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A Rare complication of epileptic seizure: two cases of neurogenic pulmonary edema

Epileptik nöbetin nadir bir komplikasyonu: iki nörojenik pulmoner ödem olgusu

Uğur Fidan, Deniz Kızılırmak, Aylin Aydilek Yılmaz, Zeynep Yılmaz Kaya, Müge Gençer Tuluy, Yavuz Havlucu

Manisa Celal Bayar University Faculty of Medicine, Chest Diseases Clinic, Manisa, Türkiye

ABSTRACT

Neurogenic pulmonary edema is a form of alveolar edema that results from an increased adrenergic response secondary to central nervous system injury. It is characterized by elevated catecholamine levels and increased pulmonary hydrostatic pressure. The causes of neurogenic pulmonary edema include traumatic brain injury, stroke, intracranial hemorrhage, acute hydrocephalus, meningitis, drug overdose, and epilepsy. Non-specific symptoms such as shortness of breath, cough, and pink frothy sputum may occur due to pulmonary edema. We present two cases of patients, aged 43 and 32, both diagnosed with epilepsy, who presented to the emergency department with respiratory complaints following epileptic seizures. Diagnostic evaluations confirmed neurogenic pulmonary edema, and clinical improvement was observed following symptomatic treatment.

Keywords: Neurogenic pulmonary edema, epilepsy, central nervous system injury.

ÖZ

Nörojenik pulmoner ödem, santral sinir sistemi hasarına sekonder olarak gelişen artmış adrenerjik cevap, katekolamin seviyesinde artış, pulmoner hidrostatik basınç artışı sonucu meydana gelen alveolar ödemdir. Nörojenik pulmoner ödem nedenleri arasında, travmatik beyin hasarı, felç, intrakranial kanama, akut hidrosefali, menenjit, aşırı doz ilaç alımı ve epilepsi yer alır. Pulmoner ödeme bağlı nefes darlığı, öksürük, pembe köpüklü balgam gibi nonspesifik semptomlar izlenebilir. Biz 43 ve 32 yaşlarında, epilepsi tanılı iki hastanın epileptik nöbet sonrası gelişen solunumsal yakınmalar ile acil servise başvurup yapılan tetkikler sonucu nörojenik pulmoner ödem tanısı alan ve sonrasında semptomatik tedaviyle klinik düzelme görülen iki vakayı sunuyoruz.

Anahtar Sözcükler: *Nörojenik pulmoner ödem, epilepsi, santral sinir sistemi hasarı.*

INTRODUCTION

Neurogenic pulmonary edema is a critical clinical condition characterized by the sudden onset of pulmonary edema following injury to the central nervous system, and it is a relatively rare occurrence (1). It can develop as a result of various central nervous system disorders, including head trauma, subarachnoid hemorrhage, multiple sclerosis, strokes, cervical spine injury, central nervous system tumors, and epilepsy (2). Although the exact pathogenesis remains unclear, the increased catecholamine levels resulting from the adrenergic response to central nervous system injury, along with elevated pulmonary hydrostatic pressure and increased capillary permeability, are considered to be the underlying cause (1).

Corresponding author: Uğur Fidan
Manisa Celal Bayar Üniversitesi Tıp Fakültesi Göğüs Hastalıkları Kliniği, Manisa, Türkiye
E-mail: *dr-ugurfidan@hotmail.com*

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The most frequently observed findings include shortness of breath, cough, hypoxemia, pink frothy sputum, rales on auscultation, and bilateral pulmonary infiltrates on chest radiography. While pulmonary edema can develop within minutes to hours following central nervous system injury, it may be resolved with the appropriate symptomatic treatment within 48-72 hours (3). This case series presents two cases of neurogenic pulmonary edema that developed subsequent to epileptic seizures.

Written consent was obtained from the patient stating that medical data could be published.

