Clinical commentary with video sequences

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# Epilepsia partialis continua of the abdominal muscles due to cerebrovascular disease

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# Introduction

Epilepsia partialis continua (EPC) is a rare form of focal motor status epilepticus that is characterized by continuous jerking of a limited part of the body, lasting from hours to weeks or years (Fernández-Torre *et al*., 2004). There is typically a predilection for face and distal limb involvement, although the trunk or abdomen may also rarely be affected (Tezer *et al*., 2008).

Cerebrovascular lesions have been referred to as one of the common causes of EPC in adults (Chalk *et al*., 1991). However, in cases of abdominal involvement, most documented causes differ and primarily include: brain tumours (Matsuo, 1984), focal cortical dysplasia (Tezer *et al*., 2008), central nervous system infections (Chalk *et al*., 1991), and subdural haematoma (Johnson, 1969).

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# Case studies

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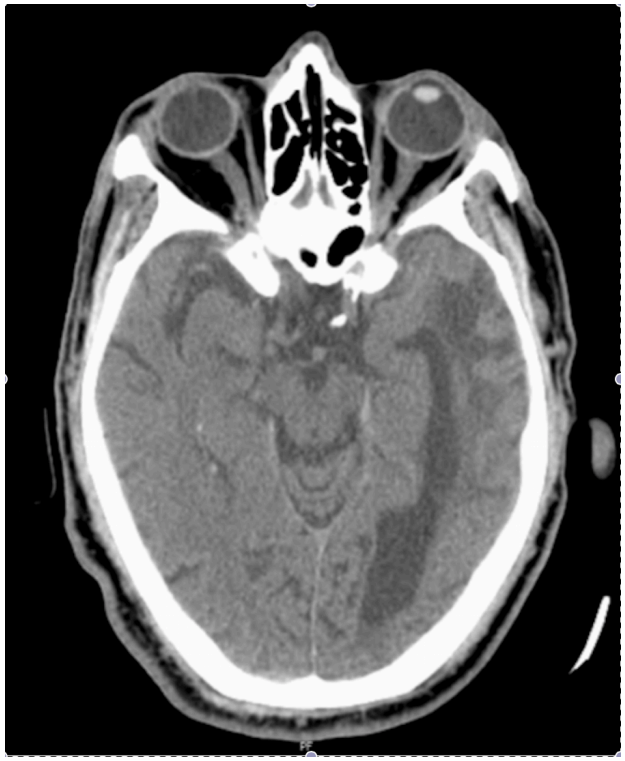
# Case 1

A 69-year-old man, with a symptomatic epilepsy due to a left hemispheric haemorrhagic stroke in 2011, was admitted to the emergency department due to disturbance of speech and involuntary movements of the right side of the body that had started three days before. The patient was being treated with oral levetiracetam at 500 mg, twice a day. On admission, he was alert, responsive and cooperative. The neurological examination revealed continuous myoclonic jerks of the right abdominal wall, without propagation to limb extremities or the face. The contractions were not painful. A mild spastic right hemiparesis was also evident.

Computed tomography imaging of the brain revealed left frontal hypodensity extending to ipsilateral temporo-occipital regions, consistent with vascular sequelae *(figure 1A)*.



