

Possible Relationship of Periodic Lateralized Epileptiform Discharges to Thalamic Stroke

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Key Words

Epilepsy Surgery

Periodic Lateralized Epileptiform Discharges
(PLEDs)

Thalamic Stroke

INTRODUCTION

Periodic lateralized epileptiform discharges (PLEDs) are seen most often in diseases that produce an acute destructive cortical lesion of the cerebral hemisphere ipsilateral to the electroencephalographic (EEG) pattern.

The most common cause in adults is an acute cerebrovascular accident.¹⁻⁷ Many other neurologic conditions, or systemic conditions with neurologic complications, are associated with PLEDs. Examples of these are central nervous system infections^{1,5} including herpes simplex encephalitis,⁸ acquired immunodeficiency syndrome,⁹ Creutzfeldt-Jakob disease,¹⁰ neurosyphilis,¹¹ and cerebral cysticercosis;¹² neoplasms,¹ multiple sclerosis,¹³ neuro-Behcet syndrome,¹⁴ subdural hematoma,^{15,16} confusional states in the elderly,¹⁷ anoxia,^{2,18} alcohol withdrawal,^{18,19} nystagmus retractorius,²⁰ toxic-metabolic disturbances,^{21,22,23} sickle cell disease,¹¹ status epilepticus,⁶ and a chronic seizure disorder.^{7,24} In addition, one case of photic induced-driven PLEDs in paroxysmal dystonic choreoathetosis has been reported.²⁵

Although cerebral infarctions are the most common causes of PLEDs, these are usually large lesions with cortical destruction. Subcortical infarctions have not yet been described in association with PLEDs. We report a case of a thalamic stroke associated with PLEDs.

CASE REPORT

A 38-year-old white female was admitted to the hospital after being found unconscious near her car. The patient was in her usual state of health the prior evening when she went out to her car. She was found the next morning (approximately 12 hours later) by her parents

on the ground by her car, with her right leg in the car. The car had not been driven the night before. She had no history of alcohol or drug abuse, or cardiac problems. Her only medication is Dilantin 400 mg. at bedtime.

She had the onset of her seizure disorder (complex partial) at age 16, a couple of weeks after the birth of her first child. Her seizures were fairly well controlled (one every 2-3 months) for the first 10 years. Over the last 10 years the seizures slowly increased in frequency to 1 a week. The patient's seizures begin with an aura of a tingling sensation involving her right upper extremity. This is followed by unresponsiveness, mouth movements, blinking, and simple hand automatisms of 1-2 minute duration. She is then post-ictal and confused for about 5 minutes. The seizures were refractory to phenytoin, phenobarbital, primidone, carbamazepine, and valproate. She underwent an epilepsy surgery evaluation 1½ years prior to this hospitalization. Diagnostic evaluation consisted of interictal and ictal continuous, video-recorded, extracranial EEG monitoring (scalp and sphenoidal electrodes), CT scan, MRI, visual field examination, neuropsychological testing, MMPI, cerebral arteriogram, intracarotid sodium amobarbital testing, and interictal and ictal continuous, video-recorded, intracranial EEG monitoring (depth electrodes). This evaluation revealed she would be a good candidate for a left anterior temporal lobectomy, and this was performed 1 year ago. She tolerated this procedure well, and has had no further auras or seizures. Pathology revealed severe loss of

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neurons and glial scarring of the hippocampal formation.

Upon admission, her temperature was 37°C, pulse was 68, and respirations were 14. The blood pressure was 120/70 mm Hg. On examination the patient was obtunded. General physical examination revealed ecchymosis on the tongue and right forearm. The remainder was unremarkable (heart was normal, no bruits). Neurologic examination revealed that she would open her eyes, and groan when painful stimuli were applied. She had no understandable speech, and could not be tested for other language functions. Oculovestibular reflexes were intact, and the fundi were normal. Both pupils were 4 mm and reacted to light. Decreased movements were noted on the lower half of the right side of the face in response to painful stimuli. No gag was elicited. The remaining cranial nerves that could be examined were normal. Motor function showed a flaccid right hemiparesis. Sensory function revealed a right-sided loss to deep pain. Gait and coordination could not be tested. Tendon reflexes were +++ on the right and + on the left; the plantar response was extensor on the right, and flexor on the left.

