

Ictal lateralized periodic discharges presenting as *epilepsia partialis continua* in a patient with chronic stroke

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Abstract

Lateralized periodic discharges (LPDs) are a frequent finding in acutely ill patients undergoing EEG recordings, but their ictal versus interictal nature is still debated. When LPDs are clearly time-locked to motor clinical manifestations, they are usually recognized as ictal and treated accordingly. We present a case of ictal LPDs presenting as *epilepsia partialis continua* in a patient with post-stroke frontal lobe epilepsy who was reducing antiepileptic polytherapy.

KEY WORDS: lateralized periodic discharges, ictal LPDs, *epilepsia partialis continua*.

Introduction

Lateralized periodic discharges (LPDs) have long been considered an interictal phenomenon, which is frequently associated with acute cerebral lesions such as acute stroke, encephalitis or other brain injuries in acutely ill patients (1). Although LPDs are associated with increased risk of clinical and/or electrographic seizures, true ictal LPDs are an uncommon finding (typically found in *epilepsia partialis continua*), and there is still considerable debate as to whether this EEG pattern warrants treatment.

We report on a case of a woman with chronic ischemic stroke admitted for *epilepsia partialis continua* (EPC), who was found to have ictal LPDs as the sole EEG correlate.

Case report

A 50 years-old woman with chronic ischemic stroke presented to the emergency department for continuous and rhythmic 0.3 Hz twitching of her left arm for 2 days, without alteration of consciousness and no other significant new neurological deficits.

Her past medical history was notable for a large right Middle Cerebral Artery (MCA) ischaemic stroke in 2015 (two years before this presentation) caused by non-traumatic dissection of the right internal carotid artery, complicated by hemorrhagic transformation with subsequent refractory intracranial hypertension which required decompressive hemicraniectomy; while in the ICU, continuous EEG monitoring revealed subclinical non-convulsive seizures and she was started on levetiracetam and lacosamide to obtain EEG-guided seizure control. After discharge from the ICU, she gradually reduced the dose of levetiracetam and gradually withdrew lacosamide during a prolonged inpatient rehabilitation programme, with no mention of further seizures.

Unenhanced head CT scan showed encephalomalacia involving almost the entire territory of the right MCA, sparing a small cortical area in right pre-motor cortex, with no new lesions (Fig. 1). A diagnosis of focal motor status epilepticus was made on clinical grounds, she was treated with diazepam 10 mg iv followed by lacosamide 200 mg iv, and her left arm twitching promptly abated. She was admitted to the Stroke Unit for close clinical monitoring.

The following day, twitching in her left arm appeared again. A 18-channel EEG with muscle polygraphic recording, showed right frontal LPDs with a frequency of 0.3 Hz time-locked to rhythmic jerking of her left arm (Fig. 2). LPDs were the only electrical correlate of the ictal motor phenomena. This finding was consistent with a diagnosis of EPC caused by ictal LPDs, a typical yet rare presentation.

A decision to restart her previous antiepileptic medications was made, and levetiracetam and lacosamide were up-titrated over the course of 1 week. Repeat EEGs showed a progressive reduction in amplitude of the discharges until they disappeared, accompanied by gradual waning and disappearance of the clonic jerks within 4-5 days.

