Ictal asystole: A rare cardiac manifestation of temporal lobe epilepsy, treated with epilepsy surgery

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Abstract

Seizures are associated with fascinatingly varied cardiac and autonomic manifestations, of which ictal tachycardia is common, and asystole and bradycardia are rare. Ictal asystole (IA), an often unsought autonomic phenomenon, occurs most commonly with temporal followed by frontal lobe seizures. Prolonged IA may lead to cerebral anoxic ischemia. As the mysteries of sudden unexplained death in epilepsy are unraveled, it is quite possible that the key to it lays within these seizure-induced cardiac rhythm abnormalities. We present a case of a young male with temporal lobe epilepsy due to left mesial temporal sclerosis with prolonged IA, which was successfully managed with epilepsy surgery.

Key Words

Anterior temporal lobectomy, ictal asystole, mesial temporal sclerosis, temporal lobe epilepsy

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Introduction

Since the recognition of occurrence of sudden unexplained death in epilepsy (SUDEP), seizure-related cardiorespiratory abnormalities have been the focus of research. These cardiac abnormalities are possibly triggered by epileptic activation of autonomic centers in the brain.[1] Most commonly reported is ictal sinus tachycardia (80–100% of all seizures).[2] A less frequently observed arrhythmia is sinus ictal bradycardia (IB), defined as an R–R interval >2 s. IB can be found in <6% of seizures. Ictal asystole (IA) is defined as the absence of ventricular complexes for more than 4 s accompanied by electrographic seizure onset.[2] IA is a rare condition, usually associated with temporal lobe epilepsy. It has a doubtful role in SUDEP but nevertheless, warrants aggressive treatment. As per a recent review of ictal cardiac arrhythmias,[3] 103 cases of IA have been reported till date, most of which have been treated by implantation of a cardiac pacemaker.

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IA associated with mesial temporal sclerosis (MTS) and temporal lobe seizures can be successfully treated with anterior temporal lobectomy, giving cure from both the ailments. This can avoid the placement of a permanent cardiac pacemaker.

Case Report

A 23-year-old man presented with a 2-year history of complex partial seizures with secondary generalization. There was no history of any childhood events such as febrile seizures, encephalitis, or head injury. At the age of 21 years, he had onset of seizures in clusters of up to six episodes per day; initially, at frequency of one cluster in 6 months. Gradually, it increased to one cluster per week. Semiology of episodes consisted of aura of epigastric discomfort followed by searching eye

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movements and a confused look; following which his right hand developed fast jerky movements with chewing and swallowing movements. There was postictal speech arrest for about 1 min. Some of the events also started with couple of jerk-like movements of both shoulders followed by fall, followed by dyscognitive seizure as described above. He had often injured himself during these falls. He was taking three antiepileptic drugs, namely, levetiracetam, lacosamide, and clobazam, in adequate doses.

He was referred to our center for the detailed workup and management of refractory epilepsy. His magnetic resonance imaging (MRI) [Figure 1] showed atrophy of the left hippocampus with T2 hyperintensity within and associated widening of the adjacent temporal horn suggestive of MTS. Hippocampal volumetry showed left hippocampal volume of 1.72 cc as compared to 2.5 cc on the right.

Neuropsychology testing showed impaired verbal memory scores, suggestive of left temporal involvement.

He underwent video electroencephalography (EEG) recording for 72 h during which six habitual seizures were recorded. His interictal EEG showed frequent focal discharges in left temporal area. His ictal semiology consisted of abdominal aura in the form of epigastric discomfort, followed by rhythmic ictal nonclonic hand (RINCH) motions of right hand[4] associated with behavioral and speech arrest lasting for couple of minutes. In one seizure he had three myoclonic jerks of both upper limbs at onset, followed by tonic posturing of whole body for few seconds. This was then followed by the habitual dyscognitive seizure. His semiology was similar in all other seizures except for the initial myoclonic jerk and tonic posturing that was not seen in rest of the seizures.

