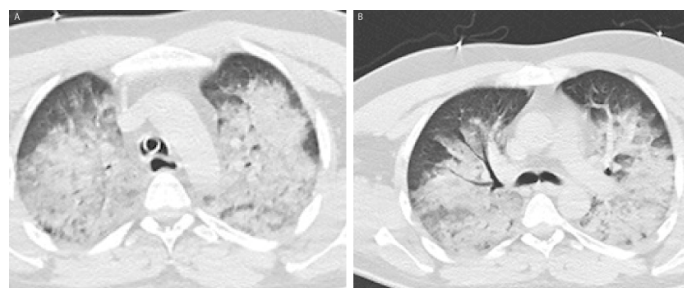


Figure 3A and 3B. The vertical section thoracic CT scans showing the bilaterally diffuse alveolar edema which consistent with neurogenic pulmonary edema(A and B)

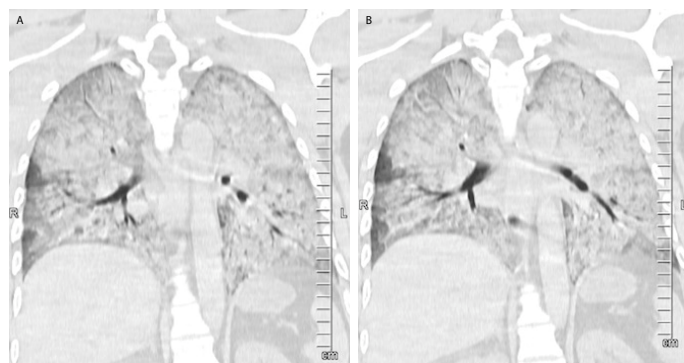


Figure 4. The coronal section thoracic CT scans showing the bilaterally diffuse alveolar edema (A and B)

sudden onset breathlessness, tachypnoea, tachycardia, hemoptysis, bilateral lung crackles on auscultation, low oxygen saturation on arterial blood gas analysis. Neurogenic pulmonary edema seen as mostly homogenous, bilateral airspace consolidations (diffuse opacities) that predominate at the apices in about half of the cases, as seen in our case. Disappearance of radiologic findings in 1-2 days supports the absence of any diffuse alveolar damage [1-7].

In conclusion; NPE is acute onset and life-threatening complication especially in patients with subarachnoid hemorrhage. If diagnosed immediately, the rapid response to treatment can be obtained.

Disclosure

The authors have no conflicts of interest to declare.

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