

Case report

Benign bilateral independent periodic lateralized epileptiform discharges

Fushimi M, Matsubuchi N, Sekine A, Shimizu T. Benign bilateral independent periodic lateralized epileptiform discharges. *Acta Neurol Scand* 2003; 108: 55–59. © Blackwell Munksgaard 2003.

Bilateral independent periodic lateralized epileptiform discharges (BIPLEDs) usually appear transiently in patients with severe disturbances of consciousness and are indicative of a poor prognosis. Recurrent BIPLEDs have not previously been reported in the literature. We report a 64-year-old patient with bilateral hippocampal lesions (cerebral infarction) who exhibited persistent periodic lateralized epileptiform discharges (PLEDs) (chronic PLEDs) associated with recurrent BIPLEDs. Electroencephalography was recorded for more than 6 months. Left hemispheric PLEDs appeared first. Next, PLEDs shifted to the right hemisphere and BIPLEDs occasionally developed. Brain magnetic resonance imaging and single-photon emission computed tomography with technetium-99-hexamethyl-propyleneamine oxime was performed before and after the appearance of BIPLEDs. The patient had no remarkable clinical symptoms aside from mild memory impairment for this period of time. This is the first case of recurrent 'benign' BIPLEDs, that is, BIPLEDs with a positive prognosis.

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Periodic lateralized epileptiform discharges (PLEDs) are a relatively rare phenomenon on electroencephalography (EEG) caused by events such as infarcts and encephalitis (1). In general, PLEDs appear transiently (1), and few reports have described persistent PLEDs (2–4). Bilateral independent periodic lateralized epileptiform discharges (BIPLEDs) are PLEDs occurring independently in both cerebral hemispheres (5). BIPLEDs are usually caused by anoxic encephalopathy and central nervous system (CNS) infections (thus the high incidence of coma), and are typically associated with a poorer prognosis than PLEDs (5).

PLEDs generally occur transiently, and recurrent BIPLEDs have not been reported previously in the literature. However, in the present case, PLEDs represented a chronic condition associated with recurrent BIPLEDs. Brain magnetic resonance imaging (MRI) and single-photon emission computed tomography with technetium-99-hexa-

methyl-propyleneamine oxime (HMPAO-SPECT) were performed before and after the appearance of BIPLEDs. Bilateral hippocampal lesions with decreased regional cerebral blood flow (rCBF) were demonstrated. This case illustrates the previously unreported EEG findings of persistent PLEDs (chronic PLEDs) associated with recurrent 'benign' BIPLEDs.

Case report

The patient was a 64-year-old man who had been treated for the past 3 years at our institution for diabetes mellitus, hypertension, hyperlipidemia and nephrotic syndrome. No history of epilepsy or alcoholism was reported. From about 1 week before admission, the patient experienced symptoms of upper airway inflammation. While at home, he suffered a suspected generalized convulsion and was brought to hospital by ambulance. On arrival, convulsions had already ceased and the

patient was fully conscious (Glasgow coma scale 14). No abnormal findings were present on neurologic examination. Pyrexia and an elevated leukocyte count were present and pneumonia was diagnosed following chest radiography. Laboratory tests revealed hyponatremia (124 mEq/l), elevated creatine phosphokinase (CPK) levels (252 IU/l), and mild hyperglycemia and he was admitted to hospital. No significant abnormalities were demonstrated on brain computed tomography (CT) or lumbar puncture.

On day 2, CPK values increased further (5140 IU/l) and myoglobinuria appeared. By day 12, CPK values had decreased (557 IU/l) and myoglobinuria had disappeared. The patient recovered from pneumonia shortly after admission, with temperature returning to normal on day 2 and abnormal findings on chest radiography disappearing by day 3. Hyponatremia resolved by day 5 and CPK values normalized by day 13. From about this time, mild signs of short-term memory disturbance [Mini-Mental State Examination (MMSE) 25] were observed. Brain MRI on day 15 revealed no abnormalities that could explain these clinical symptoms. However, on day 28, further brain MRI revealed bilateral abnormalities in the hippocampal regions. Based on the history, examination and investigations, bilateral infarction of the hippocampal regions was diagnosed. The patient was discharged on day 80, as memory impairment was only mild and he was capable of performing normal daily activities (MMSE 26, WAIS-R 89). Since his discharge, he has been visiting the hospital as an outpatient for treatment of his pre-existing conditions. No further remarkable abnormalities have been displayed other than mild memory impairment and mild hyperglycemia.

