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Diagnostic and management approach to hyperglycemic hyperosmolar state in children with type 2 diabetes mellitus in limited settings

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Abstract: Hyperglycemic Hyperosmolar State (HHS) is a triad characterized by severe hyperglycemia, significantly increased serum osmolality, and severe dehydration without marked ketoacidosis. It occurs in patients with both Type 1 and Type 2 Diabetes Mellitus. This case report discusses a 7-year and 9-month-old girl who presented to the hospital with shortness of breath and acidosis without evidence of ketonemia. The patient was moderately malnourished and had experienced increased urinary frequency over the past month. On examination, she was alert but had dry mucous membranes and visibly sunken eyes. Her laboratory results revealed a random blood glucose level of 1126 mg/dL, a blood pH of 6.97, and ketonuria of +1. Initially, the patient was misdiagnosed with diabetic ketoacidosis (DKA) at a previous hospital, leading to unresolved dehydration. Despite the absence of obesity or acanthosis nigricans, her normal C-peptide levels suggested Type 2 Diabetes Mellitus. Although DKA and HHS share some clinical features, they require distinct therapeutic approaches to reduce morbidity and mortality. The rarity of HHS in children and the lack of ketone testing in resource-limited settings pose significant challenges in its diagnosis.

Keywords: *DKA, HHS, Ketonemia, Type I DM, Type II DM.*

1. Introduction

Hyperglycaemic Hyperosmolar State (HHS) is very different from the usual form of acute decompensation in Type I Diabetes Mellitus (Type I DM) diabetic ketoacidosis (DKA), which presents with a triad of hyperglycaemia, ketonemia and acidosis. Hyperglycaemic Hyperosmolar State has a high morbidity and a mortality rate that estimated to be as high as 20%, which is about 10 times higher than the mortality in patients with diabetic ketoacidosis. Counter to diabetic ketoacidosis, in hyperglicemic hyperosmolar state there is enough circulating insulin to prevent excessive fat lipolysis and subsequent ketogenesis, The often extreme hyperglycaemia causes the plasma osmolality to rise, which in turn causes a shift in intracellular water into the extracellular space. This leads to hypovolaemia from body water loss and hypertonic dehydration due to excess sodium [1, 2].

Hyperglycaemic Hyperosmolar State is still a rare condition within children population and has high mortality and morbity, with the similarity with DKA, it is important to know how to diagnose and treat hyperglicemic hyperosmolar state appropriately, This case report presents diagnostic approach and management of hyperglicemic hyperosmolar state to prevent mortality and morbidity [3].

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2. Case Presentation

A 7-year-9-month-old female was referred to the Emergency Unit of Soetomo Hospital from a secondary hospital with a working diagnosis of Diabetic Ketoacidosis (DKA), Acute Kidney Injury (AKI), and Sepsis. The patient initially presented at the secondary hospital with shortness of breath as the chief complaint. She also experienced general weakness, abdominal pain, nausea, and vomiting for four days, with vomiting occurring five times in the last 24 hours. There was no history of fever, cough, edema, headache, diarrhea, or seizures. The patient's mother reported that the child had been urinating more than 15 times a day and drinking water more frequently over the past month. However, there was no noticeable weight loss. The patient had no prior history of hospital admission, and no family members had experienced similar symptoms.

The patient weighed 19 kg and was 128 cm tall, with an ideal body weight of 24 kg, indicating that she was moderately malnourished (79% of her ideal body weight). She appeared weak, with a blood pressure of 80/50 mmHg (P5: 82/40 mmHg), a heart rate of 150 beats per minute, and a respiratory rate of 28 breaths per minute. Her breathing did not appear Kussmaul-like. Her oxygen saturation was 98% with oxygen support via a simple mask at 5 L/min. She had sunken eyes, and her capillary refill time was <2 seconds. She did not appear anemic, icteric, or cyanotic. There was minimal chest retraction, with no rhonchi or wheezing. Cardiac examination revealed no murmurs or gallops. Abdominal examination showed no distension or tenderness.

Laboratory results from the previous hospital showed an incredibly high blood glucose level of 1121 mg/dL. A complete blood count indicated leukocytosis with a hemoglobin level of 12.4 g/dL, a white blood cell count of 27,600/µL, and a platelet count of 315,000/µL. Electrolyte levels revealed hypernatremia and hyperchloremia, along with hypokalemia, with sodium at 156 mmol/L, potassium at 3.06 mmol/L, and chloride at 120 mmol/L. The patient showed signs of reduced renal function, with a BUN of 62 mg/dL, serum creatinine of 3.29 mg/dL, and a glomerular filtration rate of 21. The patient was in an acidotic state, with a pH of 6.97 and bicarbonate levels below 3 mmol/L. The patient was also in a hyperosmolar state, with a blood osmolality of 402.

