

Case Report

Cardiac asystole associated with seizures of right hemispheric onset

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ABSTRACT

Ictal asystole is frequently underrecognized despite being a potentially lethal condition. We report two cases of ictal asystole with right hemispheric onset. These cases are unique since previous literature reports that seizures associated with bradyarrhythmias typically arise from left hemispheric foci. These cases further underscore the importance of clinical vigilance and the need of an enhanced diagnostic biomarker.

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1. Introduction

Cardiac arrhythmias in patients with epilepsy represent an important area of clinical interest, particularly given their suspected contributions to sudden unexpected death in epilepsy (SUDEP). Much remains uncertain regarding the mechanisms underlying ictal cardiac arrhythmias. Previous literature suggests that left hemispheric epileptogenic substrates are more often associated with ictal bradyarrhythmias and right hemispheric substrates with tachyarrhythmias.

We present two cases of potentially lethal asystole associated with seizures of right, rather than the expected left, hemispheric onset.

2. Methods

Case analysis with literature review was conducted.

3. Cases

3.1. Case 1

During video-EEG monitoring for seizure characterization, a 37-year-old man with nonlesional epilepsy was observed to have a seizure characterized by a feeling of *déjà vu* followed by altered awareness during which bilateral tonic extension of the upper extremities occurred (Video 1). Electrographically, the seizure demonstrated right temporal onset and remained lateralized to the right hemisphere as the seizure progressed. Ten seconds after electrographic onset, concurrent EKG

showed a slow trending heart rate that culminated in asystole for 27 s. Correspondingly, the EEG revealed diffuse and marked background attenuation during the period of asystole. Upon recovery and stabilization of the heart rate, the EEG background quickly normalized after 9 s (Fig. 1). Subsequently, the patient sustained two more seizures associated with ictal asystole of similar duration. Eventually, the patient underwent placement of a dual chamber pacemaker.

3.2. Case 2

A 60-year-old woman presented with two seizures characterized by staring and altered awareness followed by left arm and leg shaking for 30 s. Simultaneously, her EKG revealed an initial bradycardia followed by asystole lasting 15 s. She required cardiopulmonary resuscitation for both episodes. Brain MRI revealed an enhancing lesion in the right hemisphere involving the frontal and temporal lobes, insula, thalamus, hypothalamus, and pons (Fig. 2). Brain biopsy was consistent with primary CNS lymphoma. Although her seizures were not corroborated by simultaneous EEG monitoring, the ictal semiology and imaging findings strongly support a right hemispheric epileptogenic zone. The patient underwent placement of a transcutaneous pacemaker.

4. Discussion

Ictal cardiac rate changes commonly occur, with sinus tachycardia (pulse > 100 beats per minute) evident in the majority of patients [1]. While ictal bradycardia and asystole are documented in only about 2.1% of recorded seizures, many patients who experienced such arrhythmias had potentially fatal asystole and required subsequent permanent pacemaker [1].

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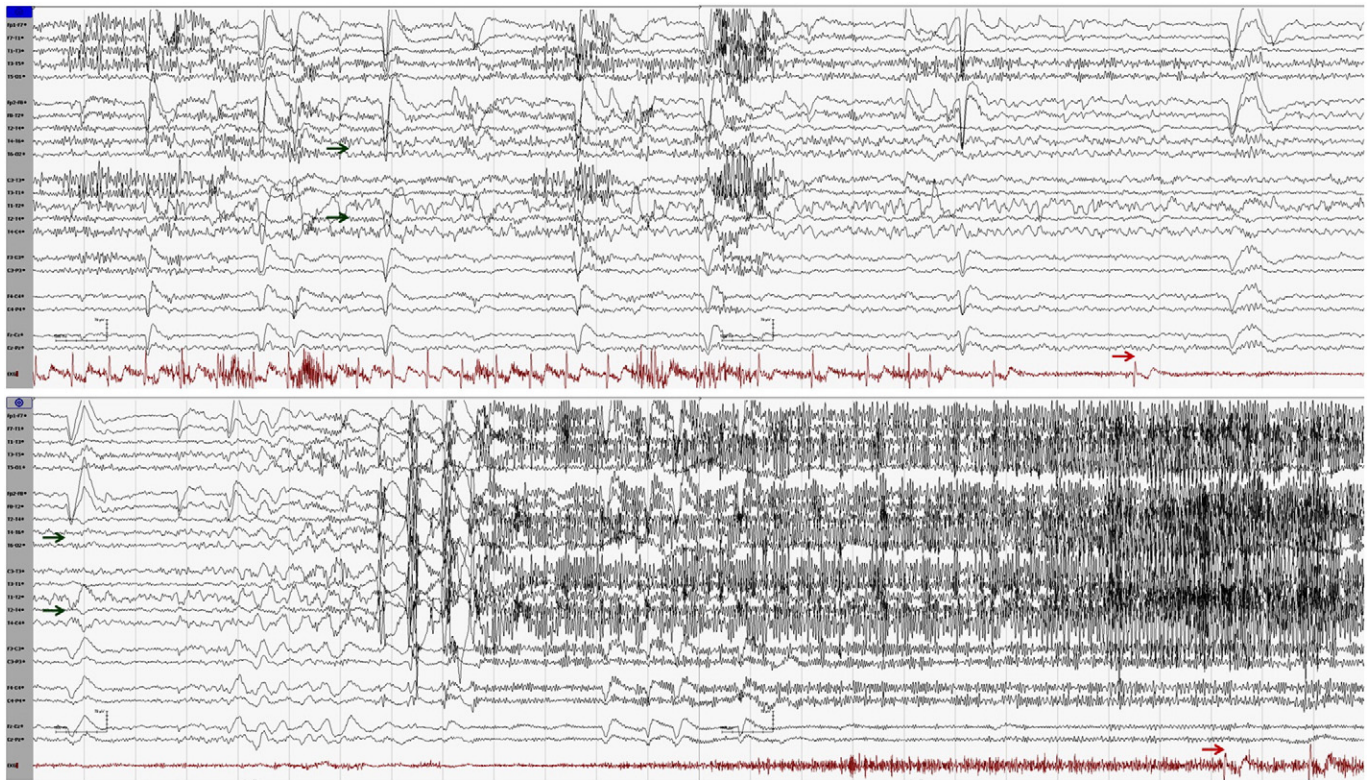


