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Diluting delirium

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We present a patient who developed delirium after correction of chronic severe hyponatremia. The delirium was reversed after treatment to lower the serum sodium concentration and allow a more gradual rise in serum sodium concentration.

Case report

A 54-year-old Tongan man with treated miliary tuberculosis and diet-treated type 2 diabetes mellitus was admitted to the emergency department with a 3-week history of increasing nausea, lethargy, and headache. Pulmonary and miliary tuberculosis had been diagnosed 3 months earlier, the CT lung scan showing miliary nodules and minor lower lobe bronchiectasis. Sputum culture was positive.

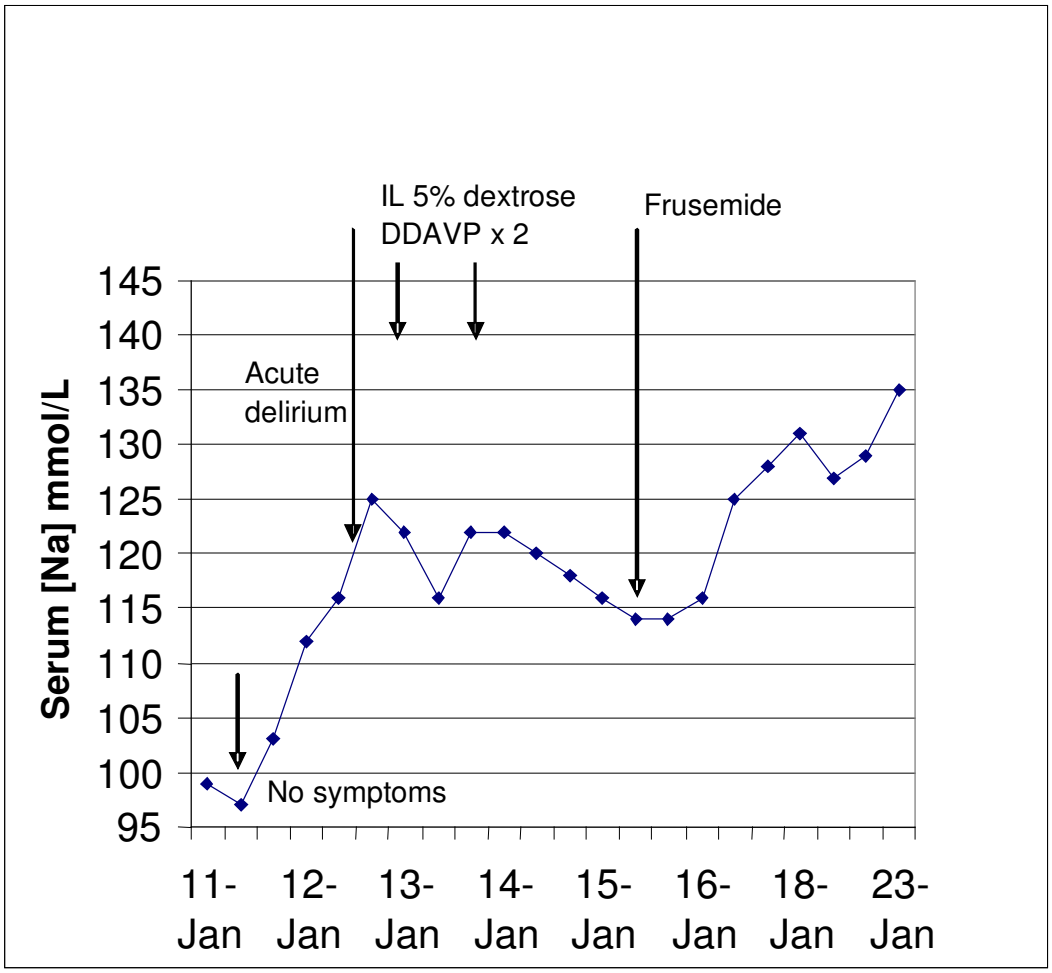
He had been treated with rifampicin 600 mg and isoniazid 300 mg daily together with a 6-week course of prednisone 30 mg daily. He was alert, hyperpigmented, and mildly hypovolemic. His neurological state was normal except for absent or reduced leg tendon reflexes. His blood pressure was 85/50 mmHg; serum sodium 99 mmol/L, potassium 5.5 mmol/L, and serum creatinine 0.12 mmol/L.

We suspected tuberculous Addison's disease with adrenal crisis induced by rifampicin[1] and withdrawal of prednisone. Despite receiving intravenous hydrocortisone (100 mg initially and 8 hourly together with 1 litre of 0.9% saline), his serum sodium fell to 97 mmol/L and he became confused. The blood glucose was 3.7 mmol/L but his confusion resolved rapidly after 50% IV dextrose.

Because of the severe hyponatremia, we elected to increase his serum sodium using 1.8% saline. He was given 240 mmol over 12 hours, and from then his fluid intake was restricted to 600 ml per 24 hours. His serum sodium rose to 112 mmol/L at 24 hours and then 122 mmol/L 48 hours post-admission. The rate of increase of his serum sodium was about 0.5 mmol/L/hour. At that time he developed an acute, agitated delirium with visual hallucinations and behavioural disturbance but without focal neurological abnormalities. Clonazepam sedation was required.

A CT brain scan was normal. Concerned that his acute delirium resulted from too rapid correction of his chronic hyponatremia and fearful of impending myelinolysis[2] we decided to lower his serum sodium again.[3] He was given 1 L of 5% dextrose over 2 hours plus dexamethasone 4 mg intravenously. The serum sodium fell to 116 mmol/L. Moreover, within 18 hours of induced hyponatremia his delirium subsided completely. As this water load was readily excreted with a rise of serum sodium to 122 mmol/L he was then given two doses of DDAVP and free fluid intake until the fifth day when the serum sodium was allowed to rise at approximately 5 mmol/L per day into the normal range. A follow-up MRI brain scan at 4 weeks was normal.

Figure 1. Serum sodium levels during the patient's treatment in 2001



Discussion

Rapid correction of chronic hyponatremia may cause fatal pontine and extra-pontine myelinolysis heralded by delirium.[4] Experimental and clinical studies by Soupart and coworkers showed that myelinolysis can be prevented and even reversed by delaying the rate and magnitude of serum sodium correction.[5] Glucocorticoids lower vasopressin levels and restore the kidney's ability to excrete free water.

The water diuresis resulting from glucocorticoid therapy in this man caused an unexpectedly rapid reversal of his chronic hyponatremia thereby provoking an acute delirium that resolved after relowering of the serum sodium. His Addison's disease was compensated initially then disguised by prednisone therapy until the enzyme-inducing effect of rifampicin led to his presentation with overt adrenal insufficiency. Subsequently primary adrenal insufficiency was confirmed by the findings of aldosterone and cortisol deficiency, markedly raised plasma renin and ACTH values, and the appearance of nodular adrenals typical of tuberculous gland involvement on a CT scan.

Hydrocortisone 100 mg IV immediately and 8 hourly led to a water diuresis. Acute delirium developed when the serum sodium was 122 mmol/L and resolved 18 hours later when the level was 116 mmol/L.

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