The urine was normal. The hematocrit was 38.2%; whitecell count was 16,900 with 92% neutrophils, 5% lymphocytes, and 3% basophils. Platelet count was 227,000; prothrombin time was 14.3 seconds, with a control of 10-12 seconds; partial thromboplastin time was 29.7, with a control of 22-35 seconds. Initial electrolytes, calcium, magnesium, and general chemistry were all unremarkable except for a CPK of 3179 mu/ml (normal, 0-225; 100% mm). The phenytoin level was 11 ng/ml, a urine drug screen was negative. The EKG and x-ray of the chest were normal. The lumbar puncture showed an opening pressure of 140 mm H₂O, glucose 81 mg/dl, protein 26 mg/dl, 11 RBC/mm³ and a nonreactive VDRL. No organisms were seen on Gram stain, cryptococcal antigen test was negative, acid fast bacilli smear was negative, and the spinal fluid grew no bacteria or fungus. The arterial blood gas on room air was a 7.37 pH, 42.9 PCO₂. The serum triglyceride was 93 mg/dl; and the total cholesterol 152 mg/dl with a VLDL cholesterol of 3 mg/dl, an LDL cholesterol of 101 mg/dl, and a HDL cholesterol of 48 mg/dl (a normal lipoprotein profile for her age).

A portable EEG (Figure 1) recording per-

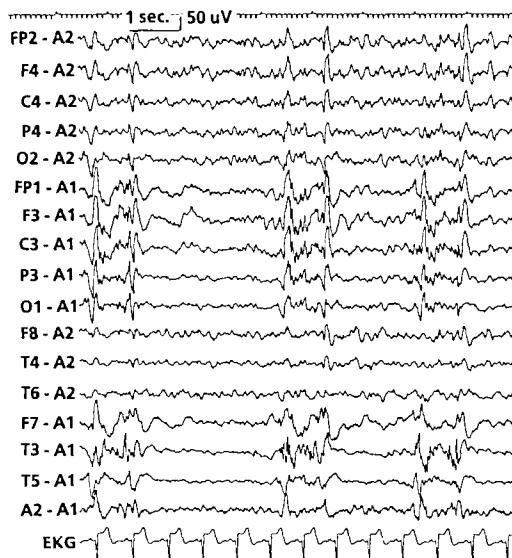
formed while she was obtunded disclosed the presence of periodic lateralized epileptiform discharges over the left hemisphere, seen maximally over the fronto-temporal area. No seizures were noted. A cranial MRI scan obtained the next day (Figure 2) disclosed evidence of the prior left temporal lobectomy and abnormal signal (increased signal intensity on the T₂-weighted images) in the left thalamus and right side of the brain stem (probably ischemic in nature). Unfortunately, no further EEGs or neuroimaging studies were performed. An aortic arch and 4-vessel cerebral arteriogram was normal except for the left vertebral arteriogram that demonstrated a focal decrease in the caliber of the distal left vertebral artery and basilar artery consistent with diffuse vertebrobasilar spasm. In addition, surgical branch occlusions in the inferior left temporal branches of the left middle cerebral artery consistent with the previous left temporal lobectomy were seen. No definite evidence of branch occlusions or vascular abnormalities were noted in the posterior fossa circulation. A fetal origin of the right and left posterior cerebral artery was noted, with both arising from their respective internal carotid arteries. The 24-hour Holter monitor was normal; a 2-D cardiac echo showed no thrombus; and the posterior circulation transcranial doppler was normal.

The patient steadily improved and by the second week was more alert, and had regained most of her strength and sensation on the right side. Her aphasia improved slowly, and consisted primarily of a severe receptive deficit with a moderate expressive disorder. She began speech therapy.

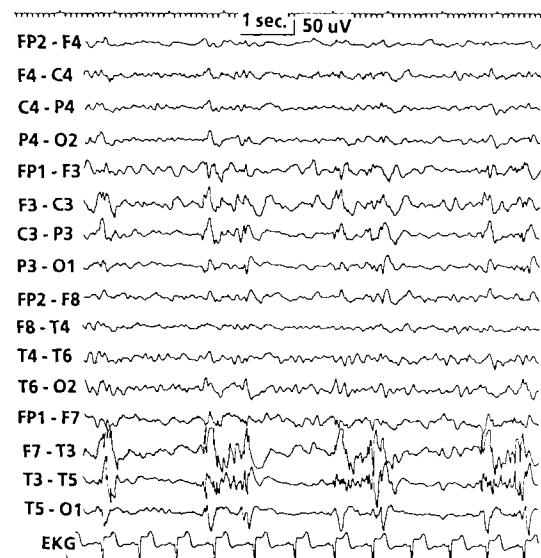
It was felt as though the patient's cerebrovascular accidents were most likely embolic in nature, even though a definite source was not identified, and she was placed on Coumadin. Three to 4 weeks after this hospitalization she had the onset of a "new" seizure type. This consisted of staring, unresponsiveness, and then focal clonic activity on her right side lasting 2-3 minutes, followed by a right-sided Todd's paralysis for 30-45 minutes.

