



Case Report

Cardiac arrest associated with epileptic seizures: A case report with simultaneous EEG and ECG



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ABSTRACT

Ictal asystole is a rare, probably underestimated manifestation of epileptic seizures whose pathophysiology is still debated. This report describes two patients who had cardiac asystole at the end of their seizure. The first patient was a 13-year-old boy with complex partial seizures. His MRI showed symmetrical signal abnormality in the bilateral parietooccipital lobe accompanied by mild gliosis and volume loss. During a 3-day long-term video-EEG monitoring, he had cardiac arrest at the end of one of his seizures that was secondarily generalized. The second one was a 42-year-old veteran with penetrating head trauma in the left frontal lobe due to shell injury. During long-term video-EEG monitoring, he had one generalized tonic-clonic seizure accompanied by bradycardia and cardiac asystole. Asystoles could have a role in the incidence of sudden unexpected death in epilepsy (SUDEP), meaning that the presence of ictal bradycardia is a risk factor for SUDEP. In cases of epileptic cardiac dysrhythmia, prolonged simultaneous EEG/ECG monitoring may be required. Cardiological investigation should be included in epilepsy management.

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1. Introduction

Epilepsy is associated with a two- to three-fold increase in mortality among patients compared with the general population [1]. Sudden unexpected death in epilepsy (SUDEP) is one of the most frequent causes of death among patients with epilepsy [2]. There is strong evidence suggesting that sudden unexpected death in epilepsy (SUDEP) is a seizure-related phenomenon [3–6]. The first description of this phenomenon was introduced by Russell in 1906 [7]. Since then, several cases have been reported in the literature presenting with a drop in heart rate or asystole during the seizure [8]. Bradycardia and asystole resulted from increased parasympathetic flow through the vagus nerve, which originates in the nucleus ambiguus and dorsal nucleus of the vagus in the medulla [9].

On the basis of one study, the incidence of SUDEP ranges from 1:1000 and 1:2000 person-years to 1:200 person-years [10,11]. According to a recent revised definition, SUDEP consists of sudden, unexpected, witnessed or unwitnessed, nontraumatic and nondrowning death in patients with epilepsy, with or without evidence of a seizure, excluding documented status epilepticus (seizure duration >30 min or seizures without recovery in between) and asphyxia; if postmortem examination does not reveal a cause of death, the diagnosis is definite SUDEP, and if there is a preexisting condition before or after autopsy, which could have contributed to the death, it is classified as SUDEP Plus [12]. Strong risk factors for SUDEP include young age, early onset of seizures, the presence of generalized tonic-clonic seizures, male sex, and bedtime occurrence. Less significant risk factors for SUDEP include the prone position, one or more subtherapeutic blood levels, sleep occurrence, and a structural brain lesion [13]. The underlying pathophysiologic mechanisms for SUDEP are not completely understood, but autonomic dysfunction [14]; ictal arrhythmias, ictal bradyarrhythmia, and asystole [15,16]; neurogenic pulmonary edema [17]; and ictal central or obstructive apnea [18–20] were introduced

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in the literature. In this report, we describe two patients with seizure-associated asystole monitored by simultaneous video electroencephalography–electrocardiography.

2. Case 1

A thirteen-year-old right-handed male with seizure disorder was admitted for a presurgical assessment. There was a history of neonatal hypoglycemia during an apparently normal vaginal delivery. No history of birth injury was recorded. He was diagnosed with epilepsy at the age of 5–6 months. The first seizure was associated with secondary generalization. He was started on antiepileptic drugs, but the second seizure appeared six months later. He was symptom-free for 1 year. Habitual seizures began at the age of 10. The seizures typically consisted of blurred vision and upward gaze followed by a loss of consciousness. Many times, there were secondary generalized tonic–clonic seizures. The results of physical and neurological exams were unremarkable. His MRI showed near symmetrical signal abnormality at parietooccipital regions bilaterally (parasagittal aspect) accompanied by mild gliosis and volume loss (Fig. 4). During monitoring, 5 clinical seizures with electrographic changes were recorded. In one episode, there was bilateral rhythmic activity maximum to the left associated with right-side clonic jerk and head and eye deviation to the right that secondarily generalized and was followed by 16 s of asystole at the end of the seizure (Figs. 1–3). Interictal abnormality consisted of bilateral spike-and-wave and bilateral slow activity maximum in the right posterior head region. At the end of monitoring, an anticonvulsant drug regimen of valproic acid and levetiracetam was started, and cardiology consultation was suggested.

