Case Report

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Case Report: Post-ictal Atrial Fibrillation Following Generalized Tonic–Clonic Seizure in a Patient with Known Epilepsy

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Abstract

Atrial fibrillation (AF) is a rare but clinically significant post-ictal cardiac arrhythmia that may complicate epileptic seizures and increase the risk of thromboembolic events. New-onset arrhythmias have been described after both focal and generalized tonic–clonic seizures; however, most published reports involve patients without pre-existing epilepsy, and the arrhythmias described are generally tachyarrhythmias. We report the case of a 47-year-old man with established epilepsy, treated with levetiracetam, who presented after a generalized tonic–clonic seizure and subsequently developed new-onset post-ictal atrial fibrillation on continuous cardiac monitoring and repeat electrocardiography. Baseline cardiac investigations, laboratory studies, and inflammatory markers were unremarkable. This case highlights the importance of systematic post-ictal cardiac monitoring, evaluation for reversible triggers, and consideration of anticoagulation with cardiology follow-up in selected patients. Clinicians should be aware of this uncommon association to avoid missed diagnoses and optimize management strategies.

Keywords: Atrial fibrillation, epilepsy, peri-ictal arrhythmia, seizure

Introduction

Cardiac arrhythmias are well-recognized post-ictal complications of epileptic seizures, ranging from benign sinus tachycardia to potentially life-threatening bradyarrhythmia and asystole. In contrast, peri-ictal atrial fibrillation (AF) and atrial flutter are rarely reported and are primarily described in isolated case reports or small case series, often labeled as "post-ictal" or "seizure-associated" AF. The underlying mechanisms remain incompletely understood but likely involve autonomic dysregulation, catecholaminergic surge, and seizure propagation to central autonomic network structures such as the insula and amygdala (1-3).

Because AF carries a well-established thromboembolic risk, even brief, seizure-triggered episodes may have important implications for stroke risk in patients with epilepsy. Existing literature suggests that seizure-associated AF often occurs in structurally normal hearts, may be transient, and is probably under-recognized in emergency settings (1,3). Here, we describe a case of post-ictal AF in a middle-aged man with known epilepsy and normal baseline systemic and cardiac evaluations.

Case Report

A 47-year-old right-handed man with a history of generalized epilepsy presented to the emergency department (ED) after experiencing a generalized tonic–clonic seizure. His epilepsy had been diagnosed several years earlier, and he was taking levetiracetam 750 mg twice daily with reported good adherence. His most recent seizure had occurred one month prior. He denied fever, systemic symptoms, substance use, or recent medication changes.

On arrival, he was fully oriented (GCS 15) and hemodynamically stable except for tachycardia (132 beats/min). Blood pressure was 125/85 mmHg, respiratory rate 17 breaths/min, and oxygen saturation 98% on room air. Neurological examination was normal, aside from a superficial 1-cm occipital laceration presumed secondary to seizure-related trauma. Systemic examination revealed no abnormalities. Initial ECG showed sinus tachycardia without ischemic changes.

Laboratory tests, including complete blood count, renal and liver function, electrolytes, and inflammatory markers (CRP 1.2 mg/dL), were within normal limits. Chest

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radiography revealed clear lung fields and a normal cardiac silhouette. Serum potassium was mildly low at 3.4 mmol/L, but no other abnormalities were detected. A non-contrast head CT scan was planned.

While awaiting imaging, the patient experienced a second generalized tonic–clonic seizure lasting approximately 45 seconds. He was treated according to institutional protocol, including airway evaluation, oxygen supplementation, and levetiracetam 1000 mg IV loading. In the immediate post-ictal period, continuous monitoring revealed an irregularly irregular rhythm with frequent escape beats. Repeat 12-lead ECG displayed atrial fibrillation with a single ventricular extrasystole and ventricular rate of 110 beats/min. No prior history of AF, hypertension, hyperthyroidism, or structural heart disease was identified.

Cardiac troponins and transthoracic echocardiography were normal, with no structural abnormalities or intracardiac thrombus. Non-contrast brain CT showed no acute pathology. Cardiology and neurology consultations were obtained. Hospitalization for further evaluation and management were planned but the patient declined admission; his scalp laceration was treated, and he was discharged with instructions for close outpatient follow-up.