Results

EEG findings over time

EEGs were based on the International 10–20 system and referential montage was used. EEGs were recorded 23 times between day 8 and day 226. In all EEGs, basic rhythm was satisfactory (e.g., no slowing down). In EEGs from day 8 to day 16, left hemispheric PLEDs were identified (Fig. 1A). On days 27 and 30, BIPLLEDs were detected (Fig. 1C). In EEGs from day 31 to day 226, right hemispheric PLEDs were seen in all EEGs (Fig. 1B) and on days 115, 136 and 170, left hemispheric PLEDs were also observed. Additional BIPLLEDs were thus documented on three separate occasions on days 115, 136 and 170 (Fig. 1D).

Brain MRI findings

Brain MRI on day 15 was unremarkable. However, MRI on day 28 using a T2-weighted image and fluid-attenuated inversion recovery revealed areas of abnormal hyperintensity in bilateral hippocampal regions (Fig. 2A), while T1-weighted imaging displayed abnormal enhancement in the right hippocampal region (Fig. 2B). However, about 2 months later, these findings had almost completely resolved and subsequent MRI revealed no additional abnormal findings.

HMPAO-SPECT findings

HMPAO-SPECT was performed before and after the appearance of BIPLLEDs on day 115. Eight days before the appearance of BIPLLEDs (only right hemispheric PLEDs appeared), rCBF was decreased in the right fronto-temporal region (Fig. 3A). Six days after the appearance of BIPLLEDs, rCBF in the right fronto-temporal region was unchanged contrary to slight decrease in rCBF in the left fronto-temporal region compared to before the appearance of BIPLLEDs (Fig. 3B).

Discussion

In general, PLEDs appear transiently and disappear within 2–3 weeks (1). A small number of reports have described persistent PLEDs (2–4). In this case, a relatively rare situation was encountered in which PLEDs occurred persistently for more than 6 months. Furthermore, a number of unusual features characterized this case. Generally, in EEGs with periodic discharges, background activity is abnormal (e.g. slowing down). In the present case, however, good basic rhythm appeared on the EEG when PLEDs or BIPLLEDs developed. This case also differs from chronic epilepsy as reported by Westmoreland et al. (3). Another form of chronic PLEDs involves the occurrence of PLEDs only during synchronized (NREM) sleep, while the EEG is normal during wakefulness (4). However, to the best of our knowledge, this is the first case of recurring BIPLLEDs over an extended period without the development of any remarkable symptoms other than mild memory impairment. In previous reports, BIPLLEDs are often associated with severe disturbances of consciousness, such as coma (5). The present case therefore demonstrates a new variant of BIPLLEDs with a relatively good prognosis.

The pathogenesis of PLEDs is not entirely clear. In the present case, lesions on MRI were in a small, restricted area. In such cases, metabolic

