

Epilepsia partialis continua possibly caused by cerebellar lesion

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The cerebellum is known to have inhibitory effects on seizures. Nevertheless, cerebellar dysplastic lesions can be epileptogenic. We report a patient presenting with epilepsia partialis continua (EPC) following a hemorrhagic cerebellar lesion. Possible pathophysiological mechanisms are discussed.

Keywords:

Epilepsia partialis continua - cerebellum - diaschisis

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Introduction

The question of whether seizures can originate in the cerebellum remains controversial. While several reports have been published, supported by intracranial ictal and interictal EEG recording and PET/SPECT findings and suggesting that cerebellar pathology may produce focal seizures, no case of epilepsia partialis continua (EPC) caused by a cerebellar lesion has been reported in the English literature.¹ We describe a patient who developed EPC following a cerebellar hemorrhage.

Case Report

A 58-year-old woman was admitted to the Department of Neurology because of involuntary rhythmic movements of her right hand and lower lip. Two months prior to this admission, she suffered from right lobar cerebellar hemorrhage and was discharged from the hospital without any neurological deficit. She was suffering from diabetes mellitus treated with Metformin and from arterial hypertension treated with beta-blockers. On admission she was afebrile, alert and cooperative. The examination of the heart, lung and abdomen was unremarkable. No signs of meningeal irritation or motor or sensory deficit were found. She presented continuous rhythmic clonic jerks with a frequency of 1–2 Hz of her right lower lip and right hand. The seizures continued during sleep with reduced amplitude and were exacerbated by voluntary movements of the affected parts. The routine laboratory work-up was normal. Repeated EEG recording disclosed intermittent epileptiform discharges

over left frontotemporal region (Figure 1). Simultaneous EMG from Right Abductor pollicis brevis showed rhythmic 1 Hz/sec myoclonic discharge. On back-averaging, the correlation between EEG spikes and EMG discharge was non-consistent. MRI of the brain showed a large hypointense lesion in right cerebellar hemisphere suggestive of post-hemorrhagic gliosis (Figure 2). Ictal PET with (¹⁸F) fluorodeoxyglucose (¹⁸F-FDG) revealed focal hypometabolism in the lesion of right cerebellar lobe. The diagnosis of epilepsia partialis continua was made. She was treated with Carbamazepine 1200 mg/day for one month, followed by Topiramate 400 mg/day for two months and afterward Lamotrigine 400 mg/day during two months. On follow-up, no significant improvement was seen.

Discussion

The main differential diagnosis in our patient included EPC, oculomasticatory/limb myorhythmia and resting/postural tremor. While clinical picture is very similar in all diseases, persistence of the movement during sleep, EEG abnormalities and hypometabolic changes on PET study is suggestive of an epileptic phenomenon.

EPC is defined clinically as a syndrome of continuous focal jerking of a body part, usually a distal part of a limb, occurring over hours, days or years.² The cause of EPC is various and includes inflammatory lesions, Rasmussen's syndrome, vascular diseases, multisystem diseases, tumors, traumas, perinatal injury, hyperglycemia, Creutzfeldt-Jakob disease,² multiple sclerosis³ and metabolic disorders.⁴

EEG in EPC is various from normal record and unspecific slowing (Bancaud's type I) to intermittent epileptic discharge and focal electrographic status epilepticus (Bancaud's type II).⁵ The correlation between EEG changes and EMG discharge may be non-consistent,⁶ as well as SPECT/PET findings.⁷

The anatomical and physiological origin of EPC is controversial. It has been argued that EPC is a form of

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Figure 1

The EEG recording shows focal epileptic discharge over left frontotemporal region with simultaneous EMG recording from Right Abductor pollicis brevis.

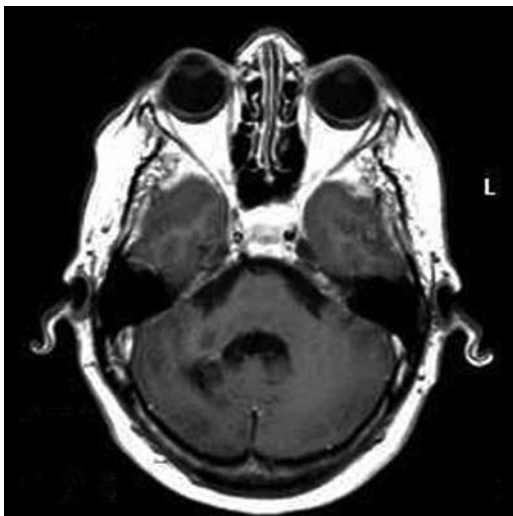


Figure 2

Flair weighted image discloses large gliotic lesion in right cerebellar hemisphere, corresponding to the previous hemorrhage.

focal cortical myoclonus, but subcortical structures have also been involved.² No reports relating EPC and cerebellar lesions were published. However several reports have described focal seizures, originating from cerebellar tumors.^{1,8} Mesiwala et al.¹ reported a child suffering from subcortical focal epilepsy with secondary generalization, refractory to antiepileptic medications in whom scalp EEG disclosed bilateral electrical anomalies. Ictal and interictal SPECT revealed a focal perfusion abnormality in the cerebellum and intraoperative EEG supported the cerebellar origin of seizure discharges that spread to the cortical surface. Ganglioglioma of the cerebellum was excised together with the cerebellar hemisphere and the patient became free of seizures. These reports suggest that at least in cases of structural lesions, the epileptic activity may indeed begin within the cerebellum.

There are extensive cerebrocerebellar connections in the human brain. Projections from the cerebellum extend to the cerebrum through relay centers in the thalamus. Thus it is possible that epileptic activity arising in the cerebellum could spread through the cerebellothalamo-

cortical projections to the cerebral surface.⁹ In two elderly patients with a structural cerebellar lesion, suffering from complex partial epilepsy, Umemura and Suzuki¹⁰ demonstrated “crossed cerebellar hyperperfusion”, a reversed crossed cerebellar diaschisis (CCD). A CCD has been described in patients suffering from stroke, infections, tumors and seizures and is characterized by depression of regional brain perfusion, metabolism and neuronal activity in the cerebellum contralateral to a cortical lesion.¹¹ This phenomenon may be documented by MRI or functional imaging such as PET or SPECT. Proposed mechanism underlying CCD include disconnection of glutamatergic corticopontocerebellar tract.¹² Conversely, the unilateral cerebellar lesion may have an influence on the contralateral cerebral hemisphere

(crossed cerebello-cerebral diaschisis).¹³ Cerebellar cortical projections are inhibitory by means of the release of the inhibitory transmitter gamma-aminobutyric acid (GABA) from the Purkinje cells. In this concept, cerebellar lesion may cause disinhibition of contralateral cortical cells and release of epileptic activity via the above pathophysiological mechanism.

EPC in our patient appeared in a strong temporal relation with a cerebellar hemorrhage and this fact together with the absence of cortical abnormalities on MRI study support a possible role of cerebellar lesion in induction of seizures. This case provides further evidence that cerebellar pathology probably may induce cortical seizures, if functional connections are retained.

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