Epileptic Disord 2019; 21 (3): 302-6

Abdominal epilepsia partialis continua in neurocysticercosis

Ajay Asranna [1], Sachin Sureshbabu [1], Gaurav Mittal [1], Sudhir Peter [2], Shobhana Chindripu [3], Leena Saini [1]

[1] Department of Neurology, St Stephen's Hospital, New Delhi
[2] Medall Heathcare, Department of Pathology, Ongole, Andhra Pradesh
[3] Department of Pathology Guntur Medical College, Andhra Pradesh, India

Received October 03, 2018; Accepted May 12, 2019

ABSTRACT – Epilepsia partialis continua (EPC) of abdominal muscles is a rare entity with variable clinical localization and aetiology. A 25-year-old man presented with sudden onset of intermittent focal myoclonic movements involving the abdominal muscles on the right side exclusively, lasting from 20 minutes to an hour. Brain MRI revealed a ring-enhancing lesion, suggestive of cysticercal granuloma over the left precentral gyrus. The patient fulfilled the revised diagnostic criteria for definitive diagnosis of neurocysticercosis. EEG did not show focal abnormalities during the events. Episodes of EPC were controlled with difficulty using 600 mg oxcarbazepine, 200 mg lacosamide, and 2,000 mg levetiracetam. The patient received antiparasitic therapy with albendazole (15 mg/kg for two weeks) and oral dexamethasone (0.1 mg/kg) for two weeks which was then tapered. The involvement of the primary motor cortex during ictal propagation may account for this curious phenomenon. This is the first report of abdominal EPC in a patient with inflammatory granuloma as a result of neurocysticercosis.

Key words: epilepsia partialis continua, inflammatory granuloma, epilepsy, neurocysticercosis

Case study

Epilepsia partialis continua (EPC) is characterised by continuous involuntary jerky movements restricted to a part of the body with preserved awareness. EPC of abdominal muscles is a rare entity with variable clinical localization and aetiology. A 25-year-old man presented with sudden onset of arrhythmic focal myoclonic movements exclusively involving the abdominal muscles on the right side, lasting between 20 minutes and an hour. He had no prior history of seizures and had a normal birth and developmental history. He had no known medical comorbidities or addictions. The neurological examination was normal except for focal myoclonus over the abdomen seen continuously with no involvement of the face, arms or lower limbs (*video 1*). Contrast-enhanced brain MRI revealed a ring-enhancing lesion, suggestive of cysticercal granuloma over the left precentral gyrus (*figure 1A, B, C*). EEG did not show focal abnormalities, either during the events or in the interictal period (*figure 2*). Serological testing for neurocysticercosis was offered to the patient but declined due to

VIDEO ONLINE

Correspondence:
Sachin Sureshbabu
St Stephen's Hospital,
New Delhi 110054, India
<drsachins1@rediffmail.com>
<sachinsuresh1977@gmail.com>