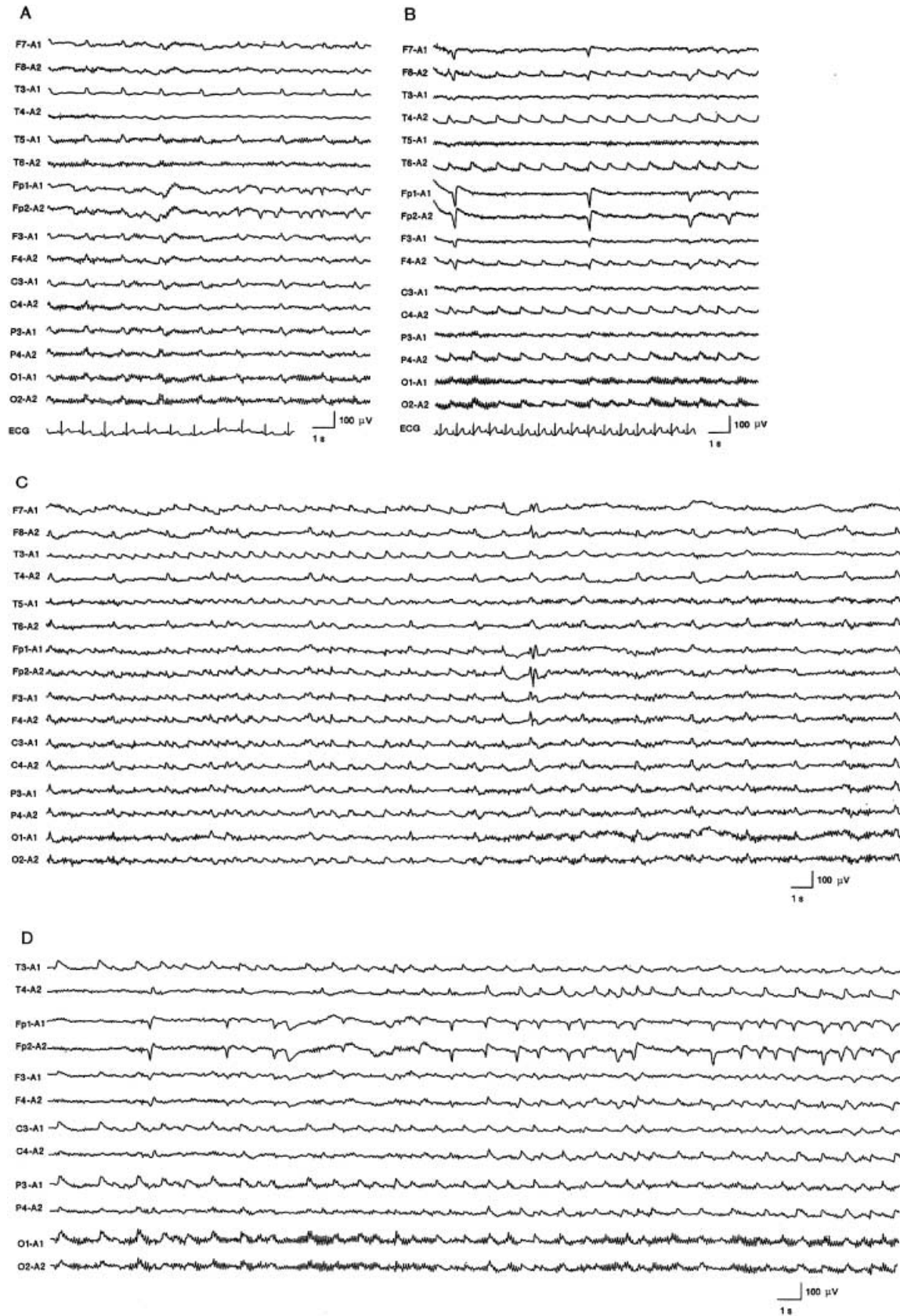


Figure 1. EEGs based on the International 10–20 system and referential montage. EEG day 9, left hemispheric PLEDs (A) and EEG day 226, right hemispheric PLEDs (B). BIPLEDs detected in EEGs on day 27 (C) and day 115 (D).

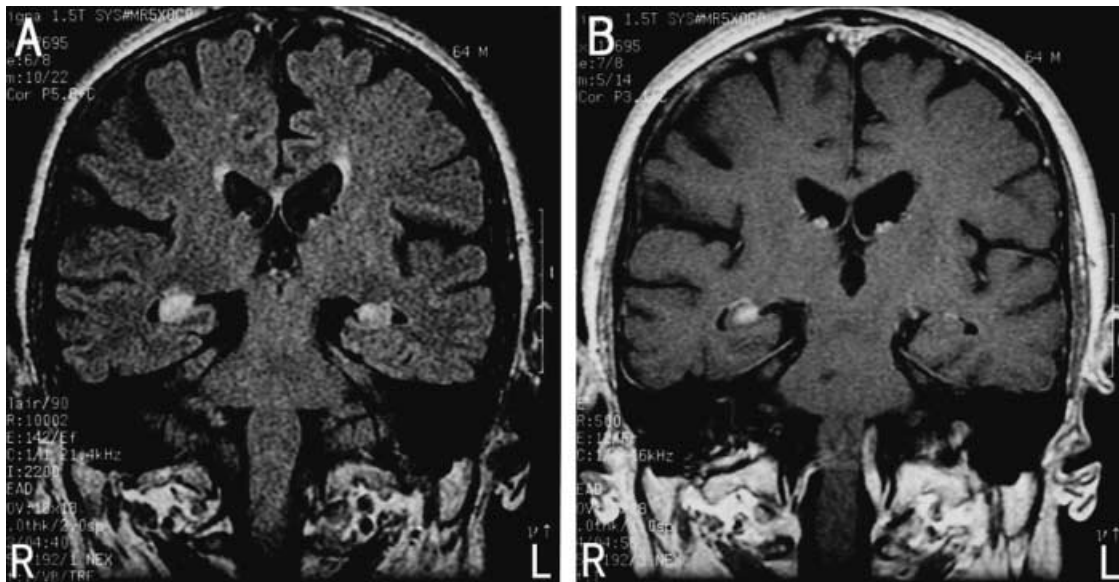


Figure 2. MRI on day 28 (coronal view) with fluid-attenuated inversion recovery reveals high intensity lesions in bilateral hippocampal regions (A) and a T1-weighted coronal image shows abnormal enhancement in the right hippocampal region (B).

