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Case report

New onset left frontal lobe seizure presenting with ictal asystole

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ABSTRACT

Ictal asystole is a presumably rare but potentially fatal complication of seizures, most often of temporal lobe origin. It is believed that at least some cases of sudden unexplained death in epilepsy (SUDEP) might be triggered by ictal bradycardia or asystole. Current standard practice is to implant a permanent pacemaker in these patients to prevent syncope and/or death. However, emerging data suggests that effective medical or surgical treatment of epilepsy might be enough to prevent cardiac asystole, eliminating the need for permanent pacemaker placement. We describe a case of new onset left frontal lobe epilepsy in a young athletic patient who presented with near-syncopal episodes but whose comprehensive work-up revealed frequent events of ictal bradycardia and asystole. He responded well to monotherapy using oxcarbazepine, avoiding a permanent pacemaker.

1. Introduction

Ictal bradycardia and asystole are rare but serious complications of seizures, most often associated with temporal lobe epilepsy.[1,2] They are believed to represent autonomic dysfunction triggered by ictal discharges involving the insula, amygdala, cingulate, or hypothalamus.[1–4] Although ictal asystole has been reported to be self-limited in most cases,[1] it is widely theorized that sudden unexplained death in epilepsy (SUDEP) might be caused, or contributed to, by ictal bradycardia or asystole. Other speculated mechanisms for SUDEP include ictal respiratory depression, cerebral depression, and autonomic dysfunction.[5] Since the incidence of SUDEP has been estimated from 1% to 12% of epilepsy patients, it is important that any potential preventive measures be explored.[6] Most reported cases of ictal asystole seem to have had permanent pacemaker placement given the potentially lethal nature of the underlying arrhythmia and the fear for potential future SUDEP.[7] However, there is emerging data in favor of medical or surgical treatment of epilepsy to prevent ictal asystole and thus avoid permanent pacemaker placement. We describe a young athletic patient with new onset left frontal lobe epilepsy presenting with near-syncopal episodes whose comprehensive work-up revealed frequent occurrences of ictal bradycardia and asystole. He became seizure free on monotherapy with oxcarbazepine.

2. Case report

A 38-year-old healthy male kickboxer presented with four brief near-syncopal episodes in the setting of aggressive athletic training, dieting and possible dehydration. The episodes were described as several seconds of staring with loss of awareness but without any abnormal motor activity or falls and a recovery free of confusion. He denied palpitations, chest pain and shortness of breath, but reported lightheadedness and a "weird sensation" immediately before and after each episode. A work-up by a local neurologist including head CT, brain MRI, and 24-h ambulatory EEG were normal. He was referred to our epilepsy center for further evaluation after he experienced a fourth episode, all within a month.

The patient was admitted to the epilepsy monitoring unit (EMU) to attempt to determine if the events were seizures or near-syncope. Scalp EEG electrodes were placed according to partial 10–10 electrode system for extra coverage of temporal and frontal areas. Continuous cardiac telemetry was included as part of our routine EMU procedures. He was not on any antiepileptic drugs before or during the recording. His baseline EEG was normal with no epileptiform discharges.

On the second day of monitoring he had three stereotyped partial onset seizures within a 12-h period while awake and lying supine in bed. Clinically, these seizures consisted of eye deviation

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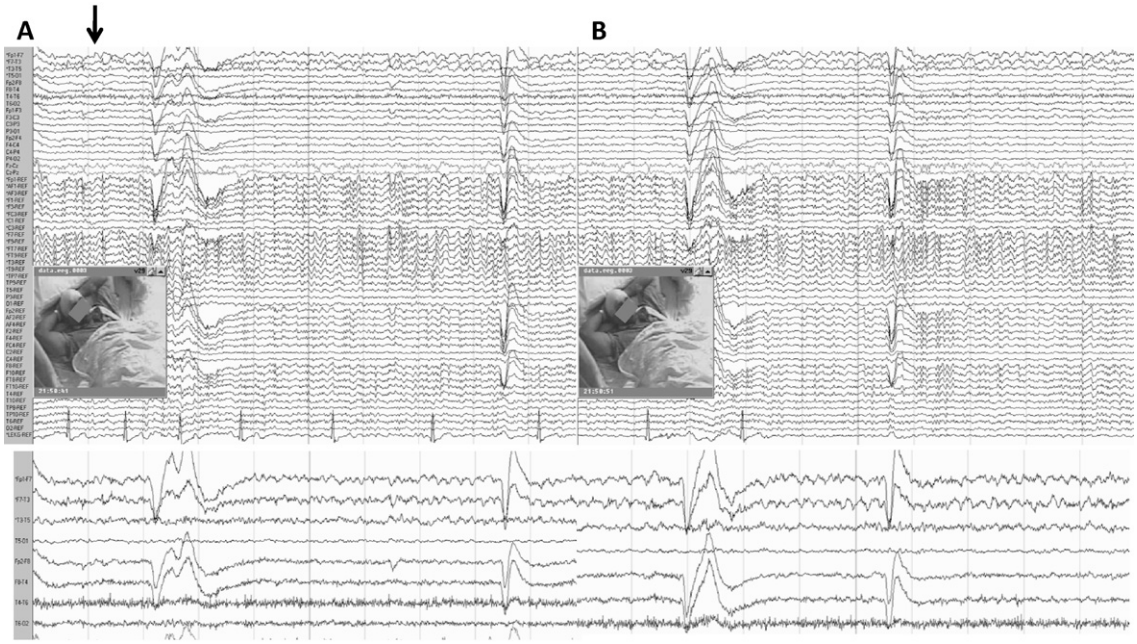

