

Continuous Electroencephalography (cEEG) Changes Precede Clinical Changes in a Case of Progressive Cerebral Edema

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Abstract

Background Continuous electroencephalogram (cEEG) is tightly linked to cerebral metabolism and is sensitive to cerebral ischemia and hypoxia. The severity of cerebral ischemia can be seen on cEEG as changes in morphology, amplitude, or frequency, and cEEG may detect neuronal dysfunction at a reversible stage.

Methods Case report and imaging.

Results We present a case of focal cerebral edema with changes seen on cEEG 24 h before clinical signs of increased intracranial pressure. cEEG showed developing asymmetry in the left hemisphere followed by burst suppression. The right hemisphere showed similar progression to burst suppression. Complete suppression of both hemispheres was noted 6 h before clinical signs of herniation. Computed tomography (CT) head confirmed a large left parietal intracerebral hematoma with mass effect.

Conclusions cEEG has applications in monitoring cerebral dysfunction in addition to detecting seizure activity in the intensive care unit. It may serve a vital role in multi-

modality monitoring for early recognition of neurological complications from brain injuries that may not be noticed clinically, which is paramount to early intervention.

Keywords Continuous electroencephalogram · cEEG · Cerebral edema · Herniation

Introduction

Continuous electroencephalogram (cEEG) reflects changes in cerebral metabolism and is sensitive to cerebral ischemia and hypoxia [1]. It is routinely used in neurological intensive care units (ICU) for detection of nonconvulsive seizures [2–4]. cEEG can also be used in cases of increased intracranial pressure (ICP) since cEEG is affected by changes in cerebral metabolism [2]. The changes in ICP on cEEG may occur at a reversible stage and be noticed before clinical findings, thus, allowing for earlier intervention [1]. Prudent monitoring of patients at risk for increased ICP should occur in ICUs. However, patients at risk for increased ICP are not routinely monitored unless there is a concern for ongoing seizure activity.

We present a case of focal cerebral edema with changes on cEEG noticed twenty-four hours before clinical signs of increased ICP.

Case Report

Our patient was a 66-year-old right-handed male with a past medical history significant for end-stage renal disease on intermittent dialysis after a failed renal transplant. He had a previous hospital course complicated by methicillin-resistant staphylococcus aureus bacteremia and infective endocarditis.

All the authors contributed equally to the writing of the case and formatting the images.

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Fig. 1 Computed tomography (CT) head 10 days before clinical herniation showing an intracerebral hemorrhage of the left temporo-parietal region measuring $50 \times 37 \times 17.5$ mm (32.3 cc) with 4 mm midline shift

