Bilateral pneumothoraces and pneumomediastinum complicating a generalized tonic-clonic seizure

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ABSTRACT

Patients often present to the emergency department following a generalized tonic-clonic seizure, particularly of new onset. Complications associated with seizures usually arise from injuries sustained from loss of consciousness or during convulsive activity. This report describes a patient with an idiopathic seizure who developed postictal alveolar edema and delayed bilateral pneumothoraces and pneumomediastinum. A literature search revealed only 1 other case of this potentially life-threatening pulmonary complication from seizures.

Keywords: seizure, pneumothorax, neurogenic pulmonary edema

RÉSUMÉ

Il est fréquent que les patients se présentent à l'urgence à la suite d'une crise tonico-clonique généralisée particulièrement lorsqu'il s'agit d'un premier épisode. Les complications des crises convulsives surviennent habituellement de blessures engendrées par la perte de conscience au cours d'épisodes convulsifs. Ce rapport décrit le cas d'un patient ayant présenté une crise convulsive idiopathique et chez qui sont survenus subséquemment un œdème alvéolaire postcritique ainsi qu'un pneumothorax bilatéral tardif et un pneumomédiastin. Une recherche dans la littérature révèle un seul cas de ce type occasionnant des complications pulmonaires potentiellement mortelles d'une crise convulsive.

Introduction

Patients often present to the emergency department (ED) after having a seizure, particularly following a first episode. Those with an established seizure disorder may only seek medical attention if the seizure results in injury or other unexpected complications. It is important for emergency physicians to recognize and appropriately manage the potential life-threatening complications of seizures.

Case report

An 18-year-old man was brought to the ED after a witnessed, generalized, tonic-clonic seizure. He had experienced an uncomplicated idiopathic seizure 1 year before presentation but was not treated with antiepileptic medication. He was a nonsmoker with no recent alcohol or illicit drug use. There was no history of febrile seizures and no family history of epilepsy.

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The initial oxygen saturation by pulse oximetry noted by paramedics was 64% on room air. On presentation to the ED, the patient was alert and oriented to person, place and time. His temperature was 37.5°C and he was hemodynamically stable, with a blood pressure of 146/66 mm Hg and a heart rate of 85 beats/minute. He was tachypneic, with a respiratory rate of 30 breaths per minute. His pulse oximetry oxygen saturation increased to 97% with a non-rebreather mask. Physical examination revealed bilateral diminished breath sounds at the bases, with no adventitious sounds. The patient's jugular venous pressure was not elevated and he had no S3 or murmurs. His abdominal and neurologic systems were normal. An arterial blood gas on a non-rebreather mask found hypoxia with an oxygen partial pressure of 71 mm Hg and a normal pH. Blood was drawn for electrolytes, complete blood count, creatinine and liver enzymes. Toxicology screening for cocaine, benzodiazepines, acetaminophen and ethanol was performed and the results of all these tests were within the normal range. An electrocardiogram showed normal sinus rhythm.

The patient's first portable chest radiograph showed bilateral alveolar edema (Fig. 1). A contrast CT scan of the chest was performed to exclude a pulmonary embolism. It showed bilateral airspace disease suggestive of either chemical pneumonitis or pulmonary edema (Fig. 2).

There were no pulmonary emboli, apical bullae or pneumothoraces noted on the CT. A repeat chest radiograph performed 6 hours after admission showed bilateral perihilar airspace opacities. Positive pressure ventilation by bag mask ventilation, continuous positive airway pressure or endotracheal intubation was not administered during any time in the prehospital or in-hospital periods. A CT scan of the patient's head was normal.

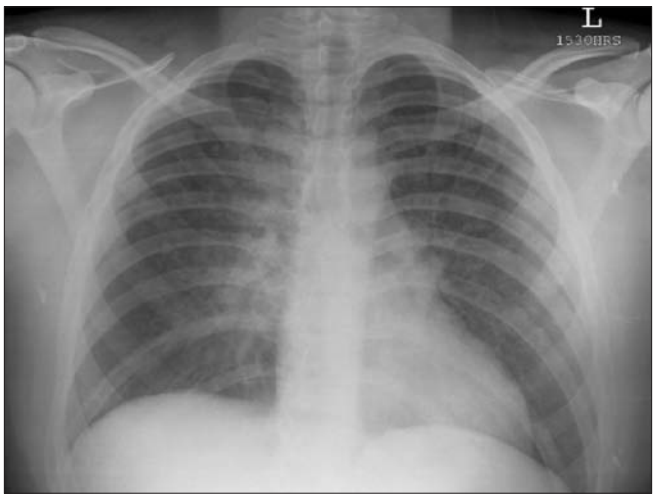

Fig. 1. The first portable chest radiograph showing bilateral alveolar edema.

The patient was admitted to the intermediate stepdown unit and was given an intravenous loading dose of phenytoin followed by daily oral maintenance dosing. He had no further seizure activity; however, he remained hypoxic with an oxygen partial pressure of 69 mm Hg despite continuous oxygen administration by a non-rebreather mask. Intravenous metronidazole and ceftriaxone were administered prophylactically for possible aspiration. A repeat portable chest radiograph 9 hours after admission unexpectedly showed small bilateral pneumothoraces and moderate pneumomediastinum in addition to bilateral alveolar edema (Fig. 3).

