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Case Report

Neurogenic Pulmonary Edema, Impaired Left Ventricular Function, and Resistant Ventricular Fibrillation Following Grade 3 Subarachnoid Hemorrhage - A Unique Case Report

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Abstract

Neurogenic pulmonary edema (NPE) is a rare but serious complication of acute brain injuries, including subarachnoid hemorrhage (SAH). The early recognition and management of NPE in the context of SAH are crucial for improving patient outcomes. This case report discusses a fatal presentation of SAH complicated by acute NPE, cardiovascular compromise, and fatal arrhythmia.

A previously healthy 40-year-old woman was urgently transferred to the Emergency Department after experiencing sudden loss of consciousness, generalized seizures, and severe respiratory distress. Initial assessment revealed a blocked airway due to frothy secretions, labored breathing with low oxygen saturation, and hemodynamic instability. Neurological examination indicated a Glasgow Coma Scale score of 7. Point-of-care ultrasound (POCUS) and imaging studies confirmed pulmonary edema, reduced cardiac ejection fraction, and a Grade IV subarachnoid hemorrhage according to the Fisher scale. Despite aggressive medical interventions, including intubation, ventilation, and inotropic support, the patient's condition deteriorated, leading to ventricular fibrillation and, ultimately, her demise.

This case underscores the critical need for heightened awareness and early intervention in the management of NPE associated with SAH. Timely, coordinated care may improve outcomes and reduce mortality in patients with this challenging clinical presentation.

Keywords: neurogenic pulmonary oedema, subarachnoid haemorrhage, resistant ventricular fibrillation

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Background

Neurogenic pulmonary oedema (NPE) is a rare but life-threatening complication of acute brain injuries, including subarachnoid haemorrhage (SAH). It results from a complex interplay of sympathetic overactivity, hemodynamic disturbances, and inflammatory responses, leading to acute respiratory distress and cardiovascular compromise [1,2]. Although NPE is relatively uncommon, its rapid onset and progression can significantly impact patient outcomes, as in our case, which presents a fatal instance of SAH complicated by acute NPE, cardiovascular instability,

and fatal arrhythmia, highlighting the challenges in its early identification and management. Similarly, reported cases in Sri Lankan literature were scarce.

Case Presentation

A previously healthy 40-year-old woman was transferred to the emergency department of a tertiary care facility within an hour of clinical deterioration. She initially presented to the nearby divisional hospital after her family found her unresponsive, exhibiting generalised tonic-clonic seizures and severe respiratory distress. The local hospital had managed her seizures before transfer.

Upon arrival, a thorough assessment was conducted. The patient presented with a blocked airway, primarily due to a substantial accumulation of frothy nasal and oral secretions, producing a distinctive gurgling sound. Her breathing was visibly laboured, with widespread crepitations in both lungs, a respiratory rate of 36, and the pulse oxygen saturation (SpO_2) as low as 60%. Immediate interventions included the placement of an oropharyngeal airway and the administration of high-flow oxygen via a non-rebreather mask.

Circulatory assessment revealed tachycardia with a heart rate of 120. Her extremities were cold and clammy, with a faint peripheral pulse. Blood pressure was 80/50 mmHg, despite receiving two pints of normal saline boluses at the local hospital. She exhibited no signs of fever or external trauma. Neurological assessment showed a Glasgow Coma Scale (GCS) score of 7 (E2 V2 M3), bilateral upgoing plantar reflexes, and no focal neurological deficits.

Bedside point-of-care ultrasound (POCUS) revealed bilateral diffuse B-lines, indicative of pulmonary oedema, a reduced ejection fraction, and an elevated ventricular rate [Figure 1]. The inferior vena cava showed less than 50% collapsibility. An electrocardiogram (ECG) demonstrated widespread ST-segment depression in the anterior and inferior territories, along with sinus tachycardia [Figure 1].

Arterial blood gas (ABG) analysis revealed metabolic acidosis with partial respiratory compensation, with a pH of 7.339 (Normal range: 7.35-7.45), pCO_2 29mmHg (Normal range: 35-45 mmHg), HCO_3 of 15.2 mmol/l (Normal range: 22-26 mmol/L), and elevated lactate levels (Lactate 5.2 mmol/L, normal range: <2 mmol/L). The patient met the criteria for acute respiratory distress syndrome (ARDS) with a PaO_2/FiO_2 ratio of 125 (normal ratio >300), alongside electrolyte abnormalities, including elevated sodium (Na^+ 153.1 mmol/L, normal range: 135-145 mmol/L) and low potassium (K^+ 2.8 mmol/L, normal range 3.5-5.5 mmol/L]) levels and negative troponin I titer (2 ng/l) and normal CRP (<5 mg/dl).

