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Rhabdomyolysis associated with cranial diabetes insipidus

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Summary: Rhabdomyolysis has been reported to be associated with hyperosmolality in diabetic ketoacidosis and non-ketotic hyperosmolal state. Whether the rhabdomyolysis was due to hyperosmolality *per se* or whether hyperglycaemia also played a role is not clear. We hereby report a case of cranial diabetes insipidus with hypernatraemia and hyperosmolality complicated by rhabdomyolysis. None of the known risk factors, such as coma, hypokalaemia, hypophosphataemia, diabetic ketoacidosis or non-ketotic hyperosmolality, were present in this patient. We believe that severe hyperosmolality *per se* is an important predisposing factor for non-traumatic rhabdomyolysis, and serum muscle enzymes should be closely monitored in the management of patients with diabetes insipidus.

Introduction

Rhabdomyolysis is an important medical condition as delayed diagnosis and management could result in acute renal failure and sometimes death. The most common causes for nontraumatic rhabdomyolysis are drugs, alcohol and muscle compression.¹ The hyperosmolar state in diabetic coma has also been cited as one of the causes for rhabdomyolysis.^{2–4} We recently encountered a patient with cranial diabetes insipidus who developed rhabdomyolysis as a result of severe dehydration and hyperosmolality. Early recognition of this complication allowed prompt treatment and prevented development of renal impairment.

Case report

A 23 year old man presented with a 6-month history of malaise, mental slowness, poor memory, and was admitted with severe dehydration. Examination also showed gynaecomastia. Investigation revealed serum sodium 187 mmol/l, serum osmolality 340 mOsmol/kg and urine osmolality 107 mOsmol/kg. His anterior pituitary functions were all normal. His cranial diabetes insipidus responded to DDAVP treatment. Subsequent investigation revealed pineal and suprasellar germinoma. He was treated with chemotherapy with complete remission.

He was admitted one year after initial presentation with a one day history of being unwell after

omission of DDAVP. Serum sodium was 180 mmol/l, potassium 4.0 mmol/l, urea 9.7 mmol/l, creatinine 0.231 mmol/l, uric acid 0.84 mmol/l, anion gap 28 mmol/l, glucose 5.4 mmol/l, serum osmolality 493 mOsmol/kg and urine osmolality 182 mOsmol/kg. His serum lactate dehydrogenase (LDH) was 623 IU/l and creatine kinase (CK) was 81 IU/l. The arterial pH was normal. He was quickly rehydrated and stabilized on DDAVP.

Two days after hospitalization, he complained of generalized muscle pain, darkening of urine, and swelling and tenderness of the right calf. The LDH level was now 3600 IU/l with CK of 60,720 IU/l. The urine was positive for heme by dipstick test and many brown pigmented urinary casts were observed. Doppler flowmetry and ultrasonogram showed deep vein thrombosis involving the popliteal vein up to the lower half of the right femoral vein, and some echogenic thrombus was seen inside the vessel. Intense forced alkaline diuresis as well as heparin infusion was commenced with close monitoring of his fluid and electrolyte balance. The muscle enzymes and creatinine level returned to normal in parallel with the serum sodium and osmolality after one week. Hypocalcaemia of 1.78 mmol/l and hyperphosphataemia of 1.58 mmol/l were noted early in the course of rhabdomyolysis for which a calcium infusion was given.

Discussion

Rhabdomyolysis with myoglobinuria due to a hyperosmolal state is a rare condition. The causes of non-traumatic rhabdomyolysis have been reviewed and none out of the 87 cases reported was

due to a hyperosmolal state.¹ Hyperosmolality causing rhabdomyolysis has been reported in only three patients. One due to prolonged coma and dehydration,² and two to diabetic nonketotic hyperosmolal coma and hypokalaemia.^{3,4} Singhal *et al.* recently reported a correlation between serum sodium and osmolality with CK level in patients with diabetic ketoacidosis and hyperosmolal coma.⁵ Whether the rhabdomyolysis was due to hyperosmolality *per se* or whether hyperglycaemia also played a role is not clear.

Rhabdomyolysis as a result of hyperosmolality due to cranial diabetes insipidus has not been reported before to our knowledge. Other metabolic derangements which are reported to be associated with rhabdomyolysis include hypokalaemia, hypophosphataemia, and myxoedema coma.¹ Our patient also had a number of poor risk factors for developing acute renal failure from rhabdomyolysis such as high serum potassium, anion gap,

phosphate and uric acid and a low serum calcium concentration.¹ Recognition of these factors allowed vigorous treatment and prevented acute tubular necrosis. The development of deep vein thrombosis in this patient is also a well known complication of dehydration.⁶

The cause of hypernatraemia and hyperosmolality leading to rhabdomyolysis is not clear, but was proposed to be due to inhibition of the sodium pump resulting in a decrease in the transmembrane potential of the muscles and subsequent muscle damage.⁷ Thus, we believe that severe hyperosmolality *per se* may predispose to the development of rhabdomyolysis, and serum muscle enzymes should be monitored closely in the management of patients with diabetes insipidus. Whether the vigorous fluid replacement of his dehydration and rapid normalization of the serum osmolality contributed to the rhabdomyolysis remains to be confirmed.

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