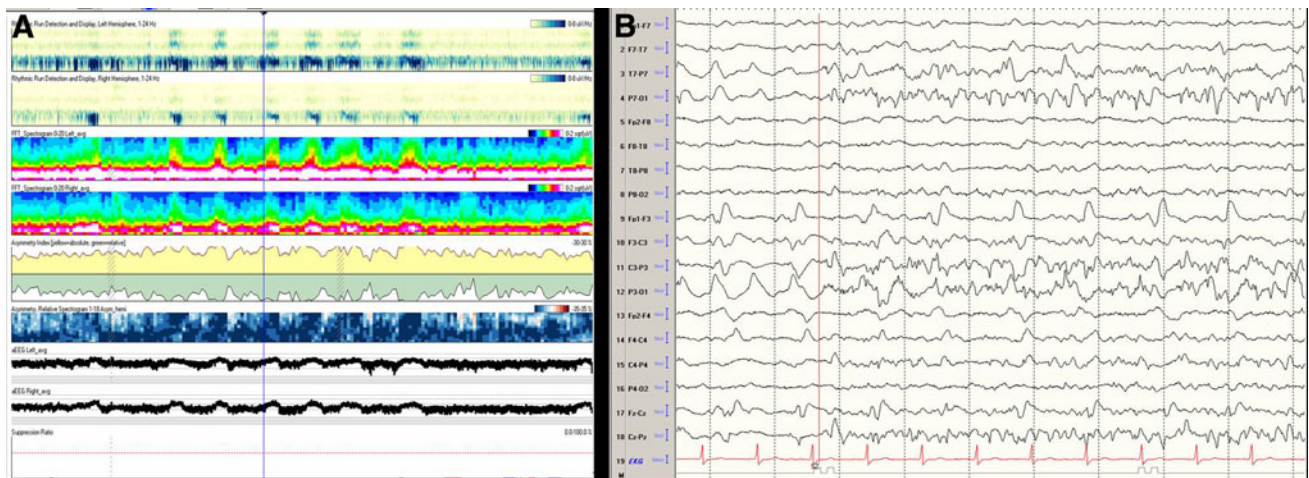
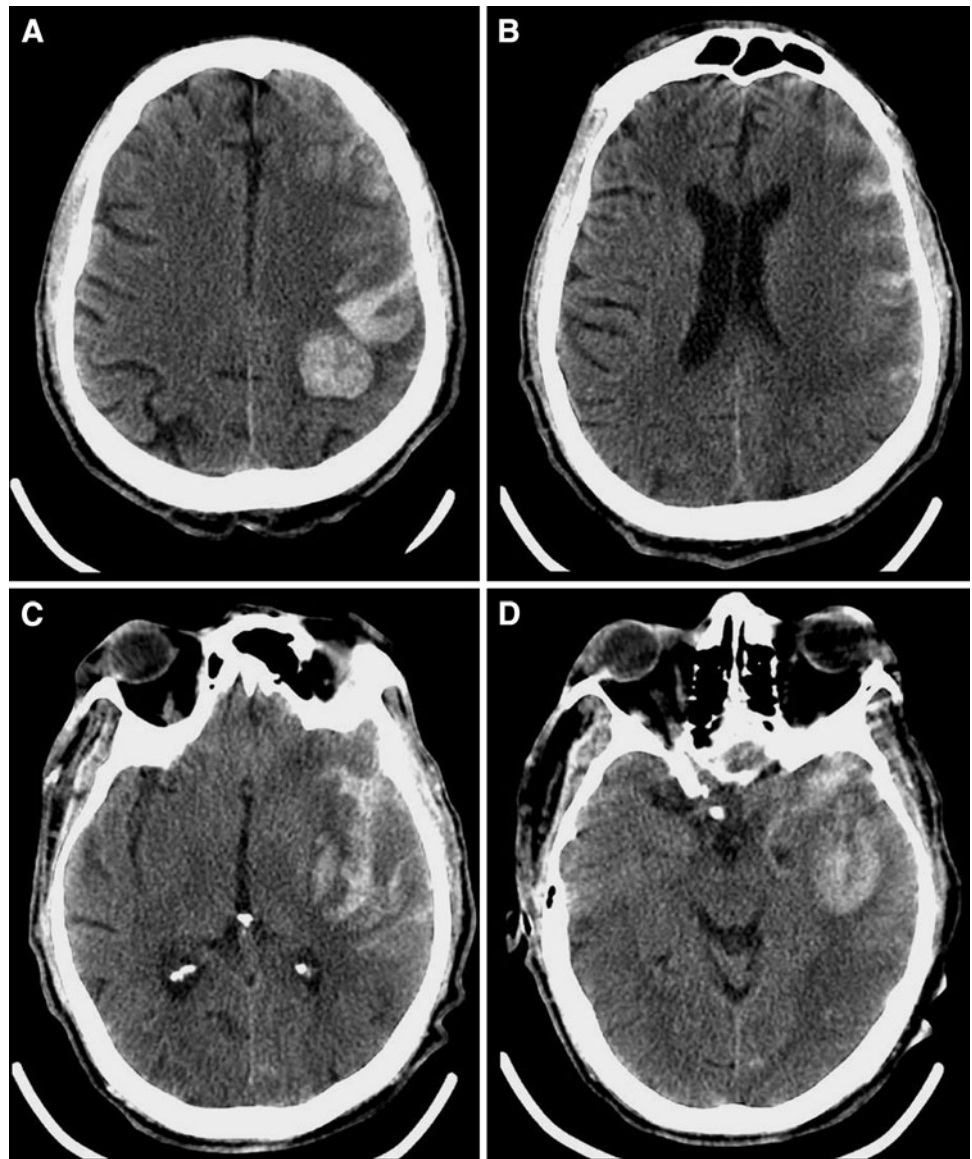
