A very uncommon clinical entity: Lansoprazole-induced symptomatic hyponatremia in a young woman

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Dear Editor,

In the literature, it has been postulated that hyponatremia is the most common electrolyte balance disorder in the younger population. Symptomatic hyponatremia owing to proton pump inhibitors is an extremely uncommon condition, especially in young people (1,2). Here, we present a young female adult with symptomatic hyponatremia caused by lansoprazole.

A 31-year-old female patient was admitted to the emergency room with nausea, weakness, and headache. Her medical history revealed sudden cardiac arrest because of Prolonged QT syndrome, followed by cardioversion 6 months ago. Coronary angiography imagery was normal but an operation was performed to insert an implantable cardioverter-defibrillator to correct abnormal heart rhythm. One week ago she was admitted to the cardiology clinic with chest pain, and acute pericarditis was diagnosed. Ibuprofen 200 mg tablet b.i.d., colchium dispert 0.5 mg tablets t.i.d., and lansoprozole 30 mg capsule b.i.d. were prescribed. Her family history was positive for sudden cardiac arrest because of Prolonged QT syndrome. She denied ever drinking alcohol, smoking, or using any kind of herbal or folk remedies. Her physical examination revealed decreased heart sounds in auscultation. Abdominal and neurologic examinations were normal. Serum sodium concentration was 115 mmol/L (135-145 mmol/L), potassium was 4.9 mmol/L (3.5-5.5 mmol/L), serum creatinine was 0.63 mg/dL (0.4-1.2 mg/dL), and chloride was 90 mmol/L (97-107 mmol/L). Blood urea nitrogen concentration and liver function tests were within the normal range. A cardiologist had reviewed her in the emergency room, and just a little cardiac effusion was noticed with echocardiogram; no other emergency cardiac pathology was diagnosed. Her abdominal ultrasonographic examination also revealed no pathology. The patient was then hospitalized.

Thyroid function tests, and adrenocorticotropic hormone and cortisol concentrations were all in the normal range. Blood and urine osmolality tests results were as 261 mOsm/L and 820 mOsm/L. Fluid restriction and fluid monitoring was planned; all medication was stopped except lansoprazole. Hypertonic saline and serum physiologic were given because of severe symptomatic hyponatremia. Despite the hypertonic saline treatment and other precautions, her hyponatremic status continued. Her medical history and medication were reviewed again and lansoprazole treatment was stopped. Within a few days her sodium level increased to normal range. However, during her clinical process, her sodium level dropped once more. She was reassessed, and it was noticed that she had again been given lansoprazole because of dyspeptic symptoms. The lansoprazole capsules were discontinued, and her sodium level increased to normal in a few days just with clinical observation (Figure 1). The patient was discharged for polyclinic follow-ups. She was observed as an outpatient without any medication and two weeks later her blood sodium concentration was 140 meq/dL.

Hyponatremia is the most commonly encountered electrolyte balance disorder in daily practice. This situation is closely associated with several health-threatening conditions, such as impaired cognitive functions, some neurologic disorders, prolonged hospital stay, and unfortunately, increased mortality rates among older patients. Lansoprazole is a commonly used proton pump inhibitor worldwide. Its common adverse effects are nausea, diarrhea, itching, and allergic reactions; however, lansoprazole-associated symptomatic hyponatremia in a young patient is a very uncommon clinical entity (3).

