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SEVERE HYPONATREMIA DUE TO ESCITALOPRAM TREATMENT IN AN ELDERLY ADULT WITH ALZHEIMER'S DISEASE

To the Editor: Hyponatremia is the most common electrolyte imbalance in all age groups, with a greater frequency in elderly adults.[1] Hyponatremia may cause nausea, vomiting, headache, loss of appetite, falls, and difficulty maintaining attention. Hyponatremic encephalopathy occurs when serum sodium concentration is <115 mmol/L and may cause serious neurological symptoms such as loss of consciousness, epileptic seizure, and coma unless treated promptly.[2] Numerous studies have revealed that drugs are one of the most common causes of hyponatremia in elderly adults.[1]

Behavioral and psychotic symptoms are frequently encountered in individuals with Alzheimer's disease, and selective serotonin reuptake inhibitors (SSRIs) are frequently recommended for the management of these symptoms and for the treatment of depression.[3] SSRIs are first-line drugs for the treatment of geriatric depression owing to their wide margin of safety, easy dose titration, and low rates of anticholinergic and cardiovascular adverse events.[4] Escitalopram is one of the most recently developed SSRIs; elderly adults tolerate it well, and it is effective in symptom control. Although SSRI-induced hyponatremia is well known, few cases of serious hyponatremia due to SSRI use have been reported.[5]

CASE

A 76-year-old woman was admitted to the emergency service with impairment in speech, walking, and perception that had begun 2 hours earlier. Her relative expressed that she had urinary incontinence, recurrent falls, and loss of appetite; was unconcerned about the people around her; and had been mixing up the rooms in the house for 3 days. Her medical history revealed hypertension, diabetes mellitus, Alzheimer's disease, and sleep disorder, and she had been taking losartan 50 mg, sitagliptin 100 mg, rivastigmine 18 mg patch, memantine 20 mg, and trazodone 100 mg for the last 9 months. Nearly 4 weeks before, she had been diagnosed with depression in the geriatric clinic, presenting with crying, anxiety, and irritability, and escitalopram 10 mg had been added to her treatment.

Mental examination revealed lethargic consciousness with impaired orientation and cooperation. Vital signs were stable. On neurological examination, deep tendon reflexes were bilaterally decreased, with positive right-sided Babinski reflex. Examination of other systems was normal. Based on these findings, cranial computed tomography was performed and demonstrated cerebral atrophy and chronic ischemic alterations with no acute neurological signs. Biochemical analyses revealed a serum sodium concentration of 113 mmol/L; renal, hepatic, and thyroid functions and blood glucose concentration were within normal ranges. Sodium chloride (3%) infusion was commenced, and speed of infusion was adjusted so that the daily increase in serum sodium concentration would not exceed 10 mmol/L. Escitalopram was discontinued because serum sodium concentration before escitalopram treatment was 138 mmol/L. On the third day of admission, when her serum sodium concentration was 128 mmol/L, lethargy had reduced, she was oriented and cooperative, and her speech had improved. Infusion was stopped during clinical follow-up, and her serum sodium concentration increased to 136 mmol/L, her appetite increasead, and her urinary incontinence was improved. She was discharged with current drug treatments except for escitalopram. During her visits to the outpatient geriatric clinic and according to laboratory analyses over the subsequent 2 months, neither her complaints nor hyponatremia recurred.

DISCUSSION

Severe escitalopram-induced hyponatremia is rarely mentioned in the literature. Moreover, the present case is important as being an individual with Alzheimer's disease. In such a case, healthcare providers and clinicians might easily interpret poor appetite, somnolence, lack of concern about the people around, irritability, and lethargy resulting from hyponatremia due to escitalopram treatment as behavioral symptoms of Alzheimer's disease or as worsening symptoms of existing depression.[3] Thus, hyponatremia might be overlooked, and the dose of the drug could be increased by mistake instead of being decreased, which

could lead to the development of life-threatening hyponatremia. For these reasons, although escitalopram is considered a safe drug, attention must be paid to hyponatremia, as with other SSRIs, in the first month of drug use, particularly in elderly adults. Serum sodium concentration should be checked before starting the drug, healthcare providers should be informed about this potential complication even though it is rare, and serum electrolytes should be rechecked in the event that the clinical picture worsens.[4] Therefore, a serious but easily treatable condition can be diagnosed easily even in the early stages by keeping the risk of escitalopram-induced hyponatremia in mind.