doi:10.1684/epd.2019.1069

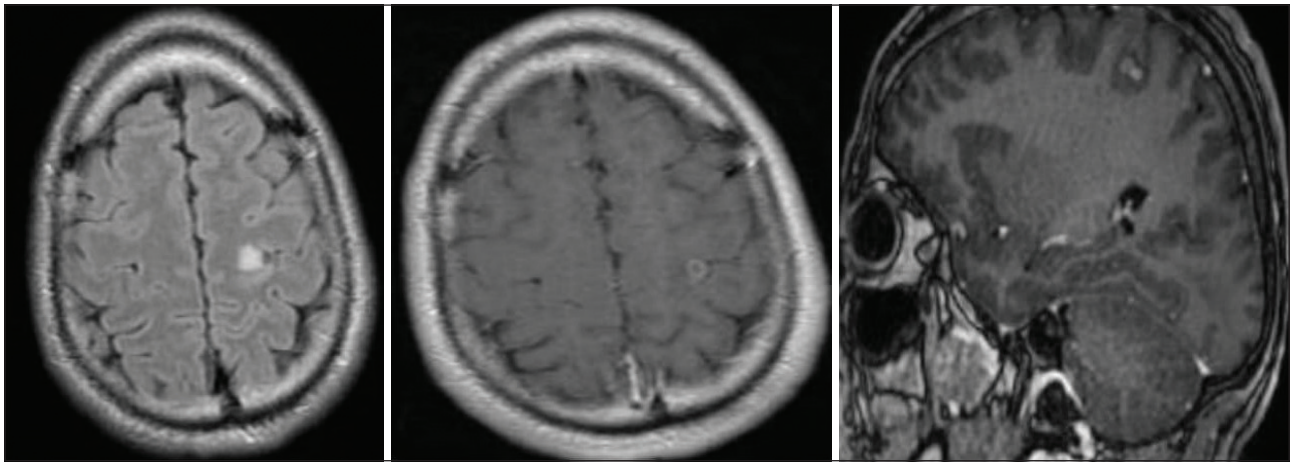

Figure 1. Brain MRI showing inflammatory granuloma in the left frontal region. (A) Flair images showing hyperintense focus over the left precentral gyrus. (B) T2 post contrast showing inflammatory granuloma over the left precentral region. (C) Sagittal images showing ring-enhancing lesion over the left frontal region.

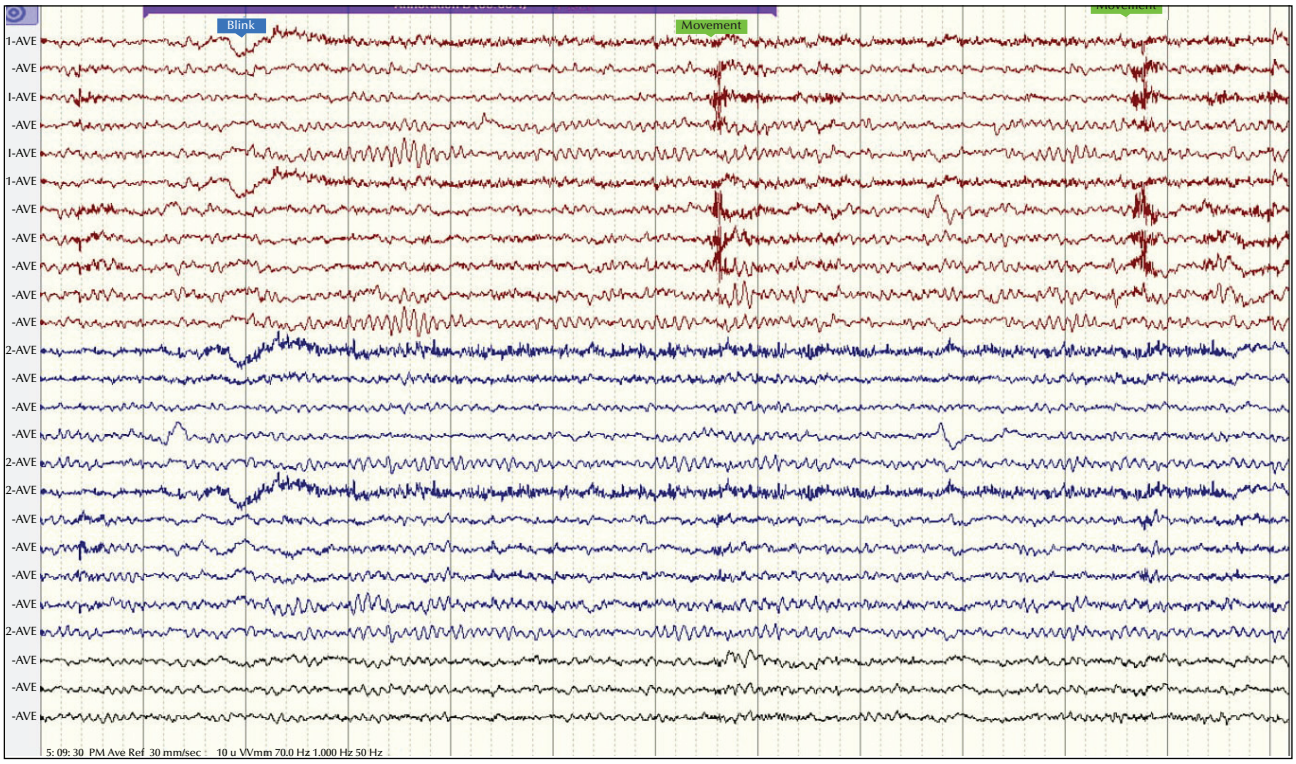

Figure 2. Ictal EEG recording showing EMG artefacts and no epileptiform discharges.