His ictal EEG [Figures 2-5] showed onset of ictal rhythm in left temporal region which then spread to the left hemisphere. Concurrent electrocardiogram (ECG) recording showed bradycardia [Figure 3] for 3–4 s along with EEG onset followed by asystole [Figures 4 and 5] lasting for 15 s, following which there was bradycardia for 5 s followed by normalization of the cardiac rhythm. In all seizures, there was bradycardia followed by asystole (ranging from 15–22 s) as soon as the ictal rhythm appeared on EEG. This asystole was probably responsible for cerebral anoxia and the anoxic myoclonic jerks that occurred in one of the seizures.

His detailed cardiac evaluation including 12-lead ECG, two-dimensional echocardiogram, and 48-h Holter monitoring was normal.

We had sufficient evidence to localize his seizures to left temporal lobe. The semiology showed RINCH in right hand and prolonged speech arrest, both localizing to temporal lobe and lateralizing to left. There were concordant data on MRI and neuropsychology tests showing left temporal lobe involvement. IB and asystole were likely to be an autonomic manifestation of temporal lobe seizure as there was no underlying abnormality of cardiac structure or rhythm.

He underwent a standard left anterior temporal lobe resection with amygdalohippocampectomy. Postoperatively on 1.5 years

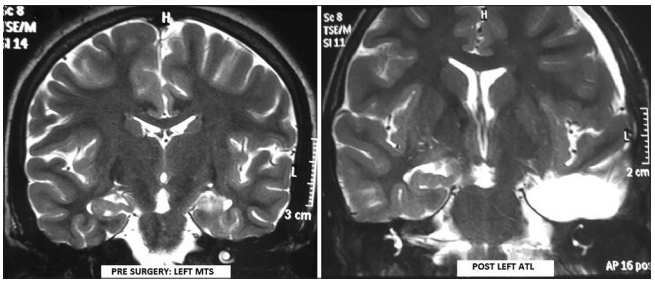

Figure 1: T2-weighted images coronal magnetic resonance imaging showing left mesial temporal sclerosis and after left anterior temporal lobectomy showing complete resection of anterior temporal lobe

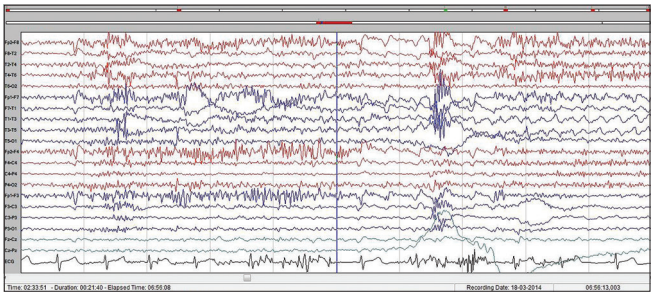

Figure 2: Electroencephalography trace showing the onset of the ictal rhythm in left temporal lobe with slowing of the heart rate towards the end of the trace

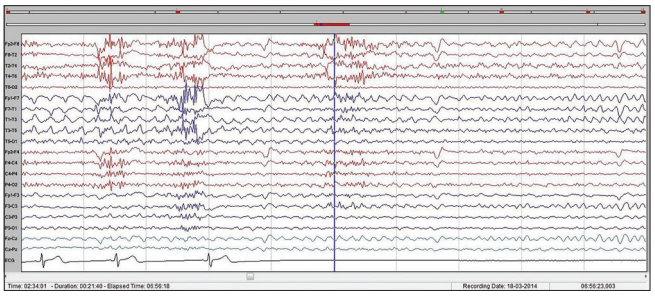

Figure 3: Electroencephalography trace showing built up of rhythm in left temporal region with progressive slowing of heart rate followed by asystole (16 s)

follow-up, the patient has been seizure-free with no further episodes of fall or syncope.

