

Chronic PLEDs with transitional rhythmic discharges (PLEDs-plus) in remote stroke

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ABSTRACT – Background. Periodic lateralized epileptiform discharges (PLEDs) are a rare phenomenon in electroencephalography, occurring in acute structural brain lesions. In general, PLEDs appear transiently in acute lesions, but a few reports have described persistent PLEDs in chronic lesions. **Case report.** An 86-year-old female was admitted, in 1999, with a left MCA stroke associated with right hand focal motor seizures. The first EEG in February of 2002 showed PLEDs over the left hemisphere associated with rhythmic discharges (PLEDs-plus). The patient was admitted on a second occasion in 2003 because of three sequential seizures and the EEG showed a similar pattern. Finally in 2006, the patient was admitted again because of sequential complex partial seizures and an EEG showed the same PLEDs-plus pattern as the EEGs of 2002 and 2003. **Discussion.** We report an unusual case of chronic PLEDs associated with rhythmic discharges in a patient with recurrent seizures and remote stroke.

Key words: chronic PLEDs, PLEDs-plus, seizures, stroke, epilepsy

The term periodic lateralized epileptiform discharges (PLEDs) was coined by Chatrian in 1964 (Chatrian *et al.* 1964). The incidence of PLEDs in unselected populations investigated in EEG laboratories ranges from 0.4% to 1% (Young *et al.* 1988, Pohlmann-Eden *et al.* 1996). PLEDs typically appear over the hemisphere involved, most often related with infarct or tumor and usually are self-limited, resolving within a few days or weeks (Pohlmann-Eden *et al.* 1996). The EEG features most often observed consist of sharp, or sharp and slow waves, less often spikes, spikes and waves or multiple spikes or complex burst, with

amplitude ranging from 50 to 150 μ V (could exceed 300 μ V). The periodicity (time interval between discharges) ranges from 0.3 to 5 seconds, being most commonly about 1 per sec (Pohlmann-Eden *et al.* 1996). PLEDs are considered an interictal pattern, but are frequently associated with seizures (Pohlmann-Eden *et al.* 1996, Schwartz *et al.* 1973). Although the physiopathology is unknown, in recent years there have been some reports considering PLEDs as an ictal and interictal EEG pattern (Terzano *et al.* 1986, Pohlmann-Eden *et al.* 1996, Terzano *et al.* 1986). A PLEDs-plus pattern was described by Reiher in



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1991 (Reiher *et al.* 1991), and was defined as the presence of rhythmic discharges in scalp EEGs, as transitional anomalies intercalated between inter-ictal PLEDs and ictal seizure discharges. Reiher (Reiher *et al.* 1991) showed that the occurrence of recorded seizures is higher in patients with PLEDs-plus than in those without these rhythmic discharges. In this report we present the case of a patient with a remote left MCA stroke and chronic PLEDs associated with transitional rhythmic discharges. Though there have been reports of PLEDs and PLEDs-plus associated with old stroke, we have not found any reports of chronic or recurrent PLEDs-plus in a patient with remote stroke.

Case report

Our patient is an 86-year-old, right handed female who, in 1999, experienced sudden aphasia and right hemiparesis. A CT scan showed a left temporal hematoma. Subsequent angiograms revealed a dural A-V fistula fed by the left ascending pharyngeal artery through the jugular foramen. The fistula was surgically disconnected but the patient developed a left middle-cerebral artery stroke. After the vascular event, the patient developed focal seizures characterized by jerking of the right arm. She was placed on phenytoin at a dose of 300 mg per day. Due to the good control of seizures, in 2002, phenytoin was discontinued, but two months after the discontinuation, she suffered two new seizures and was once again placed on phenytoin. An EEG performed in February of 2002 was consistent with PLEDs in the left hemisphere at a maximum over the left fronto-temporal region (*figure 1A*). The spike and wave discharges were seen to recur at intervals of 2-3 seconds, with rhythmic discharges consistent with a PLEDs-plus pattern. In the same year, the patient had a second EEG because of intermittent facial jerking on the left side. The EEG showed the same PLEDs-plus pattern (*figure 1B*). The neurological examination during this admission showed a mild motor deficit (4 plus of 5) in the right arm and leg, as a result of the previous stroke. In 2003, the patient was admitted to hospital because of the occurrence of three focal seizures characterized by jerking of the right hand followed by postictal Todd paralysis. There was no change in the EEG findings and the PLEDs-plus pattern persisted (*figure 1C*). While in hospital, the dose of phenytoin was adjusted to 350 and 400 mg every other day and clobazam was added at a dose of 5 mg twice a day with control of the seizures. An MRI performed in 2004 showed an extensive area of encephalomalacia in the left frontal and temporal areas consistent with the left MCA stroke (*figure 2A and 2B*). Between 2003 and 2005, the patient had occasional seizures. In 2005, the patient was seizure-free and the clobazam was decreased to 5 mg once a day. In August 2006, the patient began to experience sequential complex partial seizures characterized by oral automatism, bimanual automatism and loss of conscious-

