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

Joana Jesus Ribeiro, Mário Sousa, Rute Teotónio, Conceição Bento, Francisco Sales

Department of Neurology, Coimbra University Hospital Center, Coimbra, Portugal

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**ABSTRACT** – Two elderly men, with previous history of cerebrovascular disease, were admitted to the emergency department due to focal motor status epilepticus with persistent myoclonic jerks of one side of the body. In both cases, the clinical picture evolved into a unilateral and isolated arrhythmic myoclonus of the abdominal muscles with preserved consciousness. These involuntary movements resolved with antiepileptic drugs. Although cerebrovascular disease is one of the most common causes of epilepsia partialis continua, reported cases in the literature with predominant abdominal involvement have a different aetiology. The neuroimaging and electroencephalographic findings showed a wide spectrum of different localizations and aetiologies associated with this particular type of epileptic seizure. Indeed, the pathophysiology of focal motor seizures involving the abdominal muscles is still a matter of discussion. In our second case, we present a patient with epilepsia partialis continua of the abdominal wall with an occipital focus, which, to the best of our knowledge, has not been previously reported. [*Published with video sequences*]

**Key words:** abdominal myoclonus, epilepsia partialis continua, stroke

**Correspondence:**
Joana Jesus Ribeiro
Praceta Professor Mota Pinto,
3000-075, Coimbra,
Portugal
<joanajribeiro@gmail.com>

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).

This report describes the clinical, electrophysiological and neuroimaging features of two elderly men with continuous abdominal myoclonus in a context of cerebrovascular lesions.

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## Case studies

### 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)*. The electroencephalogram (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)*. 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.

### 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)*. 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. 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)*.

The abdominal contractions responded to intravenous levetiracetam.

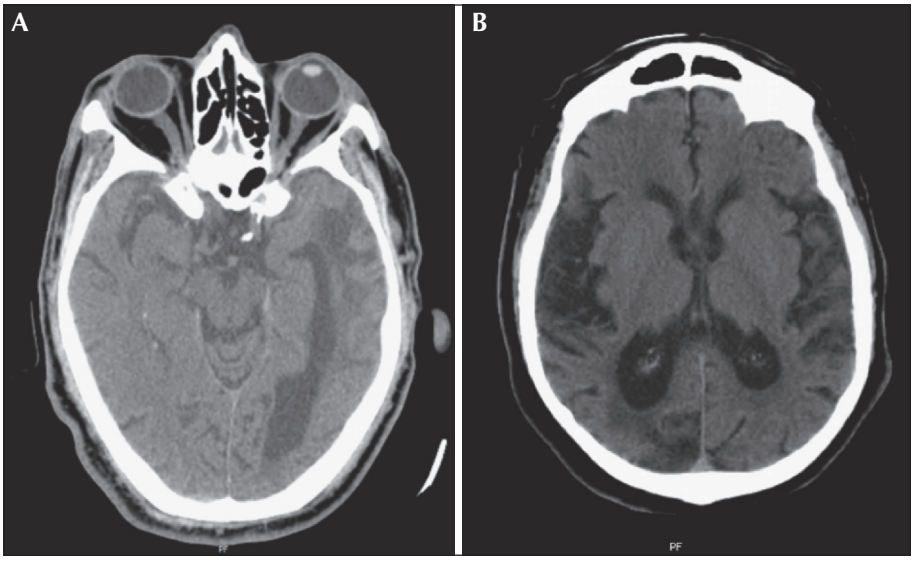


**Figure 1.** Computed tomography imaging. (A) Case 1: left frontal hypodensity extending to ipsilateral temporo-occipital regions, consistent with vascular sequelae. (B) Case 2: right occipital cortico-subcortical hypodensity, consistent with vascular sequelae.