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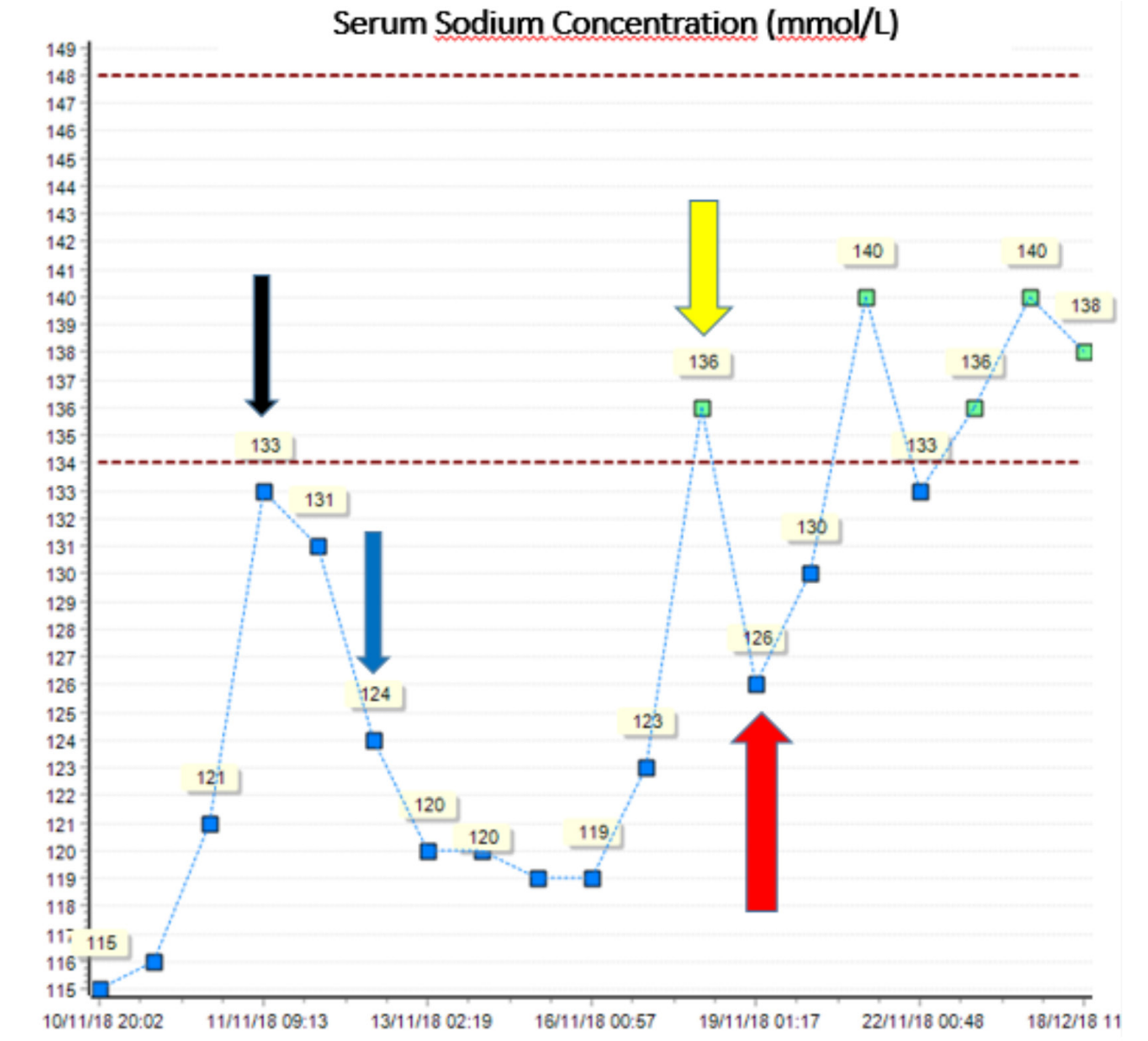

Figure 1. Sodium concentration levels of the patient during clinical progress. Black Arrow: Intravenous 3% NaCl was given to the patient. Blue Arrow: Lansoprazole was stopped. Yellow Arrow: Lansoprazole was given to the patient again because of dyspeptic symptoms. Red Arrow: Lansoprazole was stopped again.

The Naranjo adverse drug reaction scale was used to determine the adverse effect probability of lansoprazole. A score of 8 was reached, suggesting a probable causal relation between lansoprazole and hyponatremia (4). The literature suggests that the syndrome of inappropriate antidiuretic hormone secretion (SIADH) may clarify the mechanism of hyponatremia related to lansoprazole (3). However, the actual explanatory mechanism suggesting lansoprazole-induced hyponatremia remains obscure. We also are not absolutely sure of the explanatory mechanism of lansoprazole-induced hyponatremia in our patient. Though our patient had plasma osmolarity of 261 mOsm/L and urine osmolarity of 820 mOsm/L and also had normal renal, adrenal, and thyroid functions, we may

speculate that the SIADH may clarify the mechanism of hyponatremia related to lansoprazole in our patient.

Falhammar H et al. (5), in a recent study, investigated the association between PPIs and hospitalization owing to hyponatremia. They reported an association between any newly initiated PPI-treatment and hospitalization because of hyponatremia with the exception of lansoprazole. Also, ongoing PPI use was not associated with an increased risk. The first case of lansoprazole-induced symptomatic hyponatremia was reported in 2000 by Fort et al. (6). Ferreira et al. (7) reported pantoprazole-related symptomatic hyponatremia.

In our case, newly initiated lansoprazole was the probable cause of symptomatic hyponatremia, and this is the second case report on lanzoprazole-induced hyponatremia. Thus, physicians should be careful when prescribing lansoprazole to patients even in the young population, and also in patients with systemic comorbidities.

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