DISCUSSION

Our patient's seizures ceased after the left temporal lobectomy. She was seizure-free on one anticonvulsant. However, after her thalamic stroke and associated PLEDs she developed a "new" seizure type. This finding is con-



A



B

Figure 1A & 1B

Drowsy, eyes closed, HFF 70 Hz, T.C. 0.1. Periodic lateralized epileptiform discharges (spikes, polyspikes, and sharp waves) occurring every one to three seconds, over the left cerebral hemisphere, maximally over the temporal area. The background activity over that hemisphere is slower in frequency, and lower in amplitude than the right hemisphere.

sistent with prior studies that have shown the majority of patients with PLEDs go on to have recurrent seizures after hospitalization.^{22,23,26}

There are many reports on the EEG changes in cerebrovascular accidents, and their association with PLEDs. A stroke is the most common etiology in patients with PLEDs. However, in these cases PLEDs were associated with large, destructive cortical infarctions. Unilateral lesions in the thalamus may produce unilateral rhythmic delta activity,²⁷ but there are no reports of discrete thalamic lesions producing PLEDs. Schwartz reported one patient who had PLEDs (maximally seen over the left temporal area) and at autopsy had an extensive glioma spreading throughout the thalamus.⁷ This report demonstrates that PLEDs can be observed in patients with a unilateral, subcortical stroke. The pathogenesis of PLEDs is uncertain. Lombroso and Cobb hypothesized that there might be a thalamic or mesencephalic origin to periodic complexes.^{28,29} Others have hypothesized that PLEDs arise in an area of cortex anatomically or functionally deafferentated from the subcortical structure.^{17,21,25} Jacome and Risko's report of photic induced-driven PLEDs in paroxysmal dystonic cho-

reoathetosis suggested that subcortical structures participate in the generation of PLEDs.²⁵ Chu reported a patient with PLEDs and acute subdural hematoma. The autopsy revealed no underlying cerebral contusions, suggesting a possible role of subcortical structures (due to midline shift) in the pathogenesis of PLEDs.¹⁵ Chatrian et al believed that both cortical and subcortical (thalamic) structures could contribute to the genesis of PLEDs.¹ They felt that the cortex must participate in the production of PLEDs no matter what the level of origin of the discharge was. This view was reinforced by Markand and Daly and then by Kuroiwa and Celesia's more recent review of the clinical significance of periodic EEG patterns.^{3,11}

Our patient had both cortical and subcortical abnormalities that could potentially contribute to the genesis of her PLEDs. The cortical physiology surrounding a prior temporal lobectomy is altered. This is reflected as focal slowing on the EEG. It is possible that this change in local cortical physiology may lower the threshold for the production of PLEDs (by either a subsequent subcortical or cortical lesion). In addition, the embolic episode that produced the subcortical imaging abnormalities could have