3. Case 2

A 42-year-old right-handed male with seizure disorder was admitted for presurgical assessment. His epilepsy was due to penetrating

head trauma in the left frontal lobe from a shell injury. Since then, he had been experiencing episodes of intense fear followed by generalized tonic–clonic movements. The attacks mostly occurred in sleep. These episodes recurred every 3–4 months. Neurologic examination included mild paresis in the right upper extremity and in the distal part of the right lower extremity in the range of 1–2/5. He had the Babinski sign in his right side. The gait was hemiparetic. Brain CT scan showed left parasagittal encephalomalacia (Fig. 8). During video-EEG monitoring, one seizure was recorded that was compatible with his habitual attacks. The very first clinical manifestation was after the initial EEG changes and consisted of a generalized tonic–clonic seizure with the last clonic jerk of the left arm. Afterwards, the SA arrest took place and lasted about 1 min (see Figs. 5–7). The very first EEG change started with 5-Hz spike–slow waves over the left parasagittal area with the maximum amplitude on C3 and F3. The interictal abnormality consisted of delta waves seen at P3, C3, and F3. Considering his cardiac arrest during the seizure, a cardiac consultation was done, and a pacemaker was implanted. During 24 months of antiepileptic regimen, the frequency of seizures was reduced remarkably. No cardiac problem was detected upon follow-up.

4. Discussion

We present two patients with generalized tonic–clonic seizures who had cardiac asystole at the end of their seizures. Theoretically, these asystoles could have a role in the incidence of SUDEP, meaning that the presence of ictal bradycardia is a risk factor for SUDEP [21]. Furthermore, in cases of epileptic cardiac dysrhythmia, isolated EEG or ECG recording may prove insufficient, and prolonged simultaneous EEG/ECG monitoring may be required [22]. Only simultaneous EEG and ECG recording will reveal a possible cerebral origin of arrhythmias in these patients who are, typically, young [23]. Attaining the correct diagnosis is essential because appropriate treatment may prevent cardiogenic SUDEP, which is thought to be related to potentially lethal

