CASE STUDY

The Lethargic Diabetic: Cerebral Edema in Pediatric Patients in Diabetic Ketoacidosis

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Abstract

Diabetic ketoacidosis (DKA) is the leading cause of hospitalizations for pediatric patients with diabetes mellitus. The most severe complication of DKA is cerebral edema that may lead to brain herniation. We present a case report that highlights the subclinical presentation of DKA-related cerebral edema in a pediatric patient and review the acute care management of suspected cerebral edema during transport.

Diabetes mellitus is a health care problem that has been coined an "epidemic." The estimated incidence of diabetes in the United States is 24.3 per 100,000 children per year; this approximates to 15,000 children being newly diagnosed annually. The observation that children younger than 12 months of age are being diagnosed with new-onset diabetes is also alarming because diabetes is a chronic disease into adulthood and currently is the seventh leading cause of death in the US.[1] There are 2 distinct classifications of diabetes. Type 1 diabetes describes those patients who are inherently insulin deficient and must rely on lipolysis for fuel needs during times of stress. Most patients with type 1 diabetes present during childhood. Type 2 diabetes is characterized by variable degrees of peripheral insulin resistance, but these patients have inadequate cellular glucose uptake during times of stress. Patients with type 2 diabetes classically present later in life although the problem of youth obesity has been associated with earlier diagnoses during adolescence.

The most serious complication of diabetes is diabetic ketoacidosis (DKA). In children with diabetes, DKA is the leading cause of hospitalizations, morbidity, and mortality.[2,3] A single episode of DKA can place a pediatric patient at risk for developing cerebral edema with subsequent brain herniation. The occurrence of cerebral edema is rare, approximately 0.5% to 1% of all pediatric DKA cases. However, there is an estimated 40% to 90% mortality from DKA-related cerebral edema.[2-6] Risk factors for cerebral edema include first presentation, younger age, aggressive fluid administration, administration of sodium bicarbonate or bolus insulin doses, and precipitous drops in blood glucose (> 100 mg/dL/h). Additional metabolic abnormalities at presentation, namely an elevated blood urea nitrogen and low partial pressure of arterial CO_2, are also considered to be risk factors.[7,8] Medical management can be lifesaving when initiated at the time of presentation and during transport.

We present a case of a 4-year-old patient with previously diagnosed type 1 diabetes who presented at a local emergency department (ED) with severe DKA. The management of this child's acidosis was complicated by the clinical presentation of cerebral edema, which was later confirmed by computed tomographic imaging of the brain upon arrival at our institution. The goal is to encourage a high index of suspicion for the presence of cerebral edema and to provide clinicians a review of the management strategies for cerebral edema during the transport process.

Case

A 4-year-old male with a past medical history of type 1 diabetes presented to a community hospital with a 1- to 2-day history of nausea and vomiting. The mother brought the child in to the local ED for difficulty breathing and decreased responsiveness. Initial laboratory work revealed severe DKA with a pH of 6.94, glucose of 620 mg/dL, blood urea nitrogen of 15 mg/dL, creatinine of 0.99 mg/dL, and bicarbonate of 6 mmol/L. The child was documented to have been "lethargic" since presentation. After 40 mL/kg of fluid resuscitation, an insulin drip was started at 0.1 U/kg/h and transport was arranged via helicopter to our tertiary care children's hospital. No bolus doses of insulin or bicarbonate were given before transport. Within 30 minutes from the time of the call, the pediatric critical care transport team arrived. Upon initial assessment, the transport team noted the following:

1. Cardiovascular: heart rate 151 beats/min, sinus; peripherally cool with capillary refill > 2 seconds
2. Chest: accessory muscle use on inspiration, respiratory rate of 36; saturations 100% on room air
3. Neurologic: lethargic; pupils 3 mm, equal and reactive; and Glasgow Coma Scale (GCS) of 10 (E2/V4/M4)

Just before departing, his blood glucoses were noted to have decreased from 620 mg/dL to 453 mg/dL while on the

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1067-991X/$36.00
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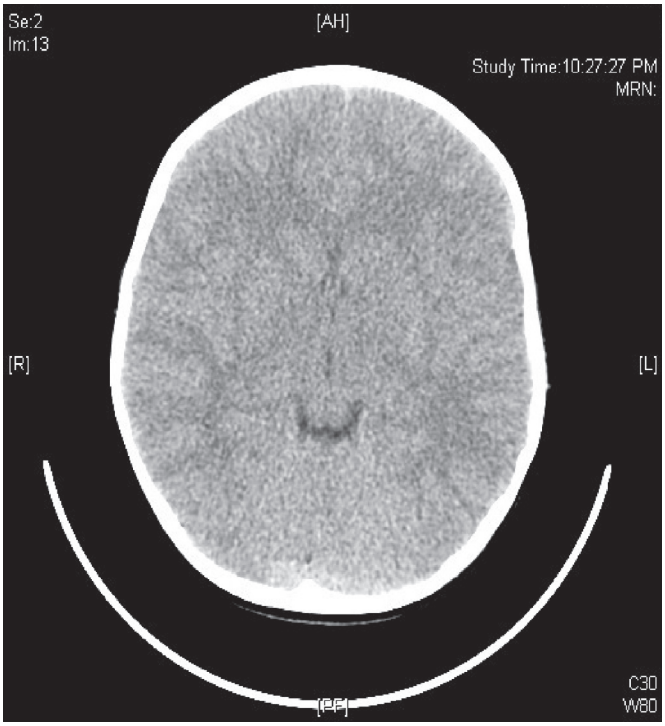
http://dx.doi.org/10.1016/j.amj.2014.10.009

