

Case Report**“NEUROGENIC PULMONARY EDEMA: A CASE REPORT”**Patra A K¹, Choudhury I², Diwakar D³

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Abstract: Neurogenic pulmonary edema (NPE) is a rare but fulminant complication of central nervous system (CNS) injury. There are no specific criteria to diagnose this condition, but in a setting of absent cardiovascular risk factors, when a patient of severe CNS injury presents with symptoms and signs of respiratory failure, NPE should be considered as one of the possibilities. NPE usually develops within minutes to hours of CNS injury and is reversible within 48-96 hrs, but may require prolonged ventilation. This article presents a case of NPE, which developed within 48h of surgical insult to the central nervous system.

Key words: CNS injury, Neurogenic pulmonary edema

Introduction: Pulmonary edema can be diagnosed with sudden onset breathlessness, tachypnoea, pink frothy sputum, crackles on auscultation in bilateral lung zones, low oxygen saturation on blood gas analysis and bilateral diffuse opacities in chest radiograph. Occurrence of such condition without any cardiovascular pathology is very rare. But literature reveals sporadic incidents of pulmonary edema in cases of severe head injury, after subarachnoid haemorrhage, epileptic episodes and in rare cases it is also noticed during shunt retrieval^[1]. Catecholaminergic surge altering the pulmonary hemodynamics is the main mechanism behind such unusual condition called “neurogenic pulmonary edema (NPE)”. It is a reversible condition and conservative management with positive pressure ventilation and high FiO₂ usually suffice to tide over the situation. This article describes a case of NPE in a young patient of head injury, where surgical insult is supposed to be the immediate cause.

Case Summary: A 31 year old male was brought to our centre with complaints of high

grade fever since two days. There was a history of a road traffic accident four weeks back for which the patient had undergone frontotemporoparietal craniotomy and evacuation of subdural haematoma. Neurological evaluation on arrival revealed Glasgow Coma Score of E1V_TM2 and pupils bilaterally 3-4mm in size, though sluggishly reacting to light. Computed tomographic evaluation of brain suggested presence of subdural haematoma and necrotic brain tissue of left temporal lobe. Immediate operative procedure was carried out and evacuation of the haematoma with debridement of necrotic tissue was done. Post-operative neurological status did not show any signs of improvement and patient continued to be in comatose state. On the 2nd post-operative day the oxygen saturation dropped, leading to repeated aspiration of profuse frothy secretion from tracheostomy port. Arterial blood gas analysis showed pH 7.30, pCO₂ 30mmHg, pO₂ 54mmHg, HCO₃⁻ 21mmol/L. Chest radiograph showed bilateral diffuse infiltration. **[Fig-1]** A pulmonary artery (PA) catheter was placed and pulmonary capillary wedge pressure (PCWP) was noted to be 08 mmHg. Bedside

Ultrasound and cardiac biomarkers ruled out any cardiac abnormality. MRI brain showed diffuse cerebral edema, midline shift and trapped deformed ventricles. Conservative management with positive pressure ventilation and high FiO₂ led to improvement of respiratory functions over next 72 hrs. Oxygen saturation and chest radiograph improved completely.



Fig-1: Chest radiograph

Discussion: Neurogenic pulmonary edema (NPE) is a clinical syndrome characterised by acute onset respiratory failure with features of pulmonary edema following a marked CNS insult.^[1] The condition was recognised in 1908 for the first time by W T Shanahan^[2]. Incidence of such condition is rare and occurrence is sporadic and unpredictable. Lack of aetiological background and specific diagnostic markers makes it difficult to diagnose and document such cases. 32% of NPE cases have been detected in isolated head injury cases dying at the scene and 50% patients dying within 96 hrs^[3]. Apart from head injury cases, NPE is also detected in cases of brainstem lesion, acoustic neuroma resection, rupture of intracranial aneurysm, leptomeningeal carcinomatosis, and excessive irrigation during ventriculostomy, angioplasty for vasospasm, post-ictal period and pneumocephalus^[4].

The clinical features of NPE remain that of pulmonary edema with a background history

of definite CNS insult. Usually the patient presents with bibasilar crackles, hemoptysis (streaks of blood in sputum), hypoxemia, radiographic pulmonary infiltrates, respiratory distress (shortness of breath, wheezing), dyspnoea, tachycardia, and tachypnoea^[5]. Evaluation should always be extended to rule out any cardiovascular conditions, volume overload or aspiration pneumonia causing such pathology. In our patient, the pulmonary function worsened suddenly leading to SpO₂-75% and PaO₂-53mmHg. He developed copious secretion of pink frothy nature raising the possibility of pulmonary edema. Measurement of PCWP and bedside cardiac evolution ruled out any cardiac cause.

Various mechanisms have been proposed for the development of NPE. Abrupt and intense increase in intracranial pressure leading to catecholaminergic surge remains the cornerstone behind the development of such fatal complications^[6,7]. Increase in intracranial pressure leads to dysfunction of the so called “NPE trigger zones” which involve nucleus tractus solitaries, area postrema, area A1 (ventrolateral aspect of medulla which projects catecholaminergic neurons to hypothalamus), area A5 (upper portion of medulla that project into the preganglionic centers of spinal cord sympathetic outflow)^[8]. Activation of NPE trigger zones causes widespread release of catecholamines which then affect the pulmonary vasculature. This is explained by many clinicopathologic paradigms: neuro-cardiac, neuro-haemodynamic, blast theory, and pulmonary venule adrenergic hypersensitivity^[1]. In our patient, the immediate MRI of brain revealed diffuse cerebral edema with trapped and deformed ventricles. Most probably, in the post-operative period the patient became susceptible for NPE following cerebral edema with trapped ventricles and activation of NPE trigger zones.

There are two clinical forms of NPE described in the literature: early and delayed^[1]. The early

form is most common and occurs within minutes to hours of neurologic injury. The delayed form usually starts 24-48 hrs after the acute CNS insult.

Management of such cases should rule out cardiogenic pulmonary. Few criteria proposed to differentiate are: (a) bilateral infiltrates (b) $\text{PaO}_2/\text{FiO}_2 < 200$ (c) no evidence of left arterial hypertension, (d) presence of severe CNS injury (e) absence of other common causes of ARDS.^[1] Conservative management involving haemodynamic and respiratory support with measures to decrease the intracranial pressure usually suffice. Positive pressure ventilation with judicious use of positive end expiratory pressure (PEEP) is recommended to improve the lung function^[9], α adrenergic blockers, Milrinone, Dobutamine may be considered in different situation to maintain the haemodynamics in an associated stunned myocardium syndrome. Diuretics, Furosemide, Mannitol, can also be given for rapid regression of pulmonary edema. Other supportive measures like 30 degree head up position and fluid management should also be considered. In our patient supportive measures helped to improve the patient's condition in three days.

Conclusion: Acute neurogenic pulmonary edema, an uncommon clinical entity can occur after virtually any severe CNS injury. Mechanism of development and progression of such condition is complex. Neurogenic pulmonary edema can cause sudden deterioration of patient unless diagnosed and supported. Usually the disease resolves if supportive measures are taken along with the treatment of the causative factor.

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