

Hyperglycemic Hyperosmolar Coma Revealing Type 1 Diabetes

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Abstract: Hyperglycemic hyperosmolar syndrome (HHS) is one of two life threatening acute metabolic complications occurring in diabetic patients. It is more commonly seen in the elderly disabled patients with type 2 diabetes and it's often due to infection or an underlying disease, its occurrence in young diabetic type 1 is extremely rare. We report the case of a 17 years old patient admitted for disorders of consciousness whose clinical and biological investigations concluded to a hyperosmolar hyperglycemic coma.

Keywords: hyperglycemia, hyperosmolar state, type 1 diabetes

INTRODUCTION

Diabetic ketoacidosis (DKA) is an acute metabolic disorder of diabetes, and the most common presentation mode of type 1 diabetes (T1D), its rates ranges from 15% to 83% in various population studies [1]. Hyperglycemic Hyperosmolar state is another acute metabolic complication of diabetes mellitus, it is a clinico-biological syndrome associating blood glucose concentration > 6g/l, serum bicarbonate >15 mmol/l, effective serum osmolarity >320 mOsm/l, absent or small ketonuria and ketonemia, with consciousness disorders such as stupor or coma [2]. This condition occurs more frequently in type 2 diabetic adults, its occurrence in young type 1 diabetic is very rare but is associated with a high rate of mortality. We report a case of a T1D patient who presented with HHS.

CASE REPORT

A 17-year-old girl, with no particular medical history, was admitted to the emergency room for consciousness disorders with a Glasgow score of 11/15, her parents have reported polydipsic polyuria syndrome for 3 months, with ingestion of large amounts of water and soft drinks, a recent undocumented weight loss estimated important despite associated overeating. On examination, the patient was dehydrated, febrile with a temperature at 39°C; her heart rate was at 130 b / min, her blood pressure at 110 / 80 mmHg and her weight was estimated at 40 kg. her capillary blood glucose was higher than 7 g/l and she had glycosuria with traces of ketonuria at the urine strips.

On blood examination, she had hyperosmolarity at 368mOsm/l, hyperglycemia at 11.38g/l, hypernatremia at 150mmol/l, her serum potassium level was at 4mmol/l, urea level at 0.51g /l, creatinine level at 10mg/l, elevated CRP level at 97 and high leukocytes level at 40000 with normal cytobacteriological examination of urine, thorax X ray and cerebral CT. The patient was rehydrated and put on continuous intravenous insulin therapy with good clinical improvement and recovery of consciousness, the evolution was marked by the occurrence of a cardio respiratory arrest and death of the patient by a probable septic shock.

DISCUSSION

Over the past several decades, the incidence of T1D among children is increasing rapidly with DKA as initial manifestation [3]. Hyperosmolar hyperglycemia syndrome (HHS) is a recognized, potentially life threatening manifestation of type 2 diabetes, occurring mainly in the elderly. It is a serious and rare complication at the presentation of diabetes in children firstly described by de Vaan GAM in 1966 [4].

Even if HHS have been distinguished from diabetic ketoacidosis (DKA), it is more appropriate to view it as one extreme in the multiples presentations of altered glucose metabolism in diabetic patients with euglycemic DKA as the other extreme [5].

In DKA, Hyperglycemia is due to the increased renal and hepatic glucose production with impaired peripheral glucose utilization leading to osmotic diuresis, while ketonemia and metabolic acidosis are the consequences of increased lipolysis and ketoacid production [6]. In contrast, HHS is due to a prolonged increasing polyuria and polydipsia which causes profound dehydration and severe electrolyte and fluid loss more than those in DKA [7].

In children with new onset T1DM, hyperglycemia causes an osmotic diuresis, and without adequate oral rehydration, hyperosmolarity can worsen. Also, ingestion of high sugar drinks will exacerbate the preexisting hyperglycemia and hyperosmolarity [8], which was probably the case of our patient whose ingestion of a large amount of high-carbohydrate solutions has precipitated the survival of HHS. Heggarty *et al.* reports that HHS is also associated with malnutrition, severe learning difficulties, drugs (phenytoin), peritoneal dialysis and gastroenteritis [9].

The presentation of HHS in children may be similar to DKA including vomiting, abdominal pain, weakness, confusion, and behavioral change [7], with the difference of a longer history of polyuria and polydipsia [10], without fruity breath odor or tachypnea because of the absence of ketosis.

Unlike DKA, HHS in children Pediatric HHS is responsible of a high rate of morbidity and mortality (37 – 75%). Serious complications included brain edema, rhabdomyolysis, arrhythmias, hyperthermia, thrombosis and renal failure [7, 11-13].

Management of HHS requires copious fluid and electrolyte replacement to expand the intra and extravascular volume restore normal renal perfusion and promote a gradual decline in serum sodium concentration and osmolality. The initial bolus should be ≥ 20 mL/kg of isotonic saline, Additional fluid boluses should be given, if necessary, to restore peripheral perfusion. Insulin administration should begin at a dose of 0.025 to 0.05 U/kg/h once plasma glucose is no longer declining at a rate of at least 0.5 g/l per hour with fluid alone [14].

CONCLUSION

Hyperglycemic hyperosmolar coma is a serious form of decompensation of diabetes mellitus. Although rare in type 1 diabetics, it must be evoked in the presence of major dehydration, unconsciousness and high plasma osmolality without ketosis, and its evolution is most often fatal.

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