Hypernatremia Associated with Severe Rhabdomyolysis

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Dear Sir,

While it is well established that certain electrolyte derangements are among the most common causes of rhabdomyolysis [1], there are only 2 cases describing its occurrence in association with severe hypernatremia [2, 3]. In another report rhabdomyolysis has been related to a nonketotic hyperosmolal state with hypernatremia [4]. We have recently seen a case supporting a role of hypernatremia in the development of rhabdomyolysis.

A 49-year-old man was admitted because of mental confusion, generalized myalgias and oliguria with dark urine. Thirteen years before he had undergone aneurys-mectomy of the anterior communicating artery, after which he developed a partial pituitary diabetes insipidus. There was no history of alcoholism or drug abuse. Physical examination revealed a dehydrated patient confused and agitated. Pulse rate was 120/min, blood pressure 170/110 mm Hg, temperature 38 °C. Neurologic examination was negative. Pulses of the lower limbs were clearly evident.

Laboratory data showed: BUN 81 mg/dl, creatinine 5.1 mg/dl, glucose 143 mg/dl, sodium 184 mEq/l, chloride 138 mEq/l, potassium 3.9 mEq/l, calcium 7.6 mg/dl, phosphate 6.0 mg/dl, uric acid 12 mg/dl, plasma osmo-lality 400 mosm/kg H₂O, creatine kinase 10,850 IU/l with MB fraction of 416 IU/l, lactate dehydrogenase 631IU/l, arterial pH 7.324, pCO₂ 32.8 mm Hg, HC0₃ 17.2 mEq/l, anion gap 29 mEq. Coagulation parameters were normal. Urine showed a 3 + positive heme test with a few red blood cells and some pigmented casts in the sediment.

Myoglobin in serum yielded values of 25,000 µg/l (normal up to 65 µg/l).

Infusion of 0.5 yVsaline was started immediately, at a rate calculated to reduce plasma osmolality of 2 mosm/ kg H₂O/h. On the second hospital day the patient suddenly developed ischemic changes of the distal third of both legs with laboratory findings of disseminated intra-vascular coagulation (DIC): platelet count was 60,000/ mm³, FDP > 160 < 320 µg/ml. Doppler examination showed no blood flow at the tibial arteries. Heparin therapy afforded no relief of the process which rapidly led to gangrene. After this event creatine kinase rose to 36,000 IU/l, creatinine to 10 mg/dl and dialytic therapy was started. On the fourth hospital day serum sodium was 145 mEq/l, creatinine 3 mg%. On the tenth day creatinine was 0.8 mg/dl.

This patient presented rhabdomyolysis, complicated by DIC, which led to the rare occurrence of macrovascu-lar thrombosis [5]. None of the most commonly reported causes of rhabdomyolysis was evident [1]. Potassium concentration was normal, however a mild potassium deficiency

could not be entirely excluded; in fact, hypokalaemia might have been lacking in the presence of acidosis, renal failure and liberation of potassium from muscle-injured cells. It has to be underlined that this patient showed a potentially fatal hypernatremia, which probably supervened because of inadequate water intake in a patient with partial pituitary diabetes insipidus.

It has been proposed [1, 6] that hypernatremia and hyperosmolality may contribute to the development of rhabdomyolysis by decreasing the extrusion of sodium ions from muscle cells through the inhibition of the electrogenic sodium pump; this in turn would result in a fall of the transmembrane potential [4]. Recent information suggests that inhibition of sodium pump and depression of membrane potential could well set the stage for irreversible cellular injury by impairing the normal exchange of extracellular sodium for intracellular calcium, thus allowing a rise of the cytoplasmic calcium. If calcium ions attain a critical elevated value in the sarcoplasm, neutral proteases are activated with consequential destruction of muscle cells [1, 6]. The case described here stresses the importance of hypernatremia as a possible cause of severe rhabdomyolysis.

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