Journal of
Pulmonology and
Intensive Care

Case Report

DOI: 10.51271/JOPIC-0045

Neurogenic pulmonary edema caused by epileptic attack: a case report

Kübra Taşkaraca Karabacak, Nilgün Yılmaz Demirci

Department of Chest Diseases, Faculty of Medicine, Gazi University, Ankara, Turkiye

Cite this article: Taşkaraca Karabacak K, Yılmaz Demirci N. Neurogenic pulmonary edema caused by epileptic attack: a case report. *J Pulmonol Intens Care*. 2024;2(4):89-90.

Corresponding Author: Kübra Taşkaraca Karabacak, kubrataskaraca@gmail.com

Received: 17/10/2024 ◆ **Accepted**: 10/11/2024 ◆ **Published**: 20/11/2024

ABSTRACT

Neurogenic pulmonary edema (NPE) is a life-threatening situation that progresses with acute respiratory distress caused by central neurological damage or injury. NPE may occur as aresult of different central nervous system diseases and disorders, such as brain malignancies,traumatic brain injuries, infections, and convulsions. This case report was designed to highlight thecauses, outcome, and treatment of NPE triggered by an epileptic attack.

Keywords: Neurogenic, pulmonary edema, catecholamines

INTRODUCTION

Neurogenic pulmonary edema (NPE) is a life-threatening situation that progresses with acute respiratory distress caused by central neurological damage or injury. NPE may occur as a result of different central nervous system diseases and disorders, such as brain malignancies, traumatic brain injuries, infections, and convulsions. Symptoms begin within minutes or hours and resolve after 48-72 hours. This study was designed to highlight the causes, outcome, and treatment of NPE triggered by an epileptic attack. NPE may also be keep in mind in the etiology of cases of acute respiratory failure and hypoxia.

CASE

A 43-year-old male patient with a diagnosis of epilepsy was brought to the emergency service by ambulance with a generalized tonic-clonic seizure. He was given a total of 10 mg of diazepam,with one administration in the ambulance and two in triage. He was known to have had one seizure in the last 3.5 months and he was using depakine at 2x750 mg. Almost immediately after the seizure, he became dyspneic and hypoxic; in light of these symptoms,a chest diseases consultation was requested. His temperature was 36.8°C, pulse was 134/min, blood pressure 130/65 mmHg, and saturation was 82% without oxygen. Oxygen was administered at 15 L/min with a reservoir mask. The PaO_2/FiO_2 ratio of 173 was indicative of severe hypoxia. Other physical examination findings were normal. Chest radiography and thoracic computed tomography (CT) showed bilateral diffuse bilateral infiltrates with preservation of the subpleural space, increased central opacity consistent with pulmonary edema and consolidation (**Figure 1: A-D**).

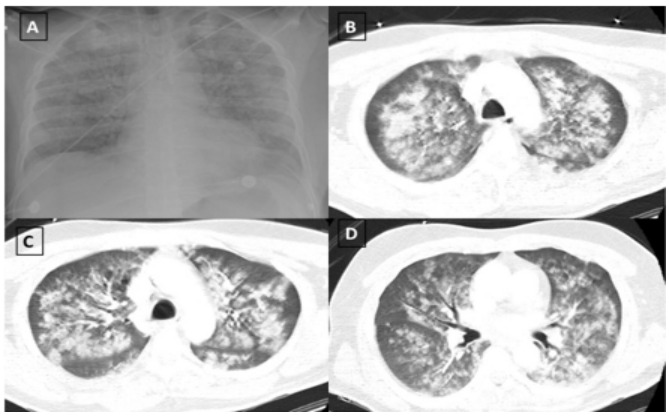

Figure 1. (**A**) Initial chest X-ray with opacity consistent with pulmonary edema, (**B-D**) bilateral alveolar filling defect and areas of consolidation consistent with pulmonary edema with preservation of the subpleural area.

Laboratory examination results were unremarkable and a polymerase chain reaction (PCR) test for COVID-19 was negative. The only abnormal electrocardiogram finding was sinus tachycardia and echocardiography was normal. No intracranial lesions were detected at cranial CT. The patient's clinical situation improved in a few hours without any further medical treatment, with saturation reaching 97% with a nasal cannula at 2 L/min. After 24 hours he no longer needed oxygen, and in an examination at 72 hours his chest radiography was completely normal (**Figure 2**). He was discharged after a consultation with neurology about his antiepileptic treatment.

