The pathophysiology of any seizure disorder includes alteration of the membrane channel and selectively included neurotransmitters. In addition, epileptogenesis of focal epilepsy has been shown to involve the selective loss of neurons and axonal reorganization.\(^1\) Focal seizures can be either motor, jacksonian, aphasis, aversive, or of the epilepsia partialis continua type.\(^2\) Different types of focal seizures may appear in the same patient. Simple partial seizures result when the ictal discharge focuses in a circumscribed area of the cortex. This type of seizure is often referred to as epileptogenic focus. Most often, patients remain fully conscious, interacting normally with their environment, except for limitations imposed by the seizure on the specific localized brain functions. Patient history is very important in focal seizures. The initial events of a seizure are the most reliable indicator in determining whether a seizure is focal. Simple partial seizures with motor signs symptoms occur with clonic (rhythmic jerking) or tonic (stiffening) movements of a discrete body part. Because of their large cortical representation, hand and face muscles are the primary structures most often involved.\(^3\)

Focal seizures can occur as an early symptom of nonketotic hyperglycemia or during an initial phase of hyponatremia and mild hyperosmolality. Seizures are common in hyperglycemia and often are the first manifestation, particularly in nonketotic hyperglycemia. Severe partial seizures may be the presenting feature of nonketotic hyperglycemia in older adults, but cases in children are rare.\(^4\) Nonketotic hyperglycemia has a definite convulsive effect, which may manifest itself in a generalized or focal manner. Focal epileptic seizures are linked with moderate nonketotic hyperglycemia with values of greater than 360 mg/dL and osmolality values of greater than 320 mOsm/kg. Higher values may lead to generalized seizures and even sometimes coma.\(^2\) Movement disorders are well known presenting signs of metabolic disorders. The case reported in this article deals specifically with focal convulsive phenomena. Focal motor abnormalities are the chief initial presentation of diabetes mellitus in the nonketotic hyperglycemic state in 0% of patients.\(^5\)

Recognition of the link between focal epilepsy and nonketotic hyperglycemia aids in the early diagnosis and treatment of this serious metabolic distur-

**Clonic Focal Seizure of the Foot Secondary to Nonketotic Hyperglycemia**

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Focal epileptic seizures can be the first manifestation of a diabetic disorder. Metabolic disturbances, including hyperglycemia, mild hyperosmolality, hyponatremia, and lack of ketoacidosis contribute to the development of partial focal seizures. A review of the medical literature for partial focal seizures is presented, followed by a case study of a patient who developed clonic seizures of the right foot secondary to hyperglycemia, hyponatremia, and hyperosmolality. (J Am Podiatr Med Assoc 92(2): 109-111, 2002)
bance. Seizures associated with hyperglycemia are often resistant to anticonvulsant treatment and respond best to insulin and rehydration. Focal seizures in adults may indicate diabetes mellitus. The therapy of choice is prompt, rapid administration of crystalloid solutions with a tonicity appropriate to the level of hyperosmolarity. The goal is for the plasma glucose level to decline within a range of 75 to 100 mg/dL/hr (4.2 to 5.6 mM/liter) with as much as 4 to 6 liters of fluid required in the first 8 to 10 hours. If hypovolemia is present, fluid therapy should be initiated with isotonic saline. In all other cases, hypotonic (0.45%) saline appears to be preferable as the initial replacement solution, switching to 5% dextrose in either water or 0.45% saline solution, or 0.9% saline solution once blood glucose reaches 250 mg/dL. The “more rapid the better” administration of solution to achieve quick consummation of the acute condition poses its own risks as well. As such, the rate of dextrose infusion should be carefully and attentively monitored to maintain glycemic levels of 250 to 300 mg/dL/hr and severe electrolyte imbalances. Patients with hyperosmolar hyperglycemic syndrome are often chronically ill, and they may have major total body deficits of potassium, phosphate, magnesium, as well as B-complex vitamins (especially thiamine). These deficits also require immediate attention and correction during therapy.

Case Report

A 74-year-old man presented to the emergency room at St Vincent Hospital in Worcester, Massachusetts with a chief complaint of abnormal foot movements occurring three separate times during the night prior to admission. The patient stated that the abnormal movements occurred from the ankle joint distal and unilaterally on the right foot in a rhythmic shaking motion. These episodes occurred in the middle of the night while the patient was asleep and he was awakened on all three occasions. Each episode lasted approximately 10 seconds. During each episode the patient tried to relieve the abnormal activity by standing up on both feet, but this was followed each time by a fall secondary to the abnormal shaking and loss of muscle tone in the right foot. After the third episode, the patient was transported to the emergency room.

