CASE REPORT

# Chest Pain in a Man With Hyponatraemia

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**ABSTRACT**

Hyponatraemia is a common electrolyte disturbance with diverse underlying aetiologies, often requiring a comprehensive diagnostic approach. We report a case of a 58-year-old man who presented to the Emergency Department with a one-day history of intermittent left-sided chest pain. He had no other significant clinical findings, aside from an elevated blood pressure. Electrocardiogram revealed no ST elevation or T-wave inversion, and high-sensitivity troponin I level was not raised. There was, however, severe hyponatraemia (120 mmol/L). Further laboratory investigations revealed presence of hypothyroidism and hypocortisolism, raising concerns about possible endocrine-related causes of hyponatraemia. Notably, the patient did not exhibit the typical neurological symptoms of severe hyponatraemia, suggesting chronicity. This case underscores the importance of a thorough diagnostic work-up for hyponatraemia, particularly when atypical symptoms such as chest pain are present, as multiple underlying causes may coexist. Early identification and management of the underlying conditions are crucial to avoid complications.
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## INTRODUCTION

Hyponatraemia is a common water and electrolyte abnormality, frequently defined as plasma sodium <135 mmol/L. It can be classified as mild, moderate and severe hyponatraemia, depending on the sodium levels [1]. The prevalence of severe hyponatraemia (plasma sodium level of <125 mmol/L), has been reported to range between 0.8% and 10.3% [1].

The clinical presentation varies in severity, ranging from asymptomatic to mild symptoms like nausea and vomiting, to more severe neurological manifestations such as seizures and coma. The clinical manifestations not only depend on the severity of hyponatraemia but also on the rapidity of plasma sodium decrease [1]. A sudden decrease in sodium levels, even in cases of mild to moderate hyponatraemia, is often symptomatic, whereas a gradual decline typically results in asymptomatic or subtle symptoms [1,2]. It is important to recognize that patients with chronic hyponatraemia may exhibit subtle symptoms that could go unnoticed during a routine clinical examination [2].

There are many causes of hyponatraemia, and a systematic approach to investigation is essential [3]. This involves evaluating the patient's volume status, exclusion of pseudohyponatraemia and conducting other relevant laboratory investigations. This case report underscores the importance of considering multiple potential causes of hyponatraemia, including endocrine-related causes, which are often under-investigated. In addition, it is important to recognise that patients may be asymptomatic or present with atypical symptoms of hyponatraemia.

## CASE REPORT

A 58-year-old man presented to the Emergency Department (ED) with a one-day history of left-sided chest pain. It was described as intermittent, with each episode lasting for 10 minutes with an intensity of 7 out of 10. He experienced three episodes that day before his presentation to the ED. He did not complain of shortness of breath, lethargy, nausea or vomiting. He was also not on any medications. An immediate electrocardiogram (ECG) revealed benign early repolarization in leads V3 and V4, with no observed ST elevation or T-wave inversion in other leads. Clinically, he was alert, there was no pallor, and he did not display signs of dehydration or fluid overload. His heart rate was 88 beats per minute, respiratory rate at 18 breaths per minute, blood pressure at 166/86 mmHg, and oxygen saturation at 99% on

ambient air. Treatment for an acute coronary event was initiated based on his clinical presentation, with the patient receiving crushed aspirin at a dosage of 300 mg, clopidogrel 300 mg, and intravenous pantoprazole 40 mg.

The initial laboratory results revealed mild anaemia with a non-raised high-sensitivity troponin I level. There was, however, severe hyponatraemia accompanied by hypochloraemia, with normal potassium level (Table I). There was a mild elevation in total cholesterol, while triglycerides, glucose, urea and the liver function test panel were within the reference interval. Further laboratory investigations aimed at identifying the cause of hyponatraemia uncovered a serum osmolality of 244 mOsm/L, a urinary osmolality of 375 mOsm/L, and a urinary sodium excretion of 85 mmol/L. Additionally, the thyroid function test revealed primary hypothyroidism (Table I). Further information provided by the patient's son indicated that the patient had a preexisting thyroid condition ten years ago and had been on medication for seven years before being discharged from a healthcare facility. Apart from that, he has no previous history of diabetes mellitus, hypertension, cardiovascular, renal, or hepatic disorders.