financial constraints. The patient fulfilled the revised diagnostic criteria for definitive diagnosis of neurocysticercosis (Del Brutto *et al*., 2017). He was initially started on oxcarbazepine and subsequently AEDs were up titrated to achieve seizure control. Episodes of EPC were controlled with difficulty using 600 mg oxcarbazepine, 200 mg lacosamide, and 2,000 mg levetiracetam. He received antiparasitic therapy with albendazole (15 mg/kg for two weeks) and oral dexamethasone (0.1 mg/kg) for two weeks which was then tapered. Follow-up EEG was performed at one month and three months which showed slowing over the left frontal region. The patient continues to have rare short-lasting episodes of abdominal EPC and focal motor seizures at a frequency of 1-2 episodes per month.

Table 1. Electroclinical and aetiological characteristics of patients with abdominal EPC.

	Unilateral/ bilateral	Distribution	Neuroimaging localisation	Aetiology	EEG changes
Johnson *et al*. (1969)	Unilateral	Left abdomen	Right parietal	Subdural haematoma	NA
Matsuo (1984)	1.Unilateral	Right lower abdomen +thigh	Left parietal	Aspergilloma	No IEDs
	2. Unilateral	Left lower abdomen	Right parietal	Meningioma	No IEDs
	3. Unilateral	Right abdomen + shoulder and face	Normal	Unknown	No IEDs, parietal slowing
Rosenbaum and Rowan (1990)	Unilateral	Left abdomen + toe	Right fronto-parietal parasagittal	Squamous cell carcinoma	Right centro-parietal continuous discharges
Chalk *et al*. (1991)	Unilateral	Left abdomen + proximal leg	Not available	Cryptococcal meningitis	Right parasagittal IEDs
Fernandez Torre *et al*. (2004)	Bilateral	Left abdomen> right	Multiple lesions- right frontal, temporal pole, cerebellar and left parasagittal frontal	Metastatic tumours	PLEDs involving the right central, mid-temporal, and parietal areas
Lim *et al*. (2004)	Unilateral	Generalised myoclonus evolving to left abdominal + lower limb EPC	Right cingulate	Venous angioma	Right anterior temporal IEDs
Dafotakis *et al*. (2006)	Unilateral	Left abdominal	Signal changes Right precentral gyrus	Idiopathic	Not available
Tezer *et al*. (2008)	Unilateral	Right abdomen, right facial and bilateral eye blinks	Left mesial parieto-occipital	Focal cortical dysplasia	PLEDs over the left fronto-centro-parietal areas
Ribeiro *et al*. (2015)	1.Unilateral	Right abdomen	1.Left frontal	1.Vascular	1. Occipital PLEDs
	2.Unilateral	Left abdomen	2. Right occipital	2. Vascular	2. Occipital IEDs
Present study	Unilateral	Right abdomen	Left frontal	Neuro-cysticercosis	No IEDs

IEDs: interictal discharges; PLEDs: periodic lateralised epileptiform discharges.

Discussion

EPC was first defined by Kozhenikov in 1894, but it was Thomas *et al*. (1977) who first defined EPC based on its phenomenology. According to this definition, episodes of EPC are spontaneous, regular or irregular, clonic muscular twitching, affecting a limited part of the body, sometimes aggravated by action or sensory stimuli, occurring for a minimum of one hour and recurring at intervals of no more than ten seconds. The ILAE task force report on status epilepticus considers EPC as a subclass of focal motor status although sensory manifestations are known to occur (Trinka *et al*., 2015). Our patient had events lasting ≥one hour which

fulfils the criteria for EPC in addition to shorter events which qualify as abdominal focal motor seizures.

