

Cerebral oedema associated with hyponatraemia in renal failure

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SUCCESSFUL treatment of cerebral oedema depends upon establishing its cause. We report the treatment of a case of presumed cerebral oedema, which appeared to have resulted from hyponatraemia.

Case report

A 47-year-old woman was admitted to the Royal Sussex County Hospital on 11 June 1968 in renal failure due to chronic pyelonephritis.

Investigations. Hb 52%, WBC 7200/m³, blood urea 346 mg/100 ml, creatinine clearance 4.2 ml/min, serum sodium 140 mEq/l, calcium 8.0 mg/100 ml, phosphate 12.2 mg/100 ml, alkaline phosphatase 16.0 KA Units; urinary sodium 26 mEq/l, potassium 22.3 mEq/l, urea 870 mg/100 ml, albumin 0.02 g/100 ml. A catheter specimen of urine cultured *Ps. pyocyaneus* and *Esch. coli*. Urinary output was 1–2 l/day. She responded well to treatment with peritoneal dialysis, sodium restriction and antibiotics.

In August 1968, however, she was readmitted in cardiac failure, conscious, but drowsy. Blood pressure 160/100 mmHg. Her investigations were as follows: blood urea 370 mg/100 ml, serum sodium 150 mEq/l, potassium 5.0 mEq/l, alkali reserve 10 mEq/l, urinary sodium 77 mEq/l, potassium 6.1 mEq/l, urea 430 mg/100 ml, blood glucose 58 mg/100 ml. There was again a heavy growth of *Ps. pyocyaneus* in the urine. She was treated with peritoneal dialysis for 2 days, and she improved initially. Peritoneal dialysis was discontinued after 2 days. Thereafter, however, her serum sodium fell, and on the morning of the fourth day after admission it was 126 mEq/l. During the evening she had grand mal fits and became deeply unconscious, and artificial ventilation was required. Her condition deteriorated rapidly, the pupils were fixed and dilated, and she developed papilloedema. At that time serum sodium was 122

mEq/l, potassium 5.0 mEq/l; blood urea 150 mg/100 ml, alkali reserve 14 mEq/l, and blood glucose 325 mg/100 ml.

Treatment. An arterio-venous shunt was inserted and a litre of 2N saline and 500 ml 10% mannitol were given intravenously and 2.5 l of fluid simultaneously removed by haemodialysis against a high venous pressure. She received a total of 875 mEq of sodium over the next 24 hr.

By the following morning she was conscious and breathing spontaneously; the pupils reacted normally. Thereafter she made an uneventful recovery and has since done well on chronic intermittent haemodialysis and sodium supplements (she is a salt-loser) for 2 years.

Discussion

Occasional patients with chronic renal failure, particularly those with pyelonephritis, may have high urinary sodium loss due to failure of sodium reabsorption. In such patients a low salt diet may result in hyponatraemia. Failure to recognize this situation may precipitate extracellular hyponatraemia with relative intracellular hyperosmolarity leading to a shift of water into the cells. Treatment was therefore directed towards reversing intracellular hyperosmolarity by extracting water from the cells to the extracellular compartment by means of infusion of hypertonic saline, expanding the plasma volume with mannitol 10% and finally removing water by haemodialysis against a high venous pressure. The rapid improvement in her condition with such a therapy suggested the above presumption may have been correct.

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