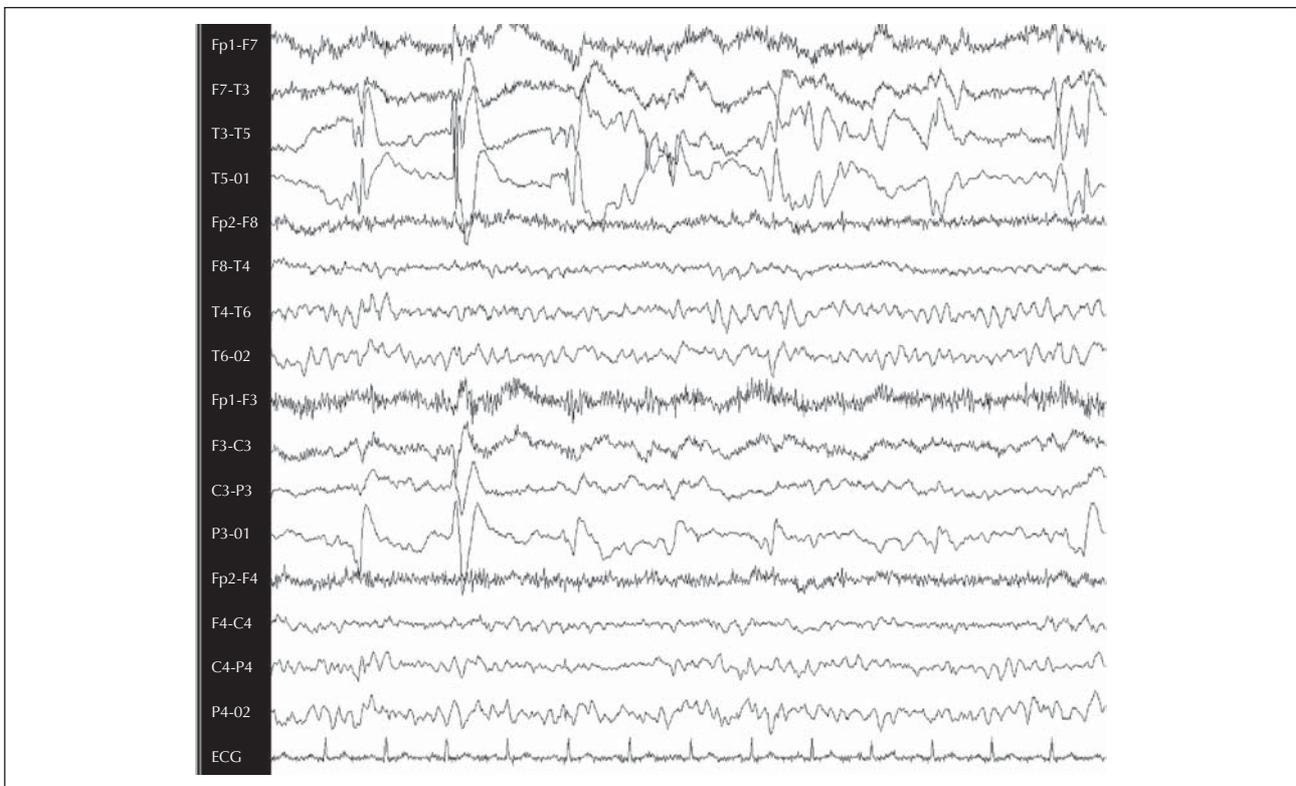
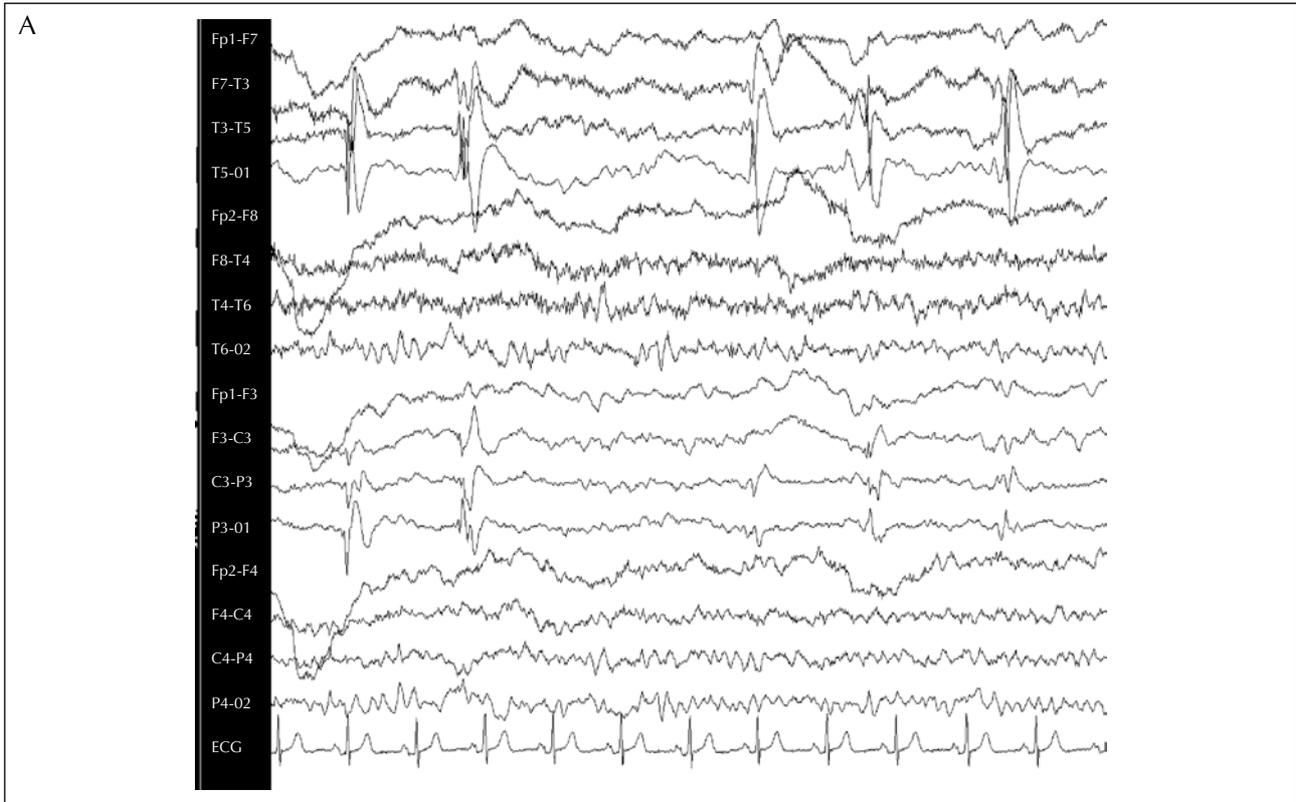
ness. Imaging revealed no evidence of any new ischemia. The patient was re-admitted with a plan to optimize her antiepileptic medication. Another EEG during hospitalisation showed PLEDs-plus over the left hemisphere with the same characteristics (*figure 1D*). While hospitalized, the clobazam was increased to 10 mg twice a day and the phenytoin was decreased to 200 and 250 mg every other day. After these changes in the dose of medications another EEG was performed and no changes in the PLEDs were identified. The neurological examination remained without changes during admissions.