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DIAGNOSIS OF LATE-ONSET TAKAYASU ARTERITIS FOR ELDERLY ADULTS USING FLUORINE-18 FLUORODEOXYGLUCOSE POSITRON EMISSION TOMOGRAPHY/COMPUTED TOMOGRAPHY

To the Editor: An 80-year-old Japanese man presented with a month's history of neck pain, low-grade fever, and fatigue. His medical history included lung tuberculosis that had been treated 15 years before. He had first consulted his primary care physician, and subacute thyroiditis was suspected on the basis of cervical pain, low-grade fever of up to 37.5°C, and high C-reactive protein levels (33 mg/dL; reference range 0–0.3 mg/dL), although thyroid function tests were within normal limits. The cervical pain had persisted for a month, so he presented to the Department of Internal Medicine, National Center for Global Health and Medicine Hospital, for further investigation. At the time of admission, his vital signs were normal, but he reported fatigue and mild pain in the left posterior neck. Goiter, lymphadenopathy, headache, muscle pain, cutaneous lesions, and rashes were not observed, and there was no muscle tenderness or weakness. There was no difference in blood pressure between his arms. The temporal arteries were pulsatile, with no tenderness on palpation, and visual acuity was normal. The chest was clear on auscultation, and heart sounds were regular, with no murmur. The examination was otherwise normal.

Enhanced computed tomography (CT) of the entire body revealed diffuse thickening of the arterial wall in the right and left common carotid arteries, aorta, brachiocephalic artery, and mesenteric arteries. Fluorine-18 fluorodeoxyglucose positron emission tomography/CT (^{18}F-FDG PET/CT) was conducted to detect the focus of inflammation, and strong linear ^{18}F-FDG uptake was observed in the same arteries (Figure 1). He was diagnosed with Takayasu arteritis (TA) before ischemia developed, and corticosteroid treatment was initiated. His symptoms disappeared, and CRP levels returned to the normal range

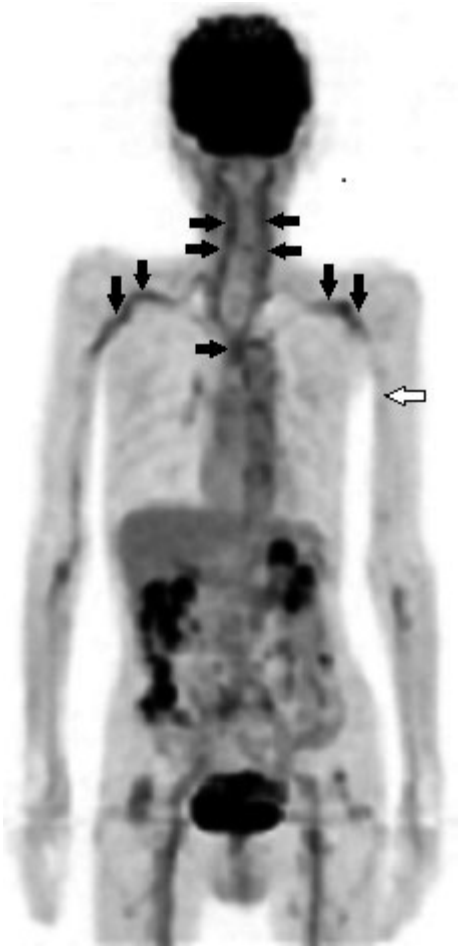

Figure 1. Fluorine-18 fluorodeoxyglucose (^{18}F-FDG) positron emission tomography/computed tomography imaging. The black arrows indicate affected vessels, and the white arrow indicates normal vessels. Strong linear ^{18}F-FDG uptake was observed in the right and left common carotid arteries, aorta, brachiocephalic artery, and mesenteric arteries.