Extreme hypernatraemia in association with renal failure following caecocystoplasty

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Summary

A case of extreme hypernatraemia (serum sodium 212 mmol/l) occurring in association with renal failure following a caecocystoplasty procedure is reported. The causative factors in extreme hypernatraemia are reviewed and an unusual reabsorptive mechanism via the transposed intestinal segment is proposed to explain the degree of hypernatraemia present in this case.

KEY WORDS: hypernatraemia, renal failure, caecocystoplasty.

Introduction

Hypernatraemia is a rare disorder, one recent study (Daggett *et al.*, 1979) identifying only 20 cases out of 16,244 hospital admissions. Cases in excess of 200 mmol/l are extremely scarce, usually fatal, and almost always the result of salt poisoning (Goldszer and Coodley, 1979). To our knowledge, no previous report of such a degree of hypernatraemia exists in the British literature.

Case report

The patient, a 75-year-old woman, first presented with an 8-month history of urinary incontinence. Her plasma biochemistry at this time demonstrated a slight elevation in urea but was otherwise normal. Urine culture was sterile but an intravenous urogram (IVU) showed marked bilateral hydronephrosis and distal ureteric hold up which was seen at cystoscopy to be due to a fibrosed, shrunken bladder of very small capacity. Biopsy demonstrated an ulcerative cystitis with chronic inflammatory cell infiltrate. Her symptoms improved on emepronium bromide therapy and a repeat IVU showed marked decrease in the obstructive features. Ten months after first presentation, she was readmitted with worsened incontinence

and plasma biochemistry consistent with early renal failure which however returned to normal with fluid therapy alone in a few days. Repeat cystoscopy confirmed the previous findings and, therefore, a caecocystoplasty was performed 1 year after first presentation to increase her bladder capacity.

Recovery was uneventful and she was allowed home 18 days postoperatively with sterile urine and, apart from a marginally elevated urea, virtually normal plasma biochemistry. However 17 days later she was readmitted comatose, dehydrated and peripherally shut down. Her plasma biochemistry was profoundly abnormal with gross elevation of sodium and chloride and established renal failure: plasma sodium 212, potassium 8.1, bicarbonate 11, chloride 151, urea 68, creatinine 0.718, glucose 4.4 mmol/l. Despite attempted resuscitation she died several hours later.

Discussion

Most cases of extreme hypernatraemia are associated with accidental sodium overload (Goldszer and Coodley, 1979), either orally, usually in hyperosmolar infant feeds (Fineberg, Kiley and Luttrell, 1963) but occasionally with saline emetics, or parenterally as a complication of saline abortion or misguided electrolyte therapy. Profound dehydration may also cause severe hypernatraemia, usually in children (Fineberg, 1973) and rarely in adults. Less severe hypernatraemia may occur in a variety of conditions causing a greater loss of water than of salt such as diabetic ketoacidosis, gastroenteritis, the diuretic phase of acute tubular necrosis and dialysis imbalances.

In this case renal failure and dehydration will undoubtedly have contributed to sodium retention and a tendency to retain sodium will have been promoted by any residual urinary obstruction (Landsberg, 1970; Gold and Roxe, 1979). However,

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these factors do not appear adequate to explain the degree of hypernatraemia present—even in combination—on the basis of previous levels reported. Reabsorption of electrolyte is well described in ureterosigmoid anastomosis (Lowe, Stowers and Walker, 1959) and in ileal ureteric replacement (Goodwin, Winter and Turner, 1959). Studies of electrolyte transport in bladders augmented with ileal segments have shown free passage of electrolyte across the new bladder wall (Pyrah *et al.*, 1955) and caecum will actively reabsorb sodium (Ganong, 1977).

Whilst this case is remarkable therefore by virtue of the sodium level recorded, it appears reasonable to propose an unusual possible mechanism which would seem to best explain the degree of sodium retention present. We suggest that equilibration of urinary to plasma sodium concentration across the caecal bladder wall segment, tending always to elevate the serum sodium towards the urine concentration and possibly beyond, could have occurred producing an excessive sodium load for the patient's impaired renal function. It is interesting to note that caecocystoplasty is not recommended in patients with impaired renal function (Skinner and Goodwin, 1975). Presumably, in normal individuals high fluid intake and adequate reserves of renal concentrating power would overcome such a sodium back diffusion effect which would not in any case become significant until some event produced a requirement for concentrated urine and therefore a need for high renal sodium excretion.

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