The pathophysiology of EPC is still unclear. A single mechanism is inadequate to explain the complex manifestations including stimulus sensitivity, however, a combination of epileptogenic motor cortex coupled with abnormal excitation of cortico-subcortical loops is likely to be responsible for producing EPC (Guerrini, 2009). Episodes are commonly distributed over the face and distal upper and lower limbs (Thomas *et al*., 1977; Sinha and Satishchandra, 2007; Mameniskiene *et al*., 2011). Abdominal EPC is a rare manifestation and only 12 cases have been reported in the literature to date (*table 1*) (Matsuo, 1984; Rosenbaum and Rowan, 1990; Chalk *et al*., 1991; Biraben and Chauvel, 1997; Fernández-Torre *et al*., 2004; Lim *et al*., 2004; Dafotakis *et al*., 2006; Tezer *et al*., 2008; Ribeiro *et al*., 2015). The largest series of patients with EPC -66 patients from India (Sinha and Satishchandra, 2007), 51 paediatric patients (Kravljanac *et al*., 2013), and a European survey of 65 patients with non-stroke and non-Rasmussen's EPC (Mameniskiene *et al*., 2011),make no separate mention of abdominal myoclonus , although six patients had hemibody involvement in the series by Mameniskiene *et al*. The limited representation of abdomen over motor homunculus may account for the rarity of this phenomenon (Catani, 2017). It is not a mere coincidence that EPC is more often seen over body parts with proportionately larger cortical representation over the sensory and motor homunculus. Abdominal EPC may occur in isolation or may involve other body parts, as seen in about half of patients in the literature. The lower limbs are most commonly involved, followed by the face, upper limbs, and shoulders. The overlapping representations of body parts, particularly the abdomen and the limbs over the superior aspect of the sensory and motor homunculus, may account for this co-existence (Catani, 2017).

Abnormal MRI is noted in a majority of patients with abdominal EPC and the localisation is commonly to parietal or frontal lobes. MRI in our patient showed a granuloma in the left pre-central gyrus which correlated fairly well with the representation of the abdomen over the motor homunculus. Though this may not be true in all cases, a contralateral parasagittal or medial location is evident in most cases. These observations point to an epileptogenic network which is highly localised and closely involves the peri-Rolandic cortex and subcortex.

The aetiology of abdominal EPC is more variable compared to EPC involving other distributions, of which Rasmussen's encephalitis is a major cause. While the commonest aetiology is vascular, other causes include FCD, aspergilloma, and tumours. This is the first report of inflammatory granuloma due to neurocysticercosis causing abdominal EPC. In a previous large series of EPC from India by Sinha *et al*., granulomas accounted for 7% although there was no abdominal EPC reported in this study (Sinha and Satishchandra, 2007).

EEG was normal in our patient, which is reported in about a fifth of patients. This is maybe because the cortical activity is very well localised or because of the unfavourable angle of orientation of the dipole in relation to the recording electrodes (Mameniškienė and Wolf, 2017).

Based on its evolution, EPC lends itself to categorisation into one of the following subtypes:

– (1) EPC as a solitary event
– (2) Chronic repetitive non-progressive EPC
– (3) Chronic persistent non-progressive EPC
– (4) Chronic progressive EPC

Our patient had an evolution resembling chronic repetitive non-progressive type. The prognosis of abdominal EPC depends on underlying aetiology and patients with a vascular aetiology generally have short-lived EPC with good response to AEDs. Our patient, at six months of follow-up, still has rare intermittent episodes of EPC despite being on three AEDs, pointing towards a slightly more protracted course for inflammatory granulomas.

To conclude, abdominal EPC is a rare manifestation, usually associated with brain lesions and varied aetiology. Inflammatory granulomas are a rare cause of abdominal EPC and may not respond readily to AEDs. □

Acknowledgements and disclosures.
We would like to thank Dr. Sudhir C Joseph, Director of St Stephen's Hospital.
None of the authors have any conflict of interest to declare.

