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# Neurogenic Pulmonary Edema: A Case Report

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**Abstract** Background: Neurogenic pulmonary edema (NPE) is a rare pulmonary edema that develops rapidly after an acute central nervous system injury. The mechanism by which neurological insult leads to pulmonary edema is not well understood. The clinical presentation of NPE varies widely and ranges from asymptomatic cases to fulminant cases. Clinical signs in pure NPE are usually those of acute pulmonary edema, without signs of left ventricular failure. Case Report: The author presents a case of NPE is a young female patient with a brief epileptic seizure in which NPE resolved within 48 hours. Patient had no respiratory symptoms but was initially placed on antibiotics because her chest x-ray showed bilateral lung patchy opacifications in a perihilar distribution suggestive of pulmonary edema and/or multifocal pneumonia. Antibiotics was discontinued when the pulmonary edema resolved. Conclusion: There is need for physicians to be aware of NPE to avoid unnecessary antibiotics in asymptomatic patients who present with bilateral pulmonary infiltrates following an epileptic seizure.

***Keywords:*** *Neurogenic pulmonary edema, epilepsy, seizure disorder, case report*

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## 1. Introduction

Neurogenic pulmonary edema (NPE) is pulmonary edema that develops rapidly after an acute central nervous system injury. The primary precipitants of NPE include epileptic seizures, intracranial hemorrhage and traumatic brain injury. [1,2]

Neurogenic pulmonary edema secondary to epileptic seizure is rare and is more likely to be seen with status epilepticus. [3] However, more than 80% of epileptics who die unexpectedly of seizure were found to have NPE at autopsy. [4]

We present a case of NPE in a young female patient with a brief epileptic seizure in which NPE resolved within 48 hours without treatment.

## 2. Case Presentation

A 44-year-old female patient presented to the emergency department with chief complaint of a seizure with onset one hour prior to arrival. The patient had a witnessed tonic-clonic seizure that lasted about 30 seconds. She stated that she was sitting on the couch watching a movie when she passed out. She stated that she didn't remember anything until she woke up and saw the paramedics. After additional questioning, she stated that she initially felt a bit confused and had bit her tongue during the seizure. The patient also stated that this was her second seizure episode. Her first seizure had occurred 6 months prior to this episode. She stated that she was not on any seizure medication.

The patient denied fever, chills, headache, head trauma, change in vision, dizziness, chest pain, shortness of breath, or urinary symptoms. She said she had started coughing post seizure, but denied chronic cough, shortness of breath, pleuritic chest pain or abdominal pain. She had no prior history of known pulmonary disease.

In the emergency department, her blood pressure was 157/102mmHg, heart rate 119, respiratory rate 18, and temperature 98.3F. Physical examination revealed a patient that was alert and oriented to person, place and time with no other remarkable findings. Laboratory tests were grossly unremarkable. Hemoglobin was 12.1g/dl, white blood cell count of 7,800/ml, and platelet of 356,000/ml. The urinalysis, blood alcohol screen and urine drug screen were normal. Hepatic and renal parameters were normal.

Arterial blood gas (ABG) showed mild hypoxemia on room air. PH 7.38, Pco2 32, PO2 53, HCO3 18.9. CT brain showed no acute intracranial hemorrhage. The chest radiograph showed bilateral lung patchy opacifications in a perihilar distribution suggestive of pulmonary edema and/or multifocal pneumonia. [Figure 1] Chest CT angiogram showed bilateral severe patchy opacification in perihilar distribution. [Figure 2 and Figure 3]

The patient received lorazepam and was started on IV ceftriaxone, IV azithromycin and Levetiracetam. She remained asymptomatic without fever or cough. A repeat chest radiograph two days later showed complete resolution of the pulmonary infiltrates [Figure 4] and IV antibiotics were discontinued.

