

Self-limiting symptomatic peri-ictal bradyarrhythmia due to junctional AV rhythm

Hanmantrao Darku Babarao, Nagappa Madhu, Sanjib Sinha, Chandrajit Prasad¹, Nagaraju Somasekhar², Naveed Ahmad³, Parayil Sankaran Bindu, Arun B Taly

Department of Neurology, ¹NIIR, NIMHANS, ²Department of Cardiology, KIMS, ³Department of Cardiology, SJIC, Bangalore, Karnataka, India

ABSTRACT

We report an elderly man with chronic uncontrolled localization-related epilepsy on three AEDs, who developed in-hospital symptomatic bradyarrhythmia due to junctional atrio-ventricular (AV) rhythm after an episode of generalized seizure during his hospital stay. On further evaluation, the cardiac enzymes were normal and 2D-echocardiogram showed mild left ventricular hypertrophy. Patient recovered from the same with supportive treatment within few hours. We emphasize on importance of prompt recognition of this event and its implication including one of the mechanism for sudden unexplained death in epilepsy.

Key words: Bradyarrhythmia, epilepsy, sudden unexplained death in epilepsy

Introduction

Sudden death is at least 20 times more common in people with epilepsy compared with the general population and may occur without a clear structural or pathological identifiable cause.^[1] Sudden unexplained death in epilepsy (SUDEP) is one of the most common causes of death in patients with epilepsy, with an annual incidence of 2.2-10/1,000 patients.^[2] Some of the postulated mechanisms for SUDEP are cardiac arrhythmias, central apnea, neurogenic pulmonary edema and asphyxia. The occurrence of asystole during a seizure is the most dramatic manifestation of ictal bradycardia and may contribute to seizure-related injury and SUDEP.^[3] There are reports of SUDEP in video-EEG monitoring unit in patients with unsuccessful resuscitation.^[4]

We report an elderly man with chronic epilepsy who developed in-hospital symptomatic ictal bradyarrhythmia

due to junctional escape rhythm and hypotension during a generalized seizure, from which he recovered with supportive treatment.

Case Report

A 66-year-old gentleman had generalized tonic-clonic seizures (GTCS) since the age of 14 years, which was well controlled for nearly two to three decades with antiepileptic drug (AED) viz., phenytoin – 300 mg/day and phenobarbitone – 120 mg/day. Cranial CT scan was reported to be normal. For the last 10 months, there was recurrence of seizures. The seizure frequency was 1-2 episodes per month in spite of polypharmacy with valproate (900 mg/day), carbamazepine (800 mg/day) and phenobarbitone (120 mg/day). He did not have any comorbid illnesses such as ischemic heart disease, diabetes mellitus or hypertension.

He was presented to us 3 days following an episode of GTCS with a confusional state. Initial examination revealed a disoriented patient with gait ataxia, fine tremor of outstretched hands, pes cavus and hammer toes and diminished ankle jerks. There were no other neurological or systemic deficits. Routine investigations including hemogram were within normal limits. Routine ECG on admission was normal. Scalp EEG at admission showed generalized slowing

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Address for correspondence:

Dr. Sanjib Sinha, National Institute of Mental Health and Neurosciences, Hosur Road, Bangalore, Karnataka, India.
E-mail: sanjib_sinha2004@yahoo.co.in

in theta range and there was no epileptiform activity. Routine serum biochemical parameters including liver and renal function tests and electrolytes were normal. Plasma ammonia was slightly raised (76.8 $\mu\text{mol/L}$; range: 11-35 $\mu\text{mol/L}$). MR imaging of brain carried out showed mild generalized atrophy with a cystic lesion secondary to gliosis of the left anterior temporal region [Figure 1]. The serum levels of phenobarbitone was elevated [53.35 $\mu\text{g/ml}$; reference range: 15-40 $\mu\text{g/ml}$], while that of carbamazepine was 7.11 $\mu\text{g/ml}$ [range: 4-12 $\mu\text{g/ml}$] and valproate was 33.58 $\mu\text{g/ml}$ [range: 50-100 $\mu\text{g/ml}$] were within therapeutic limits. A possibility of chronic uncontrolled symptomatic localization related secondary generalized epilepsy due to gliosis in the left temporal region with phenobarbitone toxicity was considered after evaluation. Tablet phenobarbitone was tapered and levetiracetam was initiated (1000 mg/day).

