CASE REPORT

Seizure Triggered by Sick Sinus Syndrome

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SUMMARY

Sick sinus syndrome (SSS) is a dysfunction of sinoatrial node resulting in symptomatic bradycardia or sinus pauses causing decreased cardiac output with cerebral hypoperfusion and usually presents as syncope, presyncope or fatigue. The occurrence of a seizure is very rare. A 69-year-old man suffered two episodes of generalised tonic-clonic seizures. MRI and electroencephalogram failed to reveal the cause of seizures. In the emergency room, he experienced presyncope simultaneous to bradycardia and sinus pauses. He was stabilised with atropine and dopamine infusion and underwent definitive therapy with a permanent dual-chamber pacemaker with complete symptom resolution. Diagnostic confounders include convulsive syncope and ictal bradycardia. Syncope may be accompanied by myoclonic jerks (convulsive syncope), but postictal confusion is absent. Bradycardia may be seen during the postictal period (ictal bradycardia syndrome), but protracted sinus dysfunction is not present. Hypoperfusion due to significant SSS triggered seizures in this patient who may have an underlying predisposition.

BACKGROUND

Generalised tonic-clonic seizure and cardiac arrhythmias are closely associated. Various cardiac arrhythmias are seen during seizure episodes and are thought to be responsible for sudden unexpected death in epilepsy.¹ On the other hand, global cerebral hypoperfusion seen with cardiac

arrhythmias presents as dizziness and syncope. Sick sinus syndrome (SSS) is a dysfunction of the sinoatrial node which results in bradycardia and sinus pauses with typical symptoms.^{2,3} It is very rare for a patient with SSS to present with seizures. The following unusual presentation will allow readers to appreciate the difference between a seizure triggered by SSS, ictal bradycardia syndrome and convulsive syncope.

CASE PRESENTATION

A 69-year-old man presented with two episodes of generalised tonic-clonic seizures. The first episode had occurred while sleeping. His wife noticed jerking movement of the body which lasted for about 1 min followed by confusion for 30 min. After 2 hours, he had another similar episode and his wife called emergency medical services. He was found to be confused but was slowly regaining his lucidity. He did not bite his tongue or lose control of his bowel or bladder. The patient denied any recreational substance use and overdose of any drug. The only medication he was taking was simvastatin 20 mg daily.

On arrival to the emergency room he was conscious, alert and fully oriented. He confirmed that he did not have any history of seizure or epilepsy. Cardiovascular examination revealed a blood pressure of 148/84 mm Hg and bradycardia with heart rate (HR) ranging from 48 to 56 beats per min. Normal S1 and S2 were auscultated over all areas with a soft holosystolic murmur over the aortic area. Jugular venous distension and leg oedema were absent. The patient revealed that he was a marathon runner and his baseline HR was in the 50s, but he had been completely asymptomatic prior to this admission.

The neurological examination revealed normal speech, language comprehension and intact short-term and long-term recall. Normal optic discs were seen on ophthalmoscopy. His visual fields were intact with both pupils reactive to light. Extraocular muscle movements were normal. Cranial nerve examination was normal. He had normal and symmetric sensations, power and tone in proximal and distal limbs. Reflexes were normal and symmetric.

INVESTIGATIONS

A basal metabolic panel was normal. Sodium, glucose, calcium, magnesium and urea were within normal limits. Serum thyroid-stimulating hormone level was also normal. Serum alcohol was not detectable. CT scan of the head did not demonstrate any haemorrhage, ischaemia or mass effect.

Figure 1 MRI demonstrating right frontal cavernous haemangioma.

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Figure 2 CT angiogram with stenosis of right internal carotid artery.

Next day, electroencephalogram (EEG) revealed a normal symmetric, moderate amplitude, 9 Hz background activity that attenuated with eye opening. Epileptiform activity was not detected. MRI confirmed absence of haemorrhage, diffusion restriction or cerebral oedema. However, a 9 mm spherical area with minimal enhancement and a haemosiderin ring representing a vascular anomaly such as a cavernous malformation was seen in the right frontal lobe (figure 1). A CT angiogram of the neck revealed 50% stenosis of the right internal carotid artery and less than 30% stenosis on the left (figure 2).