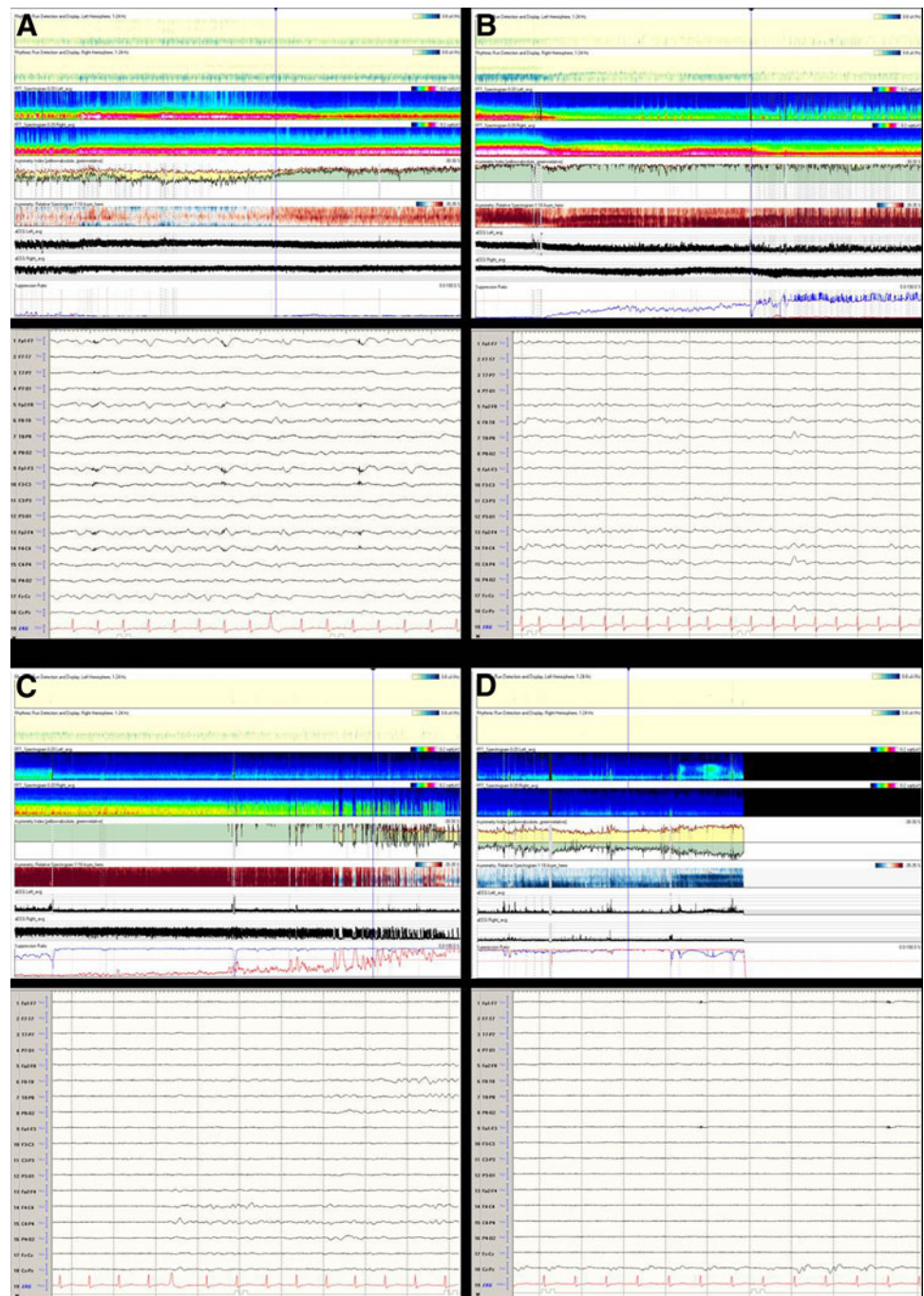


Fig. 2 Continuous electroencephalogram (cEEG) obtained 8 days before clinical herniation showing seizures lateralized to the left hemisphere. No clinical signs were seen with the seizures

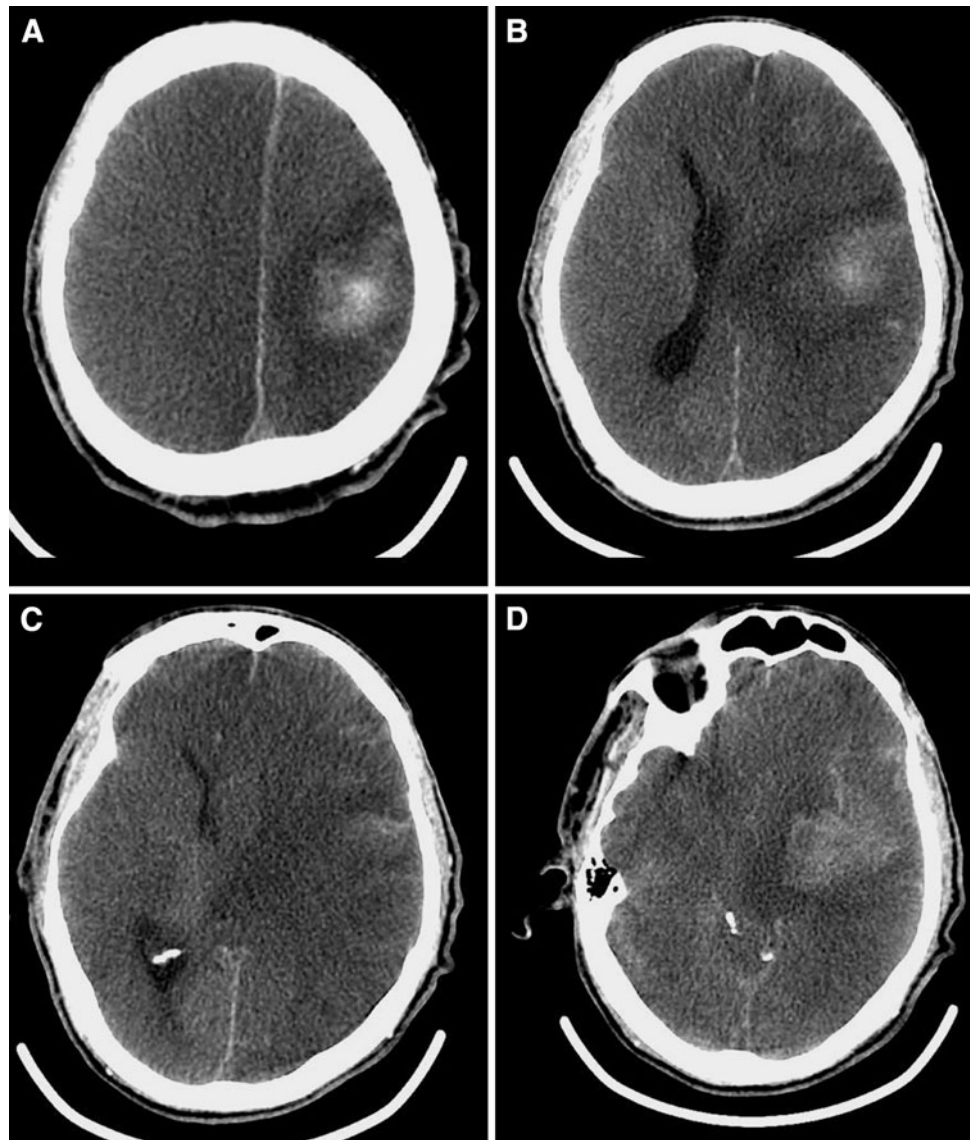
Fig. 3 The evolution of the continuous electroencephalogram (cEEG) shows **a** developing asymmetry with decreased power recorded from the left hemisphere 24 h before clinical herniation, **b** burst suppression of the left hemisphere 15 h before clinical herniation, **c** developing asymmetry with decreased power of the right hemisphere 9 h before clinical herniation, then **d** complete suppression of both hemispheres 3 h before clinical herniation



Computed tomography (CT) scan of the head performed for altered mental status showed a small right parietal intracerebral hemorrhage without mass effect. Four-vessel cerebral angiogram did not reveal underlying mycotic aneurysm or vascular malformation. He was discharged home without any motor or sensory deficits. Two months later, he was re-admitted to the ICU for acute respiratory failure and non-ST elevation myocardial infarction. Anticoagulation and antiplatelet therapy was started. Overnight, he developed right

hemiparesis. CT scan of the head showed an acute left parietal intracerebral hematoma (50 × 37 mm; 32.3 cc) with 4 mm midline shift (Fig. 1). Anticoagulation was reversed. The following day he developed left eye deviation with right arm twitching followed by cessation of movements. cEEG showed non-convulsive focal status epilepticus from the left parietal-occipital region (Fig. 2). He was loaded with lorazepam, phenytoin followed by levetiracetam and phenobarbital resulting in cessation of electrographic seizures but persistent

Fig. 4 Computed tomography (CT) head 1 h after clinical herniation again showing the intracerebral hemorrhage but now measuring 50 × 37 mm (64.3 cc) with 16 mm midline shift



periodic lateralized epileptiform discharges (PLEDs) from the left hemisphere. Over the next 24 h, the cEEG showed a developing asymmetry with decreased power recorded from the left hemisphere. This progressed to burst suppression. The right hemisphere similarly followed with developing asymmetry and decreased power progressing to burst suppression. The medical ICU team was notified. Complete suppression of the cEEG in both hemispheres was noted 6 h before his development of clinical signs of herniation with a left dilated, non-reactive pupil, and left hemiplegia. The progression of the cEEG is seen in Fig. 3. Repeat CT head confirmed a left parietal intracerebral hemorrhage with a new 16 mm shift, increased volume of 64.3 cc, and diffuse cerebral edema (Fig. 4). Medical interventions for decreasing intracranial pressure were unsuccessful. Detailed discussion with family of coexisting medical co-morbidities and poor functional prognosis ensued. The family ultimately decided to withdraw care.

Discussion

The clinical applications of cEEG in the ICU beyond the use for monitoring of non-convulsive seizures are not widely used. This case highlights the use of cEEG in following cerebral function other than monitoring for non-convulsive seizure.

Non-convulsive seizures are under-recognized in the ICU. Previous studies have shown that 8–34% of non-convulsive seizures are without clinical signs [3]. In addition, monitoring for at least 48 h is needed to identify greater than 90% of non-convulsive seizures in comatose patients [5]. Furthermore, 7–36% of patients with intracerebral hemorrhage have been found to have non-convulsive seizures [4]. Intracerebral hemorrhages can cause changes in intracranial pressure (ICP) and cerebral dysfunction. In addition to recognizing non-convulsive seizures, cEEG is

also useful in following cerebral function, such as changes in ICP.

There is a physiologic coupling between cEEG and regional cerebral blood flow allowing cEEG to be helpful in identifying early changes in cerebral metabolism before clinical recognition [2, 6]. Studies from intraoperative EEG monitoring and animal models have shown that EEG changes occur within 5 min of acute cerebral ischemia [6, 7]. In addition, in subarachnoid hemorrhage patients suspected to have vasospasm, cEEG has been used to detect onset of delayed cerebral ischemia before sonographic vasospasm [8, 9]. cEEG is sensitive to changes in cerebral blood volume and compromised blood flow [2]. cEEG abnormalities have been reported at decreased cerebral blood flows (CBF) as progressive changes in morphology, amplitude, or frequency correlating with severity of cerebral ischemia [7, 10, 11]. cEEG can detect CBF changes at the preservation of synaptic transmission and at energy failure/cell death [1, 6]. Thus, cEEG can detect neuronal dysfunction at a reversible stage [1, 6]. The morphologic and frequency changes of EEG at various CBF values and neuronal damage have been previously demonstrated [6]. This aspect of cEEG is important particularly for patients with dysautoregulation from cerebral insult who are exquisitely sensitive to changes in cerebral perfusion pressures (CPP) [1]. cEEG used in these patients can provide a real time monitoring of changes in cerebral perfusion pressures. If definitive therapeutic strategies to reverse the underlying pathology, such as osmotic agents for cerebral edema, are applied during this critical time, restoration of cerebral function may possibly be salvaged [12]. The improvements in CPP can be seen on cEEG before clinical signs [13]. This feature of cEEG could be used in patients with malignant strokes awaiting clinical indication for hemicraniectomy.

Currently, trending software compares one hemisphere to the other for level of asymmetry. This hemispheric asymmetry comparison was the predictor in our case. A more practical, but theoretical, trending software would look at the regional supply of the vasculature, rather than at a hemisphere grossly. This feature would allow for better identification of tissue at risk.

In conclusion, cEEG can add value to detecting critical events in the neuro-ICU at potentially reversible stages. It can be added to other ICU monitoring modalities for comprehensive brain monitoring. The advantage of cEEG is the availability of continuous monitoring and the ability

to be performed on patients, who are asleep, sedated, paralyzed or have altered consciousness [6]. cEEG should be utilized in monitoring events of cerebral function in addition to detecting seizure activity. It has a valuable role in early recognition of neurological complications from brain injuries that may not be noticed clinically, which is paramount to early intervention. The practical utility and feasibility of using cEEG needs further exploration. cEEG provides a noninvasive continuous set of data that can show predicted changes in a time frame allowing for intervention.

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