



Case Report

Asystole in focal epilepsy complicating a traumatic subdural hematoma

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ABSTRACT

Ictal asystole due to sinus node suppression is a cause of sudden unexplained death in epilepsy. Here, for the first time, we describe a complete atrioventricular nodal block in a patient with non-compressive traumatic subdural hematoma, who developed ictal asystole as a delayed presentation. A leadless VVI pacemaker (ventricular paced, ventricular sensed, and pacing inhibited in response to a sensed beat) was implanted as a preventive measure against seizure-related heart block.

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1. Case report

An 82-year-old male, with permanent atrial fibrillation (AF), experienced a head injury following an accidental fall. He was on metoprolol (50 mg bd) for heart rate control and warfarin for stroke prevention, but not on digitalis. An initial brain computed tomography (CT) scan revealed a left-sided 3 cm × 2 cm supratentorial hematoma, medial to the temporal lobe. The hematoma was stable on discontinuation of warfarin. Two weeks later, he was readmitted due to dizziness and headache. He was fully conscious, with electrocardiogram (ECG) analysis showing fine AF waves at a ventricular rate of 90/minute and a normal QT interval.

While being examined by a neurologist (CHL) in the intensive care unit, the patient suddenly experienced three episodes of seizure, with complete loss of consciousness. The head and eyes turned towards the right side, and the right limbs were twitching. Following each seizure, the patient developed asystole, with the longest episode lasting 30 s and the latency between seizure and asystole lasting more than five minutes. (Fig. 1) The other two asystoles lasted 11.5 and 23.5 s, with the latency between seizure and asystole lasting about 1 and 2 min, respectively. The patient regained consciousness in between seizures and oxygen saturation was 100% throughout. Intravenous levetiracetam was administered to suppress the seizures. Echocardiography showed normal left ventricular function, and the pulmonary arterial pressure was

40 mmHg, as was before the seizures. Electrolytes, including calcium, magnesium, and potassium, were at normal levels. Both ECG and cardiac marker analysis showed no infarction pattern.

Intravenous atropine 1.2 mg was administered and temporary right ventricular pacing was performed. A CT brain did not show any change in the size of the hematoma, or any evidence of brain compression or hydrocephalus (Fig. 2). A leadless VVI cardiac pacemaker (Micra™ TCP, Medtronic Inc., Minneapolis, MN) was implanted five days later. The patient remained free of seizures for six months and the administration of anticonvulsant was tailed off. The pacemaker registered 32% pacing at a programmed pacing rate of 50/minute.

2. Discussion

The novel observations in this report include (1) complete atrioventricular nodal (AVN) block induced by a seizure, (2) the long latency between the end of the seizure and the onset of asystole, and (3) asystole as a potentially fatal complication after traumatic subdural hematoma not causing brain compression.

Ictal asystole is defined as seizure-induced asystole when the RR interval exceeds three seconds and is more than twice the previous RR interval [1]. Temporal or frontal lobe seizures that activate the “operculo-insulo-mesiotemporal-orbital” complex can lead to bradyarrhythmia [2]. While left temporal basal area electrical stimulation could produce high grade AVN block [3], to our knowledge there is no existing clinical report on AVN block induced by seizure. Continuation of AF without any conducted

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ventricular complexes in the three episodes of ictal asystole demonstrated the existence of AVN block.

Head trauma can be fatal due to brain contusion, intracranial hemorrhage and raised intracranial pressure leading to cerebral herniation. However, even if the above phenomena do not occur, head trauma can have equally life-threatening consequences through more indirect ways, as our patient, who developed ictal asystole following the development of temporal lobe epilepsy after traumatic subdural hematoma, demonstrates. In our patient, the latency between seizure and asystole reached 5 min, which is much longer than the 268 s reported previously [1], emphasizing the need for more prolonged ECG monitoring after temporal lobe seizures. The long latency between convulsion and asystole suggests that

activity of the seizure might have spread to a “silent” cardioinhibitory area of the brain.

Management of ictal asystole can be achieved either by preventing the epileptic seizure or by avoiding asystole, should epilepsy develop. Anti-epileptic medications are ineffective in 35% of partial seizures [4], and such patients may require pacing [1]. A case series showed that 70% of patients who had ictal asystole received pacemakers [1]. On the other hand, pacing was reported not to be required in 6 patients with temporal lobe epilepsy [5]. In our patient, it was possible that the subdural hematoma would resolve, but scarring may have occurred in that region and developed into a seizure focus. Given the alarming and repeated ictal asystole, a permanent pacemaker was offered and was accepted by the patient.

