# A Diagnostic Dilemma

Mohammed F. Shamji, BSc, MSc, MD; and David C. Holland, MSc, MD, FRCPC

A 68-year-old female presented with known diffuse B-cell lymphoma diagnosed by femoral and posterior triangle lymph node pathology. She had a three-week history of fatigue, pain in all extremities, and progressive weakness, requiring her to use a walker to ambulate. The patient also complained of one week of increasing urinary frequency and incontinence, nocturia of four episodes every evening, and a 5 kg weight loss. Ongoing medical issues included anxiety disorder, rheumatoid arthritis, and a traumatic right hemiparesis. Past medical history included breast cancer (treated with lumpectomy), cholecystectomy, appendectomy, hysterectomy, and cesarean section. Medications on admission included amitriptylene, 100 mg orally at bedtime, celecoxib, 200 mg orally twice daily, and acetaminophen, 325 mg orally every four hours, as needed. There was no history of chemotherapy.

The patient was normocalcemic throughout admission. Computed tomography scan demonstrated a non-calcified homogenously enhancing hypothalamic mass extending to the suprasellar cistern. Results of exams are listed in Tables 1 and 2.

## *What's your diagnosis?*

a) Diabetes insipidus (DI)
b) Cerebral salt wasting (CSW)
c) Nephrogenic DI
d) Salt-wasting nephropathy
e) Syndrome of inappropriate antidiuretic hormone release

**Table 1**
### Results of the physical exam

- Blood pressure: 115/76 mmHg
- Heart rate: 102 beats/minute (tachycardia)
- Respiratory rate: 16 breaths/minute
- Temperature: 36.2 C
- Visual acuity: Decreased in the right temporal field
- Palpable adenopathy: Limited to a 1 cm, hard, non-tender, mobile lymph node in the left tonsillar region
- Heart and breath sounds: Normal
- Abdomen: Soft and non-tender, without hepatosplenomegaly or ascites
- Neurologic exam: Right-sided weakness, hyperreflexia, and increased tone

**Table 2**
### Results of the lab exam

**Blood workup:**

- Sodium: 144 mmol/L
- Osmolarity: 297 mOsm/L
- Creatinine: 58 µmol/L
- Urea: 4.4 mmol/L
- Hemoglobin: 134 g/L
- Hematocrit: 0.392

**Urine chemistry**

- Sodium: 26 mEq/L
- Osmolarity: 129 mOsm/L
- Urinalysis: Negative

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## *Answer:*
## *Concomitant central DI and CSW*

This normonatremic patient with profound volume loss was tentatively diagnosed with DI secondary to central nervous system (CNS) lymphoma. Rehydration with normal saline volume guided by urine output was initiated. Treatment with 1-deamino-8-D-arginine vasopressin (DDAVP), 10 mcg twice daily intranasally, was initiated; however this intervention did not completely improve the diuresis, and there was rapid evolution of hyponatremia. The urine also remained dilute, with an osmolarity of 220 mOsm/L. This level is inappropriate for a serum level of 256 mOsm/L. Such diuresis and sodium depletion characterize CSW, and DDAVP was discontinued.

At this point, fludrocortisone, 0.1 mg subcutaneously once daily, was introduced. Once again, this intervention did not completely improve the diuresis, and rapid progression to hypernatremia followed. Furthemore, the urine was still dilute (144 mOsm/L) compared to the serum (269 mOsm/L). Ultimate definitive management of the diuresis required both DDAVP and fludrocortisone to treat coexistent DI and CSW.

Daily electrolytes, urine output, and intervention are summarized in Table 3.

Treatment for both diagnoses continued until day 17 of admission, after which the fludrocortisone was discontinued, and the patient was discharged with intranasal DDAVP, 20 mcg twice daily, to manage ongoing DI. Radiation therapy and combination chemotherapy were planned to treat the CNS lymphoma.

Central DI and CSW can coexist, promoting diuresis by different mechanisms. Together, the afflictions can lead to an overall isotonic fluid loss, and can blur their own early differentiation. A previous report of this coincidental presentation was in the setting of pituitary adenoma, but this is the first report of such manifestation with CNS lymphoma. Indeed, managing either syndrome will allow the clinical entity of the other to manifest (*e.g.*, administering DDAVP made clearer the hyponatremia and polyuria of CSW, and administering fludrocortisone demonstrated the hypernatremia and polyuria of central DI).

Several clues that would have suggested parallel processes were initially overlooked:

1. Patient presented with an isotonic polyuria, which fits neither the pattern of sodium wasting and hyponatremia in CSW, nor free water loss and hypernatremia in DI.
2. Equivocal urine studies with sodium concentration of 26 mEq/L were neither low, nor high and, therefore, should have also been cause for questioning the mechanism driving such a profound diuresis.
3. Initial administration of DDAVP partially raised the urine osmolarity, but failed to correct it to a level greater than that of the serum. Similarly, secondary administration of fludrocortisone had the same incomplete effect. A second untreated process should have been regarded as responsible for fluid losses in each setting. Dx

**Dr. Shamji** is a resident, division of neurosurgery, The Ottawa Hospital, Ottawa, Ontario.

**Dr. Holland** is a staff member, division of nephrology, Kingston General Hospital, Kingston, Ontario.

Table 3
## Fluid, electrolytes, and interventions

| Day | Serum Na (mEq/L) | Serum Osm (mOsm/L) | Urine output (mL) | Urine Na (mEq/L) | Urine Osm mOsm/L | Management |
|---|---|---|---|---|---|---|
| 1 | 144 | 297 | | 26 | 129 | |
| 2 | 138 | | 10400 | | | |
| 3 | 145 | | 6450 | | | |
| 4 | 143 | | 8560 | | | Start DDAVP |
| 5 | 133 | | 15000 | | | |
| 6 (a.m.) | 117 | 256 | 9600 | 70 | 155 | Hold DDAVP |
| 6 (p.m.) | 130 | | 17275 | 97 | 220 | Start DDAVP |
| 7 | 122 | | 7450 | 130 | | Stop DDAVP<br>Start fludrocortisone |
| 8 | 137 | 269 | 32400 | 58 | 144 | |
| 9 | 163 | | 33725 | 93 | | Start DDAVP |
| 10 | 128 | | 6360 | | | |
| 11 | 128 | | 8235 | | | Titration of DDAVP |
| 12 | 133 | 7120 | | | | Titration of DDAVP |
| 13 | 128 | 268 | 2560 | 125 | 568 | Titration of DDAVP |
| 14 | 129 | | 2825 | | | |
| 15 | 129 | | 4275 | | | |
| 16 | 130 | | 1713 | | | |
| 17 | 129 | | 1525 | | | Stop fludrocortisone |
| 18 | 132 | | 1150 | | | |

Osm: Osmolarity
Na: Sodium
DDAVP: 1-deamino-8-D-arginine vasopressin

References available—contact *The Canadian Journal of Diagnosis* at **diagnosis@sta.ca**.

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*Our mailing address*:
955 boul. St-Jean,
Suite 306
Pointe Claire, Quebec
H9R 5K3

*Our fax number*:
(514) 695-8554

*Our e-mail address:*
***diagnosis@sta.ca***