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Case Report

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Unexpected Cause of Concurrent Hyponatremia and Hypokalaemia: A Case Report of Pituitary Adenoma

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Abstract

Rationale: In clinical practice, concurrent hyponatremia and hypokalaemia are commonly observed in the presence of thiazide diuretic use, gastrointestinal problems, or polydipsia. However, these conditions may mask rare causes, such as adrenal insufficiency, and lead to a delay in diagnosis.

Patient concerns: Herein, we present a 71-year-old female with hypertension and type 2 diabetes mellitus, who presented to our emergency department with nausea and vomiting for the past several days. Hyponatremia and hypokalaemia were noted upon admission.

Diagnosis: Initial impressions were consistent with Syndrome of Inappropriate Antidiuretic Hormone (SIADH) with water diuresis. However, even after water restriction, hyponatremia and hypokalaemia both persisted. Therefore, a pituitary lesion was strongly suspected. Further biochemical profiling of the pituitary hormones showed low Adrenocorticotropic Hormone (ACTH) levels.

Interventions: Brain magnetic resonance imaging revealed a pituitary macroadenoma, which confirmed the diagnosis of secondary adrenal insufficiency due to ACTH deficiency.

Outcomes: She was referred to the neurosurgery department for transsphenoidal removal of the pituitary adenoma.

Lessons: This case suggests that clinicians should consider a diagnosis of secondary adrenal insufficiency when presented with a patient showing concurrent hyponatremia and hypokalaemia without an obvious aetiology.

Keywords: Hyponatremia; Hypokalaemia; Pituitary Adenoma; Secondary Adrenal Insufficiency; Cortisol

Abbreviations

ACTH	:	Adrenocorticotropic Hormone
ED	:	Emergency Department
SAIDH	:	Syndrome of Inappropriate Antidiuretic Hormone
MRI	:	Magnetic Resonance Imaging
FSH	:	Follicle-Stimulating Hormone
LH	:	Luteinizing Hormone
hGH	:	Human Growth Hormone
TSH	:	Thyroid-Stimulating Hormone

Introduction

Thiazide use, gastrointestinal problems, or polydipsia are typically the main aetiologies of concurrent hyponatremia and hypokalaemia in clinical practice [1,2]. Thiazides affect the upstream portion of the distal tubule, where they restrict sodium reabsorption by inhibiting the sodium-chloride symporter, which results in hyponatremia. At the same time, thiazides also increase potassium

excretion, leading to concomitant hypokalaemia [3]. Loss of sodium and potassium from the gastrointestinal tract, caused by conditions such as diarrhoea, can also contribute to hyponatremia and hypokalaemia [4]. Further, hyponatremia and hypokalaemia present in polydipsia due to water dilution and water diuresis [5].

Hyponatremia and hyperkalaemia are the two main concurrent electrolyte imbalances that signal primary adrenal insufficiency [6]. However, patients with secondary adrenal insufficiency may also present with an altered electrolyte profile. Herein, we report a rare case presenting with hyponatremia and hypokalaemia, which were Attributed Toadrenocorticotropic Hormone (ACTH) deficiency resulting from pituitary macroadenoma.

Case report

A 71-year-old female presented to our Emergency Department (ED) with nausea and vomiting over the past two days. She had a medical history of hypertension, diabetes mellitus, and hepatitis C infection for over 10 years. Her oral medication within the previous three months included metformin (1000 mg daily), bisoprolol (5 mg daily), Exforge® (5 mg amlodipine with 160 mg valsartan, daily), and rosuvastatin (5 mg daily). The patient also complained of poor appetite, general malaise, and weight loss of 18 kg within the last year. Physical examination upon admission showed that her height and weight were 151 cm and 51 kg, respectively, and that she had a body temperature of 36.0 ˚C, a blood pressure of 135/85 mmHg, and a heart rate of 51 beats/min with a regular rhythm. Her body fluid status was euvolemic. Her consciousness was clear, and no abnormal neurological signs were found. Chest auscultation revealed normal heartbeat and clear breathing sounds. No tenderness, rebound tenderness, or abdominal muscle guarding were noted. Dry mucous membrane and pitting oedema were also absent.

