Hyponatremia Secondary to Decreased Oral Intake and SIADH and Possibly Exacerbated by Horsetail (*Equisetum arvense*)

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INTRODUCTION

Hyponatremia is defined as serum sodium less than 135 mmol/L and is the most frequent electrolyte abnormality in hospitalized and ambulatory patients.[1] Geriatric patients are especially vulnerable to hyponatremia, which occurs in 4% to 11% of these patients.[1] In up 14% of patients with hyponatremia, the problem may be drug-induced,[1,2] with thiazide diuretics being the most common cause of drug-induced hyponatremia.[1,2] Thiazide-associated hyponatremia most commonly occurs within the first few weeks of therapy but may develop months to years after initiation.[3] Several risk factors are associated with diuretic-induced hyponatremia, such as age, female sex, low body mass, and a low-sodium diet.[4]

The mechanism of drug-induced hyponatremia involves changes in the homeostasis of sodium and water. Drugs can affect water homeostasis by increasing the pituitary secretion of arginine vasopressin, thus potentiating the effect of endogenous hormone at the renal medulla and resetting the osmostat.[1,2] This results in a lower threshold for secretion of arginine vasopressin.[1,2]

Horsetail (*Equisetum*) is a plant native to Europe, North America, western Asia, and northwest Africa.[5,6] There are several different species of horsetail, including *E. arvense*, *E. bogotense, E. giganteum, E. hyemale*, *E. myriochaetum*, and *E. telmateia*.[5,6] Horsetail has traditionally been used in Europe as an oral diuretic for the treatment of edema.[5,6] The German Commission E expert panel has approved one specific species of horsetail (*E. arvense*) for this indication.[6] A small, double-blind, randomized placebo-controlled trial involving healthy male volunteers suggested that *E. arvense* has diuretic properties similar to those of hydrochlorothiazide.[7] The mechanism of action for diuresis is not entirely clear, but this effect may be attributable to flavonoids, phenolic compounds, and mineral constituents.[5,6] There is little clinical research evaluating the efficacy and safety of horsetail. When taken orally, horsetail has been reported to cause mild gastrointestinal side effects: abdominal distention, increased frequency of bowel movements, and nausea.[5] Horsetail has also been associated with seborrheic dermatitis, pancreatitis, liver failure, headache, and thiamine deficiency.[5,6]

We report a case of hyponatremia attributed to decreased oral intake and syndrome of inappropriate secretion of antidiuretic hormone (SIADH; due to nausea), possibly exacerbated by the diuretic effect of horsetail (*E. arvense*).

CASE REPORT

A 70-year-old woman presented to the emergency department with nausea and fatigue.* The patient had had decreased oral intake for 2 days before presentation. Her medical history included hypertension, hypertrophic cardiomyopathy, anxiety, and compression fractures of the thoracic spine. The patient's home medications included olmesartan 40 mg PO daily, zopiclone 7.5 mg PO at bedtime, amlodipine 10 mg PO daily, fluocinonide 0.05% cream daily as needed, and lorazepam 0.5 mg PO daily as needed. The only recent change in medication was an increase in the dosage of amlodipine, from 5 mg daily to 10 mg daily, which occurred 30 days before presentation. The patient stated that she had been taking natural supplements for approximately 10 years (Table 1). The patient reported that she did not drink alcohol or use recreational drugs and that she was a current smoker of approximately 40 years' duration. Six months before presentation, the serum sodium was 132 mmol/L.

The patient was 163 cm tall and weighed 56 kg. Her vital signs upon presentation were as follows: blood pressure 142/78 mmHg, heart rate 73/min, temperature 36.9°C, respiratory rate 18/min, and oxygen saturation 96% on room air. The physical examination showed very mild pitting edema to the ankles bilaterally. Other physical findings

*The patient gave informed consent to publish the case report.

TABLE 1. Natural Supplements before Admission

Product	Dose and Route
Apple cider vinegar 500 mg/chromium 2.2 µg	1 tablet PO daily
Vitamin K_2 120 µg/cholecalciferol 1000 units	1 tablet PO daily
Vitamin C 500 mg	1 tablet PO daily
Horsetail (*Equisetum arvense*) 15 mg	1 tablet PO daily
Multivitamin	1 tablet PO daily
Cholecalciferol 1000 units	1 tablet PO daily
Vitamin B_{12} 1000 µg	1 tablet PO daily
Kelp (iodide 553 µg)	1 tablet PO daily
Calcium carbonate 230 mg/magnesium 70 mg/vitamin D 200 units	2 tablets PO twice daily
Flax seed 1000 mg/α-linoleic acid 530 mg/oleic acid 137 mg/linoleic acid 120 mg	1 tablet PO daily

