Epilepsia Partialis Continua as a Manifestation of Hyperglycemia

Supachai Paiboonpol, MD*

** Department of Medicine, Ratchaburi Hospital, Ratchaburi*

A review of 22 patients who presented with the syndrome of epilepsia partialis continua as symptoms of hyperglycemia and occurred during the phase of hyponatremia and mild hyperosmolality. Epilepsia partialis continua persisted for an average of 9 days,and its duration correlated with the degree of hyponatremia,low blood urea nitrogen(BUN) and measured serum osmolality. In all patients,epilepsia partialis continua was the first symptom leading to the diagnosis of diabetes mellitus. The epilepsia partialis continua ceased by normalization of blood glucose level. All patients survived and did not have any complications. The majority of the patients had evidence of a localized structural brain lesions and low serum sodium. Metabolic abnormalities including hyperglycemia,mild hyperosmolality and hyponatremia contribute to the development of epilepsia partialis continua in an area of focal brain damage. It is important to determine blood glucose levels in all cases of epilepsia partialis continua.

Keywords: *Epilepsia partialis continua, Hyperglycemia*

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Epilepsia partialis continua is characterized by a simple partial motor seizure, restricted to one part of the body with repetitive regular or irregular clonic jerks without loss of consciousness. Clonic activity persists for a period of days or weeks either continuously or with interruptions and remains localized to a single muscle group or may even progress to secondary generalized convulsive status[1,2]. Epilepsia partialis continua is rather uncommon with a prevalence rate of less than one per million[2]. This condition can result from a static insult with a non progressive course including stroke,tumor,cortical dysplasia,head trauma and metabolic cause[1,3,5-7]. The second condition characterized by progressive course such as Rasmussen's encephalitis which carries a grave prognosis[1,3,4]. But a benign and treatable cause should not be forgotten. The present study aimed to describe the clinical characteristic of the patients who present with epilepsia partialis continua as the presenting symptom of diabetes mellitus with a good prognosis.

Correspondence to: Paiboopol S, Division of neurology, Department of Medicine,Ratchaburi Hospital, Ratchaburi 70000 Thailand

Material and Method

The present study reviewed the medical records of patients 14 years of age or older who presented with epilepsia partialis continua between January 1, 1993 and December 31, 2003. In Ratchaburi Hospital Thailand,which is a tertiary referral center for medical patients. The criterior for diagnosis of epilepsia partialis continua as proposed by Thomas et al[4] was used: regular or irregular clonic muscular twitches affecting a limited part of the body, occuring for a minimum of 1 hour, and recurring at intervals of no more than 10 seconds. Cases were collected from medical records. All patients underwent detailed neurological examination, blood chemistry analysis, neurologic imaging (CTscan) andelectroencephalographic studies.

Statistical analysis used the SPSS package. Comparison of total duration of seizure and different variables were assesed by Pearson moment correlation. A p-value of < 0.05 was considered significant.

Results

Twenty-two patients (19 men and 3 women) with hyperglycemia and epilepsia partialis continua were studies. All patients had epilepsia partialis

continua. The average age of the patients was 45.8 years, ranging from 33 to 65 years. The average duration of epilepsia partialis continua prior to admission to the hospital was 9.3 days (range 3 to 30 days). In 14 patients epilepsia partialis continua was localized to the right side,and in 8 to the left. Epilepsia partialis continua involved shoulder and hand more frequently than leg and distal more than proximal body parts. In the majority of patients the frequency of jerks was less than 10 per minute. Movement sensitivity was observed in nine (40.9 percent) patients. Sleep failed to suppress epilepsia partialis continua in all of them (Table 1, 2).

All patients were alert at the time of hospitalization. None of the patients were known to have diabetes mellitus. In all patients,epilepsia partialis continua was the first symptom leading to the diagnosis of diabetes mellitus. The patient's seizures, in general were resistant to anticonvulsant medication but ceased with correction of the hyperglycemia and hyponatremia. All patients survived and did not have a serious associated illness.