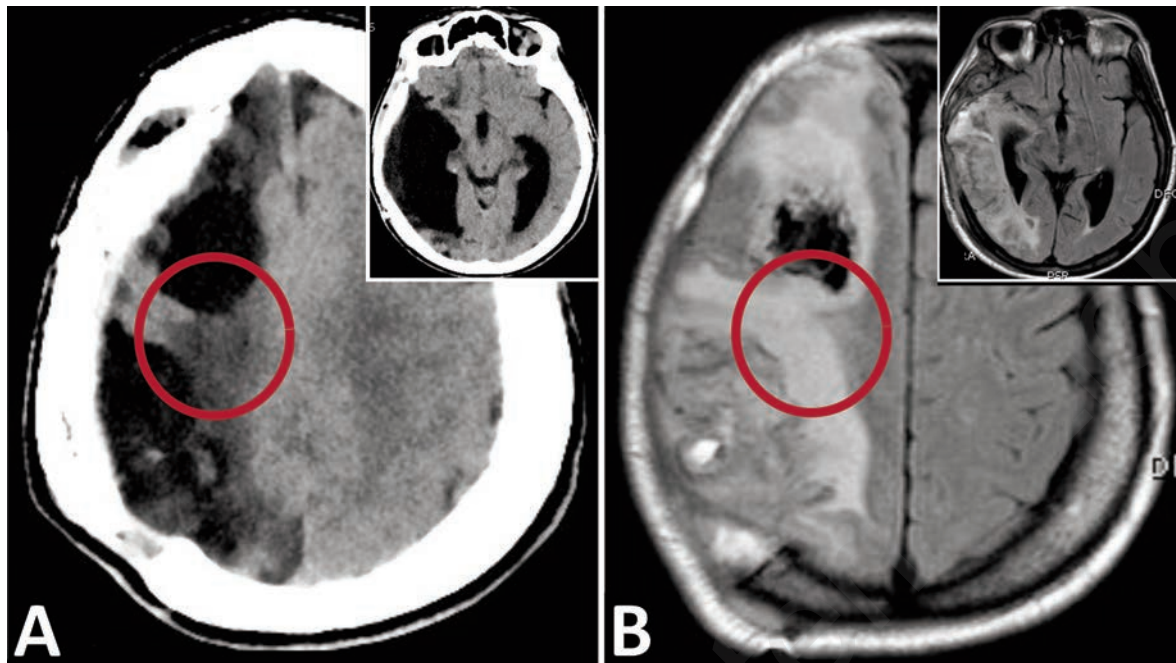


Figure 1 - A, B - A. Unenhanced head CT scan at the time of admission for EPC, showing no new lesions and large malacic areas involving nearly the entire territory of right MCA with relative sparing of cortical tissue in the right premotor area; B. MRI brain images (FLAIR sequence) obtained at the time of her stroke, demonstrating acute ischemic changes in the right hemisphere. Red circles approximately localize the cortical areas where LPDs were recorded by EEG.