Figure 1. Abnormal head computed tomographic image in our patient showing diffuse hypodensity or "graininess." There is also narrowing of the lateral and third ventricles. The cerebral sulci are poorly visualized, and there is loss of differentiation of the deep gray matter and adjacent white matter. All of these findings are suggestive of cerebral edema.



insulin drip for 30 minutes. After conferring with the ED medical control physician, the transport team decreased the patient's insulin drip to 0.05 U/kg/h. The patient was maintained on 0.45 normal saline with 20 mEq/L KCl at a maintenance intravenous fluid rate, also per medical control. En route, the patient was sleepy but had documented GCS scores of 10 and 15. The team noted intervals of "awake and crying, able to be consoled" to "sleeping, arouses easily to touch." His vitals were as follows: heart rate of 144 to 183 beats/min, respiratory rate of 36 to 44, and blood pressure of 147 to 163/66 to 78 mm Hg.

Upon arrival, the emergency medicine fellow and attending documented a GCS of 11 (not specified) and detailed their examination notes with "responsive only to pain, not interactive, not talking" and "somnolent, Kussmaul breathing, responds to painful stimuli and intermittently cries but not awake or talking." Vitals in the ED were similar to those noted during transport (heart rate = 138 beats/min, respiratory rate = 44, and blood pressure = 157/83 mm Hg). Given this worrisome clinical picture, a head computed tomographic scan was obtained and confirmed the presence of global cerebral edema (Fig. 1). A dose of 0.5 g/kg intravenous mannitol was given without improvement on the neurologic examination. The patient was then admitted to the pediatric intensive care unit (PICU) for further management.

In the PICU, the patient continued to display a severely depressed mental status. He promptly became somnolent without verbal or tactile stimulation. Although he readily withdrew from painful stimulation, he would not consistently follow simple commands. Given the concern for worsening cerebral edema, hyperosmolar fluid therapy was then initiated with a bolus dose of 5 mL/kg intravenous 3% NaCl, and intravenous fluid replacement was given slowly at 1.5 times maintenance over the next few hours. Serum sodium levels were maintained above 140 mmol/L. Hourly neurologic checks were performed. During the first 12 hours of his admission, the patient's acid/base status was also carefully corrected with an insulin drip, taking care not to decrease his blood glucose more than 50 mg/dL/h. Gradually, the patient's examinations improved. At the 24-hour mark of his PICU stay, the patient was age appropriate with a GCS of 15. At the time of transfer to the regular pediatric floor, he was back to baseline. No neurologic deficits were appreciated upon discharge to home.

Discussion

DKA is a serious event that may result in significant morbidity and mortality. Although cerebral edema is rare, it is reported to be 40% to 90% fatal if it presents.[2,4,6] Although the complications of cerebral edema are well-known, the presence of cerebral edema may be underappreciated in the transport setting and as evidenced by the transport team documenting GCS scores of 10 (E2/V4/M4) initially and 15 during transport. This scenario is complicated by the fact that pediatric patients are often resistant and uncooperative during full neurologic examinations. Moreover, in the transport scenario, pediatric patients are commonly anxious and agitated by the unfamiliar surroundings. A young patient's unwillingness to cooperate may, in reality, be early signs of the mental status changes related to cerebral edema and dehydration. Relying on the Cushing triad (hypertension, bradycardia, and abnormal breathing) to clue one in on the presence of increased intracranial pressure is a late finding and signals impeding brain herniation. Thus, in order to transport these patients safely, one must maintain a high index of suspicion and manage these patients accordingly.

From a physiologic viewpoint, the exact mechanism of cerebral edema remains uncertain. The prevailing theory is that the brain works to protect itself under circumstances of extreme dehydration. To avoid cell death, brain cells create intracellular "idiogenic osmoles" in response to dehydration. Such "osmoles" are solutes such as taurine, glycine, glutamine, sorbitol, and inositol. This compensatory mechanism allows for a favorable osmotic gradient to be created, causing water to move from the plasma and into the cells, including neurons, in order to maintain homeostasis. However, aggressive fluid resuscitation and/or the administration of hypotonic fluids (eg, 0.45 normal saline) may inadvertently cause acute cerebral swelling because fluids will continue to follow the osmotic gradient into cells. Because of this potential, one

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