Letters to the Editor

Hyponatraemia associated with the use of a selective serotonin-reuptake inhibitor in an older patient

SIR—Older patients are at increased risk of developing hyponatraemia due to age-related changes in osmoregulation, medical co-morbidity and concomitant drug use [1]. We report asymptomatic severe hyponatraemia secondary to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) in an older patient following head trauma and after prescription of a selective serotonin-reuptake inhibitor.

A 78-year-old woman was admitted to hospital because of a collapse related to severe aortic stenosis, which was complicated by trauma to the head. Her medical history revealed osteoarthritis, treated with meloxicam 7.5 mg daily. On day 18 after admission, paroxetine 20 mg daily was started for depressive symptoms. Twelve days after the prescription of paroxetine, routine laboratory assessment demonstrated that her serum sodium concentration had fallen to 109 mmol/l (it had been 134 mmol/l on admission). Mental state and cognitive function were normal. Physical examination did not reveal any new abnormalities. Serum osmolality was 226 mosm/kg (normal 280–300 mosm/kg), urine sodium excretion was 81 mmol/24 h and urine osmolality was 443 mosm/kg (normal 200–1200 mosm/kg). Renal, liver and thyroid function tests were normal. Computerized tomography of the brain showed no damage to the hypothalamic-pituitary area.

Paroxetine was discontinued and fluid intake was restricted to 500 ml/24 h. The following day, the urinary sodium excretion dropped sharply. The plasma sodium concentration gradually improved (Figure 1). The serum sodium concentration remained normal after stopping fluid restriction. Prescription of another antidepressant appeared unnecessary.

In our patient, the hyponatraemia in combination with the reduced serum osmolality and the high sodium excretion indicated SIADH, either due to cerebral injury caused by the trauma, or the use of paroxetine. Given the normal computerized tomography of the brain, the instant drop of urine sodium excretion after withdrawal of paroxetine and the persistently normal serum sodium concentration after discontinuing fluid restriction, a causal relationship with paroxetine is most probable. In addition, the use of a nonsteroidal anti-inflammatory drug may have contributed to the development of hyponatremia [2], although a direct relationship with serotonin-reuptake inhibitor-induced hyponatremia could not be demonstrated [3].

Hyponatraemia secondary to SIADH is a relatively common side-effect of serotonin-reuptake inhibitor treatment in older patients, with an incidence of about 25% [4], half of which are symptomatic. Risk factors include advanced age, comorbid use of diuretics and

Figure 1. Serum sodium concentrations (O; mmol/l, left scale) and urine sodium excretion (■; mmol/24 h, right scale) following discontinuation of paroxetine and fluid restriction.

low body weight [3, 5]. The serotonin-reuptake inhibitor-associated hyponatraemia generally develops within the first month of treatment (range 3–120 days) [5]. As this side-effect is potentially life-threatening, prescription of antidepressants to patients admitted to hospital should be carefully considered, and monitoring of the serum sodium concentration during the first month of treatment is recommended [3–5]. However, if the clinical condition of a patient deteriorates while taking a serotonin-reuptake inhibitor, hyponatraemia should be considered—irrespective of the duration of treatment.

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