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CASE REPORT

Hyperglycemic Crisis: A Serious Complication of Unknown Type II Diabetes Mellitus in Adolescent

Elisa, MD[1*] and Pratiwi Woro R, MD PhD[2]

[1]*Pediatric Intensive Care Unit, Hermina Yogya Hospital, Indonesia*

[2]*Internal Medicine Unit, Hermina Yogya Hospital, Indonesia*

***Corresponding author:** *Elisa, MD, Pediatric Intensive Care Unit, Hermina Yogya Hospital, Jln. Selokan Mataram, Meguwo, Maguwoharjo, Yogyakarta, Indonesia, Tel: +62-274-2800808, Fax: +62-274-2800909, E-mail: elisadewo@yahoo.com*

Abstract

Hyperglycemic crisis is life threatening condition in diabetic patients. Traditionally, diabetic ketoacidosis (DKA) has been associated with decompensated type 1 diabetes (T1D) and Hyperosmolar Hyperglycemic State (HHS) became a hallmark of type 2 diabetes (T2D). However, DKA is increasingly being reported in patients with T2D. We reported a case of DKA with some features of HHS, as an initial presentation, in a boy with unknown T2D at young age. Prompt diagnosis and acute critical care approach were very challenging to improve the outcome.

Keywords

Hyperglycemic crisis, Adolescent, Type 2 diabetes melitus

Introduction

Hyperglycemic crisis is an extreme manifestation of decompensated diabetes mellitus, which includes 2 spectrums of clinical states namely Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycemic State (HHS). These 2 manifestations can occur in patients with both type 1 (T1D) as well as type 2 (T2D) diabetes mellitus [1]. DKA and HHS remain to be important causes of morbidity and mortality among diabetic patients. Despite definite differences between their diagnostic criteria, DKA and HHS may still be difficult to be established as separate entities due to their similar characteristic features. Presentations with overlapping DKA and HHS account for 27% of emergency room visits of hyperglycemic crisis cases [1]. Hyperglycemia and insulinopenia, absolute or relative, are the key elements in the pathogenesis of such conditions. Absolute insulinopenia in DKA causes severe alteration of carbohydrate, protein, and lipid metabolism, that leads to major catabolic state as well as production of glucose and ketone bodies by the liver [2]. On the other hand, the presence of sufficient insulin (relative insulinopenia) in HHS prevents lipolysis and ketogenesis. Thus, DKA is characterized by Hyperglycaemia and ketoacidosis, whereas HHS is marked by hyperglycemia, hyperosmolality, with no evidence of ketosis or acidosis [2].

Here, we report a case of DKA with some clinical evidence of HHS as the first presentation of T2D in an adolescent boy who later suffered advanced complication of renal failure.

The Case

A 13-year-old obese (body weight: 74.85 Kg, height: 157.5 cm, BMI > 95th percentile) boy came to the Emergency room because of frequent vomiting and shortness of breath on the preceding night. The patient had frequent micturition, excessive drinking of sweetened beverages and eating within 1 week prior to the admission. Some weight loss and leg ulcers were noticed by his family member. Further interview revealed that his mother died from diabetic related complication and his father died from liver cirrhosis. The patient lives with extended family and mostly cared by his only elder sister. Physical examination in the Emergency room showed that the patient was in decrease of consciousness with Glasgow Coma Scale (GCS) of 11/15 and very dehydrated. He was also found with body temperature of 38 °C, normal blood pressure (100/70 mmHg), tachycardia (127 X/min), Kussmaull

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type of breathing (38 X/min), warm extremities with normal capillary refill time, and oxygen saturation of 98% in room air. His glucose level was too high to be detected with capillary glucometer.

Aggressive intravenous fluid replacement was done with 20 ml/kg of normal saline for the first hour and continued with maintenance rate with half amount of normal saline for the next 23 hours. Inferior Vena Cava (IVC) collapsibility Index and urine output were used as guidance to determine the achievement of fluid replacement therapy. Patient was then admitted to the Pediatric Intensive Care Unit (PICU).