TABLE 2. Summary of Laboratory Values on Admission

Laboratory Parameter	Measured Value	Reference Range
Serum		
Sodium	113 mmol/L	133–145 mmol/L
Potassium	3.8 mmol/L	3.5–5 mmol/L
Chloride	82 mmol/L	98–111 mmol/L
Creatinine	47 µmol/L	40–100 µmol/L
Osmolality	236 mmol/kg	280–300 mmol/kg
Urine		
Osmolality	293 mmol/kg	> 100 mmol/kg[a]
Sodium	81 mmol/L	> 30 mmol/L[a]
Morning cortisol	598 nmol/L	170–500 nmol/L
TSH	5.46 mIU/L	0.2–4 mIU/L
Free T4	16.5 pmol/L	10–25 pmol/L

TSH = thyroid-stimulating hormone.
[a]Suggests a diagnosis of syndrome of inappropriate antidiuretic hormone.

were unremarkable. The patient appeared euvolemic and did not have signs or symptoms of heart failure. The results of laboratory tests at the time of admission are presented in Table 2. The complete blood count and liver enzymes were normal. While in the emergency department, the patient received 0.9% sodium chloride at 75 mL/h for 7 hours.

The patient's herbal medications and amlodipine were held upon admission to hospital, and the olmesartan and zopiclone were continued. On day 1 of the admission, the patient received 2 doses of metoclopramide 10 mg IV for nausea. Her fluid intake was restricted to 1000 mL/day. On day 2, the patient felt much improved, as the nausea and fatigue had resolved. On day 3, sodium chloride was started (1 g PO twice daily). On day 5, the fluid restriction was discontinued. On day 6, the patient was discharged, at which time the serum sodium was 130 mmol/L (Figure 1).

The patient was instructed to discontinue the following medications: apple cider vinegar/chromium (this medication may cause hypokalemia and hyperreninemia), *E. arvense* (because this medication contributed to hyponatremia), and amlodipine (because of peripheral edema). The patient was instructed to continue sodium chloride at a reduced dose of 1000 mg PO daily. The patient's intake of fluid was not restricted at the time of hospital discharge. The patient was instructed to continue olmesartan 40 mg PO daily, zopiclone 7.5 mg PO at bedtime, and lorazepam 0.5 mg PO daily as needed. Approximately 30 days after discharge, the patient restarted the following products and medications: vitamin K_2 120 µg/cholecalciferol 1000 units 1 tablet PO daily; vitamin C 500 mg 1 tablet PO daily; multivitamin 1 tablet PO daily; cholecalciferol 1000 units 1 tablet PO daily; vitamin B_{12} 1000 µg 1 tablet PO daily; kelp (iodide 553 µg) 1 tablet PO daily; calcium carbonate 230 mg/magnesium 70 mg/vitamin D 200 units, 2 tablets PO twice daily; and flax seed 1000 mg/α-linoleic acid 530 mg/oleic acid 137 mg/linoleic acid 120 mg 1 tablet PO daily.

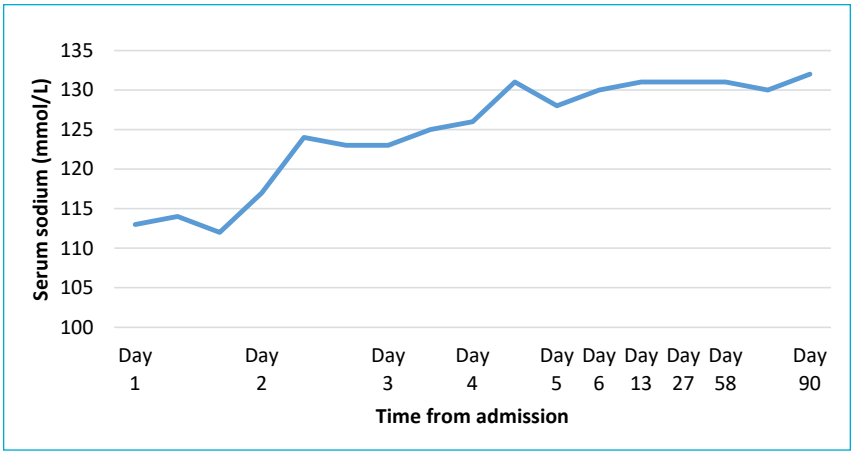

FIGURE 1. Summary of serum sodium. Note: The *y* axis is not linear.

DISCUSSION

The patient described here was euvolemic. The diagnosis of SIADH was suggested by the laboratory results, which showed serum osmolality less than 270 mmol/kg, urine osmolality greater than 100 mmol/kg, and urine sodium concentration greater than 30 mmol/L.[4] The patient was not hypothyroid and did not have adrenal insufficiency. It was thought that her low sodium was likely due to decreased oral intake and SIADH (the latter potentially triggered by nausea) and may have been exacerbated by *E. arvense*. The patient did not have so-called "tea and toast" induced hyponatremia, which may occur in elderly patients with low glomerular filtration rate, who follow a diet that is low in salt and protein combined with drinking large amounts of water.[8] Our patient had an estimated glomerular filtration rate of 55 mL/min and stated that she had been eating regular meals, except for the 2 days before admission, with no change in her fluid intake. In addition, the nausea had been present for only 2 days, which suggested that *E. arvense* may have contributed to the hyponatremia.