Following initial management, a decision was made to intubate and ventilate the patient, initiate inotropic support, and proceed with diagnostic imaging. A non-contrast computed tomography scan (NCCT) of the brain revealed a substantial SAH without intraventricular extension or intracerebral haemorrhage. Additionally, the NCCT scan depicted the characteristic 'Star Sign' in the basal regions, consistent with a Fisher Grade haemorrhage [Figure 1]. Chest X-rays revealed diffuse interstitial pulmonary infiltrates in both lung fields, consistent with acute pulmonary oedema. Neurosurgical consultation led to a plan for urgent endovascular intervention, with a follow-up contrast-enhanced CT scan once the patient's condition stabilised.

While receiving inotropic support, the patient maintained a marginally acceptable mean arterial perfusion, aided by intravenous norepinephrine and dobutamine. Despite maximal respiratory support, she continued to experience persistently low oxygen saturation (SpO_2).

Approximately one hour after initial stabilisation and continuous reevaluation, the patient suddenly went into ventricular fibrillation. Despite three rounds of defibrillation, her condition did not improve, ultimately leading to asystole. Tragically, despite an hour-long, exhaustive resuscitation effort, the patient succumbed.

Discussion

NPE is a frequently overlooked yet significant consequence of acute brain injuries. In our case, the patient exhibited the onset of NPE, confirmed by clinical signs such as frothy sputum, widespread bilateral lung crepitations, declining oxygen saturation levels, and corroborated by findings from POCUS, chest X-ray, and ABG analysis. This occurrence was concomitant with a Grade 3 SAH, assessed at Grade IV on the Fisher scale. SAH can lead to the development of acute NPE, complicating the scenario.