Laboratory data is shown in Table 1. The significant findings were hyponatremia (116 mmol/L), hypokalaemia (2.7 mmol/L), plasma hypo-osmolarity, and elevated levels of urine sodium and potassium excretion. Kidney and thyroid function were normal, albeit with a mild decrease in AM cortisol (5.9 pg/nl) with normal adrenocorticotropic hormone (ACTH) levels (5.17 pg/mL). Low urine osmolarity (160 mOsm/kg) was also noted. Based on the diagnosis of hyponatremia [1], initial impressions were consistent with Syndrome of Inappropriate Antidiuretic Hormone (SIADH) due to nausea, vomiting, and renal potassium wasting related to water diuresis. Therefore, after the abnormal electrolyte profile was corrected, the patient was discharged with water restriction education.

However, the patient returned to our ED two weeks later with the same complaint of nausea and vomiting for several days. The laboratory findings from her second admission (summarized in Table 1) also showed hyponatremia (125 mmol/L) and hypokalaemia (3.3 mmol/L), but urine osmolarity was normal (450 mOsm/kg). Strong suspicions of pituitary lesion–related SIADH were raised, which led us to arrange Magnetic Resonance Imaging (MRI) of the brain. The MRI showed a mass lesion at the sella/suprasellar region (Figure 1) that was compatible with macroadenoma. However, subsequent hormone tests showed lower levels of Follicle-Stimulating Hormone (FSH), Luteinizing Hormone (LH), Human Growth Hormone (hGH), and Thyroid-Stimulating Hormone (TSH) levels under the status of postmenopause, along with low ACTH levels (1.42 pg/ml). Therefore, a diagnosis of secondary adrenal insufficiency due to a pituitary macroadenoma with panhypopituitarism was made, and the patient was started on hormonal replacement therapy with 20 mg oral hydrocortisone daily.

The patient was then referred to the neurosurgery department for transsphenoidal removal of the pituitary adenoma, which was confirmed in the pathology report. Hyponatremia and hypokalaemia were absent in the three months following surgery.

Parameter (reference range)	First Admission	Second admission
Plasma		
pH (7.35–7.45)	-	7.48
Bicarbonate (22–26 mmol/L)	-	26.7
Blood nitrogen (7–25 mg/dl)	11	8
Creatinine (0.5–1.3 mg/dl)	0.69	0.79
Na^+ (133–145 mmol/L)	116	125
K^+ (3.3–5.1 mmol/L)	2.7	3.3
Cl^- (96–108 mmol/L)	100	-
Ionized calcium (3.68–5.6 mg/dl)	4.07	4.58
Phosphate (2.5–5 mg/dl)	2.5	3.1
Osmolality (278–305 mOsm/kg)	252	-
TSH (0.35–4.94 uIU/ml)	0.72	3.52
T4, Free (0.7–1.48 ng/dl)	0.78	-
T4 (4.87–11.72ng/dl)	-	3.8
Cortisol (6.7–22.6 ug/dl)	5.9	6.8
ACTH (5.0-77.0 pg/ml)	5.19	1.42
Renin concentration (15.7–57 pg/mL)	3.77	-
Aldosterone (78–104 pg/mL)	93.37	-
*FSH (16.74–113.59 mIU/mL)**	-	2.94
LH (10.87–58.64 *mIU/mL*)*	-	3.06
hGH (0–15 ng/mL)*	-	0.11
Spot urine		
Creatinine (mg/dl)	9	-
Na^+ (mmol/L)	53	-
K^+ (mmol/L)	10	-
Cl^- (mmol/L)	52	-
Osmolality (300–900 mOsm/kg)	160	-
TTKG (<3)**	5.83	-
K^+/Cr (mmol/mmol) (<2)**	12.22	-

*Indicates reference range for the menopause women; **Indicates reference range for normal renal response to hypokalemia. Abbreviation: TSH: Thyroid Stimulating Hormone; T4: Thyroxine 4; ACTH: Adrenocorticotropic Hormone; FSH: Follicle-Stimulating Hormone; LH: Luteinizing Hormone; hGH: Human Growth Hormone; TTKG: Transtubular Potassium Gradient

Table 1: Serum and urine biochemistry at admission.

