Severe hyponatremia associated with escitalopram

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ABSTRACT

Hyponatremia is a rare but potentially fatal complication of selective serotonin reuptake inhibitor (SSRI) therapy with only limited cases of escitalopram as the causative drug. We report the case of a 54-year-old hypertensive female who was admitted to the hospital with seizure episode and subsequently diagnosed to have severe hyponatremia due to SSRI-induced syndrome of inappropriate antidiuretic hormone (SIADH) with the cause attributed to the short history of intake of escitalopram for depression. All SSRIs, including escitalopram, can cause SIADH and should be used with caution in the depressive patients with regular monitoring of electrolytes, especially in the elderly.

Keywords: Escitalopram, hyponatremia, syndrome of inappropriate antidiuretic hormone, selective serotonin reuptake inhibitor

Introduction

Hyponatremia is a known but rare adverse effect of selective serotonin reuptake inhibitor (SSRI) with the incidence varying from 0.5% to 32% in the literature,¹ and is now being increasingly recognized due to increased use of SSRI as the first line treatment of depression.² The hypertensive patients are also advised for salt restricted diet, which may further contribute to the acute fall in the serum sodium levels. This association of hyponatremia due to SSRI has been usually reported in elderly patients (above 60 years age).³ To the authors’ knowledge, this is the second case being reported in a middle-aged female from the Indian subcontinent, the first being reported by Kar and Sharma in 2014.⁴

Case Report

A 54-year-old Indian female presented to emergency with a history of generalized tonic-clonic seizure at home, which was controlled with intravenous midazolam by the ambulance doctor. She was managed in the Intensive Care Unit. Her medical history included recent diagnosis of hypertension and depression for which she was on tablet telmisartan (anti-hypertensive), tablet escitalopram (SSRI), and salt restriction for last 3–4 days. There was no history of a headache, fever, or vomiting. Magnetic resonance imaging of the brain was done to rule out stroke, which was normal. On examination, she was drowsy, moving all limbs on pain, but not following commands and no nuchal rigidity with both pupils equal in size and reacting to light. Her blood pressure 150/80 mmHg, pulse 80/min, random blood sugar 200mg%, no pedal edema, normal jugular venous pulse, chest-bilaterally clear, heart sounds normal with no murmurs and the initial blood investigation showed severe hyponatremia of serum sodium- 116 mmol/L. Her further investigations revealed low serum osmolality of 254 mOsm/kg of water, urinary osmolality of 537 mOsm/kg of water, and the urinary sodium concentration was 119 mmol/L. Her liver function test, renal function test, echocardiogram, thyroid function test, serum lipid profile, cerebrospinal fluid study, and cortisol level was normal. A diagnosis of the syndrome of inappropriate antidiuretic hormone secretion (SIADH) was made and her serum sodium levels improved gradually with fluid restriction and discontinuation of escitalopram. The patient recovered neurologically with no focal deficit and stable hemodynamics, over the next 2 days and was transferred to a ward. The use of

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escitalopram along with salt restricted diet with the absence of other etiological factors causing hyponatremia and its subsequent correction after stopping escitalopram, suggested a cause-and-effect relationship.

Discussion

The hyponatremic effect is attributed to SSRI-induced release of antidiuretic hormone, leading to the SIADH.\[3\] SIADH is characterized by a low serum sodium concentration (<135 mmol/L), urinary osmolality exceeding 200 mOsm/kg, a urinary sodium concentration exceeding 20 mmol/L, and serum osmolality of <280 mOsm/kg.\[5\] The main modality of management in SIADH is water restriction, although in severe symptomatic cases hypertonic saline (3%) and drugs such as loop diuretics (frusemide), demeclocycline, and vaptans can be used.\[5\] Escitalopram is an active S-enantiomer of citalopram and is a highly selective and potent SSRI. The hyponatremia secondary to SSRI treatment normally occurs usually within the first 2 weeks after its initiation and is not dose dependent, with the serum sodium levels normalizing 2–20 days after stopping the drug.\[6\] There are only a few cases reported in the literature of hyponatremia due to escitalopram-induced SIADH, with the risk factors being elderly, female patients, patients on concomitant treatment with other medicines that may cause hyponatremia (thiazide diuretics), poor oral intake or malnourished, and the most common SSRI-associated with hyponatremia is fluoxetine.\[1,6,7\] Escitalopram is considered safest in regard to the adverse effect among the SSRI group.

Conclusions

This case report illustrated a known but rare adverse effect of a commonly used SSRI antidepressant escitalopram. The primary and secondary care physicians need to be aware of this adverse effect which can be potentially life-threatening while prescribing SSRI, which are now widely used drugs for depression, particularly in the elderly. A protocolized management, including regular electrolyte measurements, should be followed during the subsequent visit of the patient.

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Conflicts of interest

There are no conflicts of interest.

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