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Internal Medicine

ACANTHOCYTOSIS AND RECURRENT HYPONATREMIA UNMASKING PANHYPOPITUITARISM-A CASE REPORT

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ABSTRACT **Background:** Hyponatremia remains a common electrolyte disorder among elderly individuals, often erroneously attributed to SIADH. Central endocrine causes such as hypopituitarism are less recognized but critically important. Acanthocytosis may suggest endocrinepathology. **Case Presentation:** An 84-year-old male presented with recurrent symptomatic hyponatremia. Peripheral smear revealed acanthocytosis. Endocrine evaluation confirmed panhypopituitarism. MRI revealed a 2.2 cm pituitary macroadenoma. Hormone therapy corrected abnormalities. **Conclusion:** Recurrent hyponatremia in elderly warrants evaluation for hypopituitarism. Acanthocytosis may serve as a diagnostic clue. Hormonal therapy can reverse abnormalities.

KEYWORDS : Hyponatremia, Acanthocytosis, Panhypopituitarism, Pituitary macroadenoma, Central hypothyroidism

INTRODUCTION

Hyponatremia, a frequent electrolyte imbalance in clinical practice, is associated with heightened morbidity and mortality, particularly in the elderly. While common etiologies such as syndrome of inappropriate antidiuretic hormone secretion (SIADH) and diuretic use are frequently implicated, central endocrine disorders including hypothyroidism and adrenal insufficiency must also be considered. Among these, hypopituitarism is a particularly underrecognized cause, especially in geriatric patients where symptoms are often attributed to normal aging.

Peripheral blood smear (PBS) analysis is rarely included in the routine evaluation of hyponatremia. However, it can provide critical diagnostic clues, particularly when it reveals morphological erythrocyte changes such as acanthocytosis. Acanthocytes are classically associated with lipid metabolism disorders and neuroacanthocytosis syndromes, but reversible forms have also been reported in severe hypothyroidism. Here, we describe a unique case of an elderly male in whom refractory hyponatremia and the incidental finding of acanthocytosis ultimately led to the diagnosis of panhypopituitarism due to a nonfunctioning pituitary macroadenoma.

Case Presentation

An 84-year-old retired schoolteacher presented with recurrent episodes of vomiting and profound fatigue. He had two prior hospitalizations over four years for hyponatremia, treated as SIADH. Past medical history included medically managed coronary artery disease, dyslipidemia, and benign prostatic hyperplasia.

On admission, the patient was alert, afebrile, and hemodynamically stable. Physical examination revealed pallor and sparse axillary and pubic hair. Cardiopulmonary and neurological findings were unremarkable. Laboratory evaluation showed normocytic anemia (Hb: 10.5 g/dL), and marked hyponatremia (Na+: 118 mmol/L). Renal and hepatic parameters were within normal limits.

A PBS revealed acanthocytes along with normochromic normocytic anemia. Retrospective review of prior admissions demonstrated persistent hyponatremia (Na+: 106–127 mmol/L) with intermittent findings of acanthocytosis. Despite treatment with fluid restriction and tolvaptan, sodium levels repeatedly declined.

Extended endocrine workup showed central hypothyroidism (free T4: 7 pmol/L), secondary hypogonadism (FSH: 0.6 mIU/mL, LH: 0.3 mIU/mL, testosterone <0.05 ng/mL), and MRI revealed a 2.2 cm sellar-suprasellar mass. Hormone replacement with hydrocortisone followed by levothyroxine led to normalization of sodium and resolution of acanthocytosis. Six-month follow-up was stable.

DISCUSSION

This case highlights the diagnostic complexity of euvolemic hyponatremia in elderly individuals. While SIADH is a common consideration, persistent or recurrent cases warrant broader investigation, particularly of pituitary function. In panhypopituitarism, concurrent deficiencies of cortisol and thyroid hormones potentiate water retention, making hyponatremia resistant to standard SIADH therapies.

Inadequate suppression of ADH in adrenal insufficiency and impaired renal free water clearance in hypothyroidism are mechanisms underlying this phenomenon. Treatment aimed solely at presumed SIADH delayed the correct diagnosis. Only after comprehensive endocrine evaluation and the recognition of acanthocytosis was the central etiology identified.

Acanthocytes, spiculated red cells, are frequently linked to neuroacanthocytosis and abetalipoproteinemia, but can occur transiently in hypothyroid states. Normalization of erythrocyte morphology following hormone therapy supports a causal relationship. TSH may appear normal in central hypothyroidism, hence free T4 must be measured.

