DOI: 10.4274/Tjem.2173

Hyperosmolar Hyperglycemic State (HHS) in Newly Diagnosed Child with Type 1 Diabetes Mellitus: A Case Report

Yeni Tanı Tip 1 Diyabetik Çocukta Hiperosmolar Hiperglisemik Durum (HHD): Vaka Raporu

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Abstract

Hyperglycemic hyperosmolar syndrome (HHS), which is characterized by marked elevations in serum glucose concentrations and hyperosmolality without significant ketosis, has been rarely reported in children. However, the incidence of this disorder in children is increasing with the rise of frequently reported cases describing HHS in children. We report a 7-year-old male patient who presented with HHS with a new diagnosis of type 1 diabetes mellitus complicated by hypernatremic dehydration. The patient had a good outcome, discharged home within 7 days. *Turk Jem 2013; 17: 19-21*

Key words: Hyperosmolar,non-ketotic,diabetes

Özet

Belirgin ketozis olmaksızın ciddi serum glukoz yüksekliği, ve hiperosmolalite ile karakterize hiperglisemik hiperosmolar sendrom çocuklarda nadiren bildirilmektedir. Bununla birlikte son dönemde HHD tarif edilen vaka raporları ve serilerindeki artış çocuklarda görülen bu durumun görülme sıklığında artış olduğunu düşündürmektedir. Burada HHD ile başvuran hipernatremik dehidratasyonla komplike olmuş 7 yaşında erkek hasta sunmaktayız. Hasta 7 gün içinde iyileşti ve taburcu edildi. *Türk Jem 2013; 17: 19-21*

Anahtar kelimeler: Hiperosmolar, nonketotik, diyabet

Introduction

Hyperglycemic hyperosmolar syndrome (HHS), a rare pediatric complication of diabetes mellitus (DM), is characterized by hyperglycemia more than 600 mg/dL), hyperosmolality more than330 mOsm/L), and a mild metabolic acidosis (pH>7.2) (7.35-7.45) (1). There is a difference between HHS and diabetic ketoacidosis (DKA) where DKA presents with significant metabolic acidosis, ketosis and lower levels of hyperglycemia, although these two illnesses are acute complications of diabetes mellitus (DM)2). HHS is classically occurs in patients with type 2 DM, but there are increased reports in type 1 DM (T1DM) (3,4). DKA has a mortality rates below 1% in pediatrics with good prognosis (5).

Recent reports suggest high mortality rates in children with HHS (~10-35%) (6). The most serious complication of both HHS and diabetic ketoacidosis are cerebral edema and acute respiratory distress syndrome (7).

We present the case of a young child with newly diagnosed HHS and review of the literature.

Case Presentation

A seven-year-old Yemeni male patient presented to the emergency room with polyuria, polydepsia, weight loss, nocturnal enuresis and decreased activity for 7 days duration and history of viral infection 4 weeks prior to the presentation. There was no family history of T1DM or type 2 DM (T2DM).

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E-mail: ihabhalaby2002@hotmail.com **Recevied/Geliş Tarihi:** 01.11.2012 **Accepted/Kabul Tarihi:** 17.01.2013

The patient had a Glasgow Coma Scale score of 13 out of 15 on presentation, heart rate of 150 beats per minute, respiratory rate of 30 breaths per minute, and temperature of 36.5 C. Blood pressure was 105/85 mmHg. Both weight and height were below 5th percentile. BMI was 10 kg/m^2. Clinically, he was severely dehydrated with no acanthosis nigricans.

Initial laboratory evaluation revealed diagnosis of HHS (Table 1).

The child has received initially a bolus of 10 ml/kg of normal saline intravenously in the first hour, followed by management of 10% rehydration with deficit correction over 72 hours. He was started on continuous regular insulin drip at 0.1 U/kg/hour.

During hydration process, his sodium increased gradually up to 173 mmol/l with no change in mental status. Serum sodium normalized to a normal value of 142 mmol/l over 3 days. All other laboratory values continued to improve throughout hospitalization, including serum blood urea nitrogen, and creatinine and urine output continued to be average and a creatinie kinase was normal post-presentation. He was then shifted to subcutaneous insulin of 0.7 unit/kg/day with acceptable blood glucose readings, and discharged home.

