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Hyperglycaemic hyperosmolar state in a patient with type 1 diabetes mellitus

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Abstract

We present a thin malnourished cachectic young male with established T1DM who has multiple previous presentations with DKA requiring ICU admissions. He is on a feeding jejunostomy tube for severe gastroparesis and oesophageal stricture as a complication of gastroesophageal reflux disease (GORD).

He presented to our hospital ED with features of HHS. After initial resuscitation, he was admitted to ICU for four days then transferred to the medical ward for another six days for stabilization of blood glucose and endocrine team input.

On discharge home he had gained 5kg in weight with consistently stable blood glucose level.

Keywords

Hyperglycaemia; hyperosmolar; T1DM

Abbreviations

HHS: Hyperglycaemic Hyperosmolar State; T1DM: Type 1 Diabetes Mellitus; T2DM: Type 2 Diabetes Mellitus; DKA: Diabetic Ketoacidosis; BMI: Body Mass Index; ED: Emergency Department; ICU: Intensive Care Unit.

Background

HHS is a rare complication of T2DM in the elderly. It is Characterized by severe hyperglycaemia without ketosis, significant dehydration and decreased level of consciousness or frank coma with generally poor outcome [1,2,7].

Only one case of HHS in a patient with established T1DM who has chronic kidney disease (CKD) has been reported making our case unique [6]. However, there have been reported cases of HHS as a first

presentation of T1DM in the paediatric population [8,9].

Case Presentation

A 26 years old male patient with T1DM with multiple microvascular complications including oesophageal stricture secondary to severe gastroparesis requiring feeding jejunostomy tube, diabetic retinopathy and peripheral neuropathy but he has normal baseline renal function (baseline Creatinine is 70 µmol/L). He also has pancreatic insufficiency and limited mobility due to malnutrition.

He has had previous multiple admissions with DKA requiring ICU admissions.

His usual diet includes feeding supplement through jejunostomy tube (1000-1400 ml/day, 2.0 kilocalorie/ml) in addition to oral water (2.5 – 3 L/day).

He is on Esomeprazole, Insulin Glargine and Isophane, Pancrelipase (combination of Lipase, Amylase and Protease) and sodium bicarbonate.

He presented to ED with two days history of fatigue, drowsiness and decreased mobility with no history of infective symptoms, vomiting or diarrhoea. He had been given his regular Insulin, oral medications and was feeding as above.

He was confused on arrival and his vital signs were; a temperature of 35.9 °C, respiratory rate (RR) 22/minute, blood pressure (BP) 92/71 mmHg, heart rate(HR) 95 beat/minute and Oxygen saturation (SPO2) 87% on room air, with a Glasgow coma scale (GCS) of 14. His clinical examination was otherwise unremarkable.

Admission weight was 42 Kg with BMI of 12.9 (baseline weight is 46-47 Kg).

Investigations

His initial investigations were as follows:

Blood test	Result	Normal value
Sodium (Na)	149 mmol/L	135-145 mmol/L
Potassium (K)	6.0 mmol/L	3.5-5.2 mmol/L
Chloride (Cl)	104 mmol/L	95-110 mmol/L
Bicarbonate (HCO3)	29 mmol/L	22-32 mmol/L
Urea	41.5 mmol/L	3-8 mmol/L
Creatinine	116 µmol/L	60-110 µmol/L
Magnesium (Mg)	1.73 mmol/L	0.7-1.1 mmol/L
Calcium (Ca)	2.17 mmol/L	2.1-2.6 mmol/L
Plasma osmolality	450 mmol/Kg	275-295 mmol/Kg
Anion gap	22 mmol/L	10-22 mmol/L

Glucose	97.5 mmol/L	3-5.4 mmol/L
pH (arterial)	7.25	7.35-7.45
PCO_2 (arterial)	64 mmHg	36-45 mmHg
PO_2 (arterial)	56 mmHg	85-110 mmHg
Lactate (arterial)	0.8 mmol/L	< 1.3 mmol/L
Base excess	1 mmol/L	-3 – 3 mmol/L
Plasma ketones	1.5 mmol/L	0.05 -0.29 mmol/L
Haemoglobin	121 g/L	135-180 g/L
White cell counts (WCC)	13.3 X 10*9/L	4-11 X10*9/L
Platelets	201 X10*9/L	150-400 X 10*9/L
C reactive protein (CRP)	12 mg/L	< 10 mg/L

Urinalysis: pH 6, positive Nitrite, trace leucocytes, negative Ketones.
Chest X-Ray: No focal pulmonary pathology identified.
Urine microscopy and culture: positive for Staphylococcus aureus.

Treatment

We followed initial treatment in ED according to DKA protocol. This included intravenous fluids (normal saline), Insulin and in ICU 0.45% Sodium chloride with a switch back to a normal saline infusion.

