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Epileptic Disorders

CLINICAL VIGNETTE

Diffuse alveolar hemorrhage induced by epileptic seizure

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Diffuse alveolar hemorrhage (DAH) is a rare but life-threatening complication of generalized tonic–clonic seizures. We describe a 28-year-old woman with no prior seizure history who experienced three consecutive generalized tonic–clonic seizures, followed by acute hemoptysis and dyspnea. Imaging confirmed diffuse alveolar hemorrhage, which responded rapidly to corticosteroid therapy. Seizure-induced DAH is an uncommon and underrecognized condition. This case underscores the importance of considering DAH in patients presenting with respiratory symptoms following seizures, even in the absence of underlying systemic disease.

The pathophysiology behind postictal DAH is not fully understood; however, it has been suggested that alveolar-capillary interface disruption, akin to the mechanism underlying neurogenic pulmonary edema (NPE), may play a role.[1] The clinical manifestations of postictal DAH commonly include hemoptysis, dyspnea, and tachypnea.[2,3]

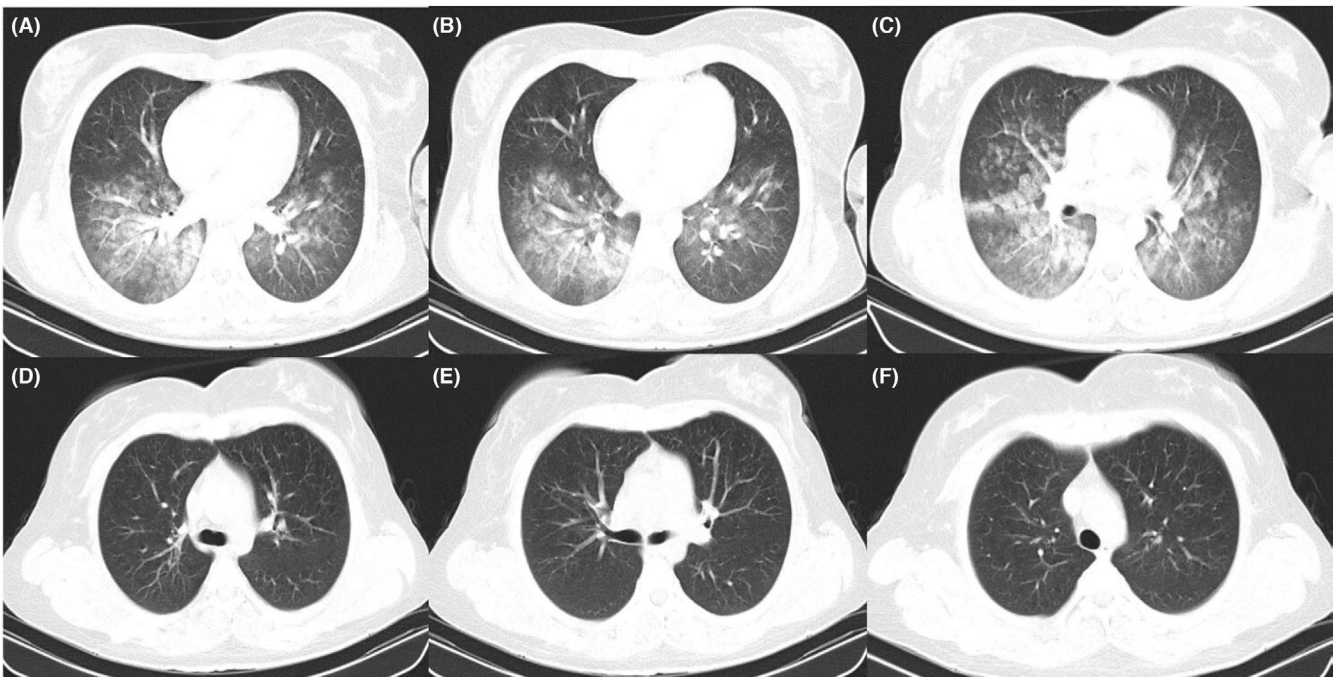

FIGURE 1 Thoracic HRCT scans demonstrating consolidated areas consistent with diffuse alveolar hemorrhage following a generalized tonic–clonic seizure (A–C) and complete resolution observed at the 1-month follow-up (D–F).

Diagnosis is based on clinical history, imaging findings revealing alveolar opacities, and the exclusion of other potential etiologies, such as autoimmune diseases, vasculitis, pulmonary tuberculosis, coagulation disorders, toxin exposure, and certain medications.[4,5] Management strategies primarily focus on seizure control, corticosteroid therapy, and respiratory support.[6,7]

A 28-year-old female presented with three episodes of generalized tonic–clonic seizures in 2 h, each lasting 2–3 min, with complete recovery between episodes. The seizures were accompanied by a loss of consciousness and occurred without a preceding aura. On the same day, during hospitalization, she developed significant hemoptysis (>100 mL) and dyspnea. The patient's past medical history was unremarkable, with no prior diagnosis or symptoms suggestive of epilepsy, pulmonary pathology, connective tissue disorders, or vasculitis. Furthermore, there was no reported family history of these conditions.