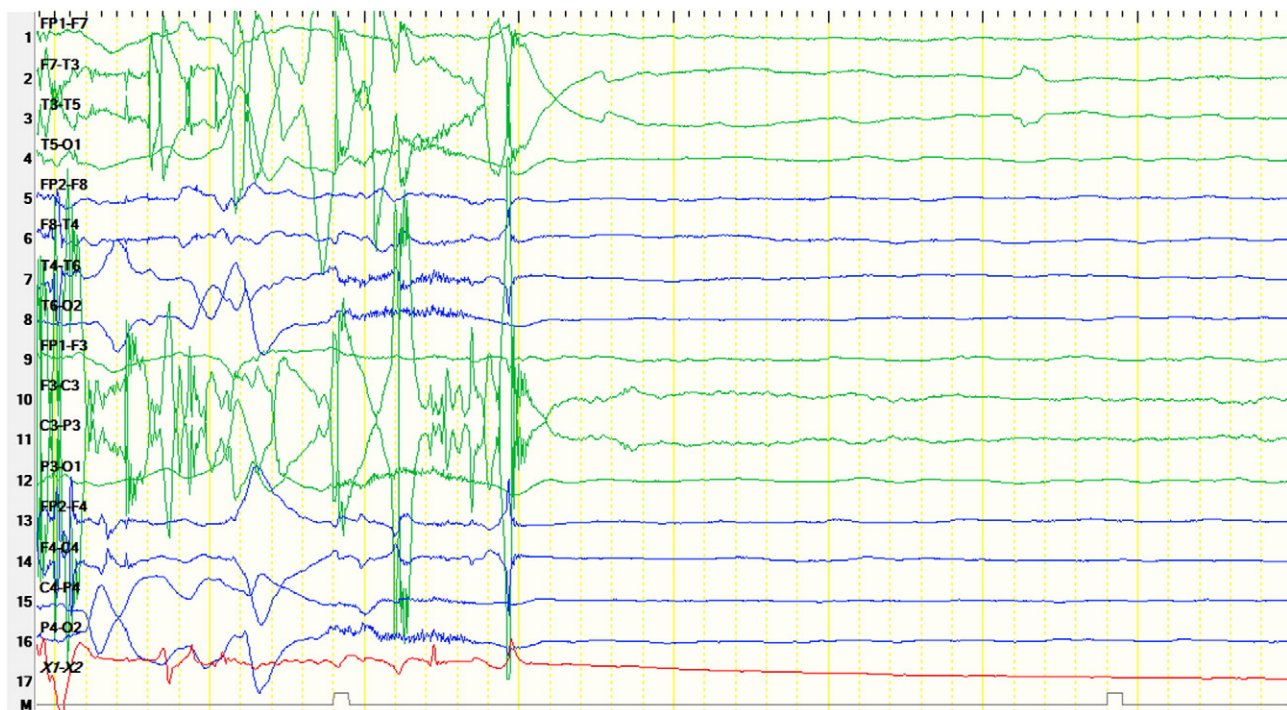


Fig. 1.

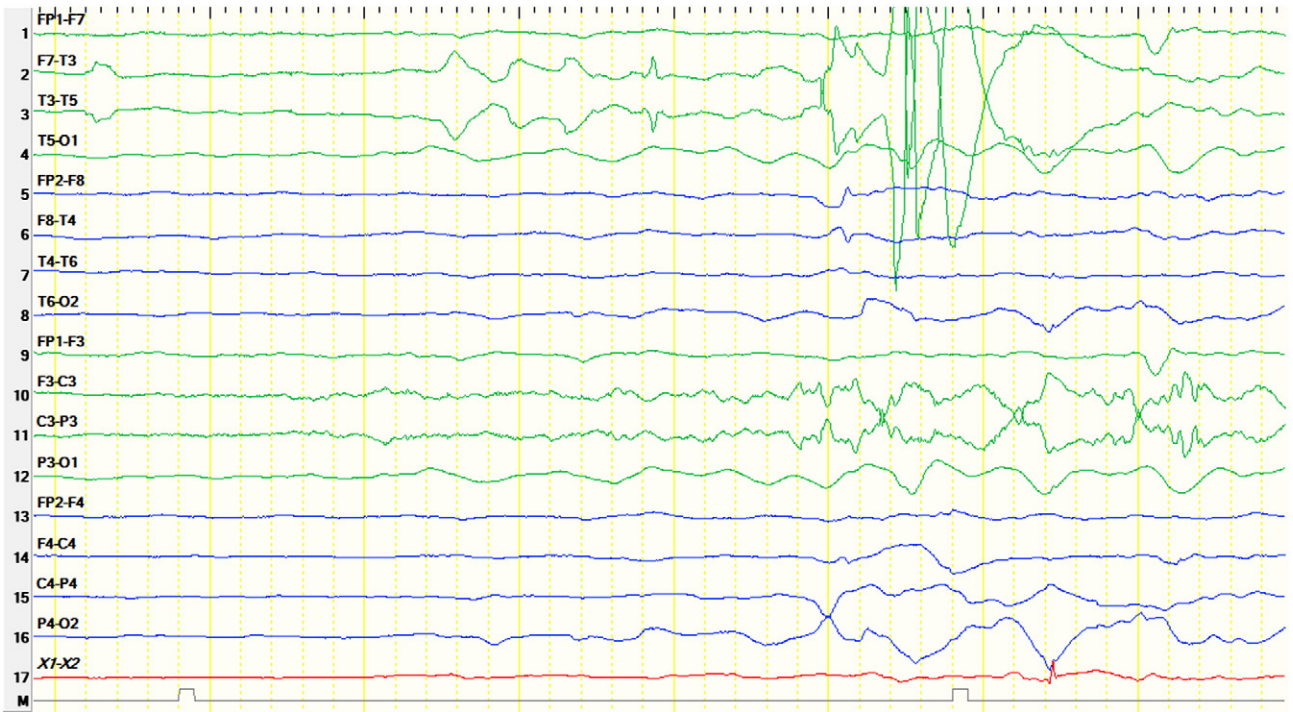


Fig. 2.