Fig. 1. Two, side by side, consecutive standard 10 s EEG traces (A and B) recorded using partial 10–10 electrode system (top panel) with magnified view of the top 8 leads (bottom panel). Buildup of semirhythmic theta (ictal) discharges in the left fronto-temporal leads (arrow) precedes bradycardia by 1–2 s. The ictal discharges continue in fronto-temporal leads, max frontal, with no clinical signs. The start of asystole is recorded on the EKG lead about 12 s later (top panel).

to the right and right arm contraction, followed by the right leg and left arm remaining in a frozen contraction–extension posture for about 5–15 s before a rapid and full recovery. The EEG associated with these events consistently showed a semirhythmic theta activity in the left anterior head regions lasting for about 8–20 s (Fig. 1) followed by a 3–10-s burst of diffuse delta slow activity, after which the EEG normalized (Fig. 2A). Both telemetry and the EKG lead on the EEG trace detected a progressively marked bradycardia upon the start of the epileptiform discharges that quickly evolved into complete asystole lasting for 22.5, 8.5 and 24.5 s with each seizure, respectively, before a return of spontaneous rhythm (Fig. 2B). The ending of each asystolic episode coincided with delta diffuse slowing on the EEG.

Immediately after the events the patient was awake and oriented. He was able to push the event button and report a "spell" described as "lightheadedness", "funny feeling", and "possibly a brief loss of awareness". There were no other documented episodes of asystole in between these seizures. These symptoms and EEG findings were most suggestive of a left frontal lobe seizure.

Cardiology was consulted for management of the asystolic episodes and possible permanent pacemaker insertion. A 2D echocardiogram demonstrated normal left ventricular size, thickness and systolic function, with an ejection fraction of 55%. The possibility of a permanent pacemaker including the risks, benefits and alternatives were discussed by the management team but deferred by the patient as it would interfere with his active lifestyle, particularly kickboxing. Further, backup pacing might have had the unintended consequence of preventing seizure termination. It has been theorized that global cerebral hypoperfusion, as manifested by bilateral hypersynchronous EEG slowing, plays a role in ending seizure discharges.[2] It seemed clear that the asystole was directly connected to inciting ictal discharges. Therefore, on the third day of admission he was started on the antiepileptic drug lamotrigine, but given the possibility of inducing conduction block, it was switched to oxcarbazepine at 150 mg twice a day (bid) to be titrated up weekly. He had no recurrences of either seizure activity or asystole during the remaining 48 h of monitoring.

The patient also had a two-week outpatient EKG telemetry monitoring within one month of hospital discharge, which showed no episodes of bradycardia or pauses as he remained seizure free during that time on medication. Two months later he reported two seizures both associated with loss of consciousness and falls; it remained uncertain whether he had any related asystole or not. The oxcarbazepine dose was increased from 450 mg bid to 600 mg bid, and after a further loss of consciousness 4 months later, to 750 mg bid. He has remained seizure free on this dose since then (last 6 months). His follow up brain MRI was normal.

3. Discussion

While the long-term benefits of cardiac pacing in preventing falls in patients with ictal asystole has been established[8] our experience with this patient suggests that effective medical therapy to control seizures might be enough to avoid permanent pacemaker insertion. In fact, most recently Strzelczyk et al. have reviewed 15 patients with ictal asystole or bradycardia, all with temporal lobe seizures, and proposed that in patients who achieve seizure freedom through medical or surgical therapy, ictal asystole may no longer be a risk thus obviating the need for cardiac pacemaker implantation. Our patient's excellent response to medical therapy and seizure freedom is further supportive of this approach.

This patient also had several other features that are heretofore uncommon in the described cases of ictal asystole, underscoring potential limitations in our current understanding and areas for possible research. Ictal asystole is often noted in patients with chronic refractory temporal lobe epilepsy.[1] Our patient is unique as he represents a new onset seizure disorder presenting with ictal asystole. Also, the incidence of frontal lobe seizures appearing as a case of near-syncope, thus obscuring the diagnosis of seizure and asystole, is rare. It is to be noted that ictal asystole is not extensively described in cardiac electrophysiology literature. This case raises the possibility of more undetected cases of ictal asystole of frontal lobe origin, where quite often ictal EEG discharges are undetectable on scalp recording. Our patient was in a seated or supine position at the time of his first few seizures, preventing collapse, which he in fact had after hospital discharge with later episodes. Had he collapsed with these initial episodes,

