International Journal of Research in Medical Sciences
Tiwary MK et al. Int J Res Med Sci. 2024 Nov;12(11):4278-4280
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20243385

Case Report

Recurrent neurogenic pulmonary edema following tonic-clonic seizures

Manish Kumar Tiwary[1]*, Yogesh Manhas[2]

[1]Department of Anaesthesiology and Critical Care, Sheikh Khalifa Medical City, Ajman, UAE
[2]Department of Anaesthesiology and Critical Care, Ibra Hospital, Ibra, Sultanate of Oman

Received: 13 September 2024
Accepted: 22 October 2024

***Correspondence:**
Dr. Manish Kumar Tiwary,
E-mail: tiwarymani@gmail.com

ABSTRACT

Neurogenic pulmonary edema (NPE) is a relatively rare life-threatening complication characterized by the acute onset of pulmonary oedema. Although many pathogenic theories have been proposed exact mechanism or aetiology is unknown. We report a case of a female patient who presented with dyspnoea following tonic-clonic seizures. Further investigations excluded cardiogenic aetiology and significant improvement in the patient within 24 h supports the diagnosis of NPE as the primary pathology. We report a case of recurrent NPE in a 24-yr old female patient with seizure disorder managed by us in our department.

Keywords: Epilepsy, Intensive care unit, NPE, Tonic-clonic seizures

INTRODUCTION

Neurogenic pulmonary edema (NPE) is a relatively rare life-threatening complication characterized by the acute onset of pulmonary oedema after a significant injury to the central nervous system.[1] The aetiology of NPE is unknown with few theories stating autonomic sympathetic discharge following central nervous system injury most likely affects systemic and pulmonary circulation by influencing pulmonary capillary pressure and permeability, resulting in pulmonary oedema.[2] We present a case of a young woman with a history of epilepsy who developed recurrent acute NPE following generalized tonic-clonic (GTC) seizures with two episodes of intensive care unit (ICU) admission.

CASE REPORT

A 26-year-old female with a history of epilepsy presented twice to the emergency department after an episode of GTC seizures. She was using tablet levetiracetam 500 mg twice daily. The patient had a seizure at home and was in postictal state on arrival. She had another GTC seizure in the emergency department which was controlled by midazolam 10 mg administered intravenously (in aliquots of 2 mg). Immediately after the seizure, she became dyspnoeic and hypoxic. She was intubated for severe hypoxia (pO_2-50 mm Hg on 100% oxygen). The ventilatory settings were: pressure-synchronized intermittent mandatory ventilation (P-SIMV): pressure control-20, respiratory rate-16/min, positive end-expiratory pressure- 6 cm water, FiO_2: titrated to 0.4 for oxygen saturation on pulse oximeter more than 92%. Chest radiograph post-intubation revealed diffuse bilateral infiltrates (Figure 1 A) and pink frothy sputum was seen accumulated in the endotracheal tube (ETT). The patient was then administered 1 gram of intravenous levetiracetam followed by 1 gram twice daily. She was admitted to the ICU, started on a course of antibiotics for suspected aspiration pneumonia (IV ceftriaxone 2 gm twice daily, IV clarithromycin 500 mg 8th hourly, metronidazole 500 mg 8th hourly), and placed on mechanical ventilation. Computed tomography (CT) of brain did not reveal any acute intracranial abnormality. Repeat chest radiographs the following day showed rapid resolution of the bilateral infiltrates (Figure 1 B). Her

PaO_2:FiO_2 (P/F) ratio improved to more than 200. Her blood, urine, and sputum cultures were within normal limits with influenza and respiratory syncytial virus polymerase chain reaction reports negative. Antibiotics were discontinued, and the patient was successfully extubated within 36 hours after intubation. She was discharged in a stable condition with instructions to take 1 gram of levetiracetam twice daily with a scheduled neurology follow-up. From parents, we got information that the patient had been diagnosed with epilepsy with non-compliance to her antiepileptic medications. The patient returned 7 months later to emergency department again after experiencing GTC seizures at home. She was in a postictal state and had a GTC seizure which controlled by 10 mg of midazolam IV (in aliquots of 2 mg). Patient was subsequently dyspnoeic and hypoxic. On auscultation, she had bilateral diffuse crackles and was started on NIV support and shifted to ICU. In ICU she had another episode of GTC seizures requiring intubation for hypoxic respiratory failure. Post intubation chest radiograph showed increased bilateral opacities (Figure 1 C) similar to the radiograph of prior admission. Pink frothy sputum was present in the ETT. She was again started on antibiotics for suspected aspiration pneumonia and started on intravenous levetiracetam.