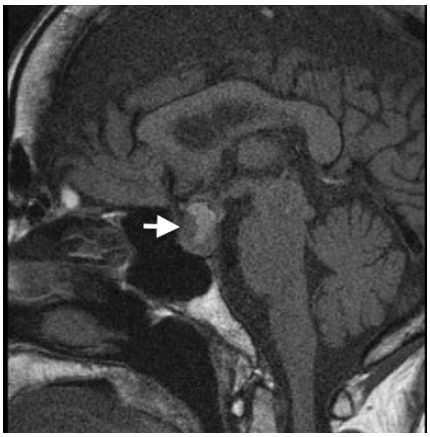

Figure 1: Magnetic resonance imaging showing a pituitary gland tumour about 1.9 cm in maximum dimension, with both intrasellar and suprasellar components (arrow).

Discussion

Pituitary adenomas comprise 15–20% of intracranial neoplasms that interfere with the hypothalamus-pituitary-target axis [7,8]. Pituitary adenomas can stimulate the excessive release of hormones from the posterior pituitary gland or destroy the gland itself, resulting in hyperpituitarism [9] or hypopituitarism [10], respectively. Hyperpituitarism induces hyponatremia, known as SIADH. Conversely, hypopituitarism is a clinical syndrome of deficiency in pituitary hormone production, wherein panhypopituitarism refers to the involvement of all pituitary hormones. Clinical manifestations depend on the extent of hormone deficiency and may be non-specific, often leading to a missed diagnosis. The progressive loss of pituitary hormone secretion is usually a slow process that occurs over a period of months or years. On occasion, hypopituitarism manifests abruptly, with rapid onset of symptoms. In general, hGH deficiency occurs first, followed by LH deficiency. The loss of FSH, TSH, ACTH, and prolactin typically follow much later. However, the deficiency of a single pituitary hormone is rarer than deficiency of multiple pituitary hormones [5,11].

Hyponatremia and hyperkalaemia are frequently and concurrently observed in primary adrenal insufficiency, and is caused by diminished secretion of cortisol and aldosterone [6]. Hyponatremia in secondary adrenal insufficiency stems from cortisol insufficiency contributing to elevated corticotropin releasing hormone, which is co-secreted with antidiuretic hormone. The ensuing water retention results in hyponatremia [12,13]. Hyperkalaemia is caused by aldosterone insufficiency and is a clinical manifestation of primary adrenal insufficiency. However, because aldosterone secretion is more dependent on angiotensin II than on corticotrophin, hypoaldosteronism is not a consequence of hypopituitarism. Thus, aldosterone–regulated secretion of renal potassium remains unaffected in secondary adrenal insufficiency, leading to normokalaemia or hypokalaemia [14].

Our case presented with concurrent hyponatremia and hypokalaemia. Her euvolemic fluid status, normal renal and thyroid function, and normal ACTH levels with mildly low cortisol levels led us to the diagnosis of nausea-related SIADH. However, persisting electrolyte imbalance indicated that the SIADH might have had an alternative aetiology, of which a pituitary lesion was highly suspect. Incidentally, we discovered a pituitary macroadenoma and performed another hormone survey, which now showed extremely low ACTH levels. Therefore, a diagnosis of secondary adrenal sufficiency was made. As discussed above, secondary adrenal insufficiency can present aldosterone–regulated hypokalaemia with renal potassium wasting. Indeed, the patient's trans-tubular potassium gradient was 5.87. Based on the diagnostic approach to hypokalaemia [2,15], profound remote vomiting was strongly suspected, which could explain how hypokalaemia, instead of hyperkalaemia, was observed in adrenal insufficiency in our case.

Early diagnosis provides the benefit of early intervention; however, early diagnosis of adrenal insufficiency is challenging due to variations in clinical presentation. Acute adrenal insufficiency leads to adrenal crisis, but the symptoms of chronic onset are often insidious and vague. Based on our case, pituitary macroadenoma related ACTH deficiency, although rare, should be considered in any patient who has unexplained severe hypokalaemia and hyponatremia.

Conclusion

Secondary adrenal insufficiency should not be overlooked when facing a case presenting with concurrent hyponatremia and hypokalaemia in the absence of polydipsia, gastrointestinal problems, or diuretic use.

Acknowledgements

Ethic approval was waived by the ethics committee at the Shin Kong Wu Ho-Su Memorial Hospital because our case study was based on chart review and the patient has been anonymized. However, the written informed consent was obtained from patient for publishing the related images and laboratory data. The authors report no conflicts of interest. The authors alone are responsible the content and writing this paper.

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