He spent a total of four days in ICU and six days on a medical ward.

During this time, he was recommenced on a jejunostomy with oral feeding and subcutaneous Insulin regimen.

Additionally, he was started on oral Cephalexin for a concomitant urinary tract infection.

He gained 5 Kg during admission (admission weight was 42kg and discharge weight was 47 Kg, admission BMI was 12.9 and discharge BMI was 14.5) and was reviewed by an endocrinologist, dietician and diabetic educator.

Outcome and follow-up: The patient was discharged in a stable condition at his baseline weight with a slight modification of his Insulin regimen and out-patient endocrinology follow up. He unfortunately deceased few months later.

Discussion

HHS represents a syndrome of acute diabetic decompensation characterized by marked hyperglycaemia, hyperosmolarity and dehydration, with decreased mental functioning that may progress to frank coma.

HHS is the most serious acute hyperglycaemic emergency in patients with T2DM [1,2].

The incidence of HHS is estimated to be <1% of hospital admissions of patients with diabetes [3]. The reported mortality is between 10 and 20%, representing ten times greater mortality rate in patients with DKA [4,5].

Most cases of HHS are seen in elderly patients with T2DM with other comorbidities (mainly chronic kidney disease). Although, it has also been reported in children and young adults, the majority of reported paediatric cases were subsequently diagnosed with T2DM, with few reported in newly diagnosed T1DM [8,9].

Recent case reports and series suggest an increasing incidence of this disorder in children and adolescents owing to increased incidence of obesity and T2DM among paediatric population [8].

The most common precipitating factor is infections in approximately 40-60% of patients. The most common being pneumonia caused by gram negative bacteria (40–60% of patients) and urinary tract infection (5–16%).

Other precipitating factors are medications (Phenytoin, glucocorticoid, Thiazide diuretics, β-blockers and atypical antipsychotics), poor medications compliance, undiagnosed diabetes mellitus(Up to 20% do not have a previous diagnosis), coexisting diseases and substance abuse [7].

The criteria for HHS include a plasma glucose concentration greater than 33.33 mmol/L (600mg/L), mild ketonuria, absent to low ketonemia, nonketotic acidosis, severe dehydration, effective serum osmolality of more than 350 mOsm/kg and stupor or coma [7].

The underlying metabolic abnormality results from the combination of absolute or relative insulin deficiency and increased amounts of counterregulatory hormones.

This suggests insulin action may be inadequate to facilitate glucose utilization by insulin-sensitive tissues but adequate for the prevention of lipolysis and ketogenesis associated with prolonged and gradually increasing polyuria and polydipsia resulting in severe dehydration. This is associated with severe electrolyte loss occurs due to prolonged duration of osmotic diuresis.

The hypertonicity of the hyperosmolar state preserves intravascular volume which may mask the clinical signs of dehydration. With treatment, serum osmolality decreases resulting in an osmotic gradient causing water to move from the intravascular to the intracellular space decreasing intravascular volume. Massive osmotic diuresis in the early hours of therapy needs aggressive fluid management to prevent shock and avoid the complications of vascular collapse (lactic acidosis, rhabdomyolysis, renal failure, thrombosis) [1,2,7]. Persistent hypernatremia, hypokalaemia, hypophosphatemia, rhabdomyolysis, pancreatitis, and thrombosis must be monitored.

Fluid resuscitation is best achieved initially with isotonic fluid (0.9% saline), which is hypotonic for the patient and followed by 0.45% saline.

In addition to insulin infusion (0.1 U/kg/hour is recommended), dextrose is added to intravenous fluids when serum glucose drops to 300mg/dl. The insulin infusion is then switched to subcutaneous insulin as per DKA protocol [1,7].

In our case the patient had mild acute respiratory acidosis which could have been due to confusion and weak respiratory muscles as a result of malnutrition and cachexia. In addition, he had acute kidney injury at presentation which is most likely due to severe dehydration.

In this case we could not explain why a patient with known T1DM progressed to HHS rather than presenting with DKA. We propose further studies are needed to fully understand the aetiology, notably the role of malnutrition, to the development of HHS in young adults with a known history of T1DM. This will help us better recognise and manage these patients in the future.

Learning Points

1) Ability to differentiate between hyperglycaemic hyperosmolar state and diabetic ketoacidosis in patients with hyperglycaemia.

2) Hyperglycaemic hyperosmolar state carries higher risk of mortality compared to diabetic ketoacidosis.

3) Clinicians should be aware of including 0.45% saline in management of hyperglycaemic hyperosmolar sates in order to avoid severe hypernatraemia.

4) Searching for potential underlying triggers in cases of hyperglycaemic hyperosmolar state and diabetic ketoacidosis should be considered during management.

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