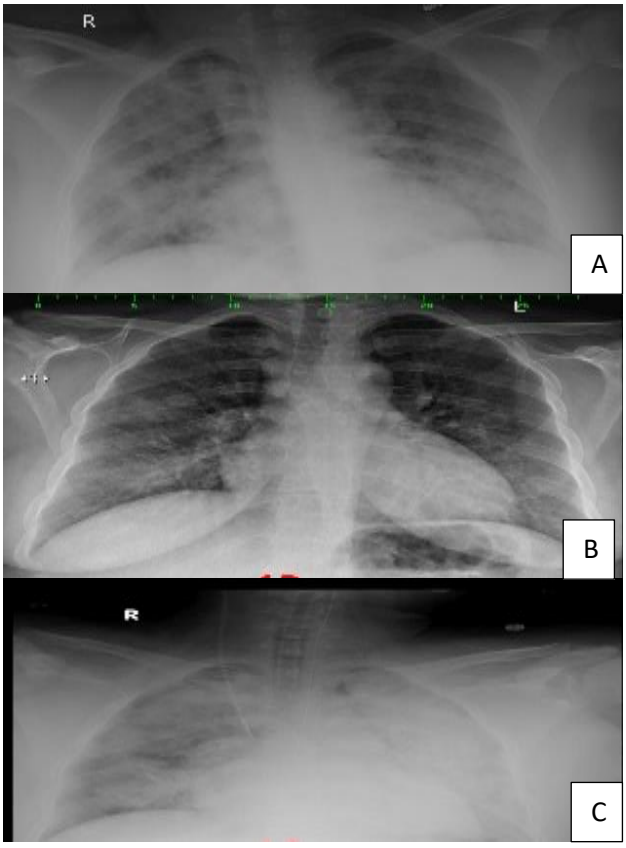

Figure 1 (A-C): Chest radiograph with bilateral infiltrates during first admission, post-treatment radiograph with complete resolution of infiltrates and chest radiograph during second admission with bilateral infiltrates.

CT Brain again showed no acute intracranial abnormalities. A repeat chest radiographs the next day showed rapid resolution of bilateral opacities. Her blood, urine, sputum cultures, and laboratory reports were normal. Antibiotics were stopped, and the patient was successfully extubated within 48 hours of mechanical ventilation. An echocardiogram was done which was essentially normal (normal ejection fraction, normal diastolic function, no significant valvular disease, and no evidence of pulmonary hypertension) ruling out any possibility of any cause of cardiac origin. We did not use lung ultrasound for B lines and ultrasound for inferior vena cava diameter. In this case, intracranial pressure was not measured quantitatively and neither an optic sheath diameter was estimated using an ultrasound.

DISCUSSION

A patient with a history of epilepsy or neurologic insult who develops shortness of breath within minutes to hours, following a GTC seizure, typically indicates NPE. Clinical findings in such a situation are tachypnoea, tachycardia, and rales or rhonchi with laboratory reports showing hypoxemia on ABG and leucocytosis.[3] Chest radiographs reveal bilateral alveolar opacities without cardiomegaly associated with a delayed onset pulmonary oedema which takes 12 to 72 hours to appear after a neurologic event.[4] In a review of NPE cases author observed that the onset of symptoms occurred less than 4 hours after the neurologic event, mostly observed in female patients (mean age- 31.6 years), one-third of patients had pink frothy sputum, chest radiographs showed bilateral diffuse infiltrates in over 90% of cases, and recovery was significantly rapid within less than 72 hours in over half of the patients.[5]

NPE is a diagnosis of exclusion, cardiac aetiology should be ruled out first. In addition, it requires the demonstration of hypoxemia and bilateral pulmonary infiltrates, in the presence of a central nervous system insult raising the ICP. Epileptic seizures, traumatic brain injury and cerebral haemorrhage are the commonest causes of NPE. Diagnostic criteria have been proposed to identify patients with NPE: bilateral infiltrates; P/F ratio less than 200, no evidence of left atrial hypertension, presence of CNS injury severe enough to have caused significantly increased ICP; and absence of acute respiratory distress syndrome due to aspiration, sepsis, or massive blood transfusion. The NPE and its pathophysiology is highly unknown and not fully understood. Increased ICP is thought to be responsible for the manifestation of NPE via catecholamine release that causes intense pulmonary and systemic vasoconstriction leading to an increase in capillary hydrostatic pressure and capillary permeability also known as “blast theory”.[4] Hypoxemia may be one of associated risk factor to the development of NPE. Severe ictal hypoxemia has been reported in different types of seizures, especially with the GTC convulsion. Treatment modalities for NPE including supplemental oxygen,

mechanical ventilation if needed, and control of pulmonary vascular pressures are primarily supportive.

CONCLUSION

Aspiration pneumonia and cardiogenic pulmonary oedema are usual differential diagnosis. However, if heart failure can be excluded and if the pulmonary oedema recovery is rapid within 12-48 hours, NPE is more likely. Negative pressure pulmonary oedema was also considered in the differential diagnosis but the most common cause is post-extubation laryngospasm following surgery and our patient developed hypoxia and respiratory failure following GTC seizures.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Tiwary MK, Manhas Y. Recurrent neurogenic pulmonary edema following tonic-clonic seizures. Int J Res Med Sci 2024;12:4278-80.