Efficacy of peritoneal dialysis in severe thiazide-induced hyponatraemia

T. CUNDY
M.A., M.R.C.P.

J. A. P. TRAFFORD
F.R.C.P.

Department of Medicine, Royal Sussex County Hospital, Brighton, Sussex

Summary
A patient with severe thiazide-induced hyponatraemia (plasma sodium 99 mmol/l) is described, who had suggestive signs of cerebral oedema. Co-existent problems of cardiac and respiratory insufficiency were thought to make treatment with hypertonic saline hazardous. A 40-hr peritoneal dialysis successfully reversed both hyponatraemia and the associated cerebral signs, plasma sodium increasing at a rate of 0·83 mmol/hr. Peritoneal dialysis appears to be a safe and efficacious method of treating severe thiazide-induced hyponatraemia.

Introduction
Hyponatraemia may occasionally be of such severity as to cause disturbance of consciousness and threaten life. In such circumstances conventional therapy with water deprivation or demeclocycline may be too slow and patients with cardiac, respiratory or renal insufficiency may be at risk from the infusion of hypertonic saline. The authors now report the use of peritoneal dialysis to reverse severe hyponatraemia.

Case report
The patient was a 62-year-old Edinburgh man whose previous history included emphysema and hypertension. His chest disease had forced his premature retirement from work in 1977. He had been investigated in 1977 for hyponatraemia which had been attributed to bendrofluazide therapy and, following the withdrawal of diuretics, his blood pressure (BP) had been controlled with methyldopa and hyponatraemia had not recurred. While on holiday in Brighton he was admitted to hospital with a one week history of disturbed behaviour, increasing unsteadiness of gait and a deteriorating level of consciousness. Four weeks before admission he had been restarted on bendrofluazide for his hypertension. On examination his consciousness was impaired and he was responding only to commands. He had symmetrical pyramidal rigidity affecting all limbs, upgoing plantar responses and nystagmus on lateral gaze. His BP was 160/90 mmHg and jugular venous pulsation was visible 2 cm above the sternal notch. There was no peripheral oedema. He had clinical evidence of severe obstructive airways disease. Investigations revealed that he had severe hyponatraemia (plasma sodium 99 mmol/l) and hypokalaemia (plasma potassium 2-9 mmol/l). The plasma osmolality was low (213 mmol/kg) and his plasma creatinine was elevated (130 μmol/l). The urine sodium content (75 mmol/l) and osmolality (534 mmol/kg) were inappropriately high.

The chest X-ray and electrocardiogram showed changes typical of severe bullous emphysema with pulmonary arterial hypertension. A 9 a.m. plasma cortisol was elevated (1089 nmol/l). Computerized tomography of the brain revealed an extensive left-sided occipital infarct.

In view of his hypertension, renal, cardiac and pulmonary insufficiency it was decided that hypertonic saline infusion would be too hazardous and so it was decided to dialyse him. Peritoneal dialysis was begun with a dialysate containing glucose 75-6 mmol/l (13-6 g/l), sodium 140 mmol/l and potassium 4-4 mmol/l and continued for 40 hr. Dialysis induced a negative fluid balance and corrected the hyponatraemia, plasma sodium rising at the rate of 0·83 mmol/l/hr. (Fig. 1). His BP fell during dialysis to 110/60 mmHg but his urine output was maintained at an average 40 ml/hr. The hypokalaemia was unaffected by peritoneal dialysis, despite increasing the dialysate potassium to 5 mmol/l. Spironolactone, 50 mg daily, was given from the third day of his admission and corrected the hypokalaemia.

Following the correction of hyponatraemia his conscious level improved and the neurological signs resolved. He developed a chest infection and respiratory failure 3 days after admission but this was successfully treated with positive pressure ventilation, physiotherapy and antibiotics. A further chest infection and respiratory failure developed 10 days later, but by this time detailed knowledge of the severity of his chest disease had been received from Edinburgh and it was decided not to ventilate him again. He died 17 days after admission. A post-mortem examination revealed severe bullous emphysema with broncho-pneumonia and right ventricular hypertrophy, and confirmed the occipital lobe infarction. There was no evidence of malignancy.
Discussion

Although pulmonary, cerebral and renal disease may have been contributory, the onset of his severe hyponatraemia seemed to be related to the re-introduction of bendrofluazide therapy. The history of hyponatraemia during previous exposure and its reversibility once the drug was discontinued also suggests that thiazide therapy was responsible. This complication of thiazides is well recognized and seems to occur particularly when hypokalaemia is present (Fichman et al., 1971).

Whatever the cause of his hyponatraemia, it was apparent that it required correction. The occurrence of cerebral signs in hyponatraemia is thought to be related to intracellular electrolyte disturbances and cerebral oedema, and is associated with a high mortality (Arieff et al., 1976). The severity of clinical manifestations depends in part upon the absolute level of serum sodium and in part upon the rate at which hyponatraemia develops. This patient, in whom profound hyponatraemia had developed over a short period, clearly had severe cerebral signs.

Controversy exists over the rate at which hyponatraemia should be reversed in these circumstances. There is concern that too rapid a reduction of brain swelling may cause cerebral venous bleeding (Levin, 1978). On the other hand, the severity of this patient’s condition suggested that correction of his hyponatraemia should not be unduly delayed. Conventional treatment for severe hyponatraemia with hypertonic saline may be hazardous in patients with cardiac or respiratory disease, whilst water deprivation and demeclocycline therapy may take several days to produce maximal effects (De Troyer and Demanet, 1975). Haemodialysis and peritoneal dialysis will reverse electrolyte abnormalities in patients with renal failure, and peritoneal dialysis has also been used in the management of acute hypercalcaemic crisis (Heyburn et al., 1980). Although some authorities mention dialysis as a possible treatment for severe hyponatraemia, there are few reports attesting to its efficacy, particularly in patients with adequate renal function (Baylis, 1980). The authors chose to dialyse using the peritoneal route in this case in order to achieve a more gradual correction of hyponatraemia than would have been obtained with haemodialysis. The resolution of the cerebral signs once hyponatraemia was corrected suggests that in this patient peritoneal dialysis was both safe and efficacious.

Although the outcome of this case was ultimately unsuccessful the correction of hyponatraemia allowed time to gather valuable information about the patient’s previous history. It is possible that the chest infection sustained after peritoneal dialysis was precipitated by the procedure itself, and this may be an important hazard with this form of treatment since many patients with severe hyponatraemia have chest disease (Thomas et al., 1978).

References


