Acute Pancreatitis Case Presented with Epileptic Seizure

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Summary

Acute pancreatitis is defined as an acute non-bacterial inflammatory condition of the pancreas. A 53-year-old female was admitted to our emergency service after the first episode of a generalized tonic-clonic seizure. Her past medical history was unremarkable. The initial laboratory findings showed a low serum calcium level (5.8 mg/dL, normal value 8.8-10.2 mg/dL), high amylase—802 U/L and lipase—489 U/L levels. Abdominal computerized tomography (CT) showed pancreatic edema and inflammation suggestive of acute pancreatitis. This case report demonstrates a rare but important differential diagnosis for generalized tonic-clonic seizures of adult onset. (Turkish Journal of Neurology 2013; 19:114-5)

Key Words: Epileptic seizure, hypocalcemia, acute pancreatitis

Introduction

Acute pancreatitis is defined as an acute non-bacterial inflammatory condition of the pancreas. It begins with pancreatic injury, elicits an acute inflammatory response, encompasses a variety of complications and generally resolves over time. There are different known causes for this disorder, although the underlying mechanisms and how they develop the disease are still unknown. The clinical expressions of acute pancreatitis show extreme variability. This variability in presentation, clinical course and complications has given rise to some confusion related to acute pancreatitis (1). Here we report a case where a generalized tonic-clonic seizure as the initial clinical presentation of hypocalcemia co-occurred with acute pancreatitis.

Case

A 53-year-old female was admitted to our emergency service after the first episode of a generalized tonic-clonic seizure. The postictal drowsiness had already subsided on admission. The physical examination showed a right lateral tongue bite and enuresis, but no other systemic abnormalities. There was no neck stiffness. There was no past medical history of epilepsy or any other disorder or surgery, and no history of alcohol abuse. The initial laboratory findings showed low serum calcium level (5.8 mg/dL, normal value 8.8-10.2 mg/dL). Thyroxin and parathyroid hormone levels were normal. Cranial CT and magnetic resonance imaging (MRI) were both normal. Serum calcium level returned to normal after the treatment with calcium was started. After 5-6
hours, however, a second generalized tonic-clonic seizure occurred. The patient’s body temperature was 39°C at this time. Control calcium level was low again. C-reactive protein (CRP) level was higher than 150 mg/L. We suspected the presence of acute pancreatitis because of persistent hypocalcemia and an infectious condition. Control blood investigations revealed WBC-18,000 cells/cubic mm, (DC- N70%, L29%, E1%), ESR- 80 mm/hr, urea-47, creatinine-1.1 mg/dL, sodium-138 meq/l, potassium-4.5 meq/l, calcium-6.3 mg/dL, bilirubin-1.1 mg/dL, SGOT-35 U, SGPT-38 U, protein (T)-5.3 g/dL, Alb- 3.3 g/dL, amylase-802 U/L, lipase-489 U/L. Abdominal CT showed pancreatic edema and inflammation suggestive of acute pancreatitis.

**Discussion**

Acute pancreatitis has a variety of clinical manifestations and can easily go undetected if there is no specific suspicion. These manifestations can involve any organ system in the body. It can be easily confirmed by any abnormalities in serum amylase and serum lipase and an abdominal CT showing features of acute pancreatitis (1). Our case presented with generalized seizures as the first symptom of hypocalcemia co-occurring with acute pancreatitis.

Electrolyte disturbances are frequently encountered in daily clinical practice and they do not usually have a clinical significance. However, they may sometimes lead to serious complications when they are overlooked or not treated appropriately. Electrolyte abnormalities may affect many organs and tissues, including the brain. Most of the clinical manifestations of these disturbances are predominantly of neurological nature and they parallel the severity of neuronal damage. Furthermore, these disorders may appear with epileptic seizures (2,3). Our case was admitted with a severe epileptic seizure due to persistent hypocalcemia.

A successful treatment of patient seizures begins by establishing an accurate diagnosis of the underlying electrolyte disturbance, since the rapid identification and correction of the disturbance is crucial in controlling seizures and preventing permanent brain damage (3,4,5). Neurological symptoms of electrolyte disorders are generally functional rather than structural and the neurologic manifestations of electrolyte disturbances are typically reversible. However, since events like seizures can lead to structural alterations, it is important to treat the underlying disturbance before the pathology becomes permanent (3,4). In our patient we initially looked at the thyroid and parathyroid hormone levels but they both were normal.

The correct diagnosis of seizures secondary to electrolyte abnormalities begins with a complete serum chemistry evaluation, including measurements of electrolytes, especially sodium, calcium, and magnesium. These assessments should always be a part of the initial diagnostic workup in adult patients with a first-time seizure (6). Hypocalcemia is defined as a plasma calcium level of <8.5 mg/dL or an ionized calcium concentration <4.0 mg/dL. Acute pancreatitis is a rare cause of persistent hypocalcemia. The symptoms of hypocalcemia are influenced by the degree of hypocalcemia and the rapidity of the decrease in the serum ionized calcium concentration (2). The initial laboratory findings of our case showed low serum calcium level such as 5.8 mg/dL.

Seizures may occur without muscular tetany in patients with hypocalcemia. Seizures occur in 20-25% of the patients with acute hypocalcemia as a medical emergency. Generalized tonic–clonic, focal motor, and (less frequently) atypical absence or akinetic seizures can occur in hypocalcemia and may be the sole presenting symptom (3). In most cases of electrolyte imbalance, treatment with an anticonvulsant is not necessary as long as the underlying disturbance is rectified. Long-term administration of anticonvulsant is not necessary (3,4,7). Indeed, anticonvulsants alone are generally ineffective if the electrolyte disorder persists. We did not give any anticonvulsant initially but we administered intravenous diazepam in the second seizure. Treatment for hypocalcemic seizures is calcium replacement; anticonvulsants are typically not needed (2).

Treatment with intravenous calcium is the most appropriate therapy. Doses of 100 to 300 mg of elemental calcium should be infused (intravenous) over a period of 10 to 20 min. Calcium-infusion drips should be started at 0.5 mg/kg/h and continued for several hours, with close monitoring of calcium levels (2). Treatment with calcium in our patient was initiated and resulted in normalized serum calcium level. But 5–6 hours later a second generalized tonic–clonic seizure occurred with fever. Control calcium level was low again. We suspected to the acute pancreatitis because of persistent hypocalcemia and an infectious condition.

In conclusion, acute pancreatitis is important for the differential diagnosis in adult patients presenting with a first-time generalized tonic-clonic seizure. This underlines the necessity of taking a careful medical history and measuring the electrolytes in patients admitted with first-time epileptic seizures.

**References**