Discussion

Hyperglycemic hyperosmolar syndrome (HHS) is one of the most severe acute complications of T2DM, but may also occur in T1DM. Similar to ketoacidosis, HHS still remains one of the major causes of morbidity and mortality in patients with DM. Children with HHS share common features, the majority of children are obese males of African-American ethnicity (2). Most of children had a family history of T2DM and had acanthosis nigricans on physical examination. The majority of children were subsequently diagnosed with T2DM which explains the increasing number of HHS reports in the pediatric literature may be due to the rising prevalence of obesity and T2DM in children, especially among ethnic minorities. HHS, may occur in children with T1DM and there is no difference in pathophysiology between children with T1DM and those with T2DM (2).

Table 1. Laboratory results of the patient

TEST	RESULT	NORMAL
Corrected sodium	152 mMol/L	136-145
Potassium	4.2 mMol/L	3.5 – 5.1
Chloride	93 mMol/L	98 – 107
Phosphorus	1.98 mMol/L	0.81- 1.
Serum glucose	72mMol/L	4 - 6.7
Serum osmolality	412 mOsm/kg	282-295
Blood Urea Nitrogen	22.3 mMol/L	2.5 – 6.4
Creatinine	210 umol/L	53 – 115
C- Peptide	0.318 nmol/L	0.37 – 1.47
HbA1c	15 %	4.8-6 %
PH	7.210	7.35-7.45
Co2	50.6 mmHg	35-45
Hco3	19.8 mMol/L	21-26
Hematocrit	49 %	34-48
Urinalysis showed 3+ glucose with no ketones		

Our patient presented with HHS as a complication of newly diagnosed T1DM. The diagnosis of T1DM was made based on the classical history of DM, thin body built, absence of acanthosis nigricans with the presence of pancreatic auto-antibodies. His presentation was similar to previously reported cases of children with HHS (8-10). Infections (pneumonia caused by gram negative bacteria, urinary tract infection and sepsis), medications, poorcompliance, undiagnosed DM, substance abuse, and coexisting diseases are precipitating factors for HHS (11,12). Infections are the leading cause of HHS (14). The most common infection is.

In HHS, insulin action may be inadequate to facilitate glucose utilization by insulin-sensitive tissues but sufficient for the prevention of lipolysis and ketogenesis associated with prolonged and gradually increasing polyuria and polydipsia, resulting in profound dehydration (7). Severe electrolyte loss occurs due to prolonged duration of osmotic diuresis. The hypertonicity of the hyperosmolar state preserves intravascular volume, which masks the clinical signs of dehydration. With hydration, serum osmolality decrases which results in an osmotic gradient leads to water move from the intravascular to the intracellular space, decreasing intravascular volume. Massive osmotic diuresis in the early hours of therapy needs agresive fluid management to prevent shock and avoid the complications of vascular collapse (lactic acidosis, rhabdomyolysis, renal failure, thrombosis) (4,7). Persistent hypernatremia, hypokalemia, hypophosphatemia, rhabdomyolysis, pancreatitis, and thrombosis must be watched for. These complications did not occur in our patient apart from hypernatremia. Fluid resuscitation is best accomplished initially with isotonic fluid (0.9% saline), which will be hypotonic for these patients, followed by 0.45% to 0.75% saline.

Management of HHS in paediatrics is the same for DKA, exogenous insulin infusion in a rate of 0.1 U/kg/hour is recommended. Dextrose is added to intravenous fl uids when serum glucose drops to 300 mg/dl to avoid overly precipitous drops in glucose levels. Upon adequate control of serum gucose, acidosis, hyperosmolality and electrolyte disturbance, insulin administration should be switched to subcutaneous regimen.

We report a case of pediatric HHS with a good outcome. Our patient had acute kidney injury, as well as a high serum glucose level complicated by hypernatremia He responded well to fluid resuscitation and gentle insulin administration. Prospective trials HHS in pediatrics need to be performed so that better management strategies, especially fluid replacement. Although HHS is a rare presentation in the pediatric population, clinicians should be aware of the manifestations of HHS and the differences in management from DKA.

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