Discussion

Among the ictal bradyarrhythmias, IA is more common as compared to ictal atrioventricular block and IB. All the three rhythms may overlap in a single patient.[3] Its incidence ranges from 0.27% to 0.4%.[2,3] IA is usually clinically relevant leading to sudden falls and unresponsiveness. It may be a possible mechanism for SUDEP; however, no reported case has been attributed to IA yet.[3]

It has been seen that most cases occur in temporal lobe or insular epilepsy (focal epilepsy)[5] without lateralization.[3] This may be related to the role of temporal lobe in autonomic control. Temporal lobe seizures stimulate insula, cingulate cortex, amygdala, and hypothalamus which may provoke

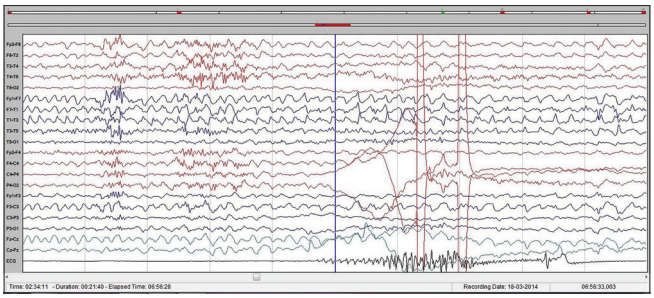

Figure 4: Electroencephalography trace showing progression of the ictal rhythm and asystole with return of cardiac activity towards the end of trace

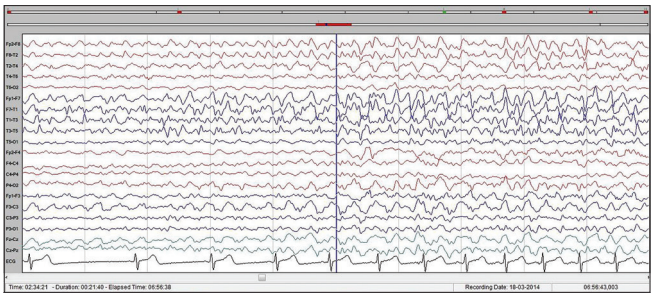

Figure 5: Electroencephalography trace showing further built up of ictal rhythm which is now left hemispheric along with return of normal cardiac rhythm

asystole through connections with the autonomic nervous system.[6,7]

As per a recent review of 103 cases,[3] the mean duration of seizure before onset of asystole was 24 (13–35) s, with the duration of asystole lasting around 20 (3–96) s. This was consistent with our patient whose asystole had a duration ranging from 15 to 22 s. However, the onset was within 3–4 s after onset of the seizure, which was much earlier. There was a bradycardia for a few seconds preceding as well as following the asystole. Usually, patients with IA show a spontaneous recovery (as seen in our patient), only rarely requiring cardiopulmonary resuscitation.[8]

Most of the cases of IA have been treated with cardiac pacemaker implantation.[2,3] In our patient, there was a clear cut concordance between the MRI findings, ictal semiology, and EEG rhythm and neuropsychological testing – all pointing to the left temporal lobe origin of epilepsy. There was no other pre-existing or baseline cardiac dysfunction. Postulating the probable cause of IA to be related to the temporal lobe epilepsy itself, the patient underwent an anterior temporal lobectomy. Potential pro-arrhythmic drugs such as carbamazepine and phenytoin[9] were avoided. Following the surgery, the patient became symptom-free with no further seizures or syncopal episodes.

This case highlights the importance of simultaneous EEG and ECG recording of focal seizures, especially those of temporal lobe etiology, which can pick up the rare but potentially life-threatening occurrence of clinically relevant IA. IA can be successfully treated permanently by epilepsy surgery and drug modulation in ideal candidates, circumventing the need for a lifelong intervention like a pacemaker. Pacemaker implantation should only be considered if other measures fail or if there is a pre-existing cardiac dysfunction.

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Conflicts of interest
There are no conflicts of interest.

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