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LETTER TO EDITOR (VIEWERS CHOICE)

NEVER FORGET GLUCOSE - A CASE OF IDIOPATHIC KETOTIC HYPOGLYCEMIA

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Idiopathic ketotic hypoglycemia (IKH) is the most common cause of hypoglycemia in non-diabetic children over 6 months.[1,2] It usually presents between 18 months and 5 years and remits spontaneously by 8 to 9 years.[3] IKH is defined by periodic episodes of hypoglycemia and ketonuria, in previously healthy children, after prolonged fasting.[3,4] Common presentation is drowsiness, loss of consciousness, seizures, fatigue and altered sensorium, most frequently in the morning, during periods of intercurrent illness, poor food intake, vomiting and diarrhea.[2,3]

A 31-month-old, previously healthy, female patient presented to the emergency room with several episodes of loss of consciousness lasting 20 seconds, that had begun the morning of admission. The patient also had fever, decreased appetite for 24 hours and two episodes of vomiting. Physical examination revealed a well-appearing child, Glasgow Coma Score 15, temperature 37ºC, normal vital signs, with no significant findings.

We considered the diagnostic hypothesis of a central nervous system (CNS) infection, performed a lumbar puncture and initiated ceftriaxone and acyclovir. The blood workup revealed reduced glucose levels (45 mg/dL). The cerebrospinal fluid was clear, glucose 30 mg/dL, without other alterations. After the lumbar puncture, we measured capillary glucose which was normal (80 mg/dL). After consultation with a Pediatric Neurologist, a cranial CT-scan was performed, that showed no alterations.

After excluding a serious CNS disease, we assumed that hypoglycemia was the cause of events. Capillary glucose was probably falsely elevated due to stress, after a difficult lumbar puncture. Urine ketone bodies were positive (+++/++++), ketonemia 3,6 mg/dL, and venous gasometry was normal. Consequently, we suspected ketotic hypoglycemia to be the causative agent. Considering that the patient displayed typical clinical presentation of IKH, was previously healthy, attained all milestones appropriate for age and had normal physical examination, we assumed the diagnosis of IKH. We suspended acyclovir and ceftriaxone, and initiated intravenous glucose with isotonic saline. The patient had no more episodes of loss of consciousness or hypoglycemia. Blood culture and microbiological/viral analysis of the cerebrospinal fluid were negative. At the third day of hospitalization the patient was asymptomatic and was discharged.

IKH's pathophysiology is not completely understood.[4] Studies indicate that inadequate hepatic glucose production, decreased capacity for hepatic gluconeogenesis and inadequate supply of ketogenic aminoacids may be implicated.[3,4] Classically, the diagnosis is based on the identification of Whipples's triad, typical clinical presentation and exclusion of other causes of ketotic hypoglycemia.[4,5] However, recent studies concluded that children with typical presentation of IKH, who are healthy, with appropriate growth and development and normal physical examination, do not need to perform work-up for endocrinopathy or inborn error.[1,4,6] Treatment consists in preventing hypoglycemia by frequent feedings of high protein, high carbohydrate diet and avoiding fasting for more than 10 hours, especially during intercurrent illness.[4] IKH can easily be mistaken for other diseases and a high index of suspicion is necessary to identify this entity.[3]

This case reminds clinicians to consider this entity as a possible cause of loss of consciousness and highlights the importance of early measurement of blood glucose in order to avoid unnecessary workup and medication.

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