Discussion

Our case is very particular considering the association of chronic PLEDs with a PLEDs-plus pattern. There are sporadic reports of chronic PLEDs in the literature. The original description of chronic PLEDs was by Westmoreland *et al.* in six patients (Westmoreland *et al.* 1986). Four patients had structural lesions two of whom had tuberous sclerosis, one patient had a porencephalic cyst, and one patient had a chronic abscess. Two patients had non-lesional complex partial seizures. The duration of PLEDs was from two to 30 years. A remarkable observation from this report is the high association of PLEDs with intractable epilepsy or frequent seizures. Three patients had complex partial seizures, two infantile spasms and one multifocal seizure onset (Westmoreland *et al.* 1986). The authors conclude in their report that patients with chronic PLEDs could be different with regards to the presence of chronic epilepsy, compared to patients with acute PLEDs where the seizures usually occurred during the acute event.

Gurer *et al.* (2004) reported the clinical findings of 71 patients with PLEDs. Five patients had chronic PLEDs. The duration of the PLEDs was from four months to five years. The etiology in two cases was subacute sclerosing panencephalitis (SSPE), one case had a chronic seizure disorder, one case had encephalomalacia secondary to surgery for an intracranial cyst, and the etiology was unclear in one patient. The two cases with SSPE had *epilepsia partialis continua* and were considered ictal; the other interictal PLEDs. There was no mention of remote stroke as an etiology in these five patients. Our patient has chronic epilepsy and a complex structural lesion in keeping with the reports of Westmoreland and Gurer (Gurer *et al.* 1986).

Gross (Gross *et al.* 1998) reported an interesting case of chronic PLEDs in a 39-year-old woman with severe left caudate nucleus atrophy and right hemi-dystonia. The patient had two thalamotomies, a pallidotomy and finally a stimulator was implanted in the centromedian thalamic nucleus in order to decrease the dystonic symptoms. EEGs performed before the first surgery and after the subsequent surgeries were consistent with left hemispheric PLEDs which were more prominent during sleep and abolished by arousal. The patients had no history of seizures.