Legend for video sequence

EPC and focal motor seizures localised to the right abdomen with no involvement of the face or limbs.

Key words for video research on www.epilepticdisorders.com

Phenomenology: focal seizure
Localisation: primary motor cortex
Syndrome: epilepsia partialis continua
Aetiology: neurocysticercosis

References

Biraben A, Chauvel P. Epilepsia partialis continua. *Epilepsy A Compr Textb* 1997: 2447-54.

Catani M. A little man of some importance. *Brain* 2017; 140: 3055-61.

Chalk CH, McManis PG, Cascino GD. Cryptococcal meningitis manifesting as epilepsia partialis continua of the abdomen. *Mayo Clin Proc* 1991; 66: 926-9.

Dafotakis M, Sparing R, Becker S, Fink GR. Epilepsia partialis continua of the abdominal muscles with transient MRI abnormalities. *Neurology* 2006; 66: 1099.

Del Brutto OH, Nash TE, White AC, *et al*. Revised diagnostic criteria for neurocysticercosis. *J Neurol Sci* 2017; 372: 202-10.

Fernández-Torre JL, Calleja J, Pascual J, Galdós P, De Pablos C, Berciano J. Epilepsia partialis continua of the abdominal muscles: a detailed electrophysiological study of a case. *Mov Disord* 2004; 19: 1375-8.

Guerrini R. Physiology of epilepsia partialis continua and subcortical mechanisms of status epilepticus. *Epilepsia* 2009; 50: 7-9.

Johnson DA. Epilepsia partialis continua. *JAMA* 1969; 210: 1756.

Kravljanac R, Djuric M, Jovic N, Djordjevic M, Zamurovic D, Pekmezovic T. Etiology, clinical features and outcome of epilepsia partialis continua in cohort of 51 children. *Epilepsy Res* 2013; 104: 112-7.

Lim ECH, Tan JJH, Ong BKC, Wilder-Smith EPV. Generalised myoclonus evolving into epilepsia partialis continua due to a cingulate gyrus lesion: case report and review of the literature. *Park Relat Disord* 2004; 10: 447-9.

Mameniškienė R, Wolf P. Epilepsia partialis continua: a review. *Seizure* 2017; 44: 74-80.

Mameniskiene R, Bast T, Bentes C, *et al*. Clinical course and variability of non-Rasmussen, nonstroke motor and sensory epilepsia partialis continua: a European survey and analysis of 65 cases. *Epilepsia* 2011; 52: 1168-76.

Matsuo F. Partial epileptic seizures beginning in the truncal muscles. *Acta Neurol Scand* 1984; 69: 264-9.

Ribeiro JJ, Sousa M, Teotonio R, Bento C, Sales F. Epilepsia partialis continua of the abdominal muscles due to cerebrovascular disease. *Epileptic Disord* 2015; 17: 72-6, quiz: 76.

Rosenbaum DH, Rowan AJ. Unilateral truncal seizures: frontal origin. *Epilepsia* 1990; 31: 37-40.

Sinha S, Satishchandra P. Epilepsia partialis continua over last 14 years: experience from a tertiary care center from south India. *Epilepsy Res* 2007; 74: 55-9.

Tezer FI, Celebi O, Ozgen B, Saygi S. A patient with two episodes of epilepsia partialis continua of the abdominal muscles caused by cortical dysplasia. *Epileptic Disord* 2008; 10: 306-11.

Thomas JE, Reagan TJ, Klass DW. Epilepsia partialis continua. A review of 32 cases. *Arch Neurol* 1977; 34: 266-75.

Trinka E, Cock H, Hesdorffer D, *et al*. A definition and classification of status epilepticus - Report of the ILAE Task Force on Classification of Status Epilepticus. *Epilepsia* 2015; 56: 1515-23.

TEST YOURSELF

(1) What is the anatomical localisation of abdominal EPC?

(2) What are the aetiological causes of abdominal EPC?

(3) What is the natural course and prognosis of patients with Abdominal EPCs?

(4) What percentage of patients with EPC have normal ictal EEG?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".