Letters to the Editor

Rhabdomyolysis associated with cranial diabetes insipidus

Sir,
I read with interest the recent article by Dr Kung and colleagues1 documenting rhabdomyolysis in association with a hypernatraemic hyperosmolal state secondary to cranial diabetes insipidus. We recently reported a patient with rhabdomyolysis and acute renal failure who had developed hypernatraemic hyperosmolality whilst on lithium therapy, presumably as a result of lithium-induced nephrogenic diabetes insipidus.2 These cases serve notice that non-traumatic rhabdomyolysis may occur in hyperosmolal states other than those associated with hyperglycaemia, as previously reported,3,4 and consequently that the hyperosmolal state per se predisposes to rhabdomyolysis. However, the cause of rhabdomyolysis in these cases remains uncertain. Concomitant deficiency of potassium and phosphate ions, also recognized predisposing factors for rhabdomyolysis,5 may be relevant, but masked by their release from damaged muscle cells. Singh et al.6 found that hyperosmolal diabetic patients developing rhabdomyolysis had lower serum potassium concentrations than those who did not, and we noted relatively low serum potassium and phosphate concentrations in our patient at presentation.2 Experimental hypokalaemia has been associated with both reduced transmembrane electrical potential and histological damage in muscle cells,6 and similarly intracellular phosphate deficiency can lead to a fall in transmembrane potential since reduced ATP levels will lead to inhibition of the energy-dependent sodium pump. Hence, hypokalaemia and hypophosphataemia may be the significant biochemical derangements leading to muscle cell injury.

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References

Hyponatraemic encephalopathy complicating thiazide reserpine preparation

Sir,
We would like to report a case of hyponatraemic encephalopathy due to excessive water drinking in association with a thiazide diuretic.

Our patient was a 54 year old man who was admitted in an acutely confused state. Physical examination revealed no focal neurological deficit. The blood pressure was 130/80 mmHg and the pulse rate was 100/min and regular. He was clinically normovolaemic with a copious urine output of about 3 litres in 12 hours. Blood biochemistry revealed Na 114 mmol/l, K 1.9 mmol/l, Cl 79 mmol/l, serum osmolality 250 mosmol/kg, urine osmolality 68 mosmol/kg, spot urine Na 34 mmol/l, and spot urine K 32.1 mmol/l.

A detailed history was obtained from the relatives about 12 hours later. The patient was an anxious individual, who had been taking Adelphane Esidrex on and off for years (one tablet of Adelphane Esidrex consists of 0.1 mg reserpine, 10 mg dihydralazine sulphate and 10 mg hydrochlorothiazide). He had recently been under great stress from his employer and from his family and combated his anxiety by drinking about 8 litres of water a day. He also became over-concerned with his health, and insisted on a low salt diet.

The diagnosis was hyponatraemic encephalopathy due to psychogenic polydipsia and thiazide diuretic. He was treated by withdrawing the diuretic and restricting the water intake to 500 ml/day. 100 mmol of 0.9% NaCl was given over 1 hour. Over 3 days, the serum sodium rose to 132 mmol/l, and the chloride to 106 mmol/l. He recovered completely.

To our knowledge, there are twelve published cases of hyponatraemic encephalopathy caused by psychogenic polydipsia combined with a thiazide diuretic.1–9 However, all these concerned patients who had a specific neurological or psychiatric illness – chronic schizophrenia in 9 patients, epilepsy complicating structural brain disease in 2 patients and psychotic depression in 1 patient. Our patient, however, had no identifiable psychiatric illness, apart from an anxiety-prone personality.

Most reports of thiazide and polydipsia induced hyponatraemia have a favourable outcome, but one reported case involved a 56 year old patient who died due to cerebral oedema with herniation.4 Thus clinicians should be alert to hyponatraemia in patients taking thiazides, especially in those at particular risk: e.g. elderly, cirrhotic, and psychiatric patients.

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Pulmonary geotrichosis

Sir,

Saprophytic fungi are known to produce opportunistic infections in immuno-suppressed individuals. Geotrichosis is a mycotic infection with oral, intestinal, bronchial or pulmonary lesions, caused by the ubiquitous fungus Geotrichum candidum.1 Pulmonary involvement simulating tuberculosis is frequently reported.2 A case of geotrichosis in an old tuberculous lung cavity occurring in an immunocompetent individual is reported.

A 45 year old male presented with cough and streaky haemoptysis of one week duration. He had been treated six years previously for pulmonary tuberculosis for a year. Examination revealed an ill looking male with extensive physical signs in the right chest. Chest X-ray showed a fibrocavitatory lesion in the right upper zone and apicogram revealed a large cavity containing a solid density mass in the right upper lobe.

Geotrichum candidum was isolated in all 10 freshly expectorated sputum specimens. The particular features of G. candidum were absence of urea utilization, assimilation of glucose and galactose but not maltose, sucrose, salicin, inositol or raffinose, thus differentiating from genus Trichosporon. The patient responded to oral administration of a supersaturated solution of potassium iodide for 3 months.

Geotrichum candidum, which belongs to the class Fungi imperfecti, is an opportunistic human pathogen.3 The repeated isolation of G. candidum with characteristic arthrospores and hyphae in freshly expectorated sputum samples and the absence of other pathogenic fungi or bacilli either by direct microscopy or culture of sputum confirms the diagnosis of geotrichosis.2,3 Radiologically, there may be patchy or fluffy infiltrates with a predilection for the upper lobes and, occasionally, cavity lesions (as in our patient). Although use of neomycin and colistin is anecdotal, nystatin and iodide preparations have been used commonly.3,4 Miconazole, clotrimazole, amphotericin B and 5-fluorocytosine have been shown to have in vitro activity at attainable concentrations.5

References


Urinary bladder carcinoma initially manifested as brain metastases

Sir,

Only 1% of transitional cell carcinomas (TCC) of the bladder give rise to brain metastasis throughout their natural history.1,2 Similarly, a bladder origin has been discovered in only approximately 1% of patients with cerebral metastases.3 We describe two cases of bladder carcinoma, the clinical presentation of which was solely due to the presence of brain metastases.

Case 1

A 67 year old male developed complete homonymous hemianopia, visual agnosia and a brief history of disorientation. Physical examination showed left supraclavicular adenopathy and tomographic (CT) scan confirmed a left occipital mass. Gland biopsy showed undifferentiated carcinoma. Further investigations revealed infiltrating bladder cancer with local and distant lymphadenopathy. There were no previous urological symptoms and haematuria only developed later. Autopsy gave histological confirmation for both TCC of the bladder and cerebral metastases.

Case 2

A 54 year old male, with a perineal urethral orifice following urethroplasty, complained of multiple urinary stones and urinary tract infection. He complained of frequent bifrontal headaches that awoke him during the night, and emotional lability and undue irritability. Generalized seizures and weakness of his left arm developed. Papilloedema was discovered and a small right periventricular mass that enhanced with contrast

References

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