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OCCULT ADRENAL INSUFFICIENCY MASQUERADING AS RECURRENT HYPONATREMIA IN A TREATED HYPOTHYROID PATIENT: THE DIAGNOSTIC CHALLENGE OF "ENDOCRINE SILENCE"

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ABSTRACT

Recurrent hyponatremia is a common yet diagnostically challenging clinical entity, particularly when classical features of underlying endocrine disorders are absent. Adrenal insufficiency remains an under-recognized but potentially life-threatening cause. We report a case of a 50-year-old female with well-controlled hypothyroidism who presented with chronic fatigue and multiple prior hospitalizations for hyponatremia over five years. She developed acute gastrointestinal symptoms with persistent hyponatremia and episodes of hypotension despite adequate fluid resuscitation. Laboratory evaluation revealed inappropriately elevated urine sodium, suggesting a non-hypovolemic etiology. The absence of hyperpigmentation, hyperkalemia, or severe hemodynamic instability masked the diagnosis. This case highlights the concept of "endocrine silence," wherein secondary adrenal insufficiency presents without classical clinical markers, leading to delayed diagnosis. Early recognition through targeted endocrine evaluation is crucial to prevent recurrent morbidity and potential adrenal crisis.

KEYWORDS: Hyponatremia, Adrenal insufficiency, Secondary adrenal insufficiency, Endocrine silence, Hypothyroidism.

INTRODUCTION

Hyponatremia is the most common electrolyte abnormality encountered in clinical practice and is associated with increased morbidity, prolonged hospitalization, and mortality. While causes such as dehydration and SIADH are frequently considered, endocrine etiologies—particularly adrenal insufficiency—are often overlooked.

Adrenal insufficiency is classically associated with hypotension, hyperkalemia, and hyperpigmentation in primary disease. However, secondary adrenal insufficiency, resulting from impaired ACTH secretion, may present subtly without these hallmark features. This atypical presentation often leads to diagnostic delay, especially in patients with coexisting endocrine disorders such as hypothyroidism.

We describe a case of recurrent hyponatremia in a treated hypothyroid patient, ultimately pointing toward occult adrenal insufficiency presenting as "endocrine silence."

CASE REPORT

A 50-year-old female presented with a one-year history of progressive generalized fatigue and an acute episode of loose stools and vomiting of one-day duration. The diarrhea was non-bloody and non-foul-smelling, occurring approximately 10 times per day, associated with postprandial, non-bilious vomiting. There was no history of fever, abdominal pain, weight loss, or cardiorespiratory symptoms.

She was a known case of hypothyroidism on stable levothyroxine therapy (50 µg/day) for five years. Notably, she had experienced multiple prior hospitalizations for hyponatremia during the same period, the etiology of which remained undetermined.

On examination, she was conscious and oriented, with signs of mild dehydration. There was no hyperpigmentation, lymphadenopathy, or edema. Axillary and pubic hair were preserved.

Vital parameters revealed a pulse rate of 92/min and blood pressure of 100/70 mmHg. During hospitalization, she developed recurrent hypotensive episodes despite resolution of gastrointestinal losses, requiring intravenous fluid resuscitation.

Systemic examination was unremarkable except for mild epigastric tenderness. Neurological examination was normal.

Laboratory evaluation demonstrated persistent hyponatremia. Thyroid function tests were within target range (TSH 3.6 mIU/L), excluding hypothyroidism as the cause. Urinary studies revealed elevated urine sodium (129 mEq/L), indicating inappropriate renal sodium loss. Autoimmune screening was negative. Stool studies, echocardiography, abdominal ultrasonography, and fundus examination were unremarkable.

Despite correction of dehydration and symptomatic management, hyponatremia persisted, and hypotensive episodes continued. This discordance between clinical improvement and biochemical abnormality prompted consideration of an endocrine etiology.

Given the combination of.

- Recurrent hyponatremia
- Inappropriately elevated urine sodium
- Persistent hypotension
- Absence of hypothyroid dysfunction
- a diagnosis of occult adrenal insufficiency, likely secondary, was strongly suspected.

DISCUSSION

Hyponatremia is one of the most common electrolyte abnormalities encountered in clinical practice and is associated with increased morbidity, prolonged hospitalization, and mortality. It may arise from a variety of etiologies including gastrointestinal losses, renal disorders, syndrome of inappropriate antidiuretic hormone secretion (SIADH), and endocrine dysfunction. Among endocrine causes, adrenal insufficiency remains one of the most under-recognized yet clinically significant conditions, particularly when it presents without classical features.[1,2] This case highlights the diagnostic complexity associated with recurrent hyponatremia and underscores the importance of maintaining a high index of suspicion for occult endocrine disorders.

In the present case, the patient had a long-standing history of recurrent hyponatremia over a period of five years, with multiple prior hospitalizations. Each episode was attributed to transient causes such as dehydration or gastrointestinal losses, without identification of an underlying unifying diagnosis. The persistence of hyponatremia despite correction of volume status, along with inappropriately elevated urine sodium levels, suggested a non-hypovolemic etiology. Furthermore, the presence of hypotensive episodes during hospitalization, even after resolution of gastrointestinal symptoms, pointed toward an underlying endocrine cause rather than simple fluid depletion.

