Fatal Hyperosmolar Hyperglycaemic Syndrome complicated by severe Rhabdomyolysis

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Hyperosmolar Hyperglycaemic Syndrome (HHS) may cause death in patients with diabetes, particularly if complicated by rhabdomyolysis and multi-organ failure.

Introduction

A 49-year-old African man, with no known past medical history, presented to the emergency department with three day history of vomiting, lethargy, confusion and drowsiness on a background of two week history of thirst, polyuria with drinking up to 10 pints of water a day. More recently he had been drinking litres of non-diet lemonade in an effort to sustain energy levels.

On examination, Glasgow Coma Score was 6 (eyes 4, verbal 1, motor 1). He was severely dehydrated with dry mucous membranes, blood pressure 75/40 mmHg, pulse rate 120 bpm and cool peripheries. He was pyrexial 38.5˚C, and hypoxic (oxygen saturations 84% on room air). Cardiovascular and respiratory examination was otherwise unremarkable. On arrival to the emergency department, he was intubated and ventilated due to hypoxaemia and poor conscious level. Baseline investigations are shown in Box 1.

Chest radiograph showed patchy consolidation in the right base, suggestive of aspiration pneumonia. A diagnosis of HHS, acute renal failure and severe mixed metabolic and respiratory acidosis was made. The patient was managed on the intensive care unit (ICU) with careful fluid resuscitation with 0.9% saline, according to central venous pressure. Slow reduction of plasma glucose was an explicit aim, using a maximum of 1 unit per hour of intravenous insulin, aiming to reduce plasma glucose by no faster than 1 mmol/L every 4 hours.1 He was fully anticoagulated in view of his high risk of stroke, and was given broad spectrum antibiotics. On ICU, he required inotropic support to maintain blood pressure, and haemofiltration in view of his acute renal failure and severe acidosis.

His condition did not improve despite full intensive care. Serum creatine kinase continued to increase to a peak of 417,907 IU/L, and a presumptive diagnosis of compartment syndrome leading to muscle infarction and acute rhabdomyolysis was made, which was supported by raised compartment pressures in the thigh and calf muscles. He underwent multiple fasciotomies, which confirmed necrotic muscle. His acidosis did not resolve despite haemofiltration, and oxygenation deteriorated, probably as a result of acute lung injury. He developed uncontrollable bleeding from disseminated intravascular coagulopathy, and after careful discussion with his family, further support was withdrawn, and he died 72 hours after arrival in hospital.

Post mortem examination showed a fatty liver, evidence of muscle infarction in the legs, but no evidence of cardiovascular or cerebrovascular events.

Discussion

Rhabdomyolysis may be defined as a clinical and laboratory syndrome resulting from skeletal muscle injury with release of muscle cell contents into the plasma.2 In addition to traumatic injuries, non-traumatic causes such as alcohol abuse, drug overdose, hypokalemia, hypophosphatemia and hypothermia may result in rhabdomyolysis.

As demonstrated in this case, rhabdomyolysis is a rare but potentially fatal complication of...
hyperosmolar states. Early cases reported an association between serum sodium, serum osmolality and plasma glucose being pivotal in initiating rhabdomyolysis in diabetic subjects with severe hyperglycaemia. To our knowledge, there are around a dozen cases reported in the literature, although it has been suggested that over 50% of patients presenting with hyperosmolar state may develop rhabdomyolysis to greater or lesser degree, although it is rarely clinically important.

Possible predisposing factors to rhabdomyolysis include hypokalemia and hypophosphatemia, both of which are common in acute diabetic emergencies, although they may be masked by potassium and phosphate release from damaged muscles.

It is noted that a linear relationship between serum creatine kinase (CK) and serum sodium and osmolarity exists, and rhabdomyolysis may aggravate hypernatraemia due to the intracellular breakdown of macromolecules into smaller molecules, promoting shift of water from extracellular fluid to muscle cells. The acute renal failure due to rhabdomyolysis secondary to muscle injury is characterized by muscle tenderness, swelling and/or necrosis, myoglobinuria and elevated CK levels. Rhabdomyolysis and acute renal failure as complications of severe hypernatraemia were seen in our patient.

**Conclusions**

We report a fatal case of HHS, which highlights complications such as hypovolemic shock, rhabdomyolysis, acute kidney injury and acute lung injury. Despite aggressive resuscitation and management, this patient had a poor outcome. Clinicians need to develop early awareness of complications such as rhabdomyolysis in patients with acute hyperglycaemic emergencies.

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