DIFFERENTIAL DIAGNOSIS

The main confounding diagnoses, in this case, were convulsive syncope and ictal bradycardia syndrome. Patients with syncope may have myoclonic jerks from transient cerebral hypoperfusion which are confused for seizures by lay witnesses. These episodes are called convulsive syncope and are sometimes encountered during tilt table testing, blood donation or post ventricular arrhythmias. The distinguishing feature is immediate lucidity with the absence of prolonged postictal confusion typically seen with a true generalised seizure.^{4 5} Ictal bradycardia syndrome is a rare disorder with a high-mortality rate manifesting with bradycardia and asystole during the immediate postictal period. Confirmation of this diagnosis requires simultaneous EEG and ECG monitoring with documentation of epileptiform spikes followed by bradycardia.⁶ Persistent rhythm abnormalities in

Figure 3 ECG at presentation with sinus pause.

Figure 4 Initial rhythm strip with 4.5 s long sinus pause.

the absence of seizure activity point to an underlying cardiac arrhythmia rather than ictal bradycardia.

TREATMENT

The patient's HR decreased to 20 with intermittent sinus pauses up to 4.5 s long with simultaneous diaphoresis, vomiting and dizziness (figures 3 and 4). He was administered atropine and then started on intravenous dopamine infusion as per the American Heart Association Advanced Cardiovascular Life Support Bradycardia algorithm. He was admitted to the Critical Care Unit for close monitoring.

He continued to have symptomatic bradycardia and sinus pauses of 3–4 s duration, and a permanent dual-chamber pacemaker was implanted with DDD mode of operation. His symptoms abated at an atrial paced rate of 60 per min.

OUTCOME AND FOLLOW-UP

The patient is symptom free, without recurrence of seizures in the absence of antiepileptic drug therapy for almost 1 year. Repeat MRI demonstrated stable appearance of vascular anomaly.

DISCUSSION

There are two other case reports of asystole triggering seizures; one being a generalised tonic-clonic seizure and the other a complex partial seizure. The case reports do not mention if the patients had a postictal phase. In both cases, antiepileptic drug therapy did not provide benefit, but permanent pacemaker implantation led to the abolition of seizures.⁷

Sinoatrial node dysfunction is typically seen due to degeneration of nodal tissue from ageing causing decreased impulse generation and inability to increase HR in response to physiological demand. The resultant reduced cardiac output causes global cerebral hypoperfusion that often presents as presyncope or syncope and is known as SSS.^{2 3} SSS may present as convulsive syncope, and the patients with SSS convulsive syncope may be needlessly treated with antiepileptic drugs.⁸

Cavernous malformations may present with seizures.^{9 10} In this case, the patient has neither been treated with antiepileptic drugs nor has he received any therapy for the cavernous malformation in the right frontal lobe. The EEG did not detect any epileptiform activity overlying the right frontal lobe. He has not had any seizures other than the presenting seizure event. Thus, it is unlikely that the structural abnormality is solely responsible for the seizure. It is likely that the partially compromised vascular flow (50% right internal carotid artery stenosis) may have resulted in worse perfusion in the territory supplied by the stenosed blood vessel. This may have precipitated a generalised tonic-clonic seizure with the structural anomaly in this vessel

Learning points

- ▶ Convulsive syncope should be distinguished from true seizure by absence of postictal confusion.
- ▶ Ictal bradycardia syndrome should be distinguished from true sick sinus syndrome (SSS) by absence of persistent sinus dysfunction.
- ▶ SSS-mediated cerebral hypoperfusion may precipitate generalised seizures in case of underlying predisposition.

territory, which would otherwise have been benign, acting as the triggering focus of the abnormal electrical discharge.

Contributors All authors contributed in data gathering, analysis and interpretation. NP and FM were primarily responsible for gathering the data while AAS and NP were responsible for analysis and interpretation of the data. All authors worked in drafting the manuscript and approve the final version. They agree to be accountable for all aspects of the manuscript.

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