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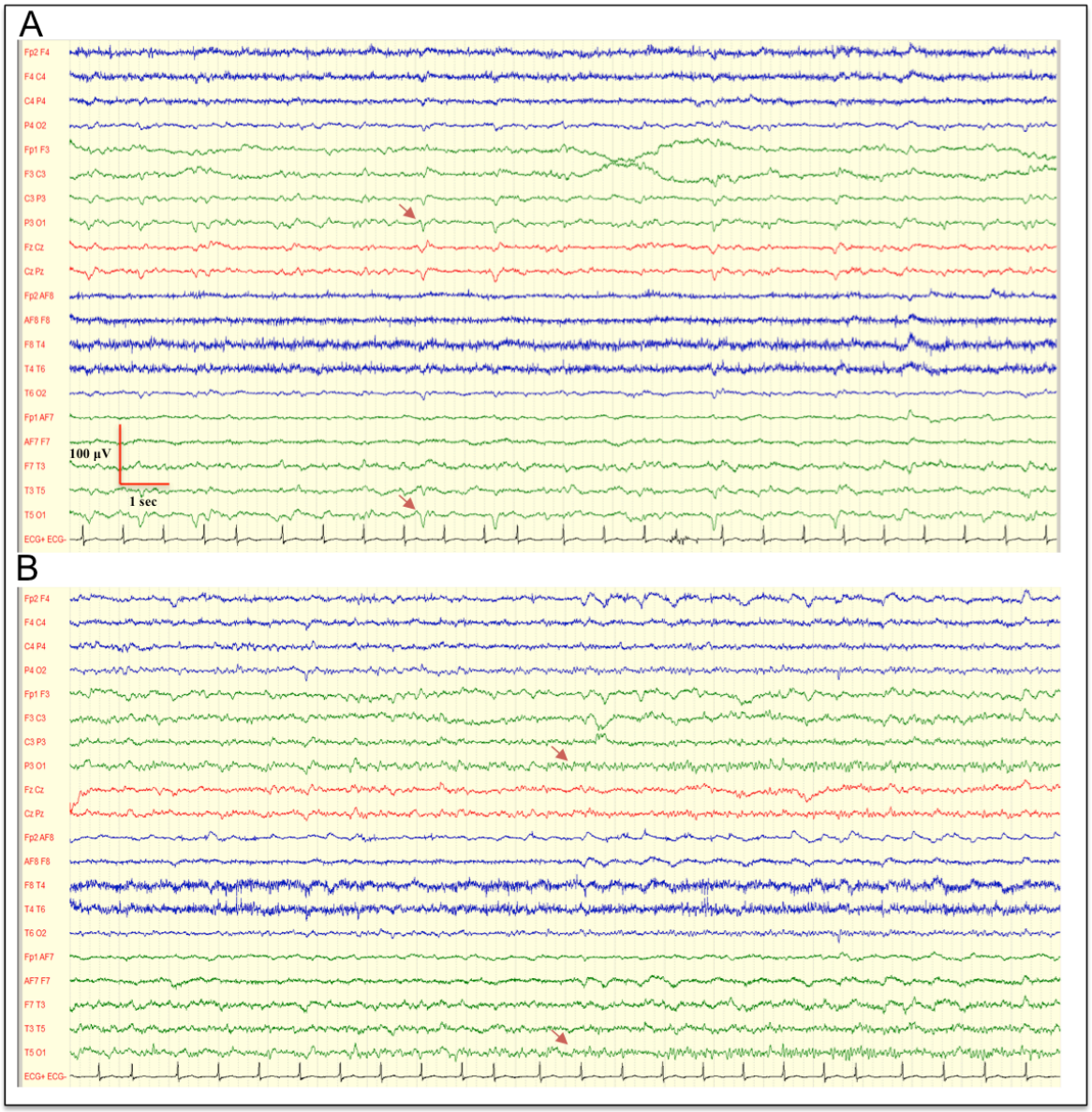
The EEG showed a disorganization of the background brain activity with no posterior alpha activity and left periodic lateralized epileptiform discharges (PLEDs) (with intervals of approximately one second). These were maximum on the occipital region *(figure 2A)* and evolved into rhythmic fast activity in the same location (occurring three times during a 30-minute recording, each with a duration of 20 to 60 seconds) *(figure 2B).*

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Clinically, this fast EEG activity was associated with an increase in the frequency of the right continuous abdominal jerking (*video sequence 1)*. The PLEDs were not synchronous with the myoclonic jerks.

After antiepileptic therapy with intravenous levetiracetam, the discharges resolved completely and no further seizures were perceived.

An EEG performed five days after seizure offset showed a mild slowing of the background cerebral activity and a more focal dysfunction on the left posterior region, associated with sporadic left temporo-parieto-occipital spikes.

