Nontraumatic rhabdomyolysis: an unusual complication of diabetic hyperosmolar nonketotic (HONK) state

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Rhabdomyolysis is an uncommon but underestimated complication of HONK.¹ We present the case of a patient presenting with HONK complicated by rhabdomyolysis.

CASE HISTORY

A 75-year-old man of Asian origin was admitted with a 2-day history of being generally unwell. He noted progressive polyuria, polydipsia for about 2 months prior to admission. There was no history of trauma, seizures, muscle pain, chest pain, drug or alcohol abuse. He had no past medical history including no known diabetes mellitus. He was a non-smoker and did not drink alcohol.

On admission his blood pressure was 150/90, pulse rate 110/minute regular, temperature 36°C. Physical examination was only remarkable for dehydration. Laboratory investigations on admission revealed venous plasma glucose 42 mmol/L; sodium 152 mmol/L; potassium 4.4 mmol/L; urea 13.4 mmol/L; creatine 147 µmol/L; arterial blood gas pH 7.36, HCO₃ 23.9 mmol/L; serum osmolality 362 mOsm/kg; serum creatine kinase 6309 iu/L with CKMB fraction of 0.9%; troponin I <0.1 ng/L; albumin 50 g/L; bilirubin 12 µmol/L; alanine transferase 60 iu/L; alkaline phosphatase 127 iu/L; serum amylase 75 iu/L; haemoglobin 16.8 g/L; leucocytes 17 × 10⁹ platelets 267 × 10⁹; C-reactive protein 8 mg/L; erythrocyte sedimentation rate 16 mm in the first hour; urine dipstick revealed 4+ glucose; the chest X-ray and electrocardiograph were unremarkable.

The patient was treated with rehydration and continuous insulin infusion for 48 h. The creatine kinase rose from 6309 iu/L on admission to a peak level of 11 275 iu/L on day 2 before reducing to 3292 iu/L on day 3. By day 3 his sodium had normalized to 137 mmol/L and creatine to 74 µmol/L. Blood and urine cultures were negative. The patient was feeling much better by day 2 when he was put on a twice-daily insulin regime. Due to important commitments at home the patient had to be discharged on day 3.

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COMMENT

Rhabdomyolysis may be defined as a clinical and laboratory syndrome resulting from skeletal muscle injury with release of muscle cell contents into the plasma. Apart from crush injuries various nontraumatic causes may also be implicated such as alcohol, drug overdose, hypokalaemia, hypophosphataemia, hypothermia.²

The exact mechanism of rhabdomyolysis in a hyperosmolar state remains unclear. Singhal *et al.* concluded that serum sodium, serum osmolality and blood glucose are the major determinants for the occurrence of rhabdomyolysis in the diabetic state.³ According to Gabow *et al.* hypokalaemia and hypophosphataemia were important predisposing factors for rhabdomyolysis.² Total body potassium deficiency, a frequent complication of hyperosmolar states, and pre-existent hypophosphataemia may be masked because of release of potassium and phosphate ions from damaged muscles.¹ The combination of all the above factors is likely to predispose to the development of rhabdomyolysis.¹

It is important to remember that subclinical rhabdomyolysis is a more common complication of the hyperosmolar state than is generally appreciated. This is especially important as the clinical manifestations are mild or absent.¹ Singhal *et al.* found that more than 50% (16/31) of his patients admitted with the hyperosmolar state developed rhabdomyolysis.¹

The clinical concern is whether renal failure is more or less common in HONK associated rhabdomyolysis and whether it is possible to predict which patients will progress to acute renal failure. For this a comparison of the clinical characteristics of rhabdomyolysis patients associated with HONK with or without acute renal failure will be required. We have not found any studies addressing this particular point. However, Gabow *et al.* did a discriminant analysis of 65 patients admitted with rhabdomyolysis (of various aetiologies) but without admission azotaemia and came out with a formula to predict high and low risk for developing acute renal failure.² The low-risk group had an *R* value of less than 0.1, where $R = 0.7(K) + 1.1(\text{creatinine}) + 0.6(\text{alb}) - 6.6$, where (*K*) is potassium concentration in mmol/L, creatine is serum creatine concentration in mg/dL and alb is serum albumin concentration in g/dL.

Hence, physicians should be alerted to this under detected complication of HONK which may lead to acute renal failure yet can be easily diagnosed by a readily available test—the creatine kinase level.

Competing interests None.

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