**Table I: Laboratory investigation results during hospitalisation**

| Parameters | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Unit | Reference Interval |
|---|---|---|---|---|---|---|---|---|
| Haemoglobin | 11.8 | 12.2 | | | | | g/dL | 13.0-17.0 |
| White cell count | 6.2 | 6.5 | | | | | $10^9$/L | 4.0-10.0 |
| Platelet | 239 | 239 | | | | | $10^9$/L | 150-450 |
| Urea | 3.2 | 3.7 | 3.5 | 3.2 | 4.9 | 4.6 | mmol/L | 3.2 – 8.2 |
| Sodium | 120 | 119 | 118 | 123 | 132 | 133 | mmol/L | 136-145 |
| Potassium | 4.2 | 3.6 | 3.9 | 3.8 | 4.6 | 4.4 | mmol/L | 3.5-5.1 |
| Creatinine | 64.3 | 65.6 | 66.4 | 64.3 | 66.7 | 67.5 | µmol/L | 62 – 115 |
| eGFR | 105 | 105 | 105 | 105 | 105 | 104 | mL/min/1.73m$^2$ | >90 |
| Chloride | 87 | 86 | 86 | 90 | 98 | 98 | mmol/L | 98-107 |
| Total protein | | 66 | | 58 | | | g/L | 58-82 |
| Albumin | 36 | 36 | | 31 | | | g/L | 34-50 |
| ALP | | 67 | | 62 | | | U/L | 46 – 116 |
| ALT | | 30 | | 33 | | | U/L | 10 – 49 |
| AST | | 82 | | 103 | | | U/L | < 34 |
| Adjusted calcium | 2.35 | 2.24 | 2.13 | 2.16 | | | mmol/L | 2.18 – 2.6 |
| Lipid profile | | | | | | | | |
| Cholesterol | | 5.21 | | | | | mmol/L | < 5.18 |
| Triglyceride | | 0.62 | | | | | mmol/L | < 1.7 |
| hs Troponin I | 34 | | | | | | ng/L | < 54 |
| Fasting glucose | | 4 | | | | | mmol/L | < 6.1 |
| Serum osmolality | | | 244 | | | | mOsm/kg | 270 - 295 |
| Urine osmolality | | 375 | | | | | mOsm/kg | 300 - 900 |
| Urine sodium | | 85 | | | | | mmol/L | |
| Random cortisol | | | 33 | | | | nmol/L | - |
| Morning cortisol | | | | 36.9 | | | nmol/L | 145.4 – 619.4 |
| TSH | | 20.49 | | | | | mIU/mL | 0.55 – 4.78 |
| fT4 | | 8.5 | | | | | pmol/L | 11.5 – 22.7 |

ALP: Alkaline phosphatase; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; fT4: free thyroxine; eGFR: estimated glomerular filtration rate based on Chronic Kidney Disease Epidemiology Collaboration; TSH: Thyroid stimulating hormone.

In the ward, he was commenced on perindopril for hypertension, along with oral sodium chloride and intravenous normal saline, with careful monitoring of urine output. Oral hydrocortisone was prescribed after two cortisol measurements (random and morning), performed as part of the hyponatraemia workup, revealed hypocortisolism (Table I). A sample for plasma adrenocorticotropic hormone (ACTH) was sent but was rejected due to improper sample handling. Synacthen test was also not performed on this patient. Oral thyroxine was initiated on day six of admission, following a counselling session with the patient and his family to support adherence to the prescribed treatment.

He remained asymptomatic for hyponatraemia and had no further episodes of chest pain throughout his hospitalisation. The patient's serum sodium gradually increased, and on discharge at day six, his serum sodium concentration was 133 mmol/L. He was scheduled for an outpatient echocardiography stress test and scheduled for a follow-up appointment at the medical clinic four months later. However, our records indicated that the patient had defaulted on the appointments.

## DISCUSSION

In the case described, the patient displayed a notable absence of typical neurological symptoms often linked with severe hyponatraemia, implying chronicity, wherein a gradual decline in sodium levels enables the brain to adapt to the intracerebral osmotic shift without progressing to cerebral oedema [3]. Considering the patient's euvolaemic state, along with hypoosmolar hyponatraemia and elevated urinary sodium (urine