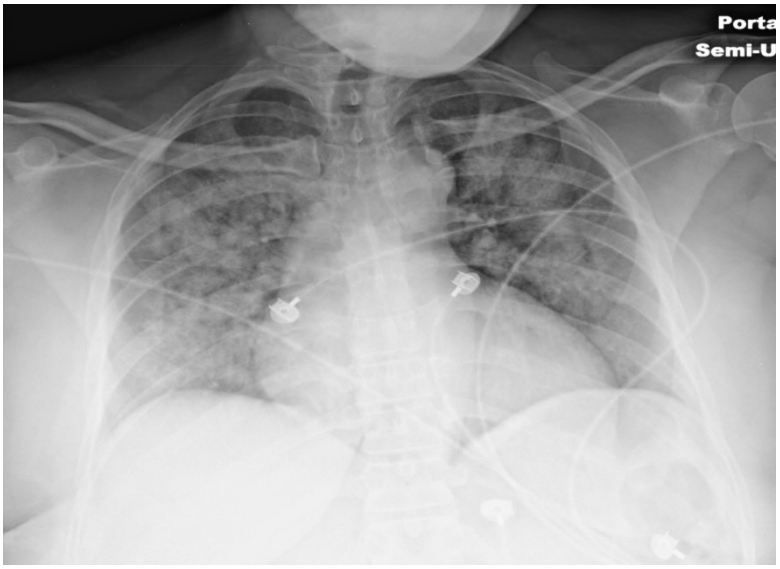

**Figure 1.** Chest x-ray on admission showing diffuse bilateral airspace opacities with normal heart size

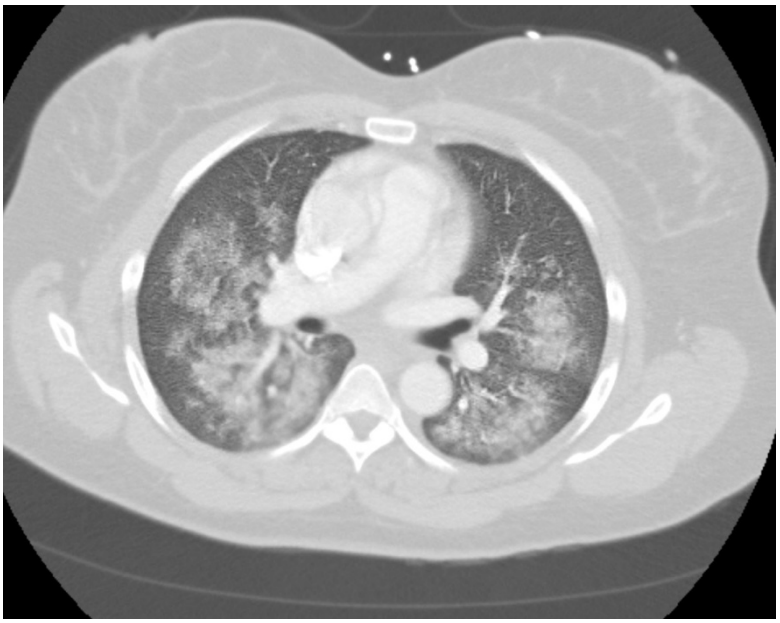

**Figure 2.** CT chest on admission showing patchy opacities typically in a perihilar distribution and/or ground glass opacities (transverse plane)

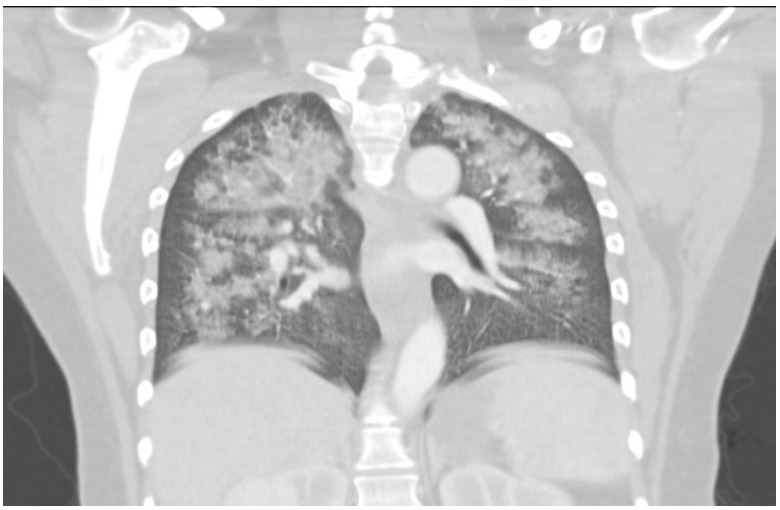

**Figure 3.** CT chest on admission showing patchy opacities typically in a perihilar distribution and/or ground glass opacities (coronal plane)