A literature search of PubMed, Google Scholar, Embase, and Reactions Weekly from inception to March 2020 using the search terms "horsetail", "hyponatremia", and the scientific names of several species of horsetail (*E. arvense*, *E. bogotense*, *E. giganteum*, *E. hyemale*, *E. myriochaetum*, and *E. telmateia*) yielded 3 citations[9-11] (Table 3). The dose, frequency, and duration of *E. telmateia* and *E. arvense* were not reported. In 1 case, hyponatremia developed after acute ingestion of an unknown species of homemade horsetail juice. Our patient had taken *E. arvense* 15 mg daily for 10 years and had lower serum sodium than 2 of the previously reported cases and higher serum potassium than all 3 of the previous cases. We suggest it may be possible for hyponatremia from *E. arvense* to

TABLE 3. Summary of Cases of *Equisetum* spp. Causing Hyponatremia

Ref	Age (yr)/ Sex	Medical History	Concomitant Medications	*Equisetum* Species	Presenting Symptoms	Laboratory Values	Treatment	Duration of Admission	Follow-Up
9	84/F	Hypertension	None	*E. telmateia*, dose and frequency not reported, duration 6 months	Falls, muscle weakness, lack of energy	SNa 120 mmol/L SK 2.3 mmol/L SCr 124 µmol/L UNa 34 mmol/L UK 11 mmol/L ADH 1.9 pmol/L UO and SO not reported	Management of hyponatremia and hypokalemia not reported; repeat SNa and SK not reported	Not reported	At 6 months, patient was asymptomatic with no electrolyte abnormalities
10	32/F	Not reported	Not reported	Unknown amount of homemade horsetail juice, species not reported Presentation 6 h after ingestion	Headache, lethargy, abnormal behaviour, tonic clonic seizure[a]	SNa 118 mmol/L SK 2.6 mmol/L SCl 85 mmol/L Glucose 10 mmol/L[b] SCr 43 µmol/L UO 702 mmol/kg UNa 68 mmol/L UK 84 mmol/L SO 240 mmol/kg TSH 1.4 mIU/L Free T4 20 pmol/L	Initially treated with 500 mL 0.9% NaCl and 14 mmol KCl After 28 h and 900 mL 2.5% NaCl, SNa was 134 mmol/L	5 days	At 12 days, patient had fully recovered
11	38/F	Not reported	Not reported	*E. arvense*, dose, frequency, and duration unknown	Presented with cardiopulmonary arrest[c] and generalized twitching	SNa not reported SK 2.8 mmol/L Low thiamine level (actual level not reported) Toxicology screen negative (details not reported)	Electrolyte and thiamine replacement (details not reported)	Not reported	Patient died

ADH = antidiuretic hormone, F = female, SCl = serum chloride, SCr = serum creatinine, SK = serum potassium, SNa = serum sodium, SO = serum osmolality, TSH = thyroid-stimulating hormone, UK = urinary potassium, UNa = urinary sodium, UO = urine osmolality.

[a]Computed tomography (CT) of the head showed diffuse cerebral edema, and the patient was transferred to the intensive care unit.

[b]Reference range for serum glucose 3.5–9 mmol/L.

[c]Cardiac catheterization performed for suspected cardiac cause showed that coronary arteries were normal. CT and magnetic resonance imaging/magnetic resonance angiography of the head excluded acute findings. CT angiography of the chest excluded pulmonary embolism, and the results of echocardiography were unremarkable.

develop after years of therapy, as may occur with thiazide diuretics.[3] Horsetail (species not reported) has been associated with serum sodium less than 116 mmol/L (n = 1) and less than 122 mmol/L (n = 3); however, details of these cases were not reported.[12] Two of the cases identified in our literature review involved young women[10,11] and one an elderly woman.[9] The patient in the current case report was a 70-year-old woman.

We have provided a more comprehensive case report than those identified by our literature search, with more information about the patient's past medical history; medication use; dose, frequency, and duration of the *E. arvense*; and hyponatremia treatment and 90-day follow-up. The hyponatremia was considered to be a possible adverse drug reaction to *E. arvense*, as assessed by the Naranjo adverse drug reaction probability scale (score of 4).[13]

CONCLUSION

We have reported a case of hyponatremia secondary to decreased oral intake and SIADH triggered by nausea, possibly exacerbated by the diuretic effect of *E. arvense*. However, the patient had decreased oral intake and nausea for only 2 days. Other causes of hyponatremia were excluded. It may be prudent for health care providers to monitor serum sodium levels within the first few weeks of starting *E. arvense* therapy and then at regular intervals, especially in elderly female patients with low body weight. More research is needed to assess the efficacy and safety of *E. arvense*.

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