Initial blood tests results demonstrated severe hyperglycemia (1,105 mg/dL), effective hyperosmolality (332.98 mMol/kg), uncompensated metabolic acidosis (pH 6.98) with high anion gap (38.5 mMol/L), Sodium corrected concentration for blood glucose of 151.88 mmol/L, hyperkalemia (6.08 mmol/L), severe glycosuria and moderate ketonuria (Table 1). Blood leucocytes count was high (37.300/uL) with neutrophil predominant.

Based on the laboratory results, the following treatments were immediately administered, (i) Regular insulin 0.1 IU/kg/hour to attain glucose reduction rate of 50-70 mg/dL, (ii) Intravenous antibiotic (ceftriaxone 25 mg/kg), and (iii) Sodium bicarbonate to normalise blood pH.

Despite the aggressive treatment and a good trend of blood glucose, electrolytes, and blood pH, the patient, however, became very restless, combative and developed acute renal failure on the second day of admission marked with oliguria, pulmonary congestion, dependent edema, high level of blood ureum (134.30 mg/dL) and creatinin (5.91 mg/dL). Patient was then sedated and intubated. Due to intolerable vital signs, the patient was subjected to Sustained Low-Efficiency Dialysis (SLED).

After 3 times of temporary haemodialysis, the patient's renal function resumed. Blood glucose, electrolyte, and pH were totally under controlled (Table 1) on the 7th day in the PICU, as he regained his consciousness. He was extubated on day 10 of admission and was on full oral diet. Insulin therapy was transformed to split rapid acting insulin.

After a 16-day hospital stay that included educations on self-monitoring blood glucose, restricted diet, physical exercise, wound care, and overall hygiene, the patient was discharged home with blood glucose of 269 mg/dL. Split rapid acting insulin therapy was given in combination with a long-acting insulin at night.

The patient was subsequently referred to the Pediatric Endocrinology outpatient department of a referral hospital and managed as a case of poorly controlled T2D. Laboratory tests showed that the patient has normal serum C peptide (7.6 ng/mL) and high titre of HbA1c (10%). Combination therapeutics of single dose daily long-acting insulin and metformin have shown to be effective in controlling the blood glucose level in this patient.

Discussion

As obesity became a growing concern in children and adolescent, T2D would be more frequently observed as its consequence. Recent studies indicate a rise in the incidence of T2D among children and adolescent accompanied by significant increase in its related complication [3]. The patient reported here presented with obese appearance, long- lasting polyuria and family history of T2D that all are very typical for T2D case [4].

Severe hyperglycemia, uncompensated metabolic acidosis, with very low serum bicarbonate, high anion gap, and moderate ketonuria in this patient were typical findings for DKA. However, the patient also developed altered mental status, increased natremia, and plasma hyperosmolality as evidences of HHS. Hence, this

Table 1: Laboratory results of the case.

Time (hour)	0	6	10	15	26	45	55	67	89	127	191
Laboratory Results											
pH	6.98	7.06	7.13	7.17		7.12	7.19			7.26	
HCO_3 (mmol/L)	3.1	4.2	6.0	9.5		11.4	13			13.9	
EB	Not detected	-24.2	-20.9	-17.6		-16.9	-14.2			-11.9	
Blood Glucose (mg/dL)	1105	729	614	479	405		311	187		377	217
Ketone urine	++					neg	neg	neg	neg		
Na^+ corrected (mmol/L)*	151.88	157.06	150.32	148.79	146.98		148.37			147.63	145.67
K^+ (mmol/L)	6.08	4.33	3.94	4.67	3.85	4.67	3.88			4.55	2.29
Osmolality (mosm/kg)**	332.98	334.5	318.31	312	306.7		307.27			307.3	308.54
Ureum (mg/dL)					112.2	134.3	104.5			96.7	68.6
Creatinine (mg/dL)	2.28				3.47	5.91	4.81			4.08	3.23

*Corrected Sodium (mmol/L) = measured sodium + [1.6 (glucose - 100)/100]; **Effective osmolality (mosm/kg) = 2 [Na(+)]+glucose/18
Shadowed part of table presented the laboratory results during hemodialysis

clinical case reaffirms the possibility of developing DKA with some manifestations of HHS in adolescent in the context of unknown T2D, which only few have been recognised and reported [5,6].