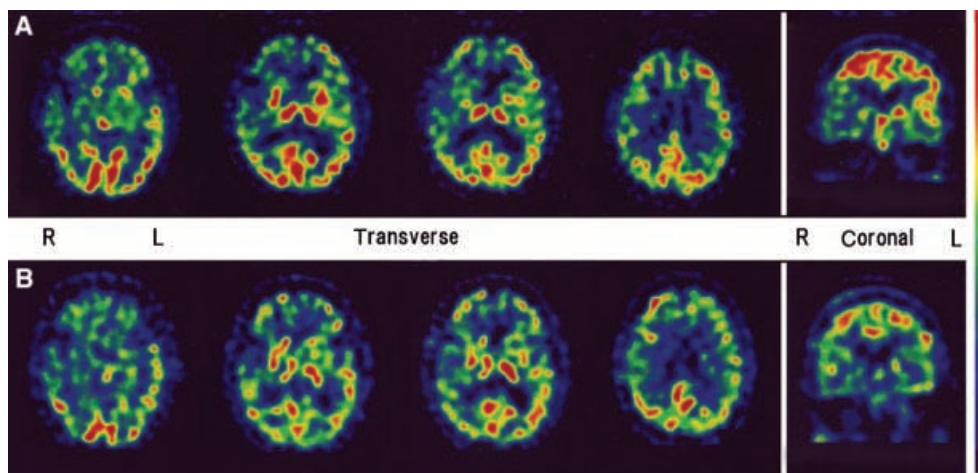


Figure 3. HMPAO-SPECT images 8 days before the appearance of BIPLEDs (only right hemispheric PLEDs had appeared), showing decreased rCBF in the right fronto-temporal region (A). Six days after the appearance of BIPLEDs, rCBF decreased slightly in the left fronto-temporal region in addition to the right fronto-temporal region (B).

abnormalities are frequently involved (6–8). Hyperglycemia probably exerted an influence in the present case.

Regarding the laterality, left hemispheric PLEDs appeared at first. PLEDs then shifted to the right hemisphere, with the occasional development of BIPLEDs. Our explanation of this pattern is based on differing levels of rCBF and differing thresholds for triggering PLEDs. Initially, ischemia may have been more severe in the left hemisphere. However, when lesions developed into infarcts, they were more severe in the right hemisphere. As a result, subsequent EEGs displayed the gradual shift of

PLEDs from left to right hemisphere. BIPLEDs probably developed during this period of transition. As a lesion was also present in the left hippocampal region, any factor (probably metabolic) inducing PLEDs would probably lead to their occurrence in both hemispheres. We therefore suspect the threshold for triggering PLEDs may have been higher in the left hemisphere than in the right. This hypothesis is based on changes in rCBF seen on HMPAO-SPECT: before BIPLEDs developed (appearance of right hemispheric PLEDs), rCBF was decreased in the right hemisphere. After had BIPLEDs developed, rCBF was also decreased

in the left hemisphere. However, whether rCBF decreases or increases at the site at which PLEDs appear remains controversial (9, 10). Further study of similar cases in the future is needed to provide firm support for this hypothesis.

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References

1. CHATRIAN GE, SHAW CM, LEFFMAN H. The significance of periodic lateralized epileptiform discharges in EEG: an electrographic, clinical and pathological study. *Electroencephalogr Clin Neurophysiol* 1964;**17**:177–93.
2. AU WJ, GABOR AJ, NAZHIYATH V, MARKAND ON. Periodic lateralized epileptiform complexes (PLEDs) in Creutzfeldt–Jakob disease. *Neurology* 1980;**30**:611–7.
3. WESTMORELAND BF, KLASS DW, SHARBROUGH FW. Chronic periodic lateralized epileptiform discharges. *Arch Neurol* 1986;**43**:494–6.
4. GROSS DW, QUESNEY LF, SADIKOT AF. Chronic periodic lateralized epileptiform discharges during sleep in a patient with caudate nucleus atrophy: insights into the anatomical circuitry of PLEDs. *Electroencephalogr Clin Neurophysiol* 1998;**107**:434–8.
5. DE LA PAZ D, BRENNER RP. Bilateral independent periodic lateralized epileptiform discharges: clinical significance. *Arch Neurol* 1981;**38**:713–5.
6. RAROQUE HG JR, GONZALES PC, JHAVERI HS, LEROY RF, ALLEN EC. Defining the role of structural lesions and metabolic abnormalities in periodic lateralized epileptiform discharges. *Epilepsia* 1993;**34**:279–83.
7. RAROQUE HG JR, PURDY P. Lesion localization in periodic lateralized epileptiform discharges: gray or white matter. *Epilepsia* 1995;**36**:58–62.
8. NEUFELD MY, VISHNEVSKAYA S, TREVES TA et al. Periodic lateralized epileptiform discharges (PLEDs) following stroke are associated with metabolic abnormalities. *Electroencephalogr Clin Neurophysiol* 1997;**102**:295–8.
9. LEE BI, SCHAUWECKER DS. Regional cerebral perfusion in PLEDs: a case report. *Epilepsia* 1988;**29**:607–11.
10. TERAOKA A, YASUDA T, KAWAI K et al. Single photon emission computed tomography (SPECT) in patients with periodic lateralized epileptiform discharges (PLEDs). *Jpn J Psychiatry Neurol* 1991;**45**:407–9.