Neurological assessment was unremarkable. Pulmonary auscultation demonstrated bilateral rales. The patient required supplemental oxygen at 4 L/min to maintain an oxygen saturation above 90%. She was transferred to the intensive care unit (ICU) for close monitoring but did not require intubation.

A chest X-ray revealed bilateral middle and lower lobe opacities. High-resolution computed tomography (HRCT) of the chest demonstrated diffuse alveolar opacities consistent with DAH (Figure 1A–C). Laboratory investigations, including complete blood count, coagulation panel, renal and hepatic function tests, and inflammatory markers, were all within normal limits. Autoimmune serology, including antinuclear antibody (ANA), anti-dsDNA, p-ANCA, c-ANCA, anti-SS-A, anti-SS-B, anti-Scl-70, anti-Jo-1, anti-centromere, anti-nucleosome, anti-Sm/RNP, anti-histone, anti-phospholipid IgG/IgM, anti-cardiolipin IgG/IgM, and anti-beta-2 glycoprotein IgG/IgM antibodies, was negative. Lupus anticoagulant and rheumatoid

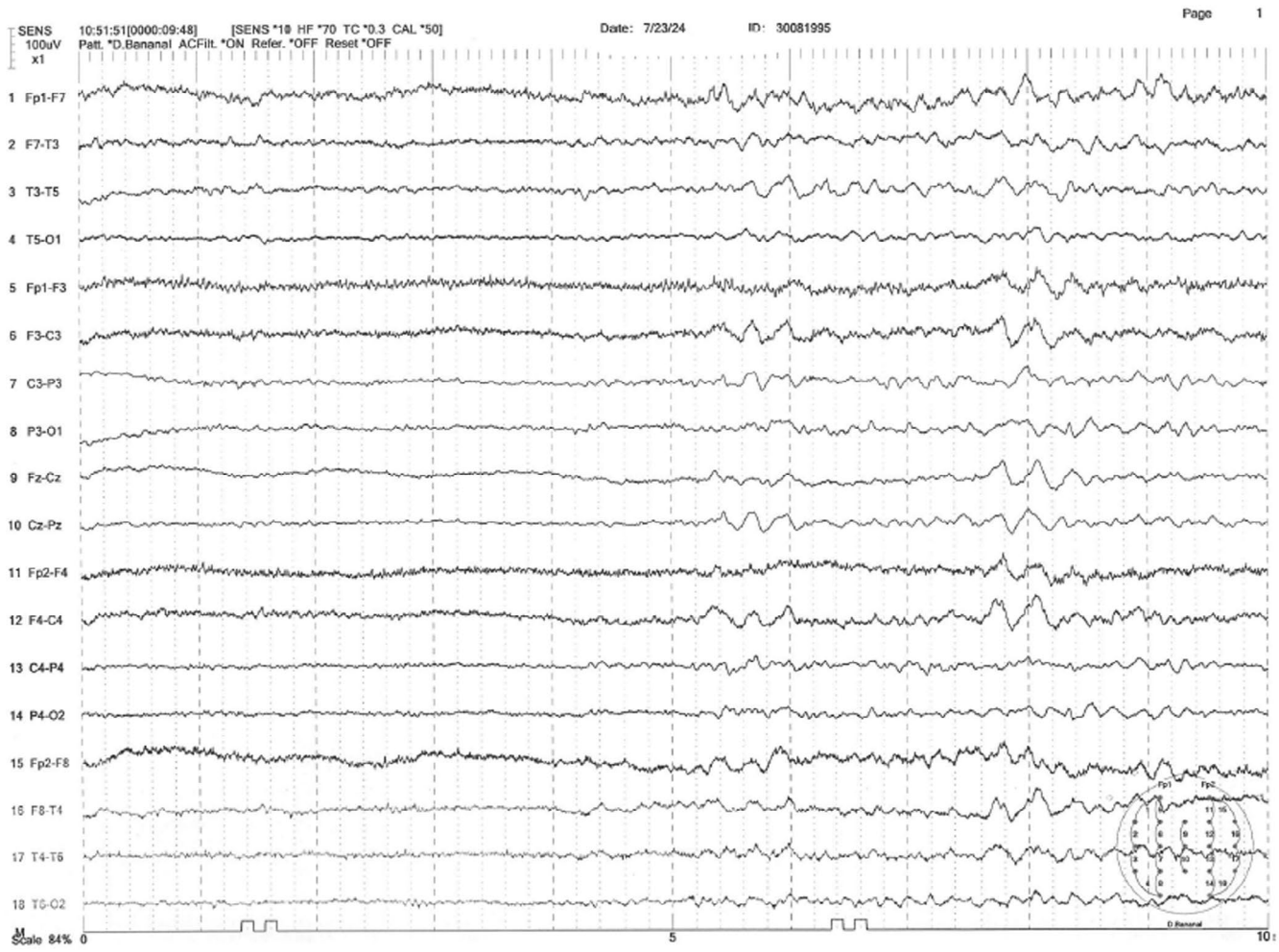

FIGURE 2 Generalized slow-wave paroxysms were observed on the interictal EEG.