On the third day of hospitalization, the patient had a generalized seizure. During the immediate postictal period, he was drowsy and diaphoretic, with shallow respiration, bradycardia [pulse rate: 52/min, feeble radial pulse] and hypotension [systolic blood pressure: 60 mmHg]. There was no cyanosis. An electrocardiogram obtained at this time showed a heart rate of 43/min, with p-wave coming after QRS complex suggestive of junctional AV rhythm [Figure 2a]. He was given supportive treatment with nasal oxygen (3 L/min), intravenous fluids and dopamine (10 $\mu\text{g/kg/min}$). About 40 minutes later, the heart rate normalized to 72 per minute and blood pressure rose to 100/80 mmHg. The 2-D echocardiogram showed only a mild left ventricular hypertrophy. The serum creatine kinase-MB [21 U/l; normal 9-25 U/l] and Troponin-I done after 4 hours was 0.6 ng/ml (ref: Up to 0.1). Repeat Troponin-I was 0.6 ng/ml. A repeat ECG carried out 2 hour later was normal [Figure 2b]. He recovered completely and at the time of discharge 11 days later, he did not have any neurological deficits.

This event led to a possibility of self-remitting in-hospital symptomatic ictal bradyarrhythmia (junctional AV rhythm) and hypotension during a generalized seizure. After 6 months of follow-up, he was asymptomatic and seizure free on levetiracetam and valproate.

Discussion

Our patient had unique presentation of symptomatic bradyarrhythmia and autonomic dysfunction in the form of severe hypotension, perspiration following a generalized tonic seizure. A more severe such event might have been fatal. Although ictal bradyarrhythmia have been documented in patients undergoing video-EEG, postictal symptomatic prolonged bradyarrhythmia with hypotension is exceptional. The possible risk factors associated with SUDEP are history of generalized seizures and frequent GTCS, severity of seizures, subtherapeutic AED levels, young adults, long duration of epilepsy, early onset of epilepsy, AED polytherapy, and Intelligence quotient < 70 among others.^[2] Our patient had many of these risk factors. Moreover, inter-ictal autonomic dysfunction have been documented in people especially those with uncontrolled seizures.^[5,6]

SUDEP has been defined as the sudden, unexpected, witnessed or unwitnessed, nontraumatic and nondrowning death of patients with epilepsy with or without evidence of a seizure, excluding documented status epilepticus, and in whom postmortem examination does not reveal a structural or toxic cause of death.^[7] It has been difficult to precisely define the causes of SUDEP because most the reports are unwitnessed. Hypothesis regarding the causes of SUDEP are based on a synthesis of what is known from a small number of witnessed cases, information gathered at the scene of unwitnessed cases, and risk factors identified in epidemiological studies. Postictal central or obstructive apnea or a cardiac arrhythmia seems to represent the most likely

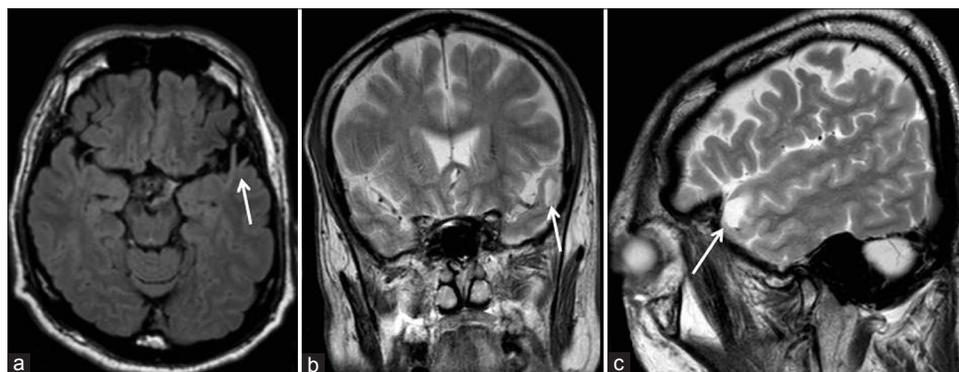


Figure 1: MRI showing a cystic gliotic area in the left anterior temporal region – (a): FLAIR axial; (b): T2W coronal; and (c): T2W sagittal