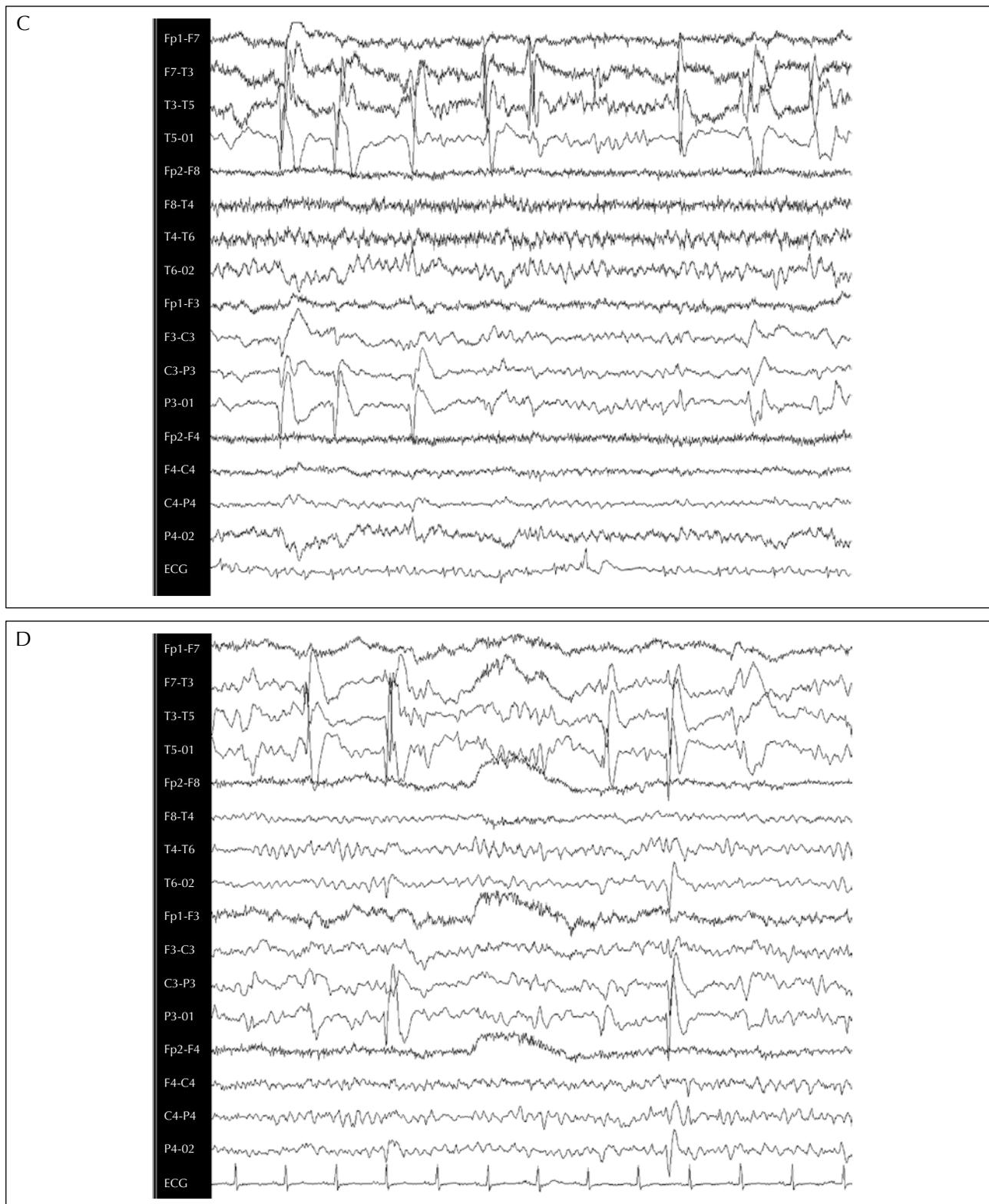


Figure 1. In this figure, we can observe the EEG evolution of this patient. Consistently, all the EEGs show PLEDs over the left hemisphere, at a maximum over the left fronto-temporal region. The repetition of the PLEDs was variable between 2-3 seconds and transitional rhythmic discharges (PLEDs-plus pattern) were identified. **A)** February 2002, **B)** October 2002, **C)** January 2003, **D)** August 2006.



Figure 2. An MRI performed in 2004 showed an extensive area of encephalomalacia in the left frontal and temporal area consistent with left MCA artery stroke (axial-FLAIR).

The authors suggest a role of the associative basal ganglia in the generation of periodic phenomena.

Transitional rhythmic discharges associated with PLEDs were originally described by Reiher *et al.* in 1991, and were referred to as PLEDs-plus (Reiher *et al.* 1991). Reiher evaluated the EEGs of 201 recordings with PLEDs from 84 patients. EEGs were divided in PLEDs-plus to describe the presence of rhythmic discharges; the PLEDs without these discharges were called PLEDs-proper. No differences were found between these two types of PLEDs with regards to the type of lesion. The significant observation in this study was the higher incidence of recorded seizures as well as the incidence of status epilepticus in patients with PLEDs-plus (Reiher *et al.* 1991). According to Reiher (Reiher *et al.* 1991), seizures were present in 74% of patients with PLEDs-plus compared to 6% in patients with PLEDs proper. In the same report, one patient with PLEDs-plus had a chronic stroke, but no repeat EEGs were done to confirm whether the PLEDs-plus were chronic. Our case is in keeping with the consideration of PLEDs-plus as a semi-ictal pattern with a great propensity for seizures as some author have suggested (Grand'Maison *et al.* 1991, Reiher *et al.* 1991). On the other hand, due to the high epileptogenic tendency of this EEG pattern, clinicians should consider more intensive treatment with antiepileptic drugs (Hirsch and Claassen, 2002).