CASE REPORT 1

A 43-year-old female patient diagnosed with epilepsy experienced three consecutive epileptic seizures following numbness on the left side of her body. The patient presented to the emergency department with complaints of shortness of breath, chest pain, and a small amount of pink sputum following the seizures. Upon admission to the emergency department, the patient's body temperature was 36.8°C, heart rate was 87 beats per minute, blood pressure was 121/68 mmHg, and peripheral oxygen saturation was measured at 82% while receiving 6 liters per minute of oxygen inhalation. Laboratory results revealed a C-reactive protein level of 0.62 mg/dL, creatinine level of 1.1 mg/dL, white blood cell count of 12,900/mL, hemoglobin level of 8.9 g/dL, and platelet count of 221,000/μL. Arterial blood gas analysis showed a pH of 7.42, pO2 of 69.4 mmHg, pCO2 of 37 mmHg, and an oxygen saturation (SO2) of 92%. The patient reports experiencing seizures intermittently over the past five years, accompanied by shortness of breath following the seizures. Upon respiratory examination, bilateral rales were audible at the bases during auscultation. Chest radiography revealed an increase in central-weighted heterogeneous opacity, more pronounced in the right lung (Figure-1a). Computed tomography of the chest revealed widespread areas of increased density in a central-weighted alveolar pattern, particularly pronounced in the right upper lobe, accompanied by bilateral peripheral focal ground-glass opacities (Figure-1b). Echocardiography demonstrated an ejection fraction of 65% and identified grade 1 mitral regurgitation. The cardiological assessment did not indicate the presence of heart failure. Neurological examination revealed no evidence of pathological findings. The neurology team-initiated treatment for epilepsy with levetiracetam.

The patient admitted to the Chest Diseases Clinic was initiated on inhaled oxygen therapy and broad-spectrum antibiotic treatment. During the subsequent follow-up period, the patient exhibited a notable improvement in respiratory symptoms, accompanied by a marked enhancement in lung radiological findings by the conclusion of the first week of follow-up (Figure-1c).

Approximately 9 months after discharge, the patient presented to the emergency department following another epileptic seizure, accompanied by shortness of breath and hemoptysis of about 200 mL. The physical examination findings and laboratory parameters were similar to those of the previous admission. Chest radiography revealed an increase in bilateral heterogeneous opacities, more pronounced in the central region of the right lung (Figure-1d). Computed tomography of the chest revealed the presence of diffuse, scattered areas of increased nodular density, which were particularly prominent in the middle and lower lobes. Additionally, focal ground-glass opacities were observed (Figure-1e).

The patient was monitored in the Chest Diseases Clinic with oxygen inhalation therapy. No pathological findings were observed on the computed tomography of the brain. The epilepsy treatment was adjusted. During follow-up, the patient's hemoptysis had completely regressed, and significant improvement was noted in respiratory symptoms and in the lesions on chest radiography (Figure-1f). The patient's condition is being monitored stably with regular follow-ups in the Chest Diseases and Neurology departments.

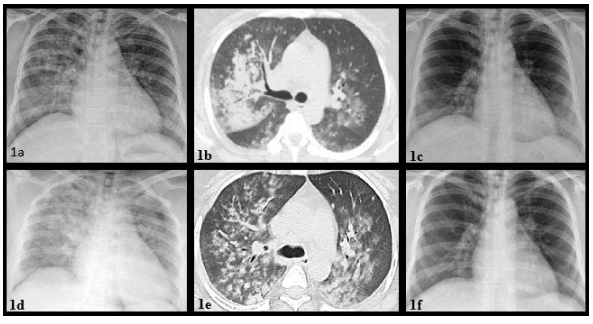

Figure-1. Figures-1a and 1b show the chest X-ray and tomography findings taken at the patient's first application; Figure-1c shows the chest X-ray findings taken at discharge. Figures-1d and 1e show the chest X-ray and tomography findings taken at the re-application 9 months after discharge; and Figure-1f shows the discharge X- ray.

