

Acute Subdural Hematoma and the Periodic Lateralized Epileptiform Discharges

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Introduction

Periodic lateralized epileptiform discharges (PLEDs) in the electroencephalogram consist of complexes of various epileptiform waves recurring at relatively regular intervals of one-half to three seconds (1). This phenomenon is often associated with an ipsilateral hemispheric structural lesion. The most common lesions are cerebral infarction and brain tumors (1-4). Other neurological diseases associated with PLEDs are intracerebral hemorrhage, encephalitis, cerebral abscess, acute cerebral trauma seizure disorders, neurosyphilis, and tuberculoma (1-7). Occasionally, a small cortical lesion appears to be responsible for the production of PLEDs under circumstances such as aminophylline intoxication (8), hypoxia (8), nonketonic hyperglycemia (9), alcohol withdrawal (10), and systemic infection (10). The occurrence of PLEDs in patients with subdural hematoma is rare. Toyonaga et al. (11) reported PLEDs in three cases of chronic subdural hematoma, but PLEDs associated with acute subdural hematoma have not been reported. During the past one and a half years at the University Hospital two cases of PLEDs were seen in patients with acute subdural hematoma among a total of 10 patients with this phenomenon. Thus, PLEDs associated with acute subdural hematoma appear to be not as infrequent as previously thought.

Case 1

The patient was a 55 year old moderately obese white female with a long history of alcohol abuse and hypertension. In 1972, the patient had an acute onset of stupor and left hemiparesis. The blood pressure was 180/115 and the cerebral angiogram revealed an avascular mass in the right temporo-occipital area. Right temporal craniotomy evacuated

25 cc of clotted blood. The patient was left with residual left hemiparesis and left homonymous hemianopsia, and continued to receive Aldomet and Hydrochlorothiazide for her hypertension. Pneumoencephalography two months later revealed moderate dilation of right occipital horn which communicated with a porencephalic cyst. On follow-up five years later, the patient had been fully functional except for the left hemianopsia.

On the day of admission, the patient tripped over the stairs of her mobile home and fell backwards striking the head on the concrete floor. She immediately lost consciousness, and in the Emergency Room a deep scalp laceration was noted on the left parieto-occipital area which was sutured. The patient was brought to the Intensive Care Unit where she was noted over a period of 30 minutes to be improving with purposeful withdrawal to painful stimulation. Subsequently, the patient became totally unresponsive with unreactive pupils which were 3 mm on the right and 5 mm on the left. Diagnostic twist drills over the temporal areas on both sides did not reveal any hematoma. The patient was stabilized with the use of diuretics and steroids. She remained stuporous, moved right extremities purposefully, but had only simple withdrawal of left side to painful stimulation. Plantar response was extensor on the left.

One and a half days later facial twitching and tonic-clonic movements of the left arm were intermittently noted for several hours. There were brisk deep tendon reflex on the left and decerebrate posturing of the left arm in response to pinching. By the next day, the

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Figure 1. Case I. Recurring discharges of sharp wave complexes in the right hemisphere. Monopolar recording with ipsilateral ear as reference. The vertical calibration is 50 μ V and the horizontal one 0.5 sec.

patient had almost continuous seizure activity involving the eyes, face, neck, and the left upper extremity. Arterial blood gas and blood chemistry were within normal limits. She was treated first with intravenous Valium, followed with Dilantin and phenobarbital. In spite of anticonvulsant treatment with Dilantin level of 12 μ g/ml and phenobarbital level of 20 μ g/ml, the patient continued to exhibit intermittent left focal motor seizures, usually lasting 5 to 15 seconds for the following two days.

EEG revealed recurring polymorphic sharp waves roughly at 1 Hz interspersed with suppression of background rhythms on the right side (Figure 1). The distribution of the sharp waves was predominantly in the frontal and central areas. A computerized tomographic (CT) scan revealed a large high density mass extending all the way from the most anterior portion of the frontal lobe to the entire occipital lobe (Figure 2). A considerable amount of mass was also present in the vicinity of right lateral tentorium. The lateral and third ventricles were shifted from right to left approximately 2 cm. The degree of shift

was more prominent anteriorly. The right lateral ventricle was considerably compressed. A right parietotemporal craniectomy was performed with evacuation of 180 cc of fresh blood clot. The seizures subsided following the surgery. Anticonvulsant levels were 22 μ g/ml for Dilantin and 29 μ g/ml for phenobarbital. The patient continued to have intermittent left decerebrate posturing, sluggish light pupillary reflex, absent oculocephalic response on the left. A few days later, her clinical course was complicated by hyperglycemia, hypokalemia, and elevated blood urea nitrogen that were gradually corrected.