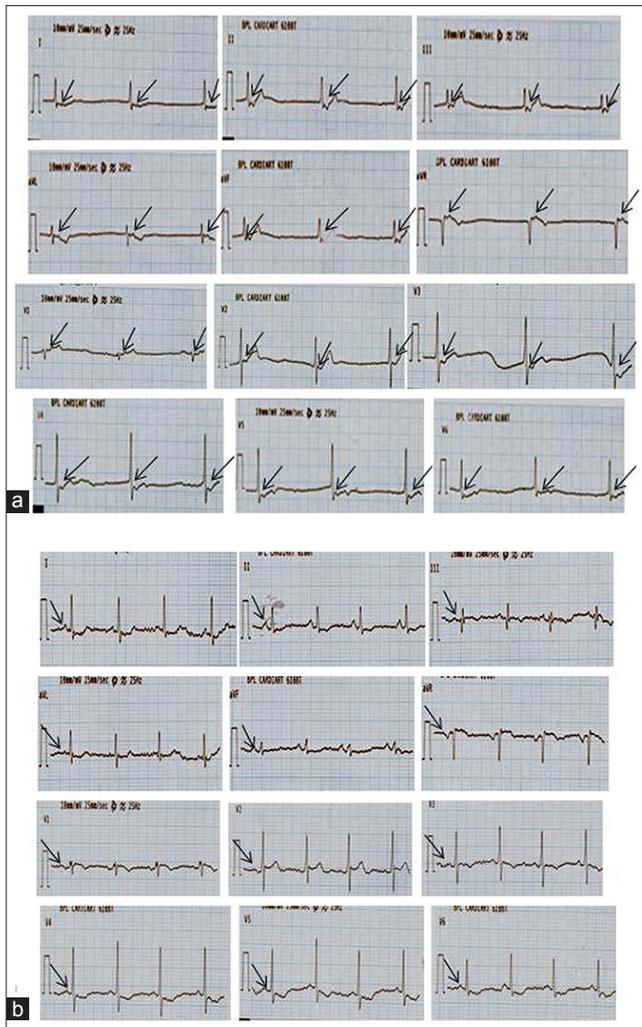


Figure 2: (a): 12-lead ECG taken during the peri-ictal period shows junctional rhythm, inverted P-wave in II, III and AVF (arrow). Note that P-wave follows the QRS complex indicating retrograde P conduction; (b): 12-lead ECG done 2 hours after the seizure demonstrates normal sinus rhythm with upright P-wave in II, III and AVF (arrow) preceding the QRS complex

mechanisms.^[8] Cardiac arrhythmogenesis in cryptogenic epilepsy could be due to ion channel dysfunction and may coexist in the same patient.

Tachycardia is the commonest cardiac abnormality observed during seizures and fatal tachyarrhythmia is one plausible cause for SUDEP.^[9] Tachyarrhythmia is more frequently observed during generalized tonic-clonic seizures. Seeck *et al.* reported a patient who developed ictal tachycardia during seizure while undergoing EEG recording and in immediate postictal period developed asystole for 31 seconds.^[10] Rarely, ictal bradycardia and asystole also may occur.^[11] Proposed mechanisms for this transient bradycardia include a direct effect of the seizure discharge or a response to apnea mediated by the cardiorespiratory reflex.^[12] It is also possible that an autonomic dysfunction during

seizure could result in a catecholaminergic surge causing slow AV junctional rhythm. Other cardiac abnormalities, including supraventricular tachycardia, atrial fibrillation (AF) and junctional rhythms, have been reported in patients undergoing video-EEG/ECG monitoring.^[13-15] Sometimes even prolonged AF has been described during the seizure.^[14]

Our patient had prolonged symptomatic postictal bradyarrhythmia without any cardiac ischemia which recovered with supportive treatment. However, it is likely that due to lack of continuous video-EEG/one lead ECG recording, an independent cardiac arrhythmia might have been missed. Reporting of such cases will increase awareness among treating physicians and thereby monitoring of such events, its early recognition and prompt treatment might prevent death.

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