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Case Report

Nonketotic hyperglycemia-related epileptic seizures ☆

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ABSTRACT

Nonketotic hyperglycemia-related seizures (NKH) are rare. We report a case of NKH-related seizures in a patient following a traumatic brain injury.

1. Introduction

Seizures related to head trauma are often encountered in clinical practice, and some of them combine with nonketotic hyperglycemia (NKH) and variable hyperosmolarity. However, seizures related to nonketotic hyperglycemia (NKH) are rare in clinical practice. Because epileptic seizures related to NKH significantly affect neurological outcomes [1] and may cause misdiagnosis or missed diagnosis, more attention should be paid to them. In this manuscript, we report a case of NKH-related seizures.

2. Case presentation

A 49-year-old male patient, without a known history of diabetes, was hospitalized because of a head trauma. Cranial CT scan showed mild subarachnoid hemorrhage without definite significance. On admission, his glucose and serum electrolyte levels were normal. Three days later, he developed partial seizures that began in his face. The average duration of the seizures in each episode was 4 min (range: 1–5 min, with one seizure lasting for 10 min) and could not be controlled by antiepileptic drugs. At the same time, his average plasma glucose level was 18.32 mmol/L (range: 15–24 mmol/L, normal range: 3.9–6.1 mmol/L), and his serum electrolyte levels were normal. When the seizures stopped, his average plasma glucose level decreased to 8.3 mmol/L (range: 4.7–11.2 mmol/L, normal range: 3.9–6.1 mmol/L). He had no family history of epilepsy and denied a history of spontaneous seizures or hyperglycemia. Under the guidance of an endocrinologist, the patient's hyperglycemia was treated with intravenous fluids and insulin and the seizures resolved. He was discharged 10 days later. He remained seizure- and antiepileptic drug-free after discharge as of his 3-month follow-up.

3. Discussion

Focal seizures induced by hyperglycemia were first reported in 1965 [2]. It was often encountered in clinical practice and was characterized by hyperglycemia, no keto-acidosis, seizures resistant to anticonvulsant treatment and seizure control associated with resolution of the hyperglycemia. Seizures related to NKH are usually partial seizures [1,2].

Nonketotic hyperglycemia-related seizures are refractory to the usual antiepileptic drugs and respond best to insulin and rehydration. These seizures can be controlled when glycemia is normal. In this case, seizures were resistant to antiepileptic drugs, but they resolved after appropriate fluid replacement and correction of the metabolic disorder by insulin therapy. Therefore, this case represents NKH-related seizures. Because the patient presented after a head injury, there was a possibility that the seizures could be misdiagnosed as post-traumatic, and therefore clinicians should consider a full differential diagnosis for seizures beginning soon after head trauma, especially when seizures do not respond to antiepileptic medications. In addition, an early diagnosis is also necessary to decrease the morbidity associated with nonketotic hyperglycemic coma.

Debates have continued about the mechanisms of seizures in NKH. Each mechanism, when considered alone, is unsatisfactory. The possible mechanisms are hyperglycemia or hyperosmolarity, a low level of gamma amino-butyric acid (GABA), and focal ischemia. Brick et al. [3] suggested that the Krebs cycle in NKH is inhibited, GABA metabolism is increased, and the levels decreased, thus lowering the threshold for seizure activity. Another hypothesis [4] involved the decrease of seizure threshold due to metabolic disturbance. Hyperosmolality and

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dehydration induced by hyperglycemia or hypo-sodium accompanying hyperglycemia were suggested to trigger focal seizures and lead to neurological deficit in some patients. Other authors suggested that a previously existing cortical lesion of an ischemic nature might lead to these seizures under altered metabolic conditions because hyperglycemia could result in reversible focal ischemia without structural damage by decreasing local blood flow in certain cerebral areas [5].

Because seizures may occur in association with NKH, early diagnosis and institution of the appropriate therapy are very important.

Conflict of interest

The authors did not receive any financial support to analyze these data and did not have any financial interest in any materials or devices described.

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Further reading

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