Case Report

A rare case of post-ictal diffuse alveolar hemorrhage due to seizures

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ABSTRACT

Diffuse alveolar hemorrhage is persistent or recurrent pulmonary hemorrhage that occurs due to a variety of causes. Here, we present the case of a 15-year-old male child who presented with chief complaints of involuntary jerking movements of the entire body in the morning followed by coughing out a massive amount of blood. At presentation, the patient's blood glucose level was high. Bronchoscopy revealed bleeding in the middle and lower lobes of both lungs. Computed tomography (CT) brain was suggestive of cerebral edema and the CT chest was suggestive of diffuse opacities in bilateral lung fields. The patient was started on corticosteroids, antiplatelet drugs, antiepileptics, insulin, and oxygen inhalation which helped the patient to recover and was discharged in a week's time.

Key words: Diffuse alveolar hemorrhage, Hemoptysis, Post-ictal, Seizures

Diffuse alveolar hemorrhage (DAH) is persistent or recurrent pulmonary hemorrhage that is most commonly caused due to autoimmune conditions. It involves widespread damage to pulmonary small vessels, leading to a collection of blood within the alveoli. Vasculitis, pulmonary tuberculosis, coagulation defects, certain drug toxicities (propylthiouracil, amiodarone, methotrexate, montelukast, and valproic acid), and toxins like cocaine are some of the well-known causes for DAH [1,2]. According to the Proceedings of UCLA Health, there might be neuronal compression and ischemia which results in a catecholamine surge [3].

In this particular case report, we observed that the patient develops DAH following a seizure which is unusual. This cause of DAH is very rare and only 13 cases have been reported so far according to the Official Case Reports Journal of the Asian Pacific Society of Respirology [4].

CASE REPORT

A 15-year-old male child who was apparently normal in the morning hours developed a sudden onset of involuntary stiffening and jerking movements of the body. The movements were tonic-clonic type, consisting of four episodes, each of which lasts for 2–3 min, and associated with loss of consciousness, uprolling of eyes, tongue bite, and pooling of secretions in the mouth. There was no preceding aura and the movements were followed by involuntary passage of urine. Following the involuntary movements, the patient complained of a single episode of coughing out fresh blood (approximately 700 mL) associated with froth and associated with shortness of breath. Now, the patient also complained of severe headache. There was a history of skipping seizure medication and insulin. The patient is a known case of seizure disorder diagnosed 10 years ago for which no documentation was available and is currently on phenytoin 100 mg twice daily. The patient is also a known case of Type 1 diabetes mellitus for 10 years and is on insulin therapy.

On general examination, the patient was conscious, coherent, and cooperative. His vitals were as follows: Blood pressure 100/80 mmHg, pulse rate 100 bpm with regular rhythm and normal volume, respiratory rate 30/min, and SpO_2 of 60%. The patient had four episodes of seizures and a single episode of massive hemoptysis at home. Post-admission, there were no new seizures and hemoptysis. The central nervous system examination showed that the patient was oriented to time, place, and person and there were no cranial nerve abnormalities detected. The bulk and tone of the muscles were normal on both sides and in both upper and lower limbs. The power was grade 5/5. Superficial and deep reflexes were intact, gait was normal, all sensations were intact, and no meningeal or cerebellar signs were seen. On respiratory examination, the trachea was central in position. The apex beat was present in the left fifth intercostal space. On palpation, there was decreased expansion of the chest on inspiration and the presence of increased vocal fremitus. On percussion, a dull note was heard in the interscapular region. On auscultation, decreased breath sounds were heard in the middle and lower lobes of the lung.

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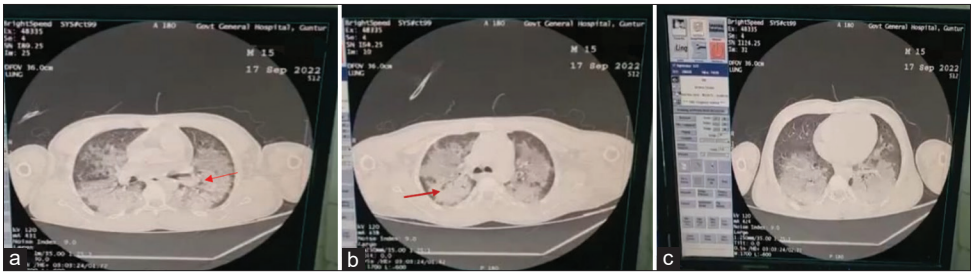

Figure 1: Axial high-resolution computed tomography of the chest (a) at the level of main bronchi shows diffuse heterogeneous opacification of bilateral lower lobes in basal segments and ground glass opacification noted in bilateral anterior segments; (b) at the level of carina shows diffuse opacification of bilateral upper lobes. Ground glass opacification in anterior segments with mild sparing of bilateral subpleural areas; (c) at the level of both ventricles showing homogenous opacification of bilateral basal zones without subpleural sparing