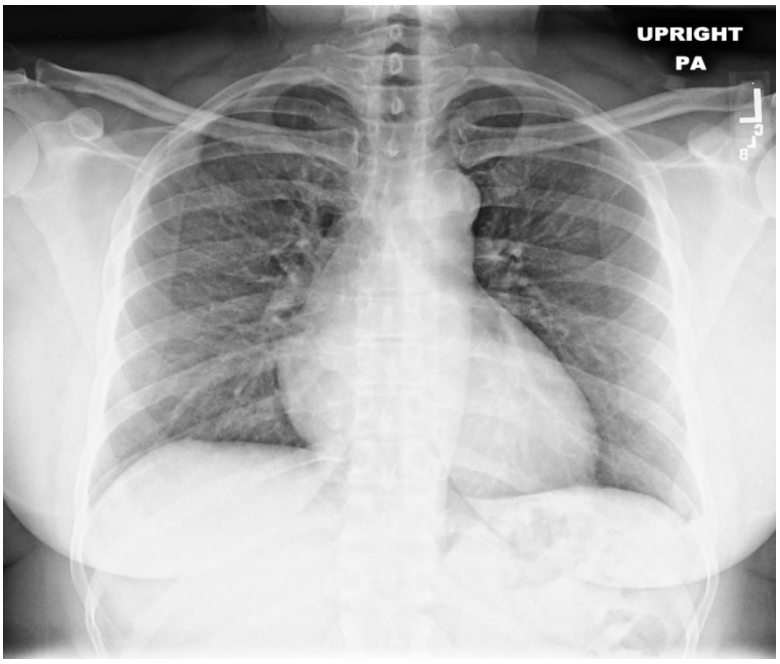


**Figure 4.** Chest x-ray after 2 days showing complete resolution of the pulmonary infiltrates

# 3. Discussion

## 3.1. Pathogenesis

The mechanism by which neurological insult leads to pulmonary edema is not well understood. However, the etiology is thought to be due to a surge in endogenous catecholamines that results in a change in cardiopulmonary hemodynamics and Starling forces in a way that increases pulmonary capillary permeability or fluid movement from the capillaries to the pulmonary interstitium, or both. [1,2]

## 3.2. Clinical Presentation

Two types of NPE have been described. Early onset NPE is the most common and typically develops within minutes to hours following a severe central nervous system insult such as status epilepticus, traumatic brain injury or intracranial hemorrhage. In contrast, delayed onset NPE develops within hours to days of a CNS insult. [2,5]

The clinical presentation of NPE varies widely and ranges from asymptomatic cases to fulminant cases. Clinical signs in pure NPE are usually those of acute pulmonary edema, without signs of left ventricular failure. [6] Sudden onset of dyspnea, tachypnea and hypoxemia are the most common symptoms. Patients also commonly present with signs of sympathetic hyperactivity such as fever, tachycardia and hypertension. Leukocytosis and mild hemoptysis/blood tinged sputum have been reported. [1,5] Physical examination usually shows bilateral crackles and rales. Chest radiographs reveal bilateral hyperdense infiltrates consistent with acute respiratory distress syndrome (ARDS). Symptoms usually resolve within 24 to 48 hours. [1]

Our patient presented with early onset NPE (NPE developed within one hour of her seizure episode) and was tachycardic, hypoxic and hypertensive on presentation. WBCs were normal on the day of presentation, were elevated the next day and returned back to normal within 48 hours. Unlike most cases of NPE, our patient was not dyspneic on presentation and her lungs were clear on auscultation. She had intermittent mild cough and two episodes of blood tinged sputum which the patient attributed to bleeding from her tongue which she had bitten during her seizure. Her chest radiograph revealed extensive bilateral infiltrates which resolved within 48 hours.