Urine examination showed ketone 1+, but unfortunately, serum ketone levels were not available from the previous hospital. At Soetomo Hospital, after 9 hours of rehydration at the previous hospital, further laboratory tests were performed. The complete blood count still showed leukocytosis with a white blood cell count of 16,280/µL. Renal function improved, with serum creatinine at 1.8 mg/dL and a glomerular filtration rate of 39. However, the patient remained in metabolic acidosis, with a blood pH of 7.19, bicarbonate at 16.4 mmol/L, and a base excess of -11.85. Sodium and chloride levels were still high at 172 mmol/L and 147 mmol/L, respectively, while potassium was 4 mmol/L. Urinalysis showed no ketones, and the serum ketone level was 0.9, later we found C-peptide was normal at 0.7 ng/mL (0.5–2.0 ng/mL).

At the previous hospital, the patient had received intravenous fluids (~20 mL/KgBW) once and was rehydrated with a total volume equal to 9% of body water and 48hr maintenance fluid based on Haliday segar formula. The patient suspected of having DKA, was started on an insulin pump at 0.1 unit/kg/hour. At Soetomo Hospital, the patient was still hypotensive and tachycardic, so another intravenous bolus of 0.9% NaCl (~20 mL/kg) was administered. Rehydration was continued with volume of 12% fluid deficit and 48 hours fluid maintenance calculated with haliday segar. Insulin therapy (0.05 unit/KgBW/hour) was maintained. Once stabilized, the patient was transferred to the Pediatric Intensive Care Unit (PICU).

3. Discussion

This case presenting patient come short of breath, sign of profound dehidration, Moderate malnutrition and high level of glucose without proof of ketonemia. Because the lack of sign of type II DM and C Peptide examination patient was treated as DKA which resulted in unresolved dehydration. HHS is more uncommon compared to DKA and has similar characteristic to DKA but HHS has higher morbidity and mortality, therefore must be treated properly to prevent fatal complications. In this

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patient, HHS could have been precipitated by undiagnosed diabetes or an infection, as indicated by the high procalcitonin level, even though blood and urine cultures were negative [3, 4].

Physical findings in patients with HHS include profound dehydration, characterized by poor skin turgor, dry buccal mucosa, sunken eyes, cool extremities, and a rapid, weak pulse. Children may present with nonspecific symptoms such as headache, weakness, and vomiting, with or without abdominal pain. Mental status can range from normal to disorientation, lethargy, or coma, depending on the effective serum osmolality. Laboratory criteria for HHS typically include plasma glucose > 33.3 mmol/L (600 mg/dL), venous pH > 7.25, arterial pH > 7.30, serum bicarbonate > 15 mmol/L, small ketonuria, absent-to-mild ketonemia, and effective serum osmolality > 320 mOsm/kg [5, 6].

This patient had a normal C-peptide level, indicating Type 2 Diabetes Mellitus (T2DM). The classic presentation of HHS in children is an obese adolescent with T2DM. However, recent studies have reported HHS in Type 1 Diabetes Mellitus (T1DM) or in non-obese patients, as seen in this case [7, 8].

The pathogenesis of HHS is similar to DKA, but with key differences. Hyperglycemia without ketosis typically occurs in patients who retain some insulin production, most commonly in T2DM. Ketogenesis and lipolysis are suppressed at lower insulin levels than those required to suppress hepatic glucose production, leading to hyperglycemia without ketonemia. Without ketoacidosis, osmotic diuresis with electrolyte and water loss may persist, resulting in severe dehydration. Impaired renal function from dehydration further exacerbates hyperglycemia due to reduced glucose excretion [5, 6, 9].