factor were within normal limits. Transthoracic echocardiography and brain magnetic resonance imaging (MRI) were unremarkable. Generalized slow-wave paroxysms were observed on the interictal electroencephalogram (EEG) shown in Figure 2.

The patient was initiated on levetiracetam (1000 mg/day) and intravenous methylprednisolone (40 mg/day). After 2 days in the ICU, her dyspnea resolved, and she was transferred to the general ward. By the fourth day, a control chest X-ray demonstrated significant resolution of the initial bilateral opacities, and the patient was discharged with stable vital signs. Methylprednisolone was scheduled for gradual tapering, while levetiracetam was continued. A follow-up chest CT at 1 month confirmed complete resolution of the alveolar hemorrhage (Figure 1D–F). Over a 6-month follow-up period, she remained seizure and hemoptysis-free.

DAH following generalized tonic–clonic seizures is an exceedingly rare entity, with only 15 cases described in the literature.[1,3,8] Clinically, DAH is characterized by hypoxia and hemoptysis. HRCT findings typically include ground-glass opacities and consolidations indicative of alveolar hemorrhage.

While DAH is often associated with immune-mediated disorders such as vasculitis, it can also result from non-immune causes, including cardiac and pulmonary disorders, coagulopathies, and toxic exposures. Accurate differential diagnosis is crucial to avoid unnecessary and prolonged immunosuppressive treatments.

The exact pathophysiology of postictal DAH remains uncertain, but it is believed to involve increased permeability and mechanical disruption of the alveolar-capillary membrane.[3]

Early recognition of this condition is essential to prevent the administration of unwarranted treatments and to focus on seizure management. Additionally, caution should be exercised when prescribing anti-seizure medications, such as carbamazepine and valproic acid, as these agents have been implicated in DAH pathogenesis.[4,9,10] Treatment primarily consists of anti-seizure therapy and supportive respiratory care, with the potential for spontaneous resolution if seizure control is achieved. Some authors advocate for the use of corticosteroids to mitigate inflammatory activity and expedite symptom resolution.[3]

This case highlights the importance of considering seizure-induced DAH in the differential diagnosis when a patient with recent seizures presents with hemoptysis or dyspnea. Accurate diagnosis is critical to avoiding unnecessary long-term immunosuppressive therapy and ensuring appropriate initiation of anti-seizure medications and respiratory support in the acute phase. The scarcity of comprehensive data on seizure-induced DAH underscores the need for further research to optimize its management and therapeutic strategies.

AUTHOR CONTRIBUTIONS

T.T. and R.I. conceived the case report, and collected and interpreted the clinical data. M.I. and T.T. performed the literature review and drafted the manuscript. N.B. critically revised the manuscript for intellectual content and supervised the work. All authors approved the final version of the manuscript.

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The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

DATA AVAILABILITY STATEMENT

The data sets used during the current study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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Test yourself

1. Why is it important to differentiate seizure-induced DAH from other causes such as autoimmune diseases or infections?
   A. Because seizure-induced DAH always requires mechanical ventilation
   B. Because other causes respond better to antibiotics
   C. To avoid unnecessary long-term immunosuppressive treatment
   D. Because seizure-induced DAH is a contagious pulmonary condition
2. Which of the following mechanisms is most likely involved in the pathogenesis of postictal diffuse alveolar hemorrhage (DAH)?
   A. Cytokine-mediated alveolar infiltration following prolonged seizures
   B. Seizure-induced transient coagulopathy and platelet dysfunction
   C. Physical disruption of the alveolar–capillary interface
   D. Immune-mediated alveolar capillaritis with perivascular granuloma formation
3. Which statement best reflects current understanding of the relationship between anti-seizure medications (ASMs) and DAH?
   A. All ASMs uniformly reduce the risk of DAH by preventing seizure recurrence
   B. Certain ASMs such as carbamazepine and valproate may themselves contribute to pulmonary hemorrhage
   C. ASMs have no role in DAH pathophysiology and are not clinically relevant to its course
   D. Levetiracetam is contraindicated due to its association with immune-mediated DAH

Answers may be found in the supporting information