DISCUSSION

NPE was first defined by Shanahan[1] in 1908 as postictal pulmonary edema in 11 epileptic patients aged between 9 and 36 years. The incidence of NPE is unknown because

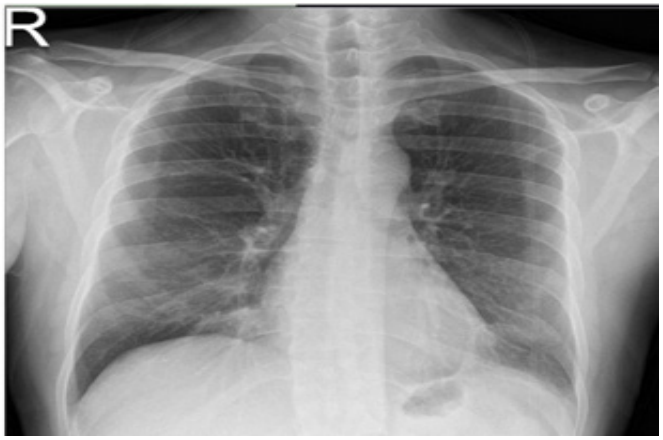

Figure 2. Control chest X-ray taken 72 hours later

it is difficult to distinguish between cardiogenic and non-cardiogenic pulmonary edema. However, it is a life-threatening situation with an unclear etiopathogenesis. The most frequent triggers are cranial trauma, subarachnoid bleeding, and epilepsy, mostly with generalized seizures. Cervical medullary trauma, electroconvulsive therapy, intoxication, postoperative complications after intracranial surgery, and meningitis are more rare triggers of NPE.[2] Due to underlying brain injury, the mortality rate is high regardless of the pulmonary effects.[3,4] Pathophysiologically, the sympathetic nervous system is the key player in the pathogenesis of NPE. Catecholamines initiate three important pathophysiological responses: systemic vasoconstriction, increased blood pressure, and increased venous return. Sympathetic activation, increased release of catecholamines and increased pulmonary capillary permeability occur.[2,5,6]

Clinical manifestations are generally nonspecific and it occurs within minutes or hours, secondary to intracranial pathologies. Dyspnea, tachycardia, hypoxemia, and hemoptysis may occur in these patients. Laboratory findings are also nonspecific and ventilation perfusion imbalance, hypoxemia, and carbon dioxide retention may be detected.[2] Acute respiratory distress syndrome (ARDS), cardiologic edema, aspiration pneumonia, and bacterial pneumonia may be involved in the differential diagnosis. NPE can be confused with many other clinical pictures from ARDS to COVID-19 pneumonia that cause similar radiological findings of diffuse infiltrations.[7,8] The radiological findings of our case were also likely with pulmonary edema. However, the COVID-19 PCR test and microbiological samples were negative. There were no remarkable laboratory results. And no pathology was detected in echocardiography.

The main principles of treatment are to prevent the increase of intracranial pressure and address the injury to the central nervous system. Providing ventilation and supportive oxygenation, as in cases of ARDS, is important along with the reduction of pulmonary vascular resistance, especially by targeting inotropic increase via β-adrenergic stimulation.[2] Experimental studies have shown that α-adrenergic blockers can be useful in the treatment of NPE by blocking the increased sympathetic response, while dobutamine increases cardiac output and reduces peripheral vasoconstriction.[9] In our case, the trigger was an epileptic seizure and antiepileptic treatment and symptomatic treatment with oxygen resolved the case. The need for oxygen supplementation was quickly reduced.

CONCLUSION

NPE is a life-threatening situation that progresses with acute respiratory distress caused by central neurological damage or injury. NPE may occur as a result of different central nervous system diseases and disorders, such as brain malignancies, traumatic brain injuries, infections, and convulsions. This case report was designed to highlight the causes, outcome, and treatment of NPE triggered by an epileptic attack.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Shanahan WT. Pulmonary edema in epilepsy. *N Y Med J*. 1908;87:54-56.
2. Al-Dhahir MA, Das JM, Sharma S. Neurogenic pulmonary edema. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; July 17, 2023.
3. Raja HM, Herwadkar AV, Paroutoglou K, Lilleker JB. Neurogenic pulmonary oedema complicating a lateral medullary infarct. *BMJ Case Rep*. 2018;2018:bcr2018225437.
4. Romero Osorio OM, Abaunza Camacho JF, Sandoval Briceño D, Lasalvia P, Narino Gonzalez D. Postictal neurogenic pulmonary edema: case report and brief literature review. *Epilepsy Behav Case Rep*. 2017;9:49-50.
5. Kerr NA, de Rivero Vaccari JP, Abbassi S, et al. Traumatic brain ınjury-ınduced acute lung ınjury: evidence for activation and ınhibition of a neural-respiratory-ınflammasome axis. *J Neurotrauma*. 2018;35(17):2067-2076.
6. Bahloul M, Chaari AN, Kallel H, et al Neurogenic pulmonary edema due to traumatic brain injury: evidence of cardiac dysfunction. *Am J Crit Care*. 2006;15(5):462-470.
7. Figueiredo EG, Oliveira AM, Almeida CE, Teixeira MJ. Subarachnoid hemorrhage and hydrocephalus causing neurogenic pulmonary edema. *Arq Neuropsiquiatr.* 2010;68(3):461-462.
8. Wonkyo K, Kim Y, Kim D, Chung R, Kim C. Fulminant neurogenic pulmonary edema developed after spontaneous cerebellar hemorrhage-a case report. *Anesth Pain Med.* 2011;6:262-265.
9. Akinci OI, Eker A, Erdem T, et al. Neurogenic pulmonary edema: a prospective study based on observation]. *Ulus Travma Acil Cerrahi Derg.* 2006;12(1):22-25.