On workup of the patient, the complete blood picture, prothrombin time, international normalized ratio, activated partial thromboplastin time, bleeding and clotting, and D-dimer levels were normal. General random blood sugar was 582 mg/dL. The liver and renal function tests were normal. The lactate dehydrogenase enzyme and serum ferritin were within normal limits but C-reactive protein was elevated (10.6 mg/dL). A complete urine examination was normal. The sputum for the cartridge-based nucleic acid amplification test and culture and sensitivity test was negative. The antinuclear antibody profile, perinuclear antineutrophil cytoplasmic antibody, and antineutrophil cytoplasmic antibody were negative.

The chest X-ray shows bilateral middle and lower lobes opacities. High-resolution computed tomography (HRCT) chest shows diffuse opacities in bilateral lung fields and CT brain shows cerebral edema (Fig. 1). Bronchoscopy revealed bloody return in the lavages. No evidence of infection or malignancy was found in bronchoalveolar lavage. The upper gastrointestinal endoscopy was normal.

The patient was started on intravenous (IV) methylprednisolone, tranexamic acid, ethamsylate, levipil, optineurin, piptaz, pantop, and ondansetron. Human mixtard (20/25) was given subcutaneously. Paracetamol and Montek LC tablets were given. Oxygen inhalation at 12 L/h was started. Nebulization with duolin and budecort was advised. IV antibiotic was used to prevent any nosocomial infection and was stopped after day 5. Oxygen inhalation was slowly veined off. No new seizure episode was reported and hemoptysis was controlled. The patient was subsequently discharged after a week with stable vitals and was advised to adhere to the seizure medication and insulin.

DISCUSSION

Post-ictal DAH is a very rare cause of DAH. Many studies have cited different causes for this association of DAH with seizures. In a previous case study done by You and Stoller, there was a seizure episode preceding the episode of massive hemoptysis, and the patient was thought to have post-ictal DAH where pulmonary capillaries ruptured due to an increase in negative pressure as the patient tried to inhale against a closed glottis (Muller's maneuver) [4]. This explanation is similar to a case study done by Lorch and Sahn which states that pulmonary edema due to upper airway obstruction can be observed in a variety of clinical situations. The predominant mechanism is increased negative intrathoracic pressure, although hypoxia, cardiac, and neurologic factors may also contribute [5].

Neurologic insults can cause both pulmonary and hydrostatic abnormalities through separate mechanisms. Excessive alpha-adrenergic-mediated arterial spasm may occur and cause transient left ventricular dysfunction. Inappropriately increased vagal tone may also contribute to the left ventricular failure, increased hydrostatic pressures, and pulmonary edema [6]. There might be a catecholamine surge that causes left ventricular pressure overload. As such, backup of blood flow into the left atrium, pulmonary veins, and pulmonary circuit contributes to pulmonary edema (neurogenic) and this is a potential diagnosis of exclusion [3,5].

Antiepileptic drugs such as valproic acid can precipitate DAH. Valproic acid has negative feedback on both intrinsic and extrinsic coagulation pathways [7]. However, this patient was never on valproate. Systemic vasculitis and collagenous diseases (e.g., Goodpasture's syndrome, antineutrophil cytoplasmic antibody-associated vasculitis, systemic lupus erythematosus), heart diseases (e.g., mitral stenosis), and exogenous factors (e.g., chemicals, drugs) are known as causes of pulmonary hemorrhage [8]. However, in our case, all these conditions have been ruled out.

Chest radiography and chest CT are of limited value in distinguishing hemorrhage from infection or other causes of diffuse alveolar opacification. Bronchoalveolar lavage is often required to identify or confirm alveolar hemorrhage and exclude infection, but it has limited utility in establishing an underlying cause. Increasing blood return in serial lavage aliquots is highly suggestive of alveolar hemorrhage and helps distinguish alveolar from the endobronchial disease [9]. Symptom relief is the mainstay of management of post-ictal DAH. Negative pressure pulmonary edema-related DAH is a rare life-threatening condition occurring primarily after a tonic–clonic generalized seizure or generalized anesthesia. Early diagnosis and recognition likely allow for successful management of this potentially serious complication, whereas ictal-DAH appears ominous in epileptic patients [10].

CONCLUSION

Epileptic seizures could induce increased pulmonary vascular permeability and structural damage to the alveolar-capillary

membrane. During the seizure episode as the patient was trying to inspire against a closed glottis, a negative pressure gradient was created thereby leading to the rupture of pulmonary capillaries.

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