EEG five days after surgery showed persistence of recurring polymorphic spike and wave complexes on the right but its occurrence was less frequent and the interval was more prolonged than in the previous tracing. Sometimes burst-suppression pattern evolved. Study of the auditory brainstem evoked responses revealed prolonged central conduction time following right-sided stimulation and the absence of

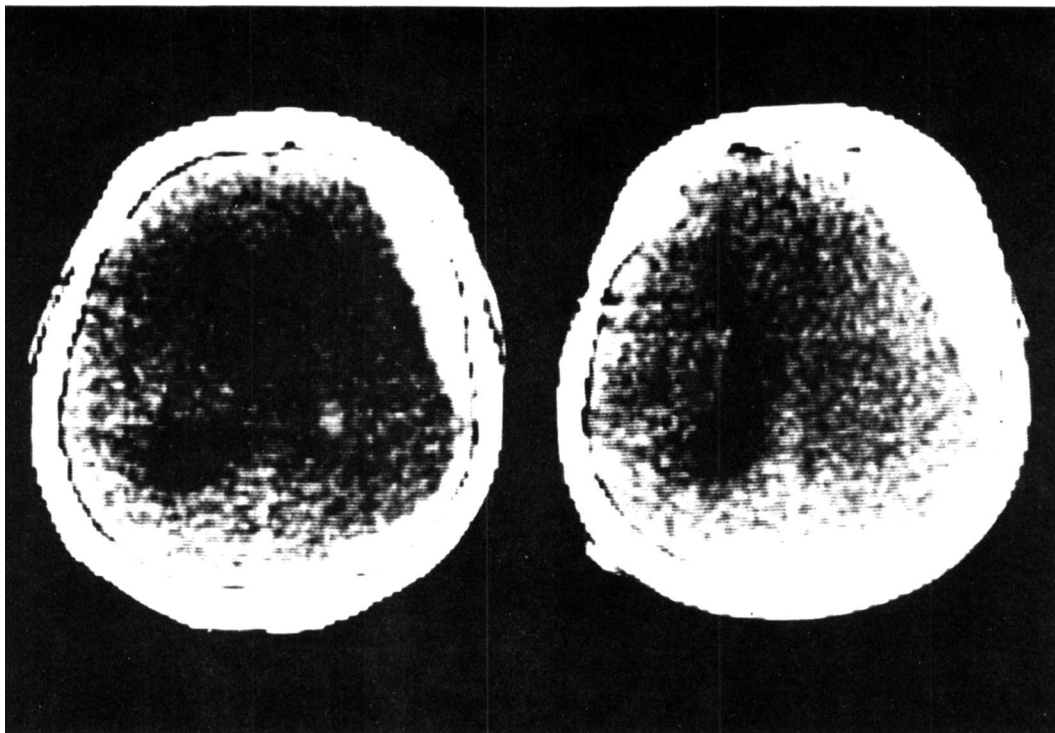


Figure 2. Case I. Computerized tomographic (CT) scan showed a large right subdural hematoma which was maximal in the frontocentral region but which also extended all the way around the convexity to the entire occipital area. This was accompanied by a marked right to left midline shift with almost obliteration of right lateral ventricle.

identifiable waves beyond Wave I on the left, suggesting marked brainstem dysfunction. Repeated CT scan showed absence of large extracerebral blood collection except for a small increased density in the frontal area and a slight right to left shift. Gradually, the patient exhibited bilateral decerebration, loss of withdrawal to pinch in the left upper extremity, and intermittent blinking movements of the eyes, clinically resembling coma vigil.

One week after the second EEG, oculocephalic response was absent bilaterally, pupillary light reflex was sluggish, but eye blinking was still occasionally noted. EEG showed repetitive sharp transients of rather low amplitudes occurring in intervals of approximately 1 to 4 seconds on the right. Phase reversal was noted in the right central area. Three days later, the patient developed bilateral pneumonia and died 4 days later following presumably septic shock and cardiac arrest. Autopsy revealed bilateral

acute bronchopneumonia with abscess formation, acute pancreatitis and left parietal skull fracture. The dura mater was tense but subdural collection of blood was absent. Cerebral hemispheres were symmetrical and had a generalized early softening. The right occipital lobe was partially obliterated with a thin membrane which had a defect of 0.5 cm communicating to the lateral ventricle. The brainstem was congested without hemorrhage and distortion.

CASE II

A 44 year old man was found on the street, confused, with multiple bruises over scalp, face, and extremities. The patient had a history of excessive alcohol drinking, and the blood alcohol level was 61.6 mg% on admission to a local hospital, where he had two generalized convulsions beginning with turning the head and the eyes to the right and possible jerking of the right arm. The convulsions were treated with Dilantin and

Paraldehyde, but the patient was left with a mild right hemiparesis and aphasia which cleared in one or two days. The patient was then transferred to the University Hospital where he remained confused. The anticonvulsant medication was discontinued, and 3 days after admission the patient had six or seven generalized convulsions with right focal onset followed again by a mild right hemiparesis and aphasia. Dilantin level was 4 µg/ml.

An EEG showed recurring medium to high amplitude bi- and triphasic wave complexes which were variably sharp and predominantly over the left hemisphere. Between the wave complexes the background was marked by suppression, and occasionally the epileptiform discharges were generalized. A CT scan revealed a high density area in the left posterior area and a rather marked left to right midline shift. The left lateral ventricle was compressed with obliteration of the posterior horn. Following cerebral angiography, a left craniotomy was performed to evacuate 65 cc of fresh blood clot. The patient was treated with Dilantin and phenobarbital.