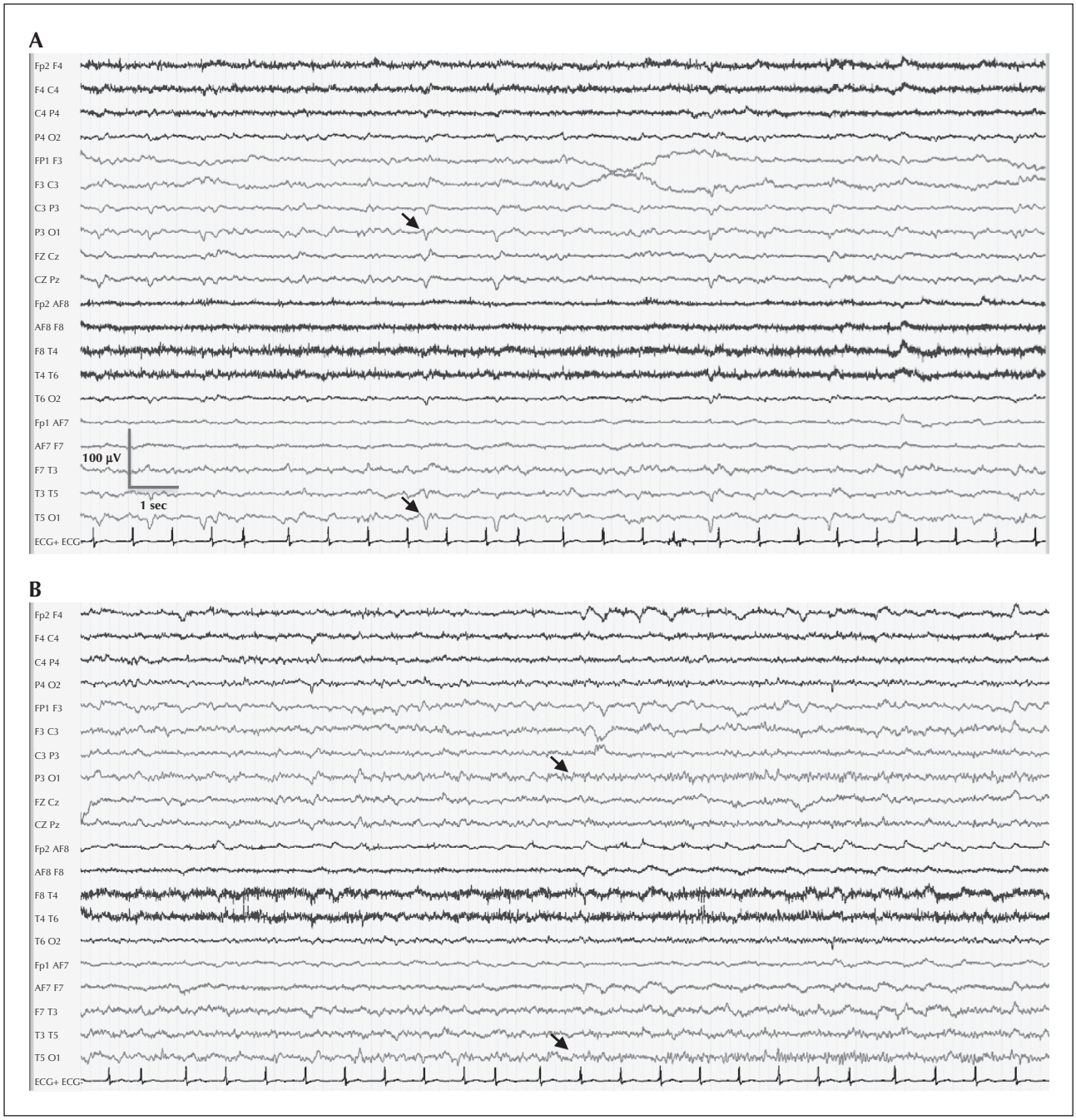


**Figure 2.** Case 1: EEG recordings were performed with cap electrodes applied according to the international 10/20 system (bipolar montage, high-pass filter at 1.0 Hz, low-pass filter at 70 Hz, gain at 40 µV/cm).
(A) PLEDs, maximum on the left occipital region. (B) Rhythmic fast activity in the posterior region.

## Discussion

We present two elderly men with an abdominal form of EPC due to cerebrovascular lesions. According to Dafotakis *et al*., one of the most common aetiologies of EPC in adults is cerebrovascular lesions (Dafotakis *et al*., 2006). However, when the predominant area involved is the abdominal wall, the reported aetiologies are of a different nature and include: brain tumours, focal cortical dysplasia, central nervous system infections, and subdural haematoma as the most frequent (Johnson, 1969; Matsuo, 1984; Chalk *et al*., 1991; Tezer *et al*., 2008). In our cases, the abdominal motor seizures were preceded by hemibody