CASE REPORT 2

A 32-year-old female patient with a known diagnosis of epilepsy was brought to the emergency department after fainting while at work. She was unable to recall the events preceding or following the incident. According to her relatives, she experienced convulsions during the episode. The patient had previously reported occasional shortness of breath and palpitations with exertion. She is currently undergoing treatment with metoprolol for mitral regurgitation. The patient has a documented history of smoking of 15 pack-years and is an active smoker. The patient's vital signs at the time of initial presentation to the emergency department were recorded as follows: body temperature 36.9°C, heart rate 119 beats per minute, blood pressure 135/90 mmHg, and peripheral oxygen saturation of 78% (room air). On respiratory examination, bilateral rales were auscultated in the middle and lower lung zones. There was no evidence of edema in the pretibial region. Neurological examination revealed a Glasgow Coma Scale (GCS) score of 15, with the patient being conscious, oriented, and cooperative. Motor strength was assessed as 5/5 in all four extremities. The echocardiogram demonstrated a normal ejection fraction and identified grade 1 mitral regurgitation. Upon examination of the laboratory values, C-reactive protein was 0.17 mg/dL, creatinine was 0.93 mg/dL, BNP was 486 pg/mL, troponin was 12 ng/L, leukocyte count was 8.980/mL, hemoglobin was 14.1 g/dL, and platelet count was 219.000/µL. The pH of the arterial blood gas was 7.40, with a PO2 of 44 mmHg, a PCO2 of 25 mmHg, and a SO2 of 79%. Computed tomography of the chest demonstrated ground-glass opacities and consolidated areas in all lobes and segments of both lungs (Figures-2a-2b). The brain computed tomography did not reveal any findings suggestive of acute intracranial stroke, hemorrhage, or mass effect. The patient was admitted to the intensive care unit with a diagnosis of neurogenic pulmonary edema. Symptomatic treatment was initiated. During follow-up, the patient did not report any respiratory complaints, required no supplemental oxygen after 48 hours, and showed significant regression on chest radiography. The patient was discharged with recommendations for follow-up in the neurology and pulmonology outpatient clinics (Figure-2c).

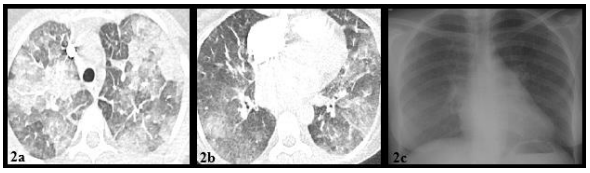

Figure-2. Figures-2a and 2b show the tomography findings taken at the patient's first application; Figure-2c shows the chest X-ray findings taken at discharge.

DISCUSSION

Both cases presented with a diagnosis of epilepsy and neurogenic pulmonary edema, which developed following epileptic seizures. A review of the literature revealed that pulmonary edema occurring after epileptic seizures is rare but can increase mortality (4).

The pathophysiology of neurogenic pulmonary edema has not been fully elucidated. The most widely accepted mechanism is non-cardiogenic alveolar edema resulting from increased adrenergic response secondary to elevated intracranial pressure, leading to increased catecholamine levels and elevated pulmonary hydrostatic pressure (5, 6). The most common causes of neurogenic pulmonary edema are head trauma, subarachnoid hemorrhage, encephalitis, brain tumors, stroke, and epileptic seizures (7). Due to the absence of established diagnostic criteria, diagnosis can be challenging. In cases, acute onset of dyspnea, tachypnea, pink frothy sputum, tachycardia, and a decrease in peripheral oxygen saturation are typically observed, often accompanied by neurological symptoms and signs. In the presence of a neurological condition that increases intracranial pressure, arterial blood gas analysis typically reveals hypoxemia, and chest radiography shows centrally weighted opacities (1). Due to the absence of specific diagnostic criteria for neurogenic pulmonary edema, it is essential to exclude other potential causes of pulmonary edema (3). The fundamental principles of treatment for neurogenic pulmonary edema include ensuring adequate oxygenation and providing sufficient ventilatory support with positive end-expiratory pressure, as well as reducing intracranial pressure. Although there is limited data regarding mortality, a study involving 21 cases reported a mortality rate of 10%. Similar to our two cases, most patients improve within 48 to 72 hours with supportive treatment (8).

CONCLUSION

Neurogenic pulmonary edema is a relatively uncommon condition, and acute pulmonary edema is predominantly of cardiogenic origin. However, in patients with central nervous system pathology and no cardiac pathology to explain the condition, the presence of accompanying dyspnea, cough, hypoxemia, and bilateral opacities on chest radiography should raise suspicion for neurogenic pulmonary edema.

Conflict of interest: None of the authors have conflict of interest to declare.

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