The patient’s medications were as follows: Prilocsec (AstraZeneca LP, Wayne, Pennsylvania) 20 mg qd, Procardia XL (Pfizer Inc, New York, New York) 60 mg qd, ASA 325 mg qd, trazadone 50 mg qh, and Isordil (Wyeth-Ayerst Laboratories, Philadelphia, Pennsylvania) 40 mg tid. The patient had no known allergies and his past medical history was significant for a cardiac catheterization 8 years earlier and a coronary artery bypass 18 years prior to admission. The patient’s social history revealed that he was a widower of 1 year. The patient lived by himself and had an exceptionally poor diet consisting mainly of foods high in sugar. He stated that he has smoked one pack of cigarettes daily for the last 50 years, and he admitted to at least one alcoholic drink per evening. His family history was significant for a grandfather with diabetes mellitus and a sister who died of a cancer of an unknown etiology.

Physical examination revealed the following findings: the patient’s blood pressure was 156/83 mm Hg, pulse 64, respiration rate 20, and temperature of 98.1°. General findings of the patient revealed an elderly male who appeared alert, awake, oriented, well nourished, and in no acute distress. The patient’s neurologic findings revealed no focal deficits and cranial nerves I to XII were intact. A motor examination found 5/5 muscle strength in all four extremities. Examination revealed a slight deficit to pinprick in both lower extremities in a stocking glove distribution. The patient’s cerebellar exam was negative. Proprioception and vibratory sensation were intact in all four extremities. Deep tendon reflexes were bilaterally equal and plantars were flexor bilaterally. Cardiac examination revealed a regular rate and rhythm with no murmurs, rubs, or gallops. The patient’s lungs were clear to auscultation and his abdominal examination was unremarkable. Extremities revealed no cyanosis, clubbing, or edema.

Upon arrival at the emergency room, the patient’s blood sugar was 720 mg/dL. The patient also was found to be hyponatremia with a value of 125 mEq/L, and hyperosmolar with a value of 337 mOsm/kg. All other chemistries and electrolytes were within normal limits. A computed tomography of the head scan did not show correlative abnormalities. A neurologic consultation was obtained in the emergency room. The neurologist elicited a clonic movement of the right foot volleying 9 to 10 times over the course of 7 to 10 seconds, starting at a fast regular rhythm and progressively slowing. The neurologist’s finding was a partial seizure involving the right foot secondary to nonketotic hyperglycemia.

Treatment was initiated with 10 units of insulin subcutaneous and normal saline infusion at 100 mL/hr. The patient was admitted to the hospital for continued hydration and insulin treatments as needed. Over the following 12 hours, the patient’s blood sugar fell to 180 mg/dL. During his admission to the hospital, the patient did not have any further episodes...
of seizure activity. Hemoglobin A1C levels were drawn and found to be elevated at 12.4%. The patient was subsequently sent home on a diabetic diet regimen and started on Glucophage (Bristol-Myers Squibb Co, Princeton, New Jersey) 500 mg bid.

Discussion

The case report presented in this article had a focal motor seizure of the foot as the first manifestation of nonketotic hyperglycemia of diabetes mellitus. The patient also presented with metabolic disturbances, including hyperglycemia, mild hyperosmolality, hyponatremia, and lack of ketoacidosis. These presenting symptoms matched the literature definition of nonketotic hyperglycemia. The treatment of prompt rehydration and adequate glucose proved to be effective.

Conclusion

Seizures are a relatively common symptom of brain dysfunction and may occur during the course of many acute medical or neurologic illnesses in which brain function is temporarily impaired. Such seizures are most often self-limited and do not persist after the underlying disorder has been resolved. The seizure presented here was characterized by stereotypic tonic changes associated with supplementary motor area seizures. Recognition of the link between this form of focal epilepsy and nonketotic hyperglycemia would help in the early diagnosis and treatment of the serious underlying metabolic disturbance. As such, patients presenting with nonketotic hyperglycemia and movement disorders may not require expensive diagnostic evaluation. However, it is always advisable to consult other appropriate medical specialties concerning systemic matters such as these.

References