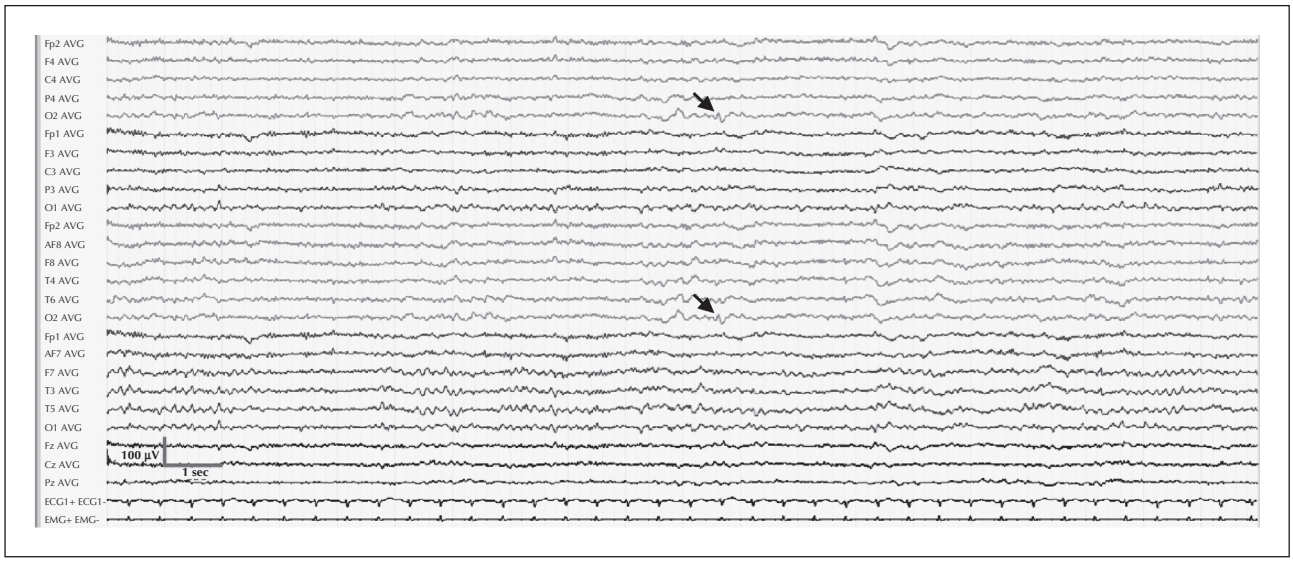


**Figure 3.** Case 2: EEG recordings were performed with cap electrodes applied according to the international 10/20 system (average referential, high-pass filter at 1.0 Hz, low-pass filter at 70 Hz, gain at 70 µV/cm). Some spikes were recorded over the right occipital area.

myoclonic movements on the contralateral side of the previous vascular event, and no other cerebral lesion was present on brain imaging. In both cases, no other precipitating factors could be found.

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). Our first case report corroborates the previous documented possible areas involved. The second case exhibited a different cortical localization with an epileptic focus in the occipital lobe that, to the best of our knowledge, has not been previously documented in EPC of the abdomen. The specific neuronal mechanism by which an occipital vascular lesion may have caused activation of the abdominal area in motor cortex is unknown. Nevertheless, 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). According to Oster *et al*., the threshold of the truncal area is postulated to be high and therefore may not be seen during seizure activity (Oster *et al*., 2011). In addition, 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. Furthermore, an antiepileptic drug was effective in controlling the abdominal myoclonus, enhancing the epileptic origin of the involuntary movements presented by the patient.

In 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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The authors have no conflicts of interests to disclose.

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## Legends for video sequences

### Video sequence 1

Continuous myoclonic jerks of the right abdominal musculature.

### Video sequence 2

Left arrhythmic myoclonic twitches of the abdominal muscles. In both cases, the abdominal movements were videotaped during EEG recordings in order to present the semiology of the episodes.

**Key words for video research on www.epilepticdisorders.com**
*Phenomenology*: myoclonic seizure
*Localisation*: frontal lobe (left); posterior cortex occipital
*Epilepsy syndrome*: epilepsia partialis continua
*Aetiology:* stroke

## TEST YOURSELF

(1) Define epilepsia partialis continua.

(2) Which are the typically affected areas in the epilepsia partialis continua?

(3) List the common causes of epilepsia partialis continua with abdominal involvement in adults.

(4) Explain the mechanism of how an occipital vascular lesion may cause activation of the abdominal area in motor cortex.

(5) Elucidate the reason for the absence of a clear ictal pattern recorded during routine scalp EEG in focal motor seizure of the abdominal muscles.