The occurrence of DKA in T2D has been believed to be due to complete loss of beta-cell function in long-standing T2D patients [7]. Other studies suggest the cause could be due to relative insulin deficiency arising from constant hyperglycemia in poorly controlled patients [8,9]. The mechanisms for the mix condition in this case might be the consequence of a more profound lack of hepatic and peripheral insulin availability in terms of isolated HHS. Hepatic steatosis induces lipolysis which causes ketogenesis and metabolic acidosis (ketosis prone T2D) [4,6]. Transient moderate ketonuria that shown in this patient supported these possible mechanisms.

This case description might give a valuable nuance for early recognition, treatment strategy, possible complication, and outcome of DKA with some HHS manifestations.

Initial fluid therapy is mandatory either in HHS or DKA that directed towards expansion of intravascular volume and maintain adequate urine output [10,11]. Standard normal saline was used at this step for 1 hour than changed to half saline subsequently to slowly correct hypernatremia. IVC collapsibility index of less than 30% and adequate urine output (1.2 mL/kg/Hour) were achieved after 3 hours of volume replacement therapy. Hydration status, electrolyte levels, blood glucose level, and urine output may become guidance for further amount and choice of replacement fluids [12]. In a hypotensive patient, normal saline of more aggressive amount should be given to optimize blood pressure.

There is no standard therapy of sodium bicarbonate in DKA, however patients who experience deterioration of pH with low bicarbonate (bicarbonate < 10 mEq/L) and pCO_2 (pCO_2 < 12) may benefit from sodium bicarbonate therapy [13]. Fifty mEq of sodium bicarbonate in 200 ml water was given to this patient over 2 hours infusion to maintain the pH at > 7.0. Potassium was not added to this therapy because the potassium serum concentration was already high.

Insulin therapy approach in this case became very challenging as there is no specific guideline in pediatric mixed DKA/HHS. In an isolated HHS case, insulin is not an essential therapy as there is no absolute insulinopenia. Fluid hydration therapy alone will reduce serum glucose level as a result of hemodilution, good renal perfusion, and improved tissue glucose uptake. Insulin treatment is considerable to resolve ketosis. Insulin continuous infusion administration is recommended after initial fluid boluses. However, insulin therapy may induce rapid declining of blood glucose which in turn may cause intravascular volume depletion, and rapid potassium influx into the cell [12]. Hence, close monitoring on hemodynamic, serum glucose concentration, and electrolyte status become crucial. There is no recommendation on insulin bolus for pediatric patients [12,14]. Therefore, a decision was made to give the patient a more conservative rate of insulin 0.1 IU/kg/hour without any priming bolus at the first hour after fluid resuscitation. Blood glucose reduction of 62.7 mg/dL/hour was successfully achieved.

There were two possible mechanisms that could induce acute renal failure in this patient i.e., severe dehydration and rhabdomyolysis. The incidence of acute renal failure in patients with Hyperglycemic crisis is 25% or may be even higher in HHS as the volume depletion is more severe [15]. However, uncorrected volume depletion was unlikely in this case as successful volume replacement therapy became evidenced with good IVC collaptibility index and adequate urination within several hours of fluid resuscitation. On the other hand, rhabdomyolysis is more likely in this case although it is less common in HHS. About 20% of patients diagnosed with HHS have rhabdomyolysis which can cause acute renal failure, disseminated intravascular coagulation, cardiac arrythmia and arrest, and significant electrolyte abnormalities. This complication induces a mortality rate of 35.5% [11]. The clinical correlation between these two conditions is yet to be clarified. Urine and serum myoglobine concentrations were not examined in this patient since the panel laboratory test was not available in the setting. Nevertheless, prompt temporary hemodialysis resolved the emergent renal problem in this case.

Conclusion

We report a case of DKA with some manifestations of HHS in an adolescent boy with T2D. Fluid replacement therapy and insulin treatment are proven to be critical step to restore intra and extracellular volume, control hyperglycemia and hypernatremia as the main causes of hyperosmolality. Sodium bicarbonate therapy was able to maintain optimum blood pH. Rhabdomyolysis is a possible cause of the diminishing renal function complication in this case that was effectively reversed by temporary Hemodialysis.

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