Cerebral infarction is one of the commonest cause of PLEDs (Gurer *et al.* 2004, Pohlmann-Eden *et al.* 1996, Young *et al.* 1988) and embolic infarction seems to carry a higher risk of PLEDs than the thrombotic infarction (Lesser *et al.* 1985). Interestingly, in previous reports on chronic PLEDs (Gurer *et al.* 2004, Westmoreland *et al.* 1986), stroke was not considered as an etiology. In the literature, some reports have mentioned the association of strokes and the recurrence of PLEDs. Schwartz (Schwartz *et al.* 1973) reported four patients where PLEDs recurred after weeks to months associated with transient ischemic attacks, and two patients with symptomatic epilepsy. Erkulvratr (Erkulvratr, 1977) reported two patients in whom PLEDs recurred after an interval of 43 and 195 days. Finally, Dauben and Adams (Dauben and Adams, 1977) described two patients with remote strokes in whom PLEDs recurred after one month. In our case, after serial EEGs, the PLEDs did not disappear, this being the first case of a patient with a remote stroke associated with chronic PLEDs.

What is unique about our case report is the association of remote stroke with PLEDs-plus and recurrent seizures which appear to be progressive. The recognition of this pattern for prognosis becomes more important with the increasing incidence of epilepsy in the elderly. We anticipate that with emergence of more aggressive stroke treatments and more survivors with remote strokes, in addition to degenerative disorders, stroke will also contribute significantly to the incidence of PLEDs-plus. The presence of chronic epilepsy in our patient is in keeping with rare descriptions of chronic PLEDs in the literature, but not commonly recognized with PLEDs-plus and previous strokes. □

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