arrhythmias, such as asystole induced by epileptic seizures, and to prevent the cardiac side effects of specific antiepileptic drugs [21]. In conclusion, cardiological investigation should be included in epilepsy

management to search for abnormalities of HR or ischemic events. In addition, it can provide an appropriate guideline in pharmacotherapy since certain types of drugs including carbamazepine, phenytoin,

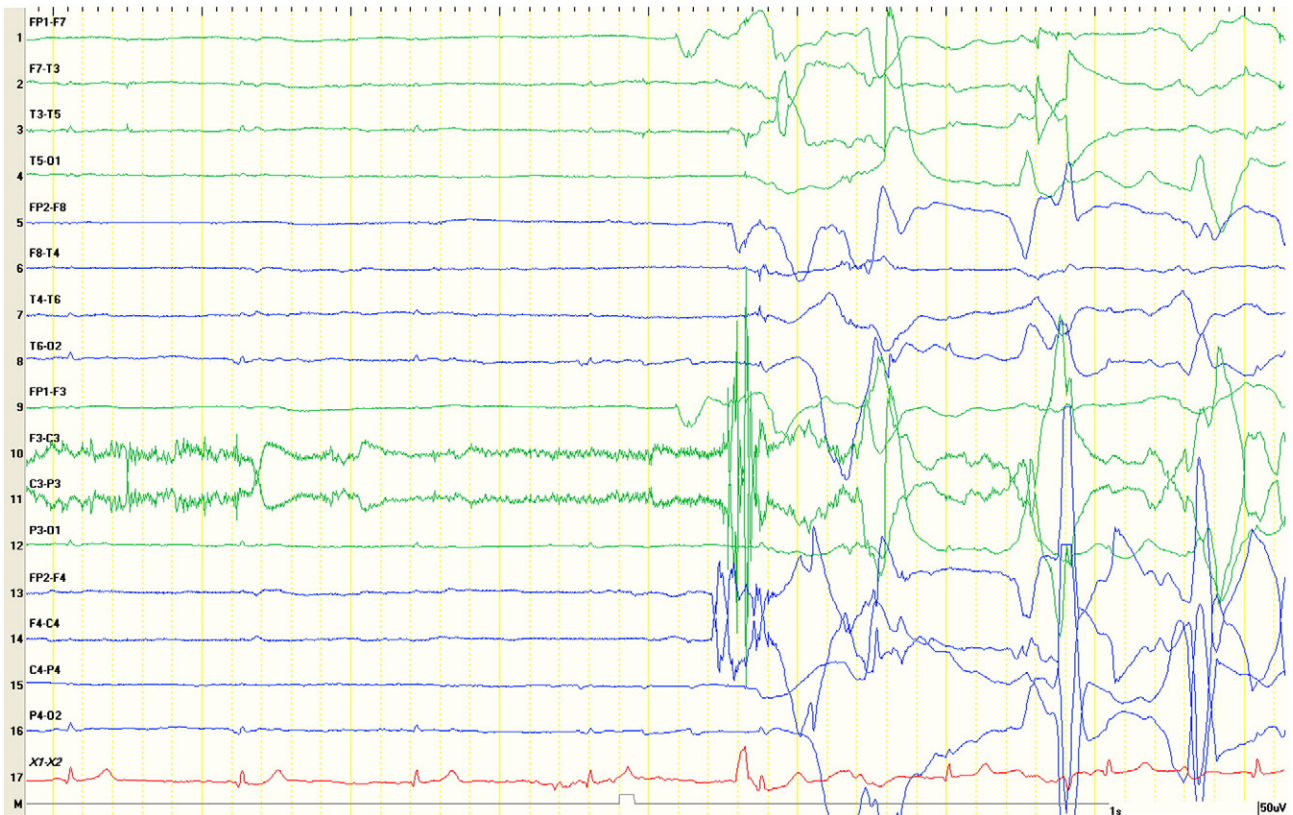


Fig. 3.

