Eastern Green Neurosurgery Case Report

egneuro,05(01):36-39,2024

Massive Flash Neurogenic Pulmonary Edema after Subarachnoid Hemorrhage

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Abstract:

Neurogenic pulmonary edema is a rare condition associated with subarachnoid hemorrhage secondary to intracranial aneurysm rupture posing high mortality rate. Management of flash pulmonary edema immediately after ictus of spontaneous subarachnoid hemorrhage is very challenging. Here, we reported a rare case of a 47- year-old lady who had massive immediate pulmonary edema following spontaneous subarachnoid hemorrhage.

Key words: Intracranial aneurysm rupture, Neurogenic pulmonary edema, Subarachnoid hemorrhage,

Neurogenic pulmonary edema is a rare clinical condition in emergency setting, presented with an acute onset of pulmonary distress syndrome following spontaneous subarachnoid hemorrhage, mainly due to intracranial aneurysm rupture. It is reported that the incidence of neurogenic pulmonary edema in aneurysmal subarachnoid hemorrhage ranges from 2 to 42.9 %.[1,2,3,4,5] Its appearance is within short interval of minutes to hours following brain hemorrhagic injury and is associated with fatal clinical outcome.[1] Since it is rare and unpredictable in nature it can be missed due to lack of clinical knowledge and specific diagnostic markers. Management of neurogenic pulmonary edema is very crucial. Treatment is mainly supportive with diuretics, vasopressor, fluid resuscitation, and mechanical ventilation for better oxygenation. Fluid resuscitation is challenging, high volume is need for brain injury whereas fluid restriction has to be kept utmost for lung edema.[2]

Case summary:

A 47-year hypertensive lady taking on and off antihypertensive medications since last 10 years, brought to emergency department with chief complains of sudden onset of loss of consciousness since she was found by her family members lying on bathroom floor with abnormal body movement and shortness of breath, blood mixed frothing from mouth. She had history of depressive

Date submitted: 21/ 05/2024
Date accepted: 02/06/2024

disorder for 6 years under medication. On examination, there was no eye opening with no verbal response. She was localizing with pain response (GCS: E1V2M5). Her pupil was B/L 3 mm in size and was non-reactive to light. Her breathing was laborious with saturation 62% with oxygen 10 L/min. On chest auscultation, bilateral decreased breath sound and generalized crackle was heard. Blood pressure was not recordable at the time of presentation. The body temperature recorded was 92°F. She was then intubated in the emergency room. Electrocardiography showed low voltage sinus tachycardia (heart rate 150 beats per minute). Initial lab test revealed PT-18.9, INR-1.54, and Creatinine-1.43. ABG showed PH- 6.877, HCO3-11.1, Lactate-10.5mmol/l. CT scan head revealed diffuse and thick basal subarachnoid hemorrhage with ventricular extension, evolving hydrocephalus and diffuse cerebral edema [Figure 1]. Chest radiograph showed diffuse bilateral patchy consolidation involving upper and middle lobe consistent with bat wing appearance and pulmonary edema [Figure 2]. Echocardiography demonstrated left ventricular global hypokinesia with all extended hypokinesia of anterior segment with severe left ventricular systolic dysfunction with ejection fraction 15-20% with mild MR and TR with IVC normal in size with reduced inspiratory collapse. After intubation she was transferred to the intensive care unit with vasopressor and other supportive care. Despite resuscitation efforts and intensive care, involving cardiac and medicine team, she passed away after 19 hours of presentation to emergency department.

Discussion:

Aneurysmal subarachnoid hemorrhage is one of the life-threatening neurosurgical emergencies that commonly affect female patients with mean age older than 55 years.[3] Clinically high grade massive subarachnoid hemorrhage due to intracranial aneurysm rupture carries higher mortality rate. Besides vasospasm, neurogenic pulmonary edema, stunned myocardium is other severe complications of spontaneous subarachnoid hemorrhage following intracranial aneurysm rupture.[4] Neurogenic pulmonary edema is defined as cardiopulmonary dysfunction due to sudden pulmonary fluid overload following damage to the central nervous system.[5] Although the exact pathophysiology is not known till now, several studies states increase in intra-cranial pressure and damage to neurons causes stress and activation of sympathetic nervous system and increased catecholamine in circulation producing direct lesion of cardiomyocyte results in developing neurogenic pulmonary edema.[6] Hypothalamus and medulla are called trigger zones of neurogenic pulmonary edema. Among which hypothalamic lesion in patient with neurogenic pulmonary edema is said to have worst prognosis. Cytokines and inflammatory mediators which are released after brain injury alter the pulmonary capillary permeability.[7] An increase in interstitial and alveolar fluid level in lung result into pulmonary edema. Traumatic brain injury, subarachnoid hemorrhage, ischemic stroke, and enterovirus induced brainstem encephalitis, subdural hematoma, epidural hematoma, and other cerebral lesions are considered as triggering factors for the development of neurogenic pulmonary edema.[8] Clinically this condition presents with dyspnea, tachycardia, tachypnea, crackles, cyanosis, or rales. On viewing the chest x-ray usually as in our case, it shows diffuse bilateral infiltrates and bilateral alveolar opacities without cardiomegaly.[8] Since the cardiac damage is present, differentiation between cardiac and pulmonary edema is difficult. It is one of the grave complications of aneurysmal subarachnoid hemorrhage. It results in severe hypoxemia despite timely management and contributes to secondary brain damage. Several theories have been

