Sertraline-induced hyponatraemia

Dear Editor,

The presence of depression in medical inpatients seems to vary from 26% to 32%. Because of the possible negative impact of psychiatric co-morbidity on the medically ill patient, the adequate treatment of depression in this setting is important. Although selective serotonin reuptake inhibitors (SSRIs), as a class, are the most frequently used antidepressants due to their better tolerability and safety in case of overdose, some SSRI-related adverse effects might be particularly serious.

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) accompanied by hyponatraemia is a potentially fatal complication of SSRI use. We report here the case of an elderly man who developed hyponatraemia when treated with sertraline.

An 81-year-old, Caucasian, recently widowed man was admitted for treatment of a parotid abscess and chronic anaemia without other clinical co-morbidities. During his hospitalization, a depressive episode was diagnosed. Treatment with sertraline (50 mg/day) was initiated with good tolerability. After 9 days of SSRI use, there was a progressive decrease in plasma levels of sodium, which reached 118 mEq/L on day 13 (normal values, 135-145 mEq/L), and the patient presented mild somnolence (Figure 1). Because no other potential clinical cause of the hyponatraemia was found, the antidepressant was suspended and water restriction was prescribed. One week after the SSRI had been discontinued, sodium plasma levels began to rise (normal values were achieved by the second week), and the level of consciousness improved. During the hyponatraemia episode, the mood disorder was treated with daily supportive psychotherapy.

The use of various medications has been associated with hyponatraemia (Table 1). Antipsychotics, benzodiazepines and promethazine are some of the psychotropic drugs associated with this clinical complication. Fluoxetine was the first antidepressant to be associated with hyponatraemia, but other SSRIs have since been implicated. Although considered a rare event (5.4 per 1000 elderly patients), incidences up to 7.7% have been reported. This discrepancy in the prevalence rates might be related to under-diagnosis, as low plasma sodium can be clinically asymptomatic.

One of the possible mechanisms associated with antidepressant-induced SIADH is the serotonin stimulatory effect of antidiuretic hormone, which is mediated by the 5HT2 and 5HT1 receptors.

In our patient, the hyponatraemia was probably associated with the sertraline use. Its occurrence at 9 days after the antidepressant treatment was initiated (other authors have reported that hyponatraemia occurred after a median of 13 days of SSRI use, together with the return of normal sodium plasma levels after the SSRI had been discontinued, reinforces the causality hypotheses. In addition, the advanced age of the patient might have favoured the antidepressant-induced SIADH by increasing antidiuretic hormone secretion and the response to osmolar stimuli.

Antidepressant use must be continuously monitored, especially in patients with accompanying clinical diseases, in the elderly and in the case of polypharmacy. Hyponatraemia resulting from SSRI use seems to be an under-diagnosed event. Commonly, discontinuation of the drug results in an improvement of the laboratory test results for sodium. Nevertheless, reintroduction of the same or another SSRI is a step that should be taken with caution due to the risk of hyponatraemia recurrence.

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References