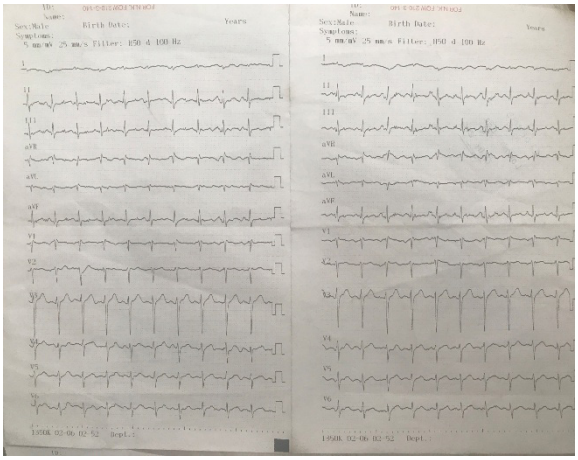

Figure 1. ECG of patient on presentation to ED.

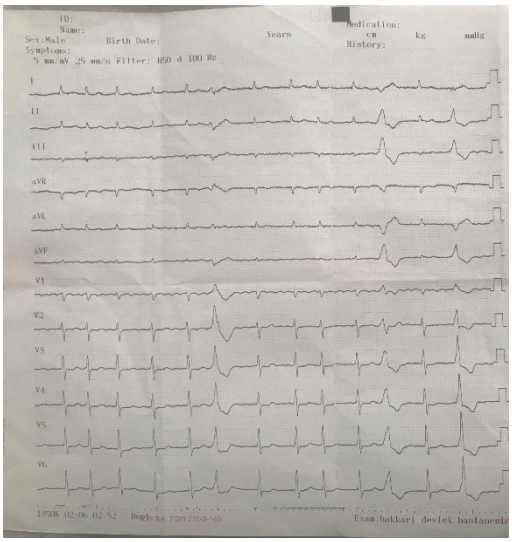

Figure 2. ECG of patient after suffering a generalized tonic-clonic seizure in the ED

At one-month cardiology review, the patient remained asymptomatic, and ECG demonstrated normal sinus rhythm. Anticoagulation was deferred based on a low CHA_2DS_2-VASc score and multidisciplinary consensus. Plans were made for ongoing cardiology and neurology follow-up, optimization of antiseizure therapy, and monitoring for recurrent arrhythmia.

Discussion

This case highlights a rare instance of post-ictal atrial fibrillation occurring after a generalized tonic–clonic seizure in a patient with established epilepsy, compliant with antiepileptic medication and with no history or new evidence of structural heart disease. Seizure-associated AF has been reported only sporadically in the literature, with most cases describing transient arrhythmias occurring during or immediately following generalized convulsive events (1,3). However, most reported cases of seizure-associated atrial fibrillation have involved patients without a known history of epilepsy. As in the present case, affected individuals frequently have structurally normal hearts, and arrhythmias often resolve spontaneously, suggesting autonomic or neurogenic mechanisms rather than primary cardiac pathology (2,4). The proposed pathophysiology involves abrupt sympathetic activation and catecholamine surges, which can shorten atrial refractory periods and promote arrhythmogenic re-entry circuits (1–3). Seizure propagation to autonomic network structures—including the insula, anterior cingulate cortex, and amygdala—may further modulate cardiac rhythm (5). Post-ictal hypoxia, metabolic acidosis, and electrolyte derangements may also contribute, although many cases demonstrate largely normal biochemical profiles (3). Our patient declined immediate hospitalization for metabolic and electrolyte work-up, hence these were not done. However, his vital signs were stable, he was hemodynamically stable and showed no signs of hypoxia during the monitorization period and in the immediate follow up. Clinically, seizure-associated AF raises concern for both immediate hemodynamic consequences and longer-term thromboembolic risk. Paroxysmal AF is independently associated with stroke, and seizure-triggered AF may go undetected without continuous monitoring. Several authors recommend routine post-ictal ECGs after generalized seizures to avoid missed diagnoses (3,6). Decisions regarding anticoagulation require individualized risk assessment using CHA_2DS_2-VASc and bleeding risk tools, particularly given increased fall risk in patients with epilepsy (4,7). In patients with low stroke risk and single, clearly seizure-related AF episodes, careful observation, seizure control, and cardiology follow-up may

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be reasonable, whereas recurrent or prolonged AF, or the presence of additional vascular risk factors, may justify anticoagulation despite the potential bleeding risk (8-10).

Conclusion

Our case underscores that seizure-associated AF can occur even in patients with established epilepsy on stable antiseizure therapy and without structural heart disease. Recognition of this association in the ED is critical, as it is uncommon and may have clinically significant complications. Maintaining a high index of suspicion for seizure-associated arrhythmias, performing post-ictal ECGs, vital signs and rhythm monitoring and collaborating with cardiology to assess thromboembolic risk and guide treatment decisions is recommended. In-hospital laboratory tests and detailed neurological evaluation may shed light onto the underlying mechanisms and triggers. Additional case reports and systematic studies are needed to better define incidence, mechanisms, and optimal management strategies.

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