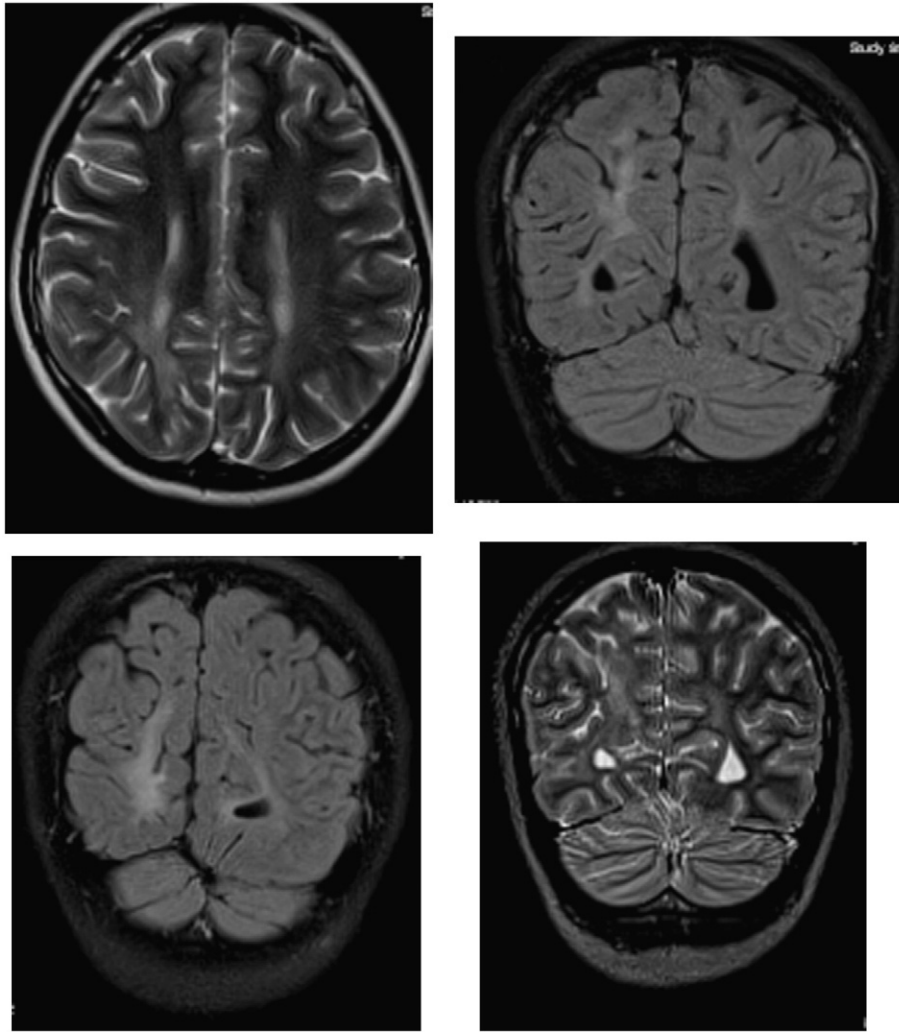


Fig. 4. MRI of Case 1. Near symmetrical signal abnormality is present in the bilateral parietooccipital lobe parasagittal aspect accompanied by mild gliosis and volume loss. It is mostly detectable in the right side, and associated diffusion restriction is not present.

benzodiazepine, and barbiturates should only be used with caution by patients with epilepsy who have cardiac dysfunction. Increasing knowledge about SUDEP risk factors can have a significant preventive role. Therefore, studies that evaluate SUDEP risk factors can provide worthwhile information. Moreover, strategies such as taking a detailed cardiovascular history to get the comprehensive clinical picture including a detailed history of symptoms, risk factors, and prior cardiac findings should be undertaken.