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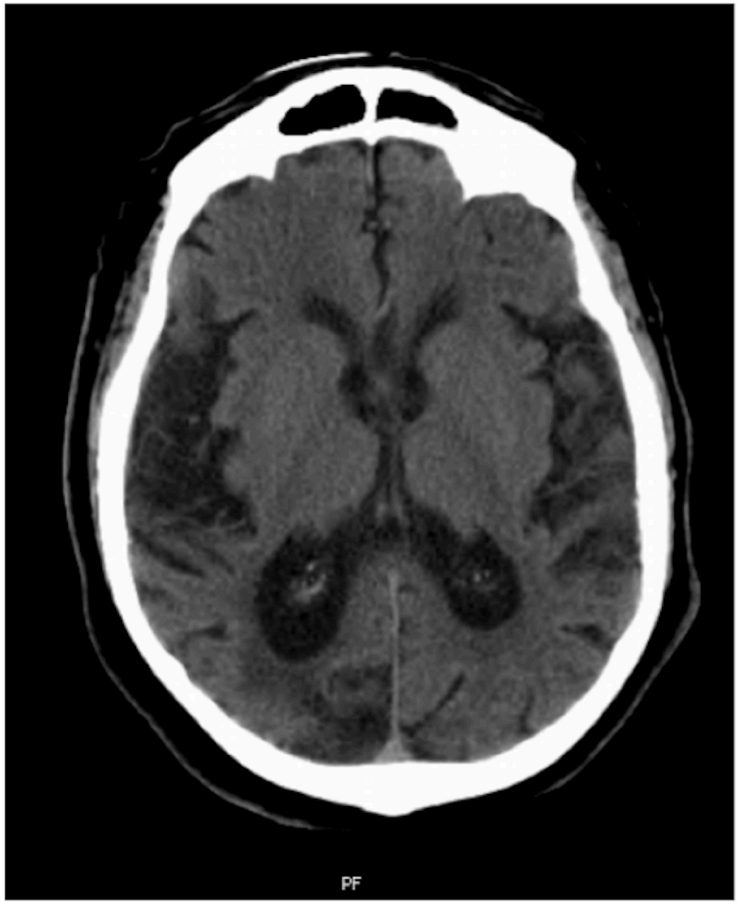
# Case 2

A 75-year-old man, with a history of right hemispheric ischaemic stroke in 2009 and subsequent seizures in the acute phase, but without a diagnosis of epilepsy or antiepileptic treatment, was admitted to the emergency department with continuous left hemibody myoclonic movements. The patient was alert but unresponsive, had a spastic left hemiparesis, and a tonic ocular deviation to the left side. Intravenous treatment with phenytoin was initiated and the seizures were resolved.

Cranioencephalic computed tomography showed right occipital cortico-subcortical hypodensity, which was consistent with vascular sequelae *(figure 1B)*.

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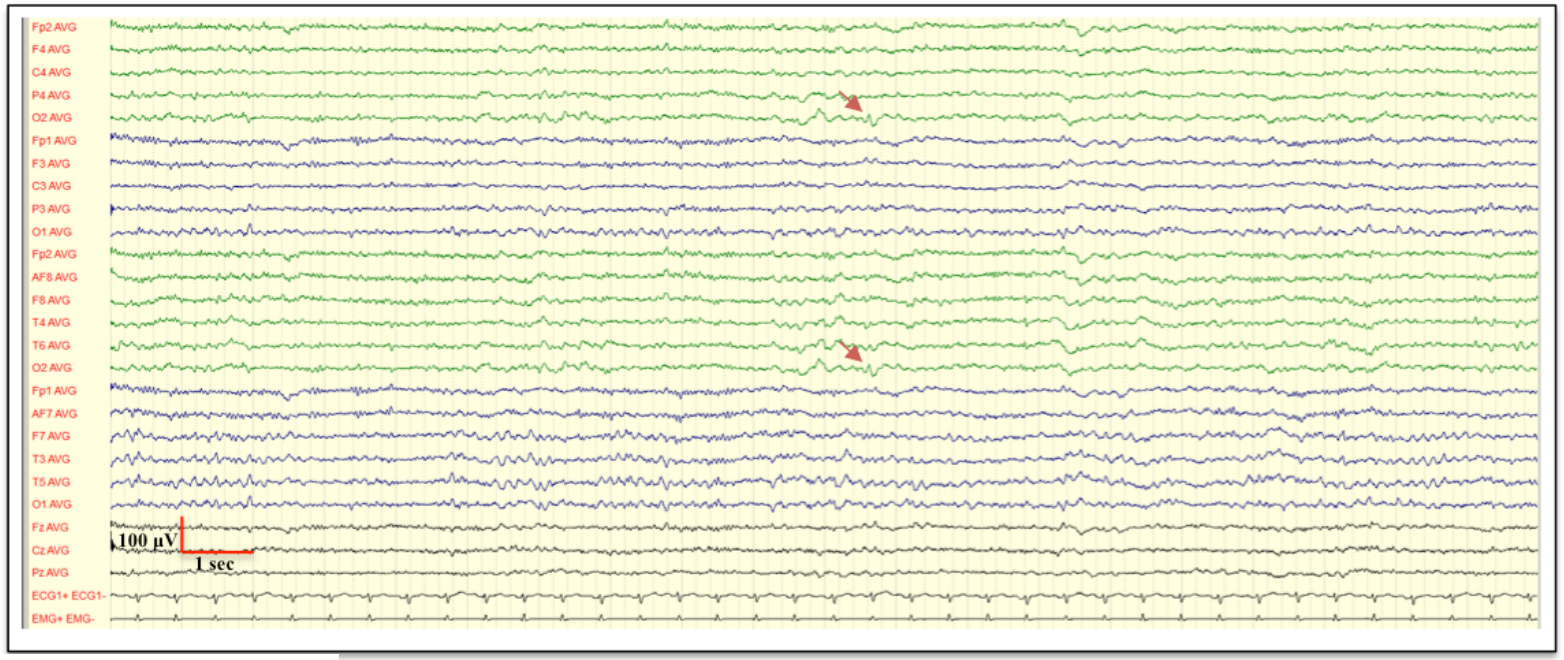
Two days after his admittance to the hospital, he developed continuous left arrhythmic myoclonic twitches of the abdominal muscles with no associated movements of the limbs, head or neck. Consciousness was preserved. The abdominal contractions responded to intravenous levetiracetam.

