Pearls & Oy-sters:

Ictal syncope in a patient with temporal lobe epilepsy

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PEARLS

- Ictal cardiac arrhythmias are common autonomic features of focal epilepsy.
- Ictal sinus tachycardia and bradycardia are the most common arrhythmias, with ictal asystole occurring infrequently.
- Ictal syncope is a late clinical manifestation of ictal asystole and may contribute to sudden unexplained death in epilepsy.

OY-STERS

- Ictal asystole and ensuing syncope can be easily misdiagnosed as vasovagal syncope, but can be correctly identified by video-EEG. There should be a high degree of suspicion in patients with epilepsy despite the lack of coexisting typical seizure semiology.

CASE REPORT We present a case of an 18-year-old Caucasian woman of normal development with a history of complex partial seizures and new-onset recurrent syncopal episodes. Her typical seizures started at 14 years of age and consisted of staring spells with motor automatisms involving both upper extremities. Her seizures were well-controlled on oxcarbazepine, and she was seizure-free for more than 2 years. MRI revealed a right temporal-occipital cavernous angioma. She presented acutely with recurrent syncopal episodes over the span of 2 weeks with increasing frequency up to multiple times per week. Witnessed episodes were described as sudden loss of consciousness with subsequent falling, without any preceding, concomitant, or postevent seizure-like activity. She denied prodromal symptoms such as palpitations, headache, sweating, abdominal discomfort, nausea, or vomiting. She denied any aura and was completely amnesic for the event. She had a quick return to baseline with brief confusion lasting only a few seconds. The events lasted less than a minute. Initial cardiac workup including EKG, 2D echo, and cardiac biomarkers revealed normal cardiac function. The cavernous angioma was unchanged on repeat imaging. General physical examination including

orthostatic blood pressure was normal. Neurologic examination was also normal. She was admitted to our epilepsy monitoring unit where a typical syncopal episode was captured on the day of admission.

Video-EEG captured her typical syncopal event and was consistent with the diagnosis of ictal syncope (IS). A right posterior temporal electrographic seizure was captured. The ictal activity induced cardiac bradycardia and 16 seconds of cardiac asystole ensued with resultant loss of consciousness and tone. This loss of consciousness lasted for 30 seconds and was followed by a quick return to baseline mental status. The figure illustrates the electrographic and EKG findings with clinical commentary.

A cardiac pacemaker was successfully placed in our patient and she was continued on one antiepileptic drug. Oxcarbazepine was switched to levetiracetam to minimize potential risk of arrhythmias. She remained seizure-free and syncope-free. The patient subsequently had excision of the cavernoma and is currently scheduled to have the pacemaker removed as well.

DISCUSSION Paroxysmal loss of consciousness commonly results in neurologic consultation to aid in the differentiation between seizure and syncope. To make this distinction, a thorough history, physical examination, and, most importantly, a reliable witness account are often required in conjunction with a well-thought-out diagnostic workup.¹ Typically, syncope is associated with a prodromal phase of autonomic dysfunction, manifested as lightheadedness, palpitations, chest pain, and sweating. At times, there are triggers of micturition, cough, or postural change. Seizures can be associated with brief abdominal or psychic auras, staring, automatisms, forced deviation of the head, convulsions, and postevent confusion. Myoclonus or convulsion, however, can occur after an episode of syncope, referred to as convulsive syncope. IS, on the other hand, results from seizure activity inducing autonomic dysregulation of the heart in the form of ictal asystole (IA).

Cardiac autonomic dysregulation and arrhythmias, most frequently tachycardia, are common during epileptic seizures.^{2,3} However, ictal bradycardia

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Figure EEG shows right posterior temporal seizure and subsequent asystole

Panels B to F are contiguous. (A) Twenty seconds prior to seizure onset, rhythmic buildup of low-voltage α and β range (8–15 Hz) and overlying δ (2–3 Hz) activity is noted in an otherwise normal EEG (arrow). (B) Clear seizure onset is demonstrated with rhythmic δ (2–3 Hz) frequency over the right temporal region with maximum discharge and phase reversal at T6 (arrow). (C, D) Evolution in morphology, increase in frequency, and spread to the left temporal region are seen. Orange bold arrow indicates first signs of bradycardia and slowing to 50 beats per minute (bpm) from baseline 75 to 100 bpm. Red bold arrow subsequently indicates last beat detected and ensuing asystole. (E) High-voltage 1–2 Hz activity is seen diffusely but maximum in the right temporal region, followed by diffuse background attenuation (F). Return of EKG beat is noted by green bold arrow in panel F with return to normal sinus rhythm. Clinical description: (A) Patient is conversing with family. (B, D) She scans the room and continues to appear normal, although formal testing of consciousness was not done. (E) Patient has upward deviation of eyes followed by head drop and loss of tone in the body (black arrow). Patient continues to be unconscious for an additional 30 seconds. She is immediately responsive to questions upon awakening.

(IB) and IA are rare occurrences, with IA reportedly occurring in 0.27%–0.4% of monitored epilepsy patients.⁴ Seizures in patients with IB and IA have been primarily associated with temporal lobe onset.⁵ However, lateralization of such events has not been consistent.^{5,6} It is plausible that seizures involving the temporal lobe result in bradycardia by stimulation of cardiac centers in insula, cingulate cortex, or amygdala. Sudden loss of postural tone usually occurs after at least 8 seconds of IA and manifests as IS. This is often preceded by epileptic auras, automotor or hypermotor activity, or a dyscognitive phase typically seen in frontal or temporal lobe epilepsy.⁷ The sequence of events leading to syncope is thought to be seizure activity causing autonomic dysfunction and asystole with resultant generalized cerebral hypoperfusion. Once the seizure ends (whether it is spontaneously or because of the hypoperfusion), normal autonomic function returns and clinical recovery occurs.

Loss of sinus nodal function or sinoatrial block without activation of an underlying escape rhythm is commonly seen in IA. However, progressive atrioventricular block has also been reported.⁸ Placement of a cardiac pacemaker is not clearly beneficial, but is common because of the theoretical risk of cardiac arrest, seizure-related falls, and IA being a possible contributory factor to sudden unexplained death in epilepsy.⁸

This case serves as a reminder of the seizure-vs-syncope diagnostic dilemma. It reinforces the idea that IS should be suspected in patients with focal epilepsy presenting with syncopal episodes. Our case was slightly more challenging due to the lack of coexisting seizure-like semiology such as auras and automatisms, and video-EEG was instrumental in making the diagnosis. We used a sequential treatment approach of first optimizing antiepileptic medications in conjunction with cardiac pacing and subsequent surgical resection of the epileptic lesion, and the patient continues to be seizure-free at 18-month follow-up.

AUTHOR CONTRIBUTIONS

Dr. Varade: contributed to acquisition of data, analysis and interpretation. Dr. Rayes: contributed to study concept, design, analysis and interpretation. Dr. Basha: contributed to acquisition of data, analysis and interpretation, revisions and editing, critical review of the manuscript for important intellectual content, study supervision. Dr. Watson: contributed to the critical review of the manuscript for important intellectual content, study supervision.

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