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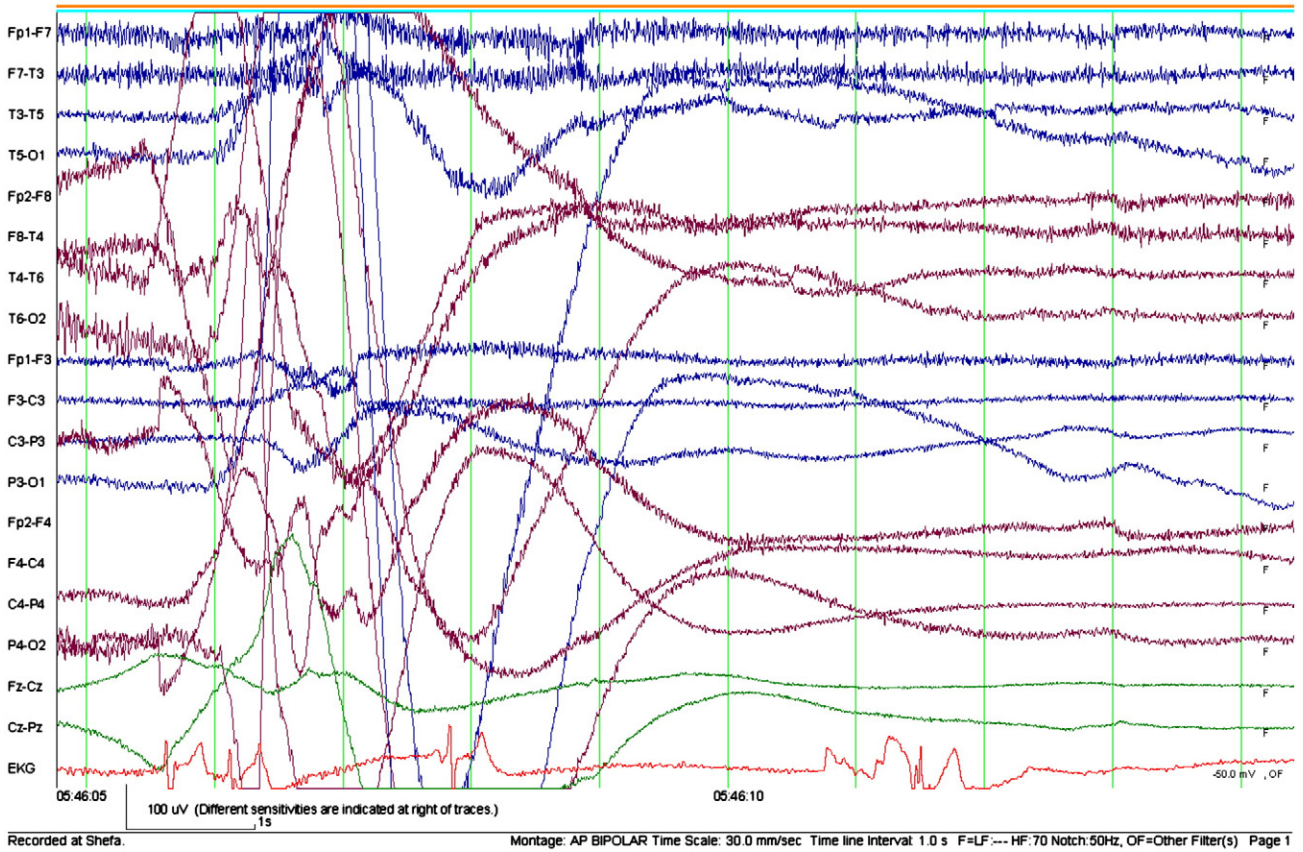


