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Case Report

A Case of Abdominal Epilepsia Partialis Continua Occurring One Year after Ischemic Stroke

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Abstract

Epilepsia partialis continua is characterized by continuous clonic contractions of a certain area of the body. One of the most common causes of Epilepsia partialis continua in adults is cerebrovascular events. Other causes include meningoencephalitis, Rasmussen encephalitis, diabetic nonketotic hyperosmolar coma, central nervous system malignancies, tuberculosis, cerebral venous thrombosis, or idiopathic. A 70-year-old male patient was admitted to the emergency department with abdominal muscle contractions for about an hour. Neurodiagnostic imaging revealed an encephalomalasia area secondary to the area of the previous infarction in the left frontoparietal region. Focal motor findings were controlled within 5 min after the VPA (valproic acid) treatment at a dose of 15 mg/kg admission, and then the treatment was continued with 1500 mg/day Valproic acid. Here, we aimed to emphasize that myoclonic jerks confined to the abdominal region is a rare motor phenomenon and may be a feature of Epilepsia partialis continua, the history of stroke should be questioned in the etiology, and seizures can be controlled with IV Valproic acid treatment.
Keywords: Epilepsia partialis continua, partial epilepsy, focal motor, Valproic acid

INTRODUCTION

Epilepsia partialis continua (EPC) is a rare form of focal motor status epilepticus, characterized by continuous clonic contractions of a certain area of the body. The contractions can be regular or completely irregular and sometimes increase with movement or sensory stimulation.[1-4] In addition to the occurrence mostly on the face or distal extremities, it can also be seen in the trunk or abdominal region in rare cases.[5] Cerebrovascular lesions are known to be one of the most common causes of EPC in adults. Tumors, vascular lesions, metastatic lesions, trauma, infections, and metabolic encephalopathies (especially hyperosmolar nonketotic hyperglycemia) are other common causes seen in adults.[1,6,7] It has been reported in the literature that frontal parasagittal or parietal region lesions may cause seizures and EPC in the trunk muscles.[5,6,8-10] There are studies reporting that Benzodiazepine, Lamotrigine, Levetiracetam, Carbamazepine, Valproic acid, and Topiramate are beneficial in its treatment.[11] Here, we present a case of EPC localized in the abdominal region, in association with stroke history.

CASE PRESENTATION

A 70-year-old male patient was admitted to the emergency department with abdominal muscle contractions. He had been having involuntary contractions for about an hour. It was ascertained that the patient had a history of diabetes mellitus, essential hypertension, coronary artery bypass operation, and ischemic stroke a year ago. In his neurological examination performed under the influence of intravenous (IV) Diazepam given at the emergency service admission, his consciousness was sleepy, his eyes were opening awakened by tactile stimuli, and there was 2–3/5 muscle strength in the right upper and lower extremities as a sequelae of a previous stroke. Babinski sign was positive on the right side. Complete blood count (CBC), biochemistry, and arterial/venous gas values taken at the emergency service were within normal limits. Neurodiagnostic imaging (brain CT and MRI) showed chronic ischemic changes without any newly occurring pathology (Figures 1 and 2).

Cranial MRI revealed an encephalomalasic area secondary to the area of the previous infarction in the left frontoparietal region. The patient was admitted to our clinic for further examination and treatment. Intravenous Valproic acid treatment at a dose of 15 mg/kg was started in the patient whose abdominal contractions were refractory to IV diazepam. Focal motor findings were controlled within 5 min after the VPA administration, and then the treatment was continued with 1500 mg/day valproic acid. Myoclonic feature of these contractions was the main reason for giving VPA as the first choice in the treatment.

EEG examination was within normal limits without any ictal activity (Figure 3). The patient, whose treatment was regulated with VPA 1500 mg/day, was discharged and advised to follow up in the outpatient clinic.

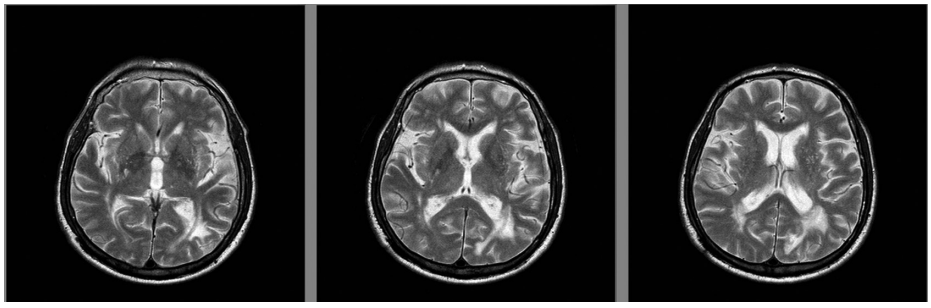

Figure 1. Chronic encephalomalasia areas secondary to previous infarction in the left frontoparietal region.