An EEG was performed to determine if there were any EEG correlates. Myoclonic jerks occurred during EEG monitoring (*video sequence 2)* but were unaccompanied by a clear ictal pattern. However, a slight asymmetry of the posterior background activity, with slower activity on the right side and some spikes over the right occipital area, was observed *(figure 3)*.

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# Discussion

Several anatomical localizations have been presented as epileptogenic zones in cases of EPC involving abdominal musculature, such as the parietal lobe (Matsuo, 1984; Tezer *et al*., 2008), frontal lobe and parasagittal areas (Rosenbaum and Rowan, 1990; Chalk *et al*., 1991; Fernández-Torre *et al*., 2004).

None of these were restricted to the somatotopic representation of abdominal musculature. The notion of multiple localizations associated with a common clinical manifestation of abdominal contractions may be justified by the complex organization of the homunculus and some individual variability (Tezer *et al*., 2008) .

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The second case exhibited a different cortical localization with an epileptic focus in the occipital lobe. The specific neuronal mechanism by which an occipital vascular lesion may have caused activation of the abdominal area in motor cortex is unknown.

It is well recognized that seizures originating in the occipital lobe can spread, anteriorly generating symptoms from temporal, parietal and frontal lobes (Panayiotopoulos, 2010). Thus, supracalcarine foci tend to propagate to the parietal and frontal regions, giving rise to predominantly motor seizures (Panayiotopoulos, 2010).

Following a brain lesion, changes in other regions are also documented to occur at different post-lesion times, with altered post-stroke activation patterns (Johansson, 2000). A long period between the vascular event and seizure onset favours the plasticity of the cerebral cortex as the underlying phenomenon rather than an alternative propagation pathway and a functional reorganization of the adjacent cortical tissue (Johansson, 2000).

Involvement of the abdominal musculature as a clinical manifestation of a focal motor seizure is rare
(Fernández-Torre *et al*., 2004).

The threshold of the truncal area is postulated to be high and therefore may not be seen during seizure activity
(Oster *et al*., 2011).

The trunk has a small topographic representation on the motor cortex (Rosenbaum and Rowan, 1990). This small cortical representation may explain the absence of a clear ictal pattern recorded during routine scalp EEG, concomitant with ictal semiology in our second case.

An antiepileptic drug was effective in controlling the abdominal myoclonus, enhancing the epileptic origin of the involuntary movements presented by the patient.

# Conclusion

Cerebrovascular disease may cause a rare clinical manifestation of EPC with abdominal seizures.

Various anatomical locations have been associated with EPC of the abdominal wall. However, to the best of our knowledge, an occipital lobe lesion has not been previously reported.

The neuroimaging and EEG studies were of major importance in understanding the relationship between these two entities.

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