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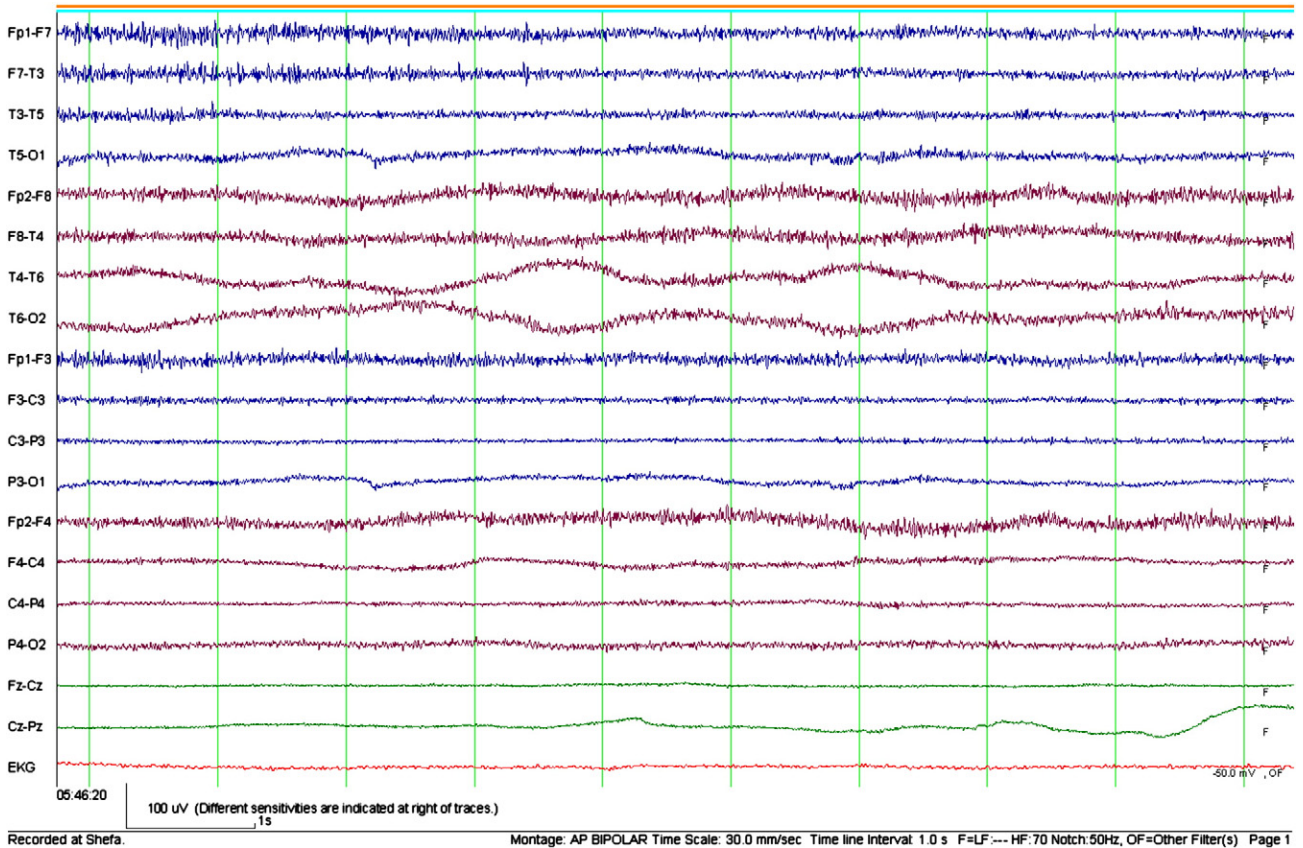