Although hypothyroidism is a recognized cause of hyponatremia, it is typically associated with severe and untreated disease, and its contribution in adequately treated patients is minimal.[3] In this patient, thyroid function tests were within the therapeutic range, effectively excluding hypothyroidism as the primary cause. This necessitated consideration of alternative endocrine etiologies, particularly adrenal insufficiency. Cortisol deficiency leads to increased secretion of antidiuretic hormone (ADH), resulting in impaired free water excretion and dilutional hyponatremia.[4] In primary adrenal insufficiency, concomitant aldosterone deficiency contributes to sodium loss and hyperkalemia; however, in secondary adrenal insufficiency, aldosterone secretion is preserved, and hyperkalemia is often absent.[2,4]

A notable feature in this case was the absence of classical clinical signs such as hyperpigmentation, severe hypotension at presentation, or hyperkalemia, which are typically associated with primary adrenal insufficiency. This atypical presentation raises the possibility of secondary adrenal insufficiency, wherein adrenocorticotropic hormone (ACTH) deficiency leads to cortisol deficiency without affecting mineralocorticoid function.[2] Such presentations are often subtle and may be easily overlooked, particularly in the presence of coexisting endocrine disorders like hypothyroidism, which may contribute to diagnostic anchoring and delay appropriate evaluation.

Similar atypical presentations have been described in the literature. Liamis et al. reported cases of endocrine-related hyponatremia where adrenal insufficiency presented without classical biochemical abnormalities, leading to initial misdiagnosis as SIADH.[3] In another report, Hahner et al. highlighted that secondary adrenal insufficiency frequently manifests with isolated hyponatremia and nonspecific symptoms such as fatigue and gastrointestinal disturbances, often resulting in delayed diagnosis and repeated hospital admissions.[2] These reports closely parallel our case, where the absence of hyperkalemia and hyperpigmentation masked the underlying diagnosis.

The differentiation between SIADH and adrenal insufficiency is particularly critical, as both conditions can present with euvolemic hyponatremia and elevated urine sodium. However, the presence of hypotension and recurrent episodes, as seen in this patient, favors adrenal insufficiency over SIADH. Current clinical guidelines

emphasize that adrenal insufficiency must be excluded before diagnosing SIADH, as failure to do so may result in inappropriate management.[1]

This case exemplifies the concept of "endocrine silence," wherein significant hormonal deficiency exists in the absence of overt clinical manifestations. Such presentations pose a diagnostic challenge and often lead to repeated hospital admissions and increased morbidity. Early recognition through targeted hormonal evaluation, including measurement of morning serum cortisol and ACTH levels, is essential for timely diagnosis and appropriate management.[2,5]

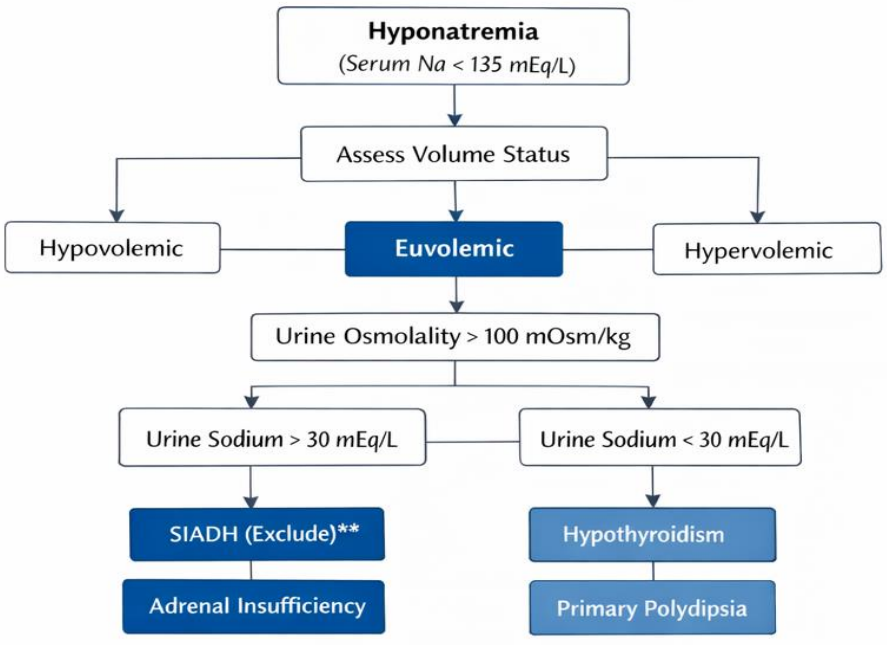
Overall, this case emphasizes the importance of considering adrenal insufficiency in all patients presenting with recurrent or unexplained hyponatremia, particularly when accompanied by hypotension and inappropriate urinary sodium loss. A systematic and physiology-based approach is crucial to avoid misdiagnosis and to ensure optimal patient outcomes.

Occult Adrenal Insufficiency Masquerading as Recurrent Hyponatremia in a Treated Hypothyroid Patient: The Diagnostic Challenge of "Endocrine Silence"

WHAT IS NEW IN THIS CASE:

- This case emphasizes the concept of "endocrine silence" where adrenal insufficiency presented without hyperpigmentation or hyperkalemia.
- Highlights the pitfall of recurrent hyponatremia misdiagnosed as SIADH due to absent classical signs.
- Underscores the necessity of adrenal evaluation in patients with unexplained hyponatremia and hypotension.

Diagnostic Approach to Hyponatremia



** *SIADH Diagnosis: Check Serum Cortisol First*

ACTH = Adrenocorticotroppic Hormone

AI = Adrenal Insufficiency

CONCLUSION

Recurrent hyponatremia should never be labeled idiopathic without exclusion of adrenal insufficiency. This case underscores the importance of recognizing subtle endocrine dysfunction in the absence of classical signs, particularly in patients with coexisting endocrine disorders.

The presence of hypotension with inappropriately elevated urine sodium should immediately prompt evaluation of adrenal function. Early diagnosis is critical to prevent recurrent morbidity and life-threatening adrenal crisis.

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