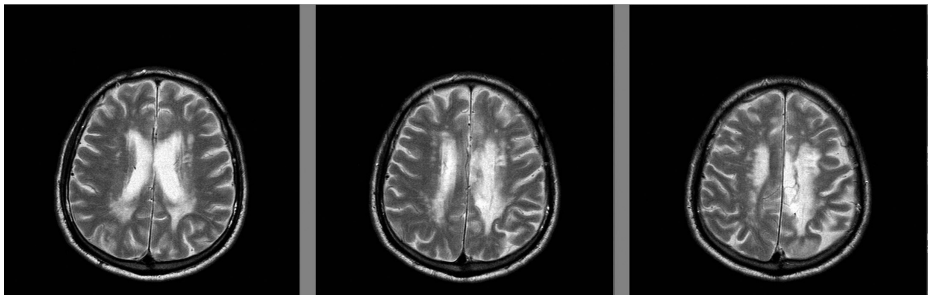

Figure 2. Chronic ischemic changes in the left cerebral hemisphere.

DISCUSSION

Epilepsia partialis continua was first described by Kozhenikov[8] in 1894 as a special form of cortical epilepsy. In 1977, Thomas et al[12] defined EPC as regular and irregular muscle twitches affecting a limited body portion, lasting at least 1 h and recurring at intervals not longer than 10 s. Obeso et al[13] defined EPC as clonic twitches of cerebral cortical origin, spontaneously regular or irregular, sometimes triggered by action or sensory stimuli, and lasting for hours, days, or weeks, limited to a part of the body. In the study of Mameniskiene et al. EPC cases were classified as types 1, 2, and 3 according to clinical course: an isolated single episode as type 1, chronic repetitive and non-progressive cases as type 2, and chronic persistent and non-progressive