The mean blood glucose level was 488.3 mg/dl (range 324 to742 mg%); serum sodium concentration, 129 mEq/L (range115.3 to 139 mEq/L); serum potassium, 3.8 mEq/L (range 2.9 to 4.4 mEq/L); serum carbondioxide, 21.2mEq/L (range 20.1 to24.4 mEq/L); serum blood urea nitrogen (BUN), 25.6 mg/dl (range 15.3 to 39.2 mg/dl); serum chloride, 93.5 mEq/L (range 92 to 102 mEq/L); measured serum osmolality, 301.7 mOsm/L (range 268.6 to 328 mOsm/L).

For statistical analysis,the correlation of total duration of epilepsia partialis continua and plasma glucose, serum sodium, serum potassium, serum carbondioxide, serum blood urea nitrogen (BUN), serum chloride and measured serum osmolality were analysed by Pearson moment correlation. There was no significant difference in the plasma glucose, serum chloride and serum potassium level to the duration of epilepsia partialis continua. The serum sodium, serum blood urea nitrogen (BUN) and serum osmolality level were significantly lower in patients with a longer duration of epilepsia partialis continua ($p < 0.05$) (Table 3). Ten patients had considerable evidence of a structural lesions on the appropriate site of the brain. In 9 patients, had focal area of decreased density on CT scan. All of the patients did not have residual neurololgical deficit after cessation of epilepsia partialis continua. Twelve patients had complications of microangiopathy from diabetes mellitus (Table 4).

Electroencephalograms were obtained during seizure in 4 patients. In 2 of these the record was so distorted by movement artifact that no reliable investigation was possible. Two others had focal discharge in the temporal region,and slow wave frequency. The clinical seizures were not always fully correlated with the electrical discharge. Seven patients had electro-

Table. 1 Patient characteristics

Patient characteristics	n = 22 (%)
Male	19 (86.4)
Female	3 (13.6)
Aged range (years)	33-65
Mean aged (years)	45.8

Table. 2 Clinical manifestation

Clinical manifestation	n = 22	Percentage
Part of the body		
- Face and arm	3	13.6
- Shoulder, arm and hand	10	45.5
- Hand only	8	36.4
- Leg only	1	4.5
Frequency of seizures		
- <10 jerks /min	16	72.7
- 10-20 jerks /min	6	27.3
Jerk sensitivity		
- Stimulus sensitive	7	31.8
- Movement sensitive	9	40.9
- Unknown	6	27.3
Duration of seizure		
- <7 days	13	59.1
- 8-30 days	9	40.9

Table. 3 Comparison of data in relation to total duration of seizures by Pearson's product moment correlation

Determination	r	p-value
Blood glucose (mg/dI)	0.066	0.771
Serum sodium (mEq/L)	-0.509	0.016
Serum chloride (mEq/L)	0.041	0.856
BUN (mg/dI)	-0.783	0.000
Serum osmolality (mOsm/L)	-0.536	0.010
Serum potassium (mEq/L)	0.053	0.650

Table. 4 Neuroimaging findings

CT scan	n = 22	Percentage
normal	10	45.5
Abnormal		
- cerebral infarction	9	40.9
- brain atrophy	3	13.6

encephalogram only after cessation of seizure activity. Of these, two showed a spike focus in the appropriate location,two had evidence of a slow-wave focus,while three were normal. No electroencephalogram was obtained in the remaining eleven patients (Table 5).

Table. 5 EEG findings

EEG finding	n = 9	Percentage
Interictal EEG		
-Focal spike and sharp waves	2	22.2
-Focal slowing	2	22.2
-normal	3	33.3
Ictal EEG		
-Spike and slow wave discharges	1	11.1
-Slow wave activity	1	11.1

Discussion

The syndrome of epilepsia partialis continua has been described in many causes[4,8,11,12,16]. In the literature the epilepsia partialis continua occurs in the early stage of nonketotic hyperglycemia, prior to the onset of coma, in a setting of moderate hyperglycemia, hyponatremia and mild hyperosmolality. The pathogenesis of epilepsia partialis continua in hyperglycemia remains uncertain but it is clear that most of these patients have a definite organic brain lesion in the appropriate location. The metabolic abnormalities most likely influence the epileptic threshold, enhancing the epileptogenic effect of the lesions. This hypothesis is supported by the finding of the present study. One of the patients, who presented with epilepsia partialis continua involved the left extremity. The CT scan showed an area of infarction in the right posterior temporal region. The patient recovered with correction of hyperglycemia and hyponatremia.