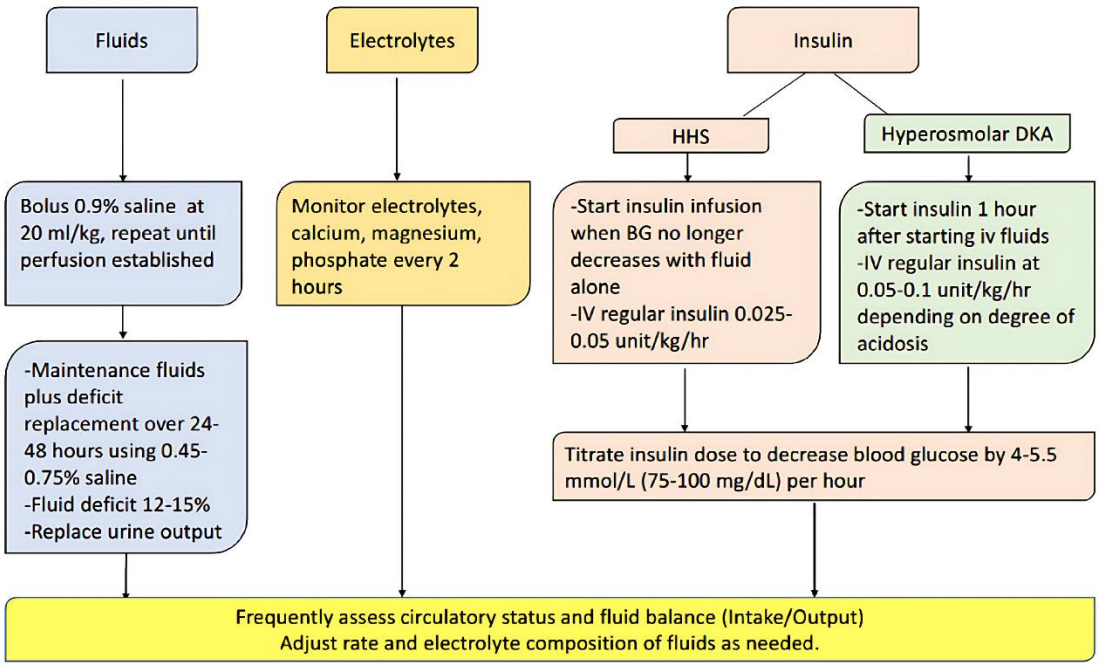

Figure 1.
Management of hyperglicemic hyperosmolar state [5].

Management of HHS includes general resuscitation, rapid restoration of intravascular volume, correction of fluid and electrolyte deficits, and normalization of hyperglycemia and hyperosmolality. All children and adolescents with HHS should be admitted to a high-dependency or pediatric intensive care unit for frequent monitoring, as complications are common. In this case, the patient had a patent airway and remained alert despite vomiting more than five times, but a nasogastric tube was inserted as a precaution. The patient was given 100% oxygen via face mask. Circulation should be monitored for T-

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wave changes (to detect hypokalemia), blood pressure, and heart rate. Resuscitation fluids should be administered urgently, equal to 12–15% of body weight, due to profound dehydration [7, 10, 11].

Patients with hyperglycemic hyperosmolar state (HHS) develop severe dehydration due to prolonged osmotic diuresis, leading to hypertonic dehydration. Treatment focuses on restoring intravascular volume with 0.9% sodium chloride solution (20 mL/kg bolus), repeated as needed until hemodynamically stable. Once stable, 0.45–0.75% sodium chloride is given over 24–48 hours to replace an estimated 12–15% fluid deficit, adjusting sodium concentrations to gradually lower plasma sodium (0.5 mmol/L per hour). Plasma glucose should decline by 50–70 mg/dL per hour with fluids alone; if it falls too fast (>70 mg/dL per hour), 5% glucose should be added to prevent complications. Insulin should not be started immediately in HHS—only when glucose levels stop decreasing with fluids alone—since rapid glucose reduction can lower serum osmolality too quickly, increasing the risk of cerebral edema. Unlike diabetic ketoacidosis (DKA), HHS requires a slower glucose reduction (50–75 mg/dL per hour) to avoid complications [5, 7, 10].

The prognosis of hyperglycemic hyperosmolar state (HHS) in children is dependent on timely diagnosis, appropriate fluid resuscitation, and meticulous management of glucose and electrolyte levels. However, HHS is frequently misdiagnosed as diabetic ketoacidosis (DKA), resulting in improper insulin administration and exacerbation of dehydration. Limited access to essential diagnostic tests, such as serum osmolality and ketone measurements, further contributes to delayed recognition, particularly in resource-constrained settings. Children with Type 2 Diabetes Mellitus (T2DM) are at an increased risk of future metabolic complications. Preventing recurrence necessitates adherence to lifestyle modifications, proper medication management, and comprehensive diabetes education to improve long-term outcomes and reduce morbidity [5].

4. Conclusions

The successful management of this case highlights the essential role swift diagnostic approach and correct management of HHS, despite the similarity with DKA the same treatment as DKA would not resolve HHS properly. The absent obesity and nigricans acanthosis should not rule out posibility of T2DM and eventhough it is rare we should always consider HHS to be the differencial diagnosis of DKA. The rarity of condition itself and absence of ketone examination in limited setting is one and many pitfalls of hyperglicemic hyperosmolar state diagnosis.

Abbreviations:

DKA: Diabetic Ketacidosis; HHS: Hyperglycaemic Hyperosmolar State; Type I DM: Type I Diabetes Mellitus; Type II DM: Type II Diabetes Mellitus.

Transparency:

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained. This study followed all ethical practices during writing.

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