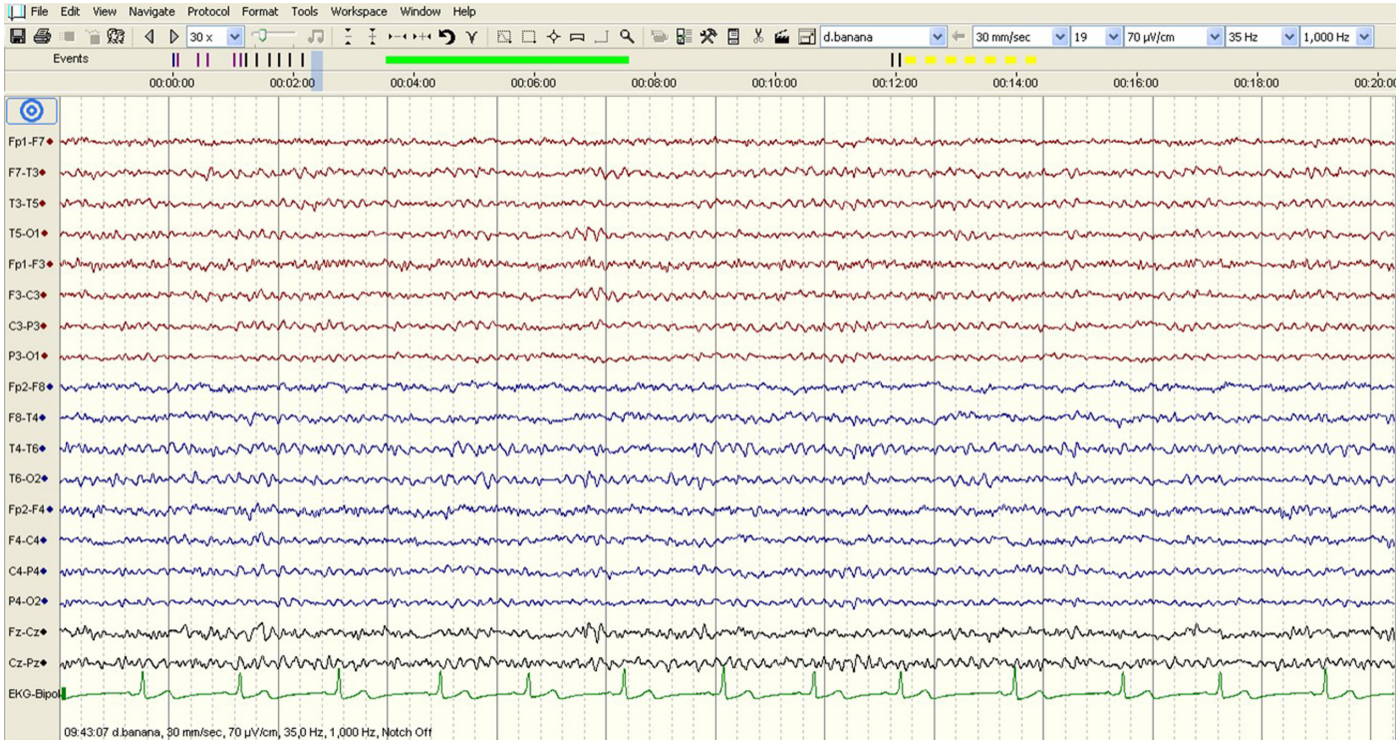

Figure 3. EEG image of the patient.

cases as type 3. It has been reported that the duration varies between 36 h and 3 months in type 1 EPC cases in this classification.[11] The prevalence of EPC is less than 1 in a million and hence the face and distal extremities are typically affected, while the trunk and diaphragm may also be affected.[5,14] The underlying etiology mainly determines the prognosis of EPC.[8]

One of the most common causes of EPC in adults is cerebrovascular events. Other causes include meningoencephalitis, Rasmussen encephalitis, diabetic nonketotic hyperosmolar coma, central nervous system malignancies, tuberculosis, and cerebral venous thrombosis, or idiopathic.[1,6,7] Following a vascular brain lesion after a stroke, activation patterns are known to increase. Why the period between the vascular event and the onset of a seizure is longer can be explained by the plasticity of the cerebral cortex.[1] EPC may rarely occur in association with focal cortical dysplasia.[2] One of the major causes seen in children is Rasmussen encephalitis.[15] EPC was defined by Chrevie et al[4] in two cases diagnosed with MELAS (mitochondrial encephalomyopathy, lactic acidosis and stroke-like episodes).

Abdominal muscle involvement is quite a rare entity in focal motor seizures. EPC cases with only abdominal muscle involvement without distal extremity and facial involvement have been rarely reported.[5,8-10] The cortical projection areas of the abdominal muscles are considerably smaller than the distal extremity and facial muscles. Many anatomical localizations have been described in EPC cases with abdominal muscle involvement. Among these localizations, frontal parasagittal or parietal lesions are the best-documented ones.[5,6,8-10] Three cases with truncal onset partial seizure and parietal lobe origin were described electrographically and neuroradiologically by Matsuo.[9] Subsequently, Rosenbaum et al also reported a case of EPC with abdominal muscle involvement with frontal and parasagittal ictal epileptiform activity in ictal EEGs.[10] None of these anatomical regions are involved in the somatotopic organization that fits the abdominal muscles. This difference between anatomical localizations is thought to be due to the complex organization of the homonculus.[1,2,5] It has been reported that the bilateral involvement of the abdominal muscles in general is due to the reasons such as bilateral corticospinal connection pathways, fast-spreading to the contralateral cortex, difficulty in distinguishing the abdominal muscles from each other anatomically, and unilateral twitches leading to diffused and chaotic movement of the abdominal wall.[5,9]

Spinal or segmental myoclonus must be considered in the differential diagnosis. However, while unilateral abdominal contractions are observed in segmental myoclonus, the contractions we see in EPC are bilateral. In addition, the presence of extremity and face involvement, irregular rhythm, increased severity with movement and arousal, and absence of spinal involvement should suggest cortical myoclonus.[5] Based on clinical, electrophysiological, and radiological findings, EPC due to previous ischemic stroke was considered in our case.

No specific EEG finding regarding EPC has been defined in the literature.[7,8] Interictal and ictal EEGs can be lateralizing and localizing and may be associated with lateralized periodic discharges (LPD). In some cases, EEG features are accused of being false lateralizing.[8] In our patient, the EEG taken in the interictal period was within normal limits.

Topiramate, oxcarbazepine, valproic acid, levetiracetam, carbamazepine, pregabalin, and steroids have been shown to be effective in the treatment of EPC according to cases in the literature. In our patient, seizure control is maintained by IV VPA and then continued with oral VPA prophylaxis.

In conclusion, we aimed to emphasize that myoclonic jerks limited to the abdominal region are a rare motor phenomenon and may be a feature of EPC. It should be kept in mind that past stroke history must be questioned for the etiology, and seizures can be controlled by IV VPA administration.

Informed Consent: Informed consent was obtained from the patient.

Peer-review: External independent.

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