The role of serum hyperosmolality has been stressed as a cause of epilepsia partialis continua in hyperglycemia. Experimental and clinical observation by Vastola,Maccario,and Homan suggest that serum hyperosmolality and brain dehydration are a sufficient explanation for the occurrence of seizures, given the condition of a potentially epileptogenic process[13]. The majority of patients in the present study had only minimum hyperosmolality eventhough they had had epilepsia partialis continua for a few days prior to admission. Hyponatremia, ranging between 115.3 to 139 mEq/L, was a major abnormality in most of the 22 patients, especially in those with a prolonged duration of epilepsia partialis continua. Low serum sodium levels in the syndrome of nonketotic hyperglycemia have also been reported by others[7,8-10,14-17,19]. In experiments to differentiate between the roles of serum sodium and of hyperosmolality in producing focal neurological deficit in animals model of cerebral infarction as a result of ligation of middle cerebral artery, Espinas and Poser concluded that the neurological deficit is related to hyponatremia rather than to changes in osmolality[18]. Marked hyperglycemia in the presence of hyponatremia with normal serum osmolality was not accompanied by neurological dysfunction in non diabetic patients who developed hyperglycemia due to intravenous glucose infusion[14]. It appears that hyperglycemia and hyponatremia precipitate epilepsia partialis continua in patients with nonketotic hyperglycemia only when where is an associated structural brain lesion[14].

Brain glucose utilization is reduced in nonketotic hyperglycemia, an increased rate of GABA utilization via the GABA shunt may be one of the sources of energy requirements,thereby further lowering the GABA level and reducing the threshold for seizure activity[20]. It seems, that an acute cerebral infarct or an old infarct, a scar formation, is triggered to produce focal epileptic activity by the superimposed metabolic disturbances especially hyperglycemia and hyponatremia[19,20]. Practically all, patients who present with epilepsia partialis continua should have immediate determination of blood glucose levels. This suggestion may not only help in the diagnosis of previously unsuspected diabetes mellitus, but also to the appropriate management of the seizure disorder and prevention of further neurological complications.

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อาการชักกระตุกของร่างกายอย่างต่อเนื่องบางส่วนเป็นอาการนำของ ระดับน้ำตาลในเลือดสูง

ศุภชัย ไพบูลย์ผล

จากการศึกษาผู้ป่วย 22 ราย ซึ่งมีอาการชักกระตุกของร่างกายต่อเนื่องบางส่วนเป็นอาการนำของระดับน้ำตาลในเลือดสูง ซึ่งเกิดขึ้นในระยะแรกของเกลือโซเดียมในเลือดต่ำและความเข้มข้นสารละลายสูงเล็กน้อย อาการชักกระตุกของร่างกายอย่างต่อเนื่องบางส่วนมีอาการเฉลี่ยประมาณ 9 วัน ระยะเวลาที่มีอาการชักมีความสัมพันธ์กับระดับเกลือโซเดียมต่ำ สารไนโตรเจนในเลือดและความเข้มข้นสารละลายที่ต่ำกว่า ผู้ป่วยทุกรายที่มีอาการชักกระตุกของร่างกายอย่างต่อเนื่อง บางส่วนมีระดับน้ำตาลในเลือดสูง ไม่มีภาวะแทรกซ้อน และเสียชีวิต ในผู้ป่วยที่ได้รับการรักษา ผู้ป่วยส่วนใหญ่มีความผิดปกติของสมองตำแหน่งที่สัมพันธ์กับการชัก และระดับเกลือโซเดียมต่ำ ฉะนั้นผู้ป่วยทุกรายที่มาด้วยอาการชักกระตุกของร่างกายอย่างต่อเนื่องบางส่วน ควรตรวจหาระดับน้ำตาลในเลือด