proposed for the pathophysiological changes in lung after subarachnoid hemorrhage owing to the intracranial aneurysm rupture.[4] They are (1) Blast/burst theory, (2) Neurocardiac theory, (3) Theory of Neuro-hemodynamic change and pulmonary venule adrenergic hypersensitivity.[5,9] Blast/burst theory explains about the acute increase in capillary pressure results in injury to alveolar capillary membrane causing vascular leakage and subsequent pulmonary edema in no time.[5] Neurocardiac theory defends sympathetic hyperactivity with catecholamine surge in the blood circulation. It then directly injures cardiac cells leading to neurogenic pulmonary edema.[5] Neuro-hemodynamic change theory says about alteration of myocardium by sudden change in pulmonary pressure due to acute lesion in brain. Sudden shifting of blood from high resistance systemic circulation to low resistance pulmonary circulation causes rapid neurogenic pulmonary edema so in our patient. Pulmonary blood volume rapidly increases, injures alveolar tissues, increases hydrostatic pressure thus affecting pulmonary capillary permeability mechanism.[5] The mechanism of adrenergic hypersensitivity in pulmonary venule explains sudden rise in sympathetic activity following sudden brain insult injures endothelium of pulmonary vascular network carrying alfa and beta receptors. [9]

On view of different articles published in English literature, diagnosis is made based on clinical and radiological features of acute respiratory distress syndrome in presence of neurological emergencies. However cardiac and pulmonary cause for the development of rapidly occurring neurogenic pulmonary edema must be excluded. Wet lung sign or bat wing appearance is typically seen in chest x-ray due to bilateral infiltration. The ratio between partial pressure of oxygen and FIO2 is usually less than 200. There should not be any evidence of left atrial hypertension or other causes of acute respiratory distress like aspiration pneumonia, sepsis, or transfusion related lung injury.

Conclusion:

Massive flash neurogenic pulmonary edema is relatively rare presentation within hours of subarachnoid hemorrhage predominantly due to intracranial aneurysm rupture. Such patients possess very minimal chance to revive due to abrupt multi-system involvement and intensive care management in such cases appears very challenging.

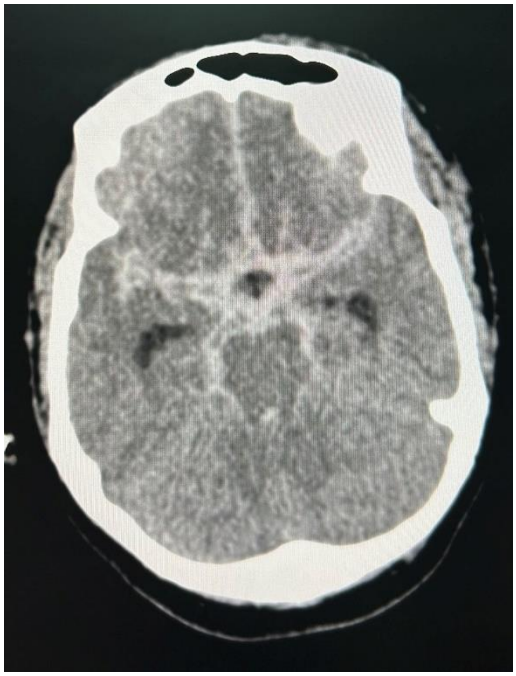

Figure 1: Non contrast CT head axial view showing diffuse and thick basal subarachnoid hemorrhage with evoking hydrocephalus, diffuse cerebral edema

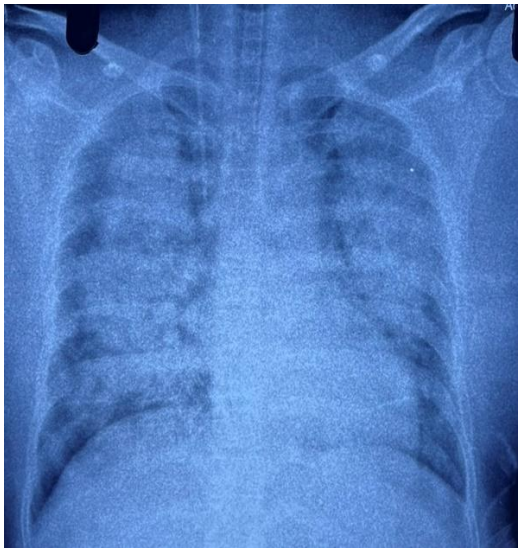

Figure 2: Chest x-ray AP view showing diffuse bilateral patchy consolidation involving upper and middle lobe consistent with bat wing appearance and suggestive of massive pulmonary edema

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