Two days after the surgery the EEG revealed left-sided PLEDs with background suppression, although clinical seizures were not observed. The right hemiparesis gradually resolved, but a dense aphasia persisted. Two weeks later, seizures recurred. Determination of serum anticonvulsant levels revealed 4 µg/ml for Dilantin and 10 µg/ml for phenobarbital. Repeated CT scan showed persistence of a marked left-to-right midline shift, but the extracerebral blood collection had completely regressed. The dosage of anticonvulsants was increased, and the EEG repeated 3 days later revealed left hemispheric slowing, maximal in the temporal and parietal areas.

Two generalized seizures with right focal onset were again observed with Dilantin level of 1 µg/ml. The EEG showed almost continuous spike and slow wave discharges occurring at about one per second over the entire left side. The slow waves were located in the posterior temporal and occipital areas. With readjustment of anticonvulsant dosages, the patient had been free of seizures for the next two weeks until he was discharged. However, the patient was still left with some impairment of left-right discrimination, visual motor perception, auditory comprehension,

and word finding.

Discussion

This report demonstrates that PLEDs can be observed in patients with acute subdural hematoma. Both patients had a history of alcoholism and recent head injury. They presented with focal motor seizures with or without secondary generalization, concomitant with EEG manifestations of recurring discharges of polymorphic sharp wave complexes over the cerebral hemisphere where subdural hematoma was located. CT scan revealed extracerebral collection of dense fluid with marked midline shift. In spite of the evacuation of subdural hematoma, PLEDs persisted or recurred for days or weeks.

One of the complications associated with subdural hematoma is seizures which are more often seen with acute subdural hematoma (12). Although EEG alterations in subdural hematoma are variable, asymmetry of alpha activity or ipsilateral slowing with suppression of background amplitude has been stressed (13). The occurrence of PLEDs is uncommon. Toyonaga et al (11) reported PLEDs in 3 patients with chronic subdural hematoma although in one patient both old and recent hemorrhages were present. All the patients had a history of alcoholism and their seizures were focal contralateral to subdural hematoma except for one patient who had in addition secondary generalized convulsions. PLEDs disappeared after evacuation of hematoma, but in one patient PLEDs persisted 3 days after the surgery.

Brignolio et al (14) observed a patient with bilateral chronic subdural hematomas who presented with left focal motor seizures and left hemiparesis. EEG study revealed recurring bilateral sharp wave discharges but maximal over the right frontal area where the larger hematoma was located. Johnson (15) reported a case of subdural hematoma of unknown chronicity with right-sided continuous focal seizures predominantly involving the abdominal wall, but the EEG was not obtained. Bilateral periodic slow wave discharges resembling hepatic encephalopathy were observed in two patients with unilateral chronic subdural hematoma (16). Thus, PLEDs associated with acute subdural hematoma have not yet been adequately documented.

The pathogenesis for PLEDs is still unsettled, although anatomical or functional deafferentation of cerebral cortex has often been implemented (1,3). The latter hypothesis, however, makes it difficult to explain the fact that in patients with focal epilepsy or a small cortical lesion, PLEDs occur under various toxic-metabolic circumstances such as hypoxia (8), aminophylline intoxication (8), alcohol withdrawal (10), etc. In acute subdural hematoma, compression of the underlying cerebral cortex from the expanding subdural mass could be an important factor for the production of PLEDs, but there was also a marked midline shift to suggest a significant dysfunction of subcortical structures. It could be argued that it was the underlying cerebral contusion but not the subdural hematoma that was responsible for the occurrence of PLEDs. Autopsy study in Case I fails to support this contention, but this possibility cannot be completely ruled out in Case II for there was persistent marked midline shift. However, in cases of chronic subdural hematoma, midline shift was either absent or minimal and PLEDs disappeared following surgery, thus arguing against the possibility of underlying cerebral contusion.

In these two cases, the persistence or recurrence of PLEDs following evacuation of acute subdural hematoma appears to occur under different circumstances. In Case I, PLEDs persisted for two weeks in spite of

adequate anticonvulsant medication and absence of clinical seizures. It is possible that the porencephalic cyst in the right occipital lobe aggravated by the underlying metabolic derangements of hyperglycemia and hypokalemia could be responsible for persistent PLEDs. However, PLEDs continued in spite of correction of the metabolic conditions and there was a phase reversal in the EEG in the central area suggesting a possible structural etiology. In Case II, the recurrence of PLEDs was apparently related to inadequate anticonvulsant treatment. In any event, when PLEDs and focal motor seizures with or without secondary generalization are observed in patients with recent head trauma, acute subdural hematoma should be strongly suspected. Appropriate tests such as CT scan and cerebral angiography should be performed to ensure prompt treatment.

SUMMARY

Focal motor seizures associated with periodic lateralized epileptiform discharges (PLEDs) were observed in two patients with acute subdural hematoma. Following the surgery, one patient continued to exhibit PLEDs but clinical seizures were absent. PLEDs recurred in the second patient due to inadequate anticonvulsant medication. Autopsy study in one patient suggests that underlying cerebral contusion was not responsible for the occurrence of PLEDs.

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