## 3.3. Differential Diagnosis

There are other conditions that mimic NPE. The diagnosis of NPE requires documentation of non-cardiogenic pulmonary edema following neurological injury and exclusion of other causes of pulmonary edema. [5] Aspiration pneumonia and pulmonary edema are conditions that could mimic NPE and can occur following a CNS injury.

Aspiration pneumonia differs from NPE because NPE tends to develop more rapidly with diffuse bilateral opacities on radiograph, while fever and focal opacities in the dependent portions of the lungs favor aspiration pneumonia. [5,6] In addition, NPE tends to resolve more rapidly (1 to 3 days) than aspiration pneumonia (1 to 3 weeks). [6] Pulmonary edema from acute respiratory distress syndrome (ARDS) and heart failure should also be considered. A diagnosis of NPE should be made in the absence of other causes of ARDS such as aspiration, sepsis and massive blood transfusion and the absence of pulmonary edema secondary to heart failure. [5]

## 3.5. Treatment

Treatment for NPE is usually supportive and is focused on treating the underlying neurological injury. Seizures are treated with anticonvulsants, clots are evacuated in intracranial hemorrhage and intracranial pressure is reduced in head trauma, in order to stop the sympathetic discharge responsible for causing the lung injury. Most NPE patients are hypoxemic and may require supplemental

oxygen or mechanical ventilation. [1,2] Medications such as dobutamine, milrinone, and chlorpromazine have been used to treat patients with severe hypoxemia secondary to NPE, but their efficacy has not been proven. [7,8,9,10] Most cases of NPE resolve within 48-72 hours. [1,2,6]

## 4. Conclusion

NPE secondary to epileptic seizure is rare and most cases resolve within 48-72 hours. However, physicians need to be aware of the condition in order to avoid unnecessarily giving antibiotics to asymptomatic patients who present with bilateral pulmonary infiltrates following an epileptic seizure.

## References

[1] Davison DL, Terek M, Chawla LS. Neurogenic pulmonary edema. *Crit Care*. 2012;16(2):212. Published 2012 Dec 12.

[2] Wemple M, Hallman M, Luks AM. Neurogenic pulmonary edema. Parsons PE, Finlay G, editors. UpToDate [Internet]. Waltham, MA: UpToDate Inc. Available from https://www.uptodate.com (Accessed on December 04, 2019.).

[3] Simon RP: Neurogenic pulmonary edema. Neurol Clin 1993, 11: 309-323.

[4] Wayne SL, O'Donovan CA, McCall WV, Link K: Postictal neurogenic pulmonary edema: experience from an ECT model. Convuls Ther 1997, 13: 181-184.

[5] Colice GL. Neurogenic pulmonary edema. Clinics in chest medicine. 1985 Sep; 6(3): 473-89.

[6] Baumann A, Audibert G, McDonnell J, Mertes PM. Neurogenic pulmonary edema. Acta Anaesthesiologica Scandinavica. 2007 Apr; 51(4): 447-55.

[7] Knudsen F, Jensen HP, Petersen PL. Neurogenic pulmonary edema: treatment with dobutamine. Neurosurgery 1991; 29:269.

[8] Deehan SC, Grant IS. Haemodynamic changes in neurogenic pulmonary oedema: effect of dobutamine. Intensive Care Med 1996; 22: 672.

[9] Chi CY, Khanh TH, Thoa le PK, et al. Milrinone therapy for enterovirus 71-induced pulmonary edema and/or neurogenic shock in children: a randomized controlled trial. Crit Care Med 2013; 41: 1754.

[10] Wohns RN, Tamas L, Pierce KR, Howe JF. Chlorpromazine treatment for neurogenic pulmonary edema. Crit Care Med 1985; 13:210.

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