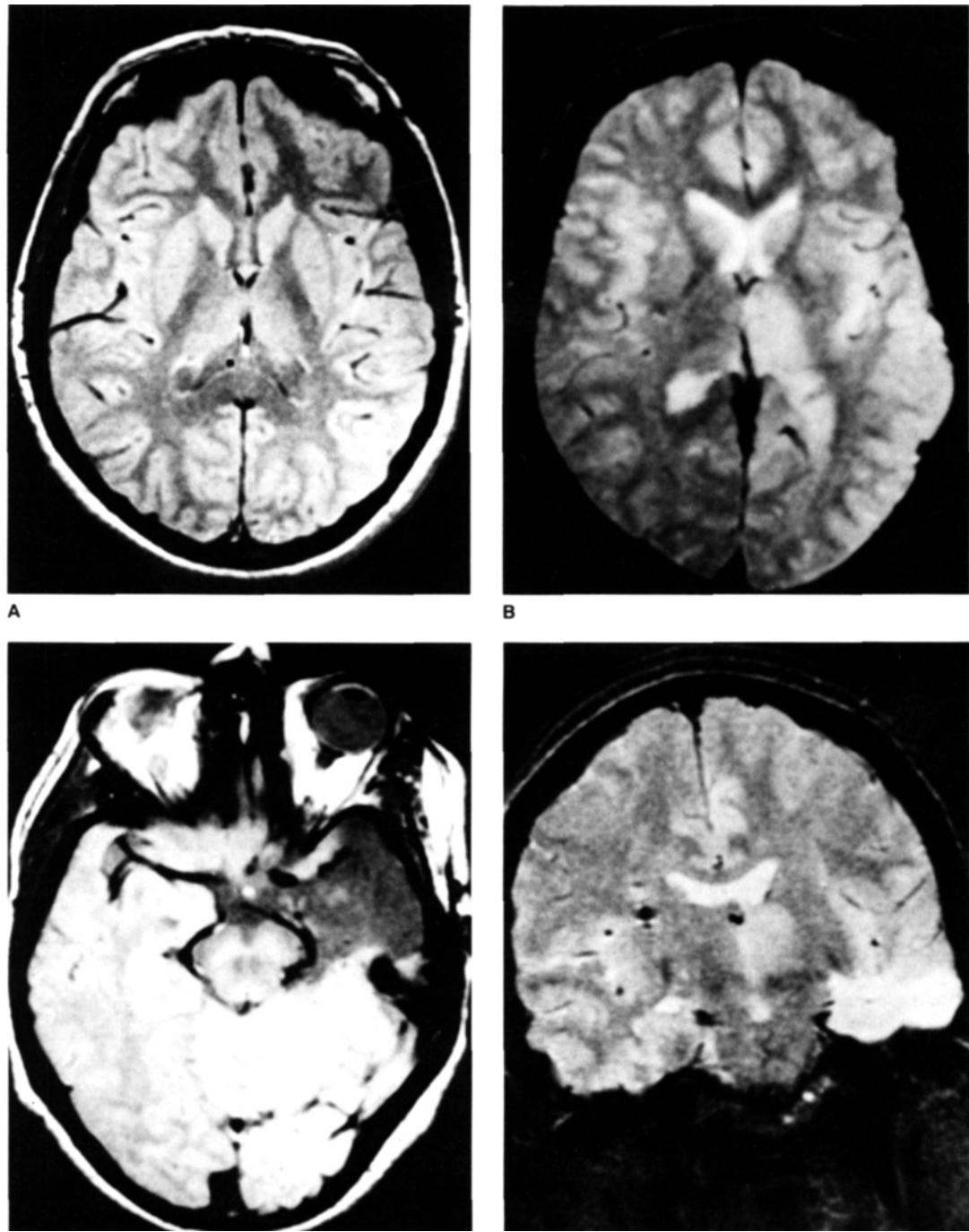


Figure 2 A - MRI obtained in the axial plane (TR 2000, TE 20) prior to the temporal lobectomy is normal. (The left cerebral hemisphere is on the right side of each photograph.)
B - MRI obtained in the axial plane (TR 2000, TE 70) during this hospitalization reveals a left thalamic abnormality (increased signal).
C - MRI obtained in the axial plane (TR 2000, TE 20) reveals the prior left temporal lobectomy.
D - MRI obtained in the coronal plane (TR 2000, TE 20) during this hospitalization reveals a left thalamic abnormality (increased signal) and evidence of the prior left temporal lobectomy.

caused mild hypoxic-ischemic changes in the cortex adjacent to the prior lobectomy. However, it is very unlikely that significant cortical hypoxia occurred since no signal abnormalities were seen on an MRI performed more than 24 hours after the event. This occurrence of the thalamic stroke ipsilateral to an area of prior cortical resection may have allowed the genesis of PLEDs without a new cortical abnormality being seen. This suggests a possible association between subcortical structures and the generation of PLEDs. With improved neuro-

imaging (MRI) it will be possible to further characterize the relationship of discrete subcortical lesions to PLEDs, something that was not previously possible.

SUMMARY

A patient with a unilateral thalamic infarct and associated periodic lateralized epileptiform discharges is described. The implications of this for the pathophysiology of PLEDs is discussed.

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