

A Case of Pulmonary Edema Induced By Seizures

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Abstract: A 44-year-old woman suffered from tonic-clonic seizures while eating lunch. She had chronic renal failure due to acute glomerular nephritis, idiopathic thrombocytopenic purpura, hypertension and glaucoma. On arrival at the hospital, she had clear consciousness and peripheral oxygen saturation of 96% under 2 L/minute of oxygen through a nasal cannula. Chest X-ray, electrocardiogram and cardiac echo findings were negative. However, whole-body computed tomography (CT) showed a sporadic, patchy ground-glass appearance, suggesting pulmonary edema. She received a diagnosis of convulsion with neurologic lung edema and underwent observational admission with 2 L/minute of oxygen. On the second day, she had no complaints, and her peripheral oxygen saturation increased to 99% without oxygen therapy. On the third day, chest CT demonstrated almost clear lung fields. We herein report a rare case of pulmonary edema induced by seizures and discuss the complexity of the mechanisms underlying pulmonary edema induced by seizures.

Keywords: seizure; lung edema; mechanism.

INTRODUCTION

Pulmonary edema induced by epilepsy has been reported only rarely [1]. However, it is important to understand the mechanisms underlying this entity. We herein report a case of epilepsy-induced pulmonary edema and discuss the etiology.

CASE REPORT

A 44-year-old woman suffered from tonic-clonic seizures while eating lunch. She had chronic renal failure due to acute glomerular nephritis, idiopathic thrombocytopenic purpura, hypertension and glaucoma, which were treated by steroids, eltrombopag, telmisartan, 1-hydroxycholecalciferol, and rebamipide. When emergency medical technicians (EMTs) checked her, she was in a disoriented state and had a systolic blood pressure of 139 mmHg, heart rate of 81 beats per minute, respiratory rate of 30 breaths per minute and peripheral oxygen saturation of 82% under room air, so she received 6 L/minute of oxygen through a face mask. On arrival at the hospital, she had clear consciousness, blood pressure of 175/107 mmHg, heart rate of 100 beats per minute, respiratory rate of 22 breaths per minute and peripheral oxygen saturation of 96% under 2 L/minute of oxygen through a nasal cannula. Her physiological findings were negative, including respiratory sounds and neurological findings. Chest X-ray, electrocardiogram and cardiac echo findings were negative. However, whole-body computed tomography (CT) showed a sporadic, patchy ground-glass appearance, suggesting pulmonary edema (Figure 1).

The results of a blood gas analysis on admission (room air) were as follows: pH, 7.336; PCO₂, 34.4 mmHg; PO₂, 62.0 mmHg; HCO₃⁻, 17.9 mmol/L; base excess, -6.6 mmol/L; Lactate, 7.0 mmol/L; and oxygen saturation, 88.5%. A complete blood count revealed the following findings: white blood cells, 12,700/mm³; hemoglobin, 12.8 g/dl and platelets, 4,900/mm³. Serum biochemical analyses revealed the following findings: total bilirubin, 0.4 mg/dl; aspartate aminotransferase, 20 IU/L; alanine aminotransferase, 10 IU/L; total protein, 7.2 g/dl; glucose, 110 mg/dl; blood urea nitrogen, 10.1 mg/dl; creatinine, 1.43 mg/dl; creatine phosphokinase, 74 IU/L; sodium, 141 mEq/L; potassium, 4.6 mEq/L; chloride, 106 mEq/L; C-reactive protein, 0.3 mg/dl; prothrombin time; 12.3(12.0) sec, activated partial thromboplastin time; 30.7 (27.4) sec; and D-dimer, 1.9 µg/ml. She received a diagnosis of convulsion with neurologic lung edema and underwent observational admission with 2 L/minute of oxygen. On the second day, she had no complaints, and her peripheral oxygen saturation increased to 99% without oxygen therapy. On the third day, chest CT demonstrated almost clear lung fields (Figure 1). She was discharged without sequelae.



Fig-1: Chest computed tomography (CT) on arrival (upper) and at the third day (lower). CT showed a sporadic, patchy ground-glass appearance, suggesting pulmonary edema these lesions resolved spontaneously

DISCUSSION

The differential diagnosis of pulmonary edema is inflammation, hypo-osmolality, overhydration, heart failure and neurogenic edema. As she had normal values for leukocytes, C-reactive protein and total protein, the possibility of inflammation and hypo-osmolality was low. As her history did not include heart or liver diseases, the possibility of overhydration was also denied. As she had thrombocytopenia and temporary unconsciousness, she might have had aspiration pneumonia or alveolar hemorrhaging [2, 3]. However, such diseases usually did not improve within a few days without specific treatment. In addition, aspiration pneumonia is typically characterized by the posterior segment of the upper lobes and the superior segment of the lower lobes being the involved lung sites when aspiration occurs in a recumbent patient. In an erect patient, aspiration is more likely to involve the bilateral basal segments, middle lobe and lingula, findings that were not noted in the present case [2]. However, she was under a stressed condition due to her tonic-clonic seizures. Accordingly, we concluded that she most likely had neurologic lung edema, which can resolve spontaneously in a short period of time [1].

Seizures can cause Takotsubo cardiomyopathy following lung edema [4]. However, our examinations on arrival did not indicate complication with Takotsubo cardiomyopathy. Neurologic lung edema tends to be caused by any of three major mechanisms. The first mechanism involves inflammatory mediators secondary to endothelial damage, which increase the capillary permeability, leading to edema [1]. The second mechanism involves hypoxia induced by seizures, which can cause lung edema [5]. Hypoxia leads to pulmonary vasoconstriction and increased permeability of the pulmonary vasculature [5]. The third mechanism involves intense pulmonary and systemic

vasoconstriction as a response to adrenergic release by brain injury, leading to an increase in the capillary hydrostatic pressure and capillary permeability [1]. The second and third mechanism is the one most compatible with the present case.

CONCLUSION

We encountered a rare case of pulmonary edema induced by seizures and discussed the complexity of the mechanisms underlying pulmonary edema induced by the seizures.

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