ABSTRACT – We report on the preliminary clinical and electrophysiological aspects of an in-patient possibly presenting epilepsia partialis continua (Koshevnikov). We discuss the different etiologies and emphasize on the possible idiopathic form in this case.

KEY WORDS: epilepsia partialis continua, electrophysiology, symptoms, idiopathic form.

Epilepsia parcial contínua (Kojevnikov): relato preliminar de um caso

RESUMO – Relatamos os aspectos clínicos e eletrofisiológicos preliminares de um paciente hospitalizado que possivelmente apresenta crises de epilepsia parcial contínua (Kojevnikov). Discutimos as diferentes etiologias dessa condição e salientamos a possível forma idiopática, nesse caso.

PALAVRAS-CHAVE: epilepsia parcial contínua, eletrofisiologia, sintomas, forma idiopática.

Epilepsia partialis continua was firstly described by Koshevnikov in 1895 as a condition characterized by a continuous focal jerking of a body part, usually localized to a distal limb, and occurring over hours, days or even years. Several causes have been implicated in the genesis of this condition. This variety of etiologies is linked to variable EEG expression of this condition from normal to abnormal one as well as to a variable and refractory therapeutic response.

Epilepsia partialis continua is a rare condition, and in this paper, we aim at describing the clinical and electrophysiological characteristics of an in-patient from the University Hospital of Fortaleza (Brazil).

CASE

ARC, 19 years old, female, mullatoe, single, born in Solonópoles, Ceará State, Brazil. The patient reports that her disease began 40 days before admission to hospital with continuous rhythmic jerking movements in the
left shoulder. She reported no pain or other symptoms. Her antecedents were irrelevant for her clinical condition. The physical examination was normal. The neurological examination revealed continuous rhythmic movements in the left shoulder, absence of atrophy or fasciculations, normal tonus, force, reflexes, coordination and sensibility. Her gait was normal.

Among the exams carried out for diagnostic and etiologic investigation, the CT scan and both waking and sleep EEGs were normal. However, the study of the somatosensory evoked potentials (SSEP) showed that the amplitude of the right somatosensory evoked potential (N20 P23) was significantly higher than the left side, and that there was no significant difference in latency of the SSEP and central conduction time (N9-N20) in both sides.

The patient was treated with Clonazepam (2 mg) twice a day, with control of the symptoms in a period of 10 months up to now. The patient has been followed up as outpatient and a complete remission of the clinical picture has been observed, so that the patient spontaneously decided to stop her medication, without recidive.

**DISCUSSION**

Epilepsia partialis continua is most often seen in children under 16 years, with no gender predominance. The discharges involved in the genesis of epilepsia partialis continua seem to arise from cortical or subcortical structures. It may also involve bilateral brain hemispheres with stable neurological deficits. Clinically, it is characterized by persistent rhythmic clonic movements of one muscle group, usually of the face, arm or leg, with duration of hours, days, weeks or months, without spreading to other parts of the body. In our patient, these rhythmical movements were limited to the proximal (mostly) and distal parts of the left arm, which differs from the literature, which shows a predominance of the distal part of one extremity.

Epilepsia partialis continua is etiologically related in children to Rasmussen encephalitis, Alpers Huttenlocher disease, Leigh syndrome and partial deficit of cytochrome c oxidase, and neuronal migration anomalies. In adults, it is related to vascular disease, tumor and inflammatory lesions involving the sensorimotor cortex, non-ketotic hyperglycemia, HIV and liver insufficiency, and in some cases, it is of idiopathic origin.

Possibly, our patient could be included among the idiopathic group. However, the exams which were carried out in our patient are not yet sufficient to definitely show or exclude an etiology, so that further follow-up analysis, and the use of magnetic resonance imaging, for its important contribution in the diagnosis of the epilepsies, will be certainly of help to define its cause.

The EEG may be normal or abnormal. In our case, both waking and sleep EEGs were normal. However, SSEP were abnormal and consistent, with a decrease in local cortico-cortical inhibition and expression of hyperexcitability of motor cortex. Moreover, the remission of myoclonies in a few months gives additional support to the hypothesis of an idiopathic form. However, further analyses of this case will possibly define this etiological question.

**REFERENCES**

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