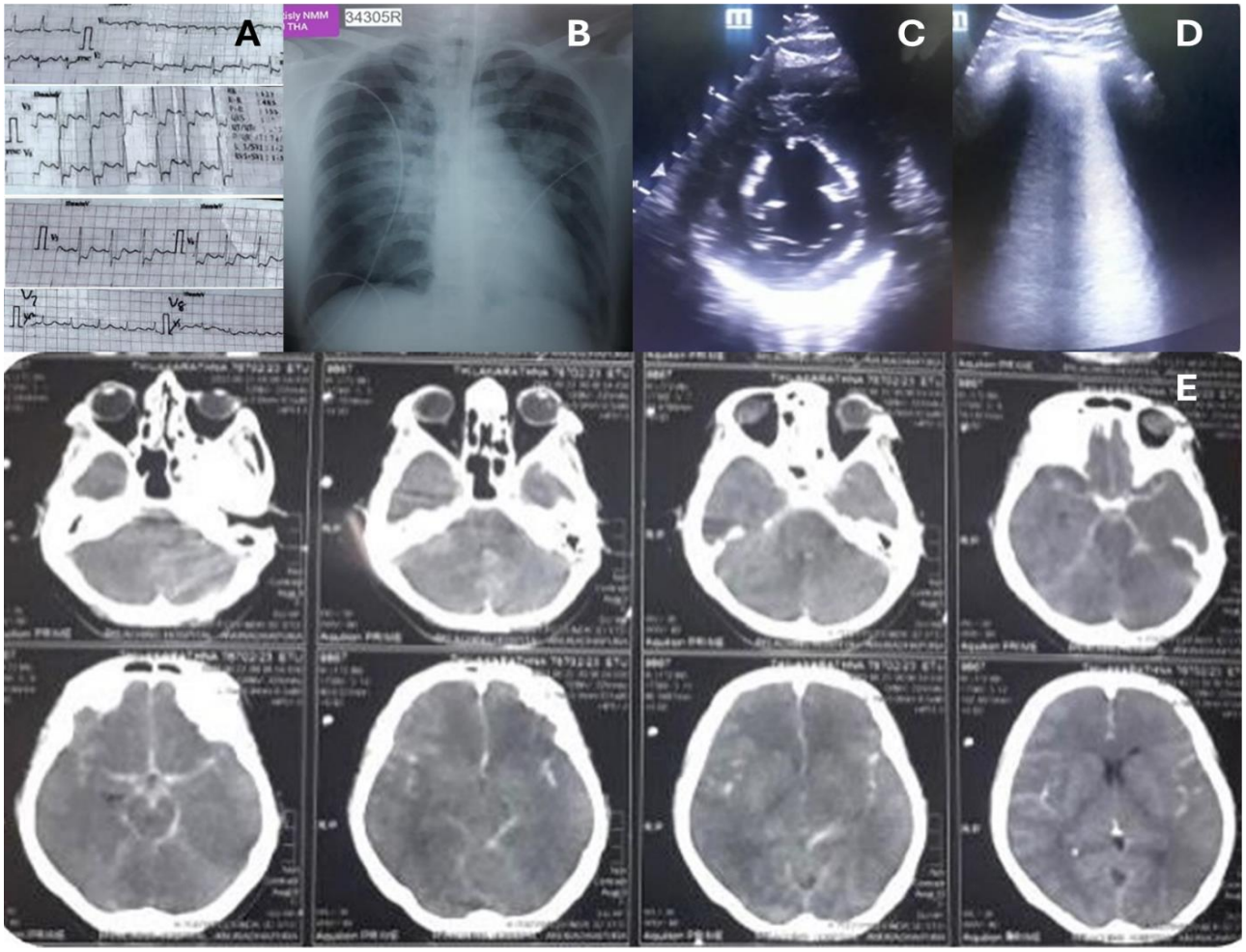

Figure 1: A: Initial electrocardiogram, B: Chest X-ray posteroanterior, C: point-of-care echocardiogram; D: point-of-care lung ultrasound, E: Non-contrast computed tomography brain (star sign of SAH evident on left lower slice)

POCUS has proven to be a valuable tool for accurately identifying pulmonary oedema in SAH patients, as in our case. In NPE due to SAH, vertebral artery dissection and severe World Federation of Neurosurgical Societies (WFNS) grades upon admission are considered to have worse outcomes [2].

The pathophysiology of this condition encompasses several intricate mechanisms. One proposed cause involves the overactive sympathetic discharge triggered by elevated intracranial pressure, subsequently causing alterations in pulmonary vascular pressure and fluid seepage. Additionally, the adrenergic surge induced by SAH can initiate a chain of reactions that contribute to the development of both NPE and stress-induced hemoconcentration [3]. Hemodynamic alterations, including elevated extravascular lung water index and pulmonary vascular permeability index, have been documented in individuals experiencing both SAH and pulmonary oedema [4]. Furthermore, individuals with low-grade SAH are even at a higher risk of experiencing acute cardiopulmonary complications, such as NPE and conditions resembling takotsubo cardiomyopathy [6,7]. In our case, the patient demonstrated hemodynamic instability and impaired left ventricular function, as evident in bedside 2D Echo findings.

In this case, the patient also exhibited widespread ST-segment depressions in the anterior and inferior regions, ultimately culminating in refractory ventricular fibrillation. Studies have described the prevalence and multiple variations of ECG abnormalities in SAH patients and their potential to worsen clinical outcomes. These ECG changes in SAH patients can stem from autonomic neural stimulation originating in the hypothalamus or elevated levels of circulating catecholamines. Furthermore, ECG anomalies, including atypical Q or QS waves and nonspecific ST- or T-wave deviations, hold promise as predictive indicators of the onset of NPE within a 24-hour timeframe in adult patients with spontaneous SAH [8,9,10].

The most effective treatments for subarachnoid hemorrhage in patients with aneurysms encompass a range of options, including endovascular treatment

(Both single-stage and multiple-stage endovascular treatments), surgical intervention (initially clipping the ruptured aneurysm and subsequently addressing intact aneurysms in a later phase), and medication therapy (such as nimodipine, dexmedetomidine, Selective Serotonin Re-uptake Inhibitors, and DL-3-n-butylphthalide have been prescribed) [11,12,13,14]. Moreover, delayed treatment of bystander aneurysms after initial stabilisation in SAH patients has exhibited safety and efficacy, with no observed growth or bleeding during follow-up [15]. Although many of the options are less widely available in a resource-poor setting like Sri Lanka, Early recognition, stabilisation, and neurosurgical intervention may help achieve better outcomes.

Conclusion

Early recognition and prompt management of NPE in subarachnoid haemorrhage are crucial for improving outcomes. A multidisciplinary approach, protocol-driven care, and quality improvement initiatives aimed at reducing delays in seeking tertiary care services in complicated scenarios, along with heightened clinical awareness, are key to timely interventions and reducing associated morbidity and mortality.

Consent and Ethical Considerations

Informed verbal consent was obtained from the spouse of the patient for the publication of this case report. No personal information is published, and all the clinical images are deidentified.

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