Fig. 1. Ictal asystole. Sample EEG from case 1 demonstrating an electrographic seizure (green arrows) arising from the right temporal region, which is followed by cardiac bradycardia and then asystole (first red arrow). Cardiac rhythm returns after 27 s (second arrow).

According to the lateralization hypothesis, seizures with a left-sided focus result in bradyarrhythmias, whereas seizures with a right-sided focus result in tachyarrhythmias [2]. Selective activation of parasympathetic or sympathetic centers via the electrical propagation of seizures may explain ictal cardiac changes. In one study, electrical stimulation of the left insular cortex resulted in bradycardia, implying that a parasympathetic-mediated pathway was involved. Conversely, stimulation of the right insular cortex induced tachycardia, implying a right hemispheric predominance in cardiac sympathetic regulation [3]. In contrast to this hypothesis, the two cases of ictal asystole presented here occurred with right-sided seizure foci. Other studies have shown

that autonomic alterations may be more prominent in patients with right-sided versus left-sided epileptogenic zones [4]. Interestingly, the second case demonstrated an extensive lesion involving the thalamus and cingulate — structures which have been shown to influence cardiac rhythms [5]. Furthermore, more recent studies involving 13 patients with ictal bradycardia failed to show any consistent lateralization of seizures. In fact, 9 of the 13 patients showed bilateral electrographic activity at seizure onset [6]. Based on these previous studies as well as our present cases, ictal asystole may be driven by a mechanism that is more intricate than the strictly lateralized cardiomotor representation of either sympathetic or parasympathetic function.

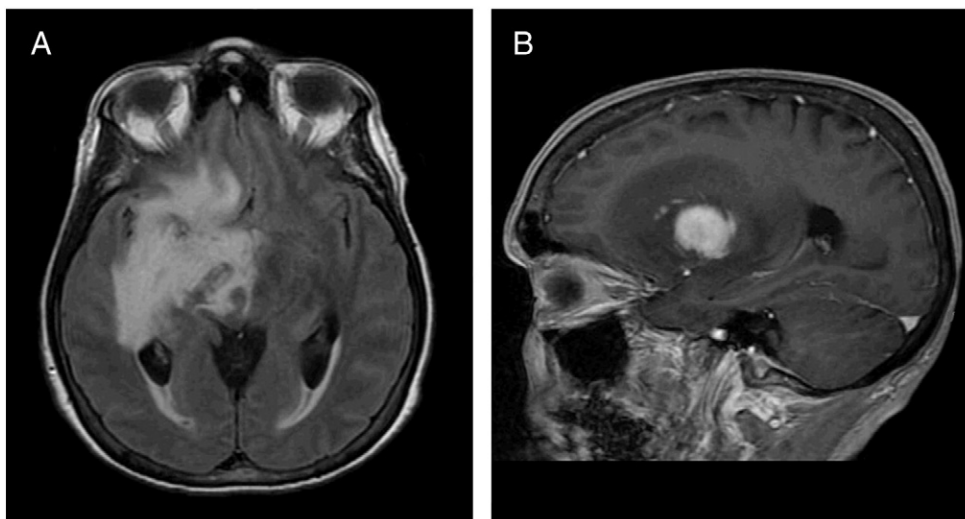


Fig. 2. MRI of the brain. A) Axial T2 FLAIR with an enhancing lesion that extends along the right lateral ventricle and into the right frontal and temporal lobes, insula, thalamus, hypothalamus, right cerebral peduncle, and pons. B) Sagittal T1 postcontrast with lesion that extends along the right temporal lobe and insula region.

The mechanisms underlying ictal cardiac arrhythmias remain elusive, and most patients have no underlying cardiac risk factors. Frequently, ictal arrhythmias are detected incidentally during simultaneous EEG and ECG monitoring. This diagnostic elusiveness, in the midst of a potential lethal condition, underscores the importance of clinical vigilance and the need of an enhanced diagnostic tool for early detection of this condition such that appropriate intervention may promptly ensue. Genomic biomarkers are in development, aiming to reliably detect channelopathies which may subserve dual roles in epileptogenesis as well as cardiorespiratory pathophysiology (i.e., neurocardiac genes) [7]. There are currently no clinical guidelines pertaining to the type of epilepsy syndrome or the extent of ictal bradycardia/asystole and seizure control for which the placement of a permanent pacemaker becomes indicated. However, even in cases of apparently good seizure control, it may be intuitive to place pacemakers in patients with ictal asystole considering the gravity of a potentially lethal alternative outcome. Important investigations on the mechanisms underlying ictal cardiac arrhythmias and their clinical management are ongoing.

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.ebcr.2014.05.001>.

Conflict of interest

The authors have no conflict of interest to disclose. The patient depicted in the supplementary materials provided permission for his face to be shown in the images.

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