Transsphenoidal surgery is standard for symptomatic macroadenomas; however, conservative hormone therapy can be effective in elderly patients without mass effect.

Clinical Pearls

1. Consider central endocrine causes in elderly patients with recurrent or refractory euvolemic hyponatremia.
2. Peripheral smear findings like acanthocytosis can offer diagnostic clues.
3. Do not rely solely on TSH to rule out hypothyroidism—check free T4.
4. Always administer glucocorticoids before initiating thyroid hormone therapy.
5. Conservative therapy may suffice for pituitary macroadenomas in select elderly patients.

CONCLUSION

This case emphasizes the importance of considering hypopituitarism in the evaluation of unexplained, recurrent hyponatremia, particularly in elderly patients. Peripheral smear findings like acanthocytosis, although uncommon, can provide critical diagnostic clues. Timely and properly sequenced hormone replacement can reverse both biochemical and hematologic abnormalities, improving outcomes.

Table 1: Baseline Laboratory Results

Parameter	Result	Reference Range
Hemoglobin	10.5 g/dL	13.5–17.5 g/dL
Total leukocyte count	5,500/µL	4,000–11,000/µL
Platelet count	150,000/µL	150,000–450,000/µL
Serum creatinine	1.1 mg/dL	0.6–1.3 mg/dL
Serum sodium	118 mmol/L	135–145 mmol/L
Serum potassium	4.0 mmol/L	3.5–5.0 mmol/L
Serum osmolality	271 mOsm/kg	280–295 mOsm/kg
Urine osmolality	455 mOsm/kg	300–900 mOsm/kg
Urine sodium	76 mmol/L	< 20 mmol/L(if hypovolemic)
Serum cortisol (8 AM)	102 ng/mL	100–250 ng/mL
TSH	2.1 µIU/mL	0.4–4.2 µIU/mL

Table 2: Summary Of Prior Admissions (2017–2021)

Year	Presenting Symptoms	Na+ (mmol/L)	Cortisol (8 AM)	TSH	PBS Findings	Initial Diagnosis
2017	Fatigue, giddiness	127	Not done	Normal	Not reported	Hypovolemic hyponatremia
2019	Vomiting episodes	106	Normal	Normal	Acanthocytosis	SIADH
2020	Fatigue, decreased intake	106	Normal	Normal	Acanthocytosis	SIADH
2021	Vomiting, fatigue	118	102 ng/mL	2.1 µIU/mL	Acanthocytosis	Uncertain

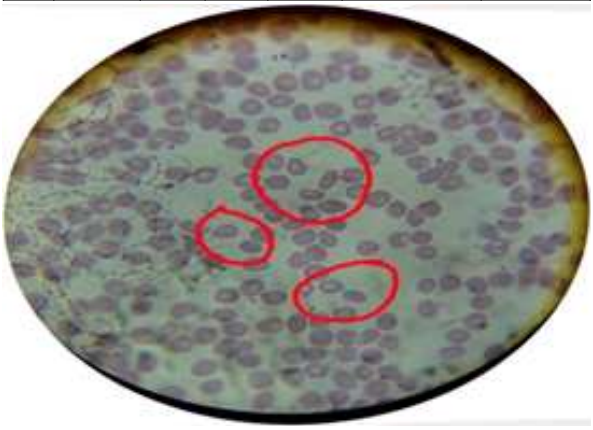

Figure 1: Peripheral Blood Smear Showing Acanthocytes (Spiculated Erythrocytes).

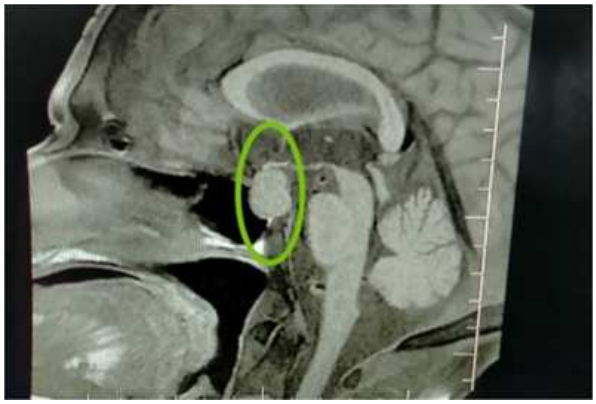

Figure 2: Sagittal T1-weighted MRI showing a 2.2 cm sellar-suprasellar lesion compressing the optic chiasma.

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