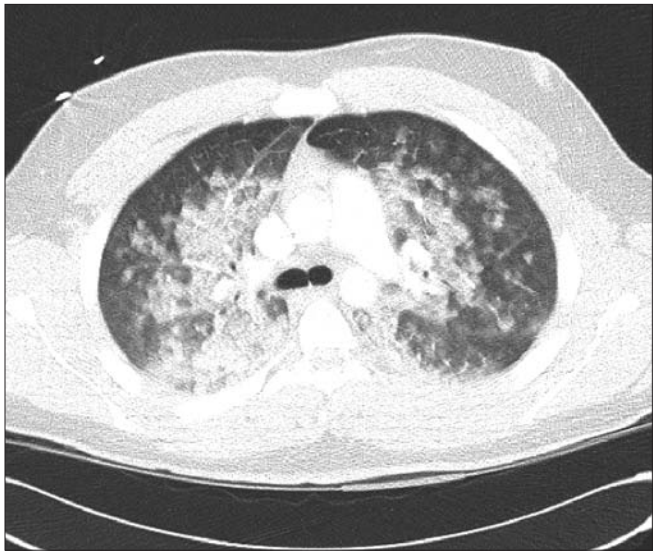

Fig. 2. A contrast CT scan of the chest showing bilateral airspace disease.

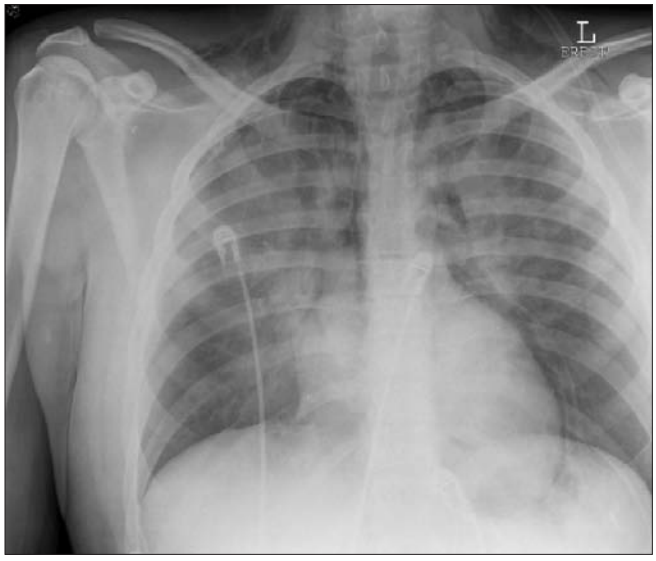

Fig. 3. A repeat chest radiograph 9 hours after admission to the emergency department showing alveolar edema, small bilateral pneumothoraces and pnuemomediastium.

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Bilateral chest tubes were inserted and the oxygen requirements subsequently reduced to 2–4 L/minute by nasal prongs. A modified gastrograffin swallow was performed and showed no evidence of an esophageal tear or rupture. There was progressive resolution of the pneumothoraces and pneumomediastinum, with subsequent improvement in oxygenation over the ensuing days. The chest tubes were removed uneventfully and a full course of antibiotics was completed. The patient was discharged with neurology follow-up 10 days after admission. One year after discharge, he reported being well with no further seizures or pulmonary sequelae.

Discussion

Complications of seizures are most often related to direct injuries sustained during convulsive activity or as a result of an abrupt reduction in the level of consciousness.[1,2] Head injuries, fractures, cognitive decline, motor vehicle collisions, falls, burns, drowning and sudden unexplained death are described consequences of seizures.[3] Described pulmonary complications of seizures include the aspiration of gastric contents and neurogenic pulmonary edema.[4–7]

A pneumothorax can occur spontaneously in an individual with no underlying pulmonary disease and is termed primary in such situations. Risk factors associated with a primary pneumothorax include smoking, family history, Marfan syndrome and a tall thin body habitus.[8] In contrast, a secondary pneumothorax is one that occurs as a complication of underlying pulmonary disease. These are associated with a variety of conditions, including chronic obstructive pulmonary disease, bronchogenic carcinoma, turberculosis, sarcoidosis and idiopathic pulmonary fibrosis.[9] The combined annual incidence of primary and secondary pneumothorax is approximately 24 per 100 000 for men and 9.8 per 100 000 for women.[9]

The association between pneumothorax and generalized tonic-clonic seizures is poorly described. We identified only 1 case report in the French literature that describes bilateral pneumothoraces and pneumomediastinum immediately following a seizure. The authors proposed that the likely mechanism related to increased intra-alveolar pressure generated by expiratory effort against a closed glottis during seizure activity.[10] This pressure was hypothesized to cause rupture of alveoli at the lung periphery with a subsequent escape of air into the mediastinum and pleural space.

When the chest CT of our patient was reviewed again, in light of the findings on subsequent plain radiographs, no pneumothoraces were identified. We speculate that an intense Valsalva manouver during a generalized tonic-clonic seizure, combined with bilateral airspace disease, may have predisposed our patient to develop this pulmonary complication.[11] The pulmonary edema, which was noted on both the chest radiograph and the CT scan, was likely neurogenic in origin.[12] However, the delay between the seizure and the subsequent development of the pneumothoraces and pneumomediastinum is very unusual.

Conclusion

We describe an uncommon occurrence of delayed bilateral pneumothoraces after a seizure complicated by alveolar edema. Our case highlights the need for physicians to be aware of this potentially life-threatening pulmonary complication from seizures and, when faced with postictal hypoxia, to regularly review the differential diagnosis to reduce the likelihood of missing a treatable problem.

Competing interests: None declared.

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