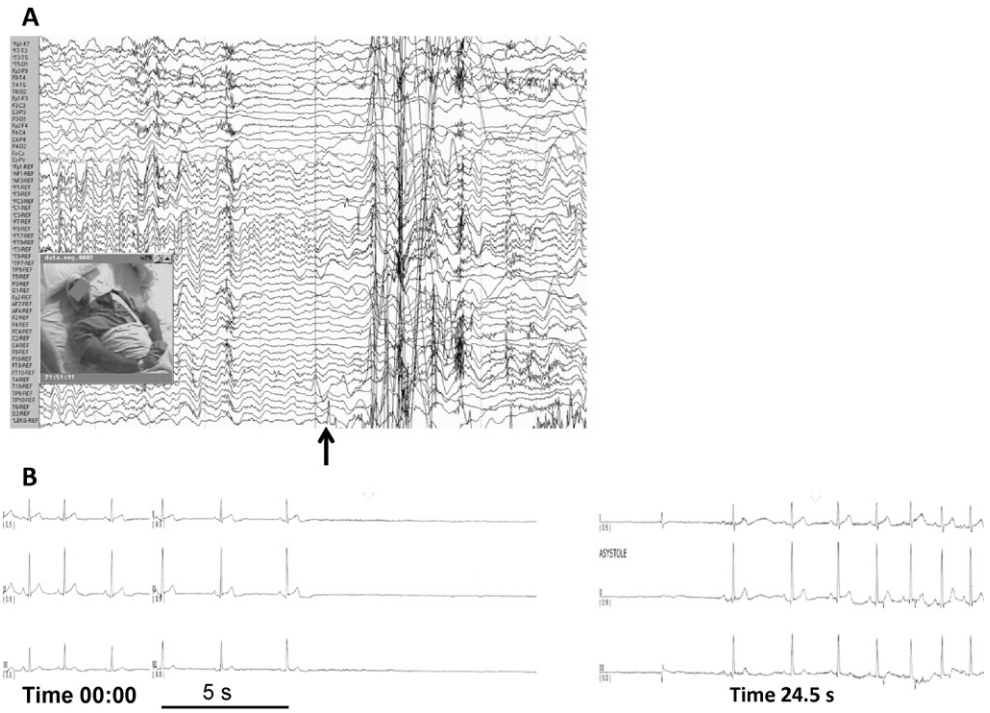

Fig. 2. Diffuse delta slow activity more pronounced on the left, as well as lower voltage faster discharges in the left fronto-temporal head regions are present towards the end of the prolonged asystole (A), before the normal cardiac rhythm returns (arrow). The start and end of asystole is recorded on the cardiac telemetry strip (B).

the events would have been more difficult to distinguish from syncope and potentially delayed the diagnosis of epilepsy. Moreover, such cases could easily be misdiagnosed by a non-neurologist as asystole-induced brain anoxia with secondarily triggered seizures, which would further delay appropriate therapy.

This case also points out the importance of cardiac telemetry as part of epilepsy monitoring since the EKG lead used as part of EEG recording would not trigger an alarm in case of asystole. We propose including cardiac telemetry and pulse oximetry during epilepsy monitoring as affordable safety measures to minimize the risk of missing cardiac or respiratory arrest, which – as this case highlights – can be caused even by subtle seizures that might not be detected based on EEG or clinical presentation alone. Finally, initiating antiepileptic drugs and achieving seizure freedom in ictal asystole could also minimize the risk of SUDEP, which remains an obscure and lethal complication of epilepsy.

References

1. Lanz M, Oehl B, Brandt A, Schulze-Bonhage A. Seizure induced cardiac asystole in epilepsy patients undergoing long term video-EEG monitoring. *Seizure* 2011;**20**:167–72.
2. Schuele SU, Bermeo AC, Alexopoulos AV, Burgess RC. Anoxia-ischemia: a mechanism of seizure termination in ictal asystole. *Epilepsia* 2010;**51**:170–3.
3. Leung H, Kwan P, Elger CE. Finding the missing link between ictal bradyarrhythmia, ictal asystole, and sudden unexpected death in epilepsy. *Epilepsy Behav* 2006;**9**:19–30.
4. Strzelczyk A, Cenusa M, Bauer S, Hamer HM, Mothersill IW, Grunwald T, et al. Management and long-term outcome in patients presenting with ictal asystole or bradycardia. *Epilepsia* 2011;**52**:1160–7.
5. Shorvon S, Tomson T. Sudden unexpected death in epilepsy. *Lancet* 2011. July 5 [Epub ahead of print].
6. Donner EJ. Explaining the unexplained; expecting the unexpected: where are we with sudden unexpected death in epilepsy? *Epilepsy Curr* 2011;**11**:45–9.
7. Zubair S, Arshad AB, Saeed B, Luqman S, Oommen KJ. Ictal asystole—late manifestation of partial epilepsy and importance of cardiac pacemaker. *Seizure* 2009;**18**:457–61.
8. Moseley BD, Ghearing GR, Munger TM, Britton JW. The treatment of ictal asystole with cardiac pacing. *Epilepsia* 2011;**52**:16–9.