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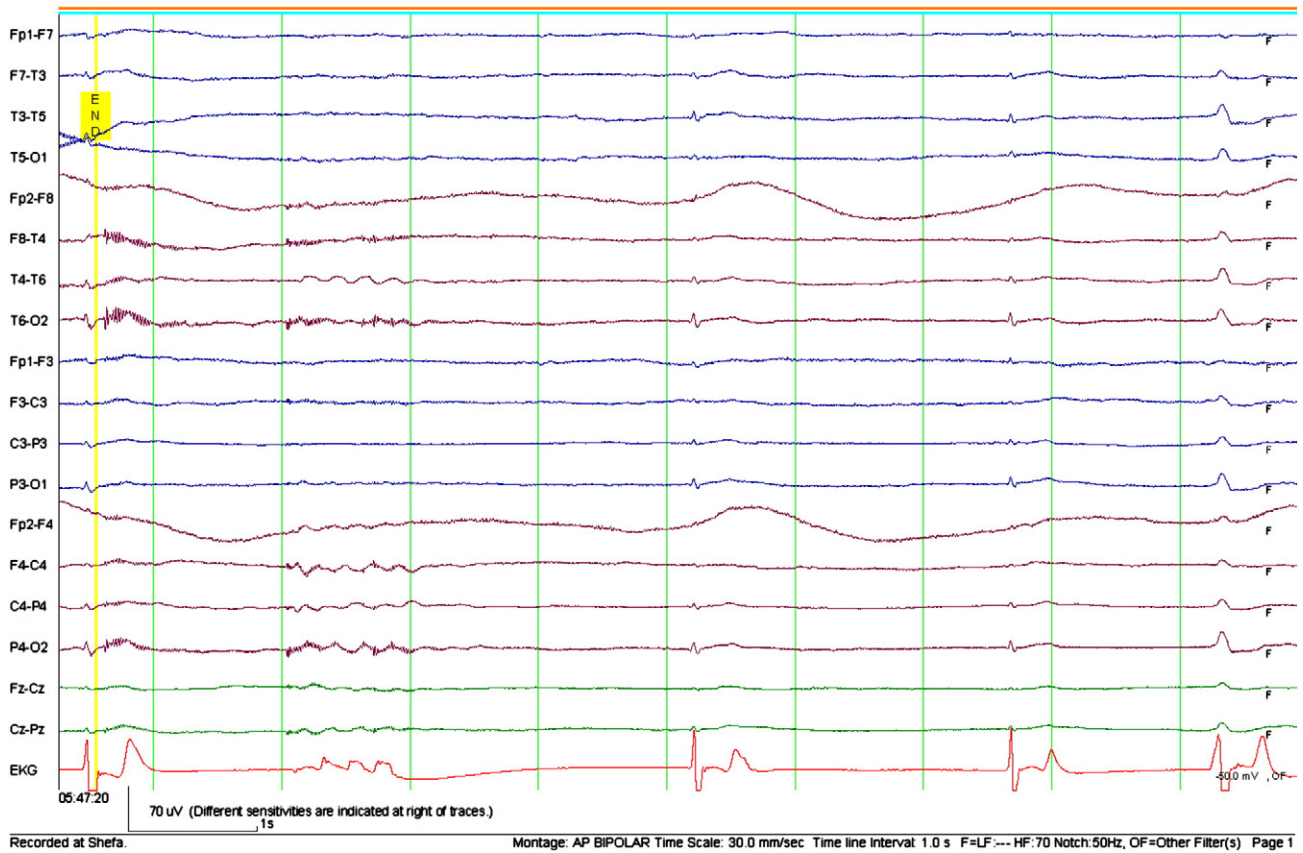


Fig. 7.

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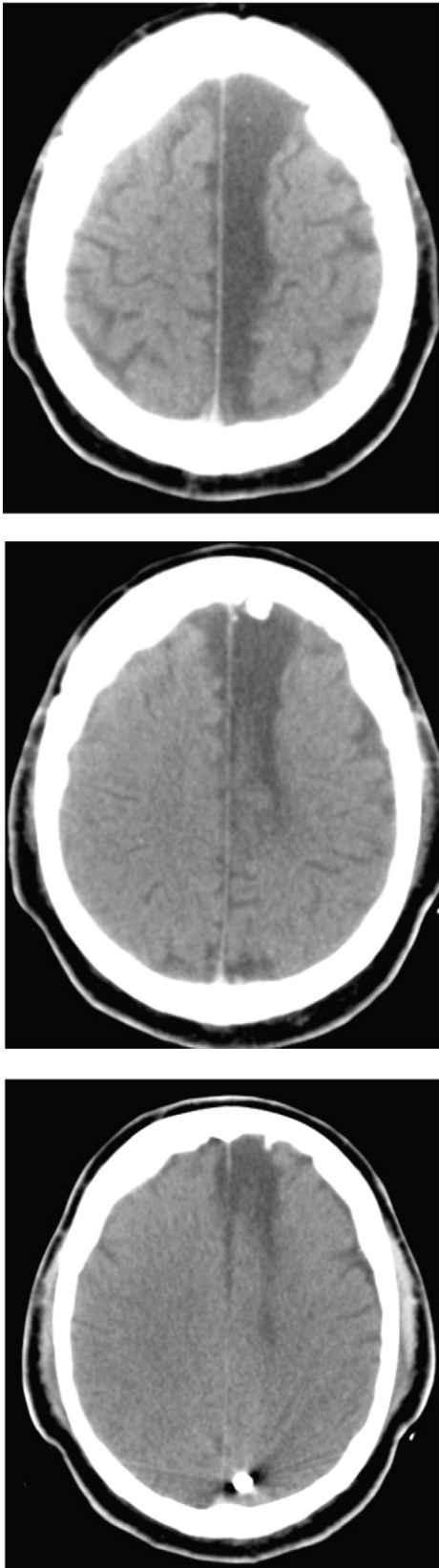


Fig. 8. Case 2: Brain CT scan showed left parasagittal encephalomalacia.