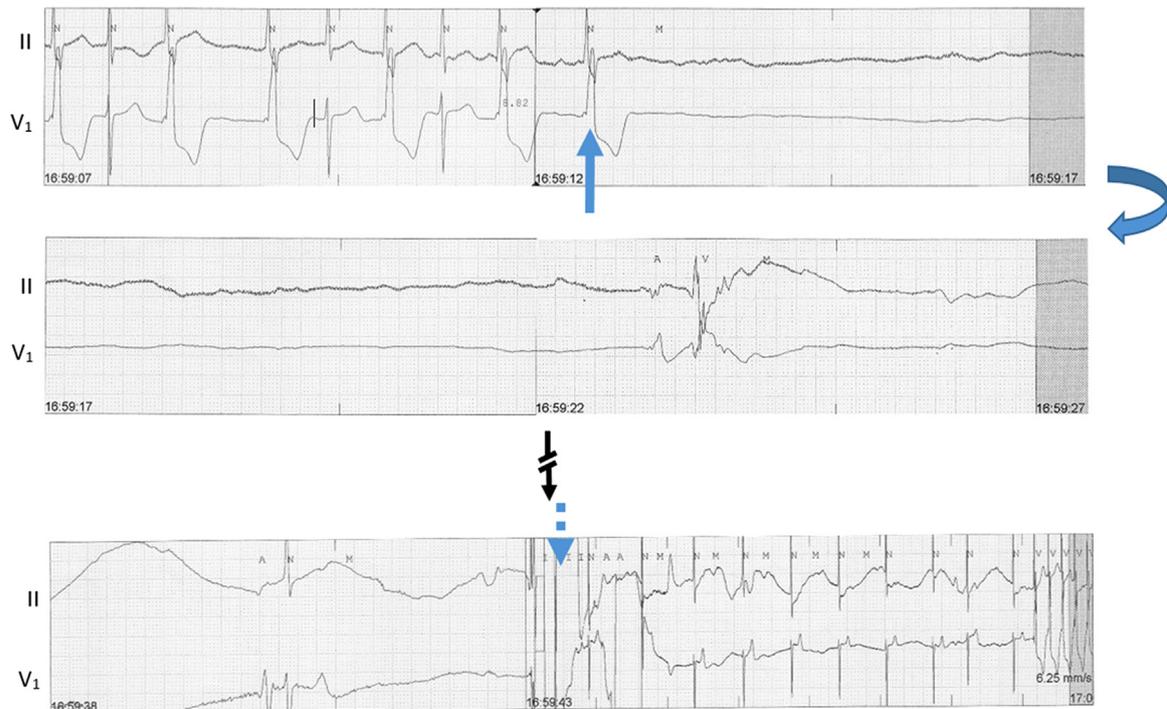


Fig. 1. Telemetry strips of lead II and V₁. The patient was in permanent atrial fibrillation with a ventricular rate of about 90/minute with intermittent wide QRS complex ventricular beats. At 16:59:13 (solid arrow) to 16:59:43 (dotted arrow), 30 seconds of ventricular asystole occurred (Each of the top 2 strips represent 10 seconds bottom strip, 30 seconds).

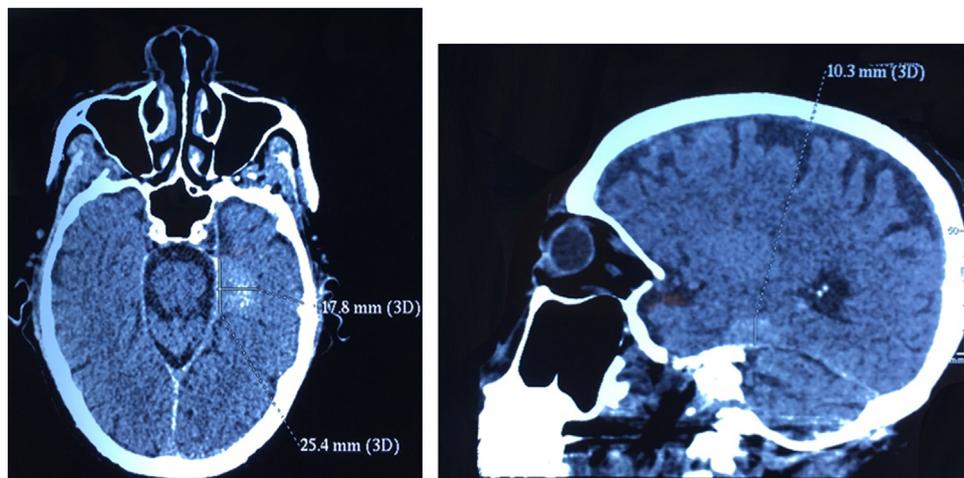


Fig. 2. Axial (left) and sagittal (right) views of computed tomography of the brain showing the hematoma (the hyper-dense area) at the left medial temporal lobe region.

3. Conclusions

This case highlights the importance of recognizing ictal asystole as a complication of temporal lobe subdural hematoma. Life-threatening risk could be due to asystole or the complications that arise from it. Apart from sinus node suppression, our case demonstrates that ictal asystole may also be due to complete AVN block. Patients with temporal lobe subdural hematoma should be closely monitored not only for any seizure, but also for asystolic events after seizures. Pacing, either temporary or permanent, may be necessary.

Conflict of interest

All authors declare no conflict of interest related to this study.

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