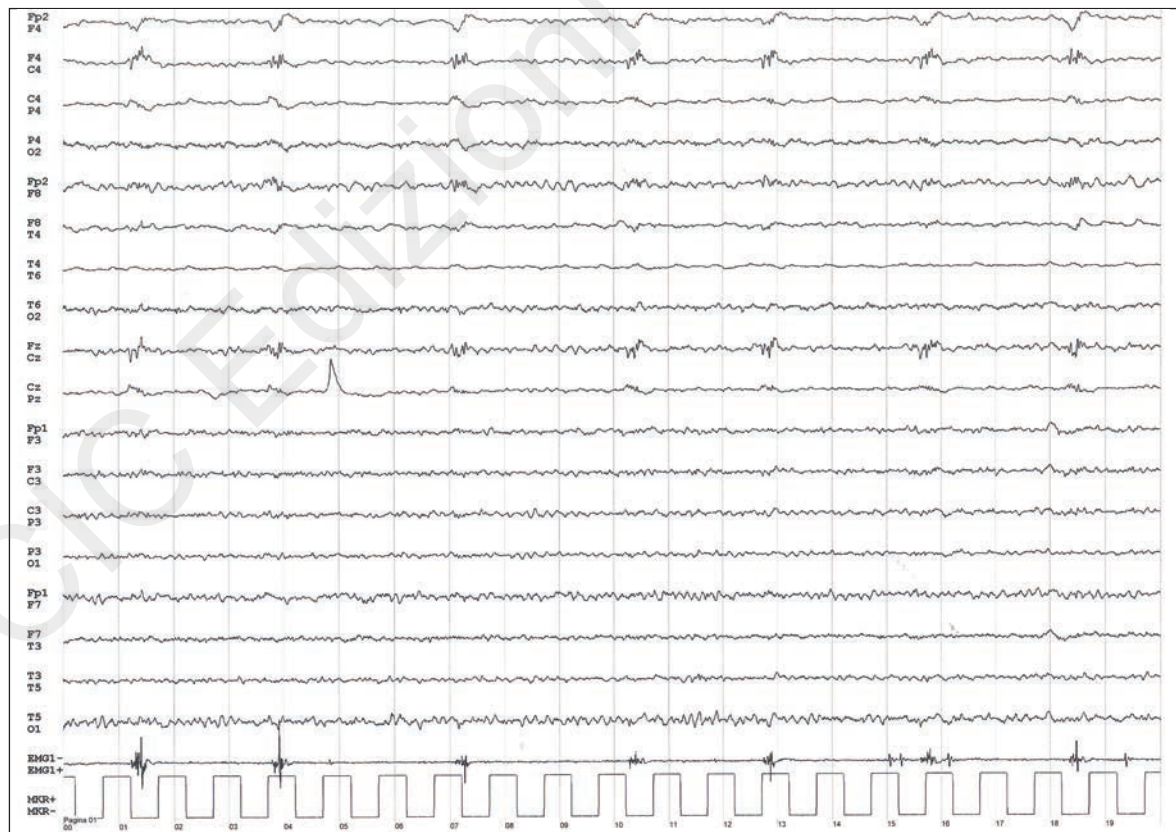


Figure 2 - EEG epoch (20 sec) obtained after 3 days of EPC, showing 0.3 Hz LPDs in the right central areas, clearly time-locked with EMG potentials in the left deltoid and with clinically visible brief clonic movements of the left arm.

She was discharged home with levetiracetam 2000 mg/day and lacosamide 400 mg/day, no further seizures have been reported in 6 months follow-up.

Discussion

LPDs are defined as “waveforms with relatively uniform morphology and duration with a quantifiable inter-discharge interval between consecutive waveforms and recurrence of the waveform at nearly regular intervals”, confined to one hemisphere (2)

They were previously known as PLEDs (periodic lateralized epileptiform discharges), but were renamed LPDs in the last version of the Standardized Critical Care EEG Terminology of the American Clinical Neurophysiology Society (2) because the morphology of periodic complexes often lacks clear epileptiform characteristics.

LPDs are commonly seen in acutely ill subjects undergoing EEG recordings. Their true nature is still unresolved and they are considered among the patterns in the ictal-interictal continuum (3). LPDs are generally viewed as an acute interictal EEG pattern not to be treated aggressively (4).

However, in rare cases LPDs may be ictal in nature and be the only EEG correlate of clinically apparent seizures, specifically when LPDs are time-locked to contralateral ictal motor phenomena. In our case, 0.3 Hz LPDs in the right hemisphere (largely affected by chronic post-stroke damage) were time-locked with clinically observable clonic movements in the left arm. Despite polygraphic recording of muscles involved by the ictal discharge was available, we believe that visual clinical observation was sufficient to establish a time-locked association at this low frequency. Ictal LPDs are classically described in motor *Epilepsia Partialis Continua* (EPC) (5), but have also been described in cases of sensory or cognitive seizures, or subtle motor manifestations (6-8). In critically ill patients with altered mental status these correlations may be easily overlooked or not recognized at all.

A case series published in 2014, exploring the utility of classifying ictal versus non-ictal LPDs on the basis of time-locked clinical phenomena, challenged this practical assumption and cast doubt on the concept that they represent different populations: LPDs defined as ictal were more likely to be associated with central head regions or lesions involving the clinically eloquent sensorimotor cortex compared to non-ictal LPDs, and the Authors concluded that time-locked criteria probably convey more a cerebral localization rather than a functional distinction, calling for caution in treatment deci-

sions based solely on clear clinical correlates (9). Notably, our case is consistent with these findings, since ictal LPDs were generated by right central areas spared by encephalomalacia.

Epilepsia partialis continua is classically and narrowly considered a subgroup of focal motor status epilepticus (as defined by the ILAE Task Force in 2015) (10), with prolonged muscle jerks ranging from single muscles to an entire hemibody. EPC recognizes a wide range of etiologies, the most studied being Rasmussen's encephalitis, but stroke is considered a typical cause too. Treatment is influenced by etiology. In our case, initial treatment of EPC as a focal status epilepticus (intravenous diazepam followed by intravenous lacosamide) was only transiently effective. After relapse, polytherapy was gradually increased and this was followed by gradual disappearance of both clinical motor phenomena and LPDs. Since etiology in our case was reduction of antiepileptic drug polytherapy in a chronic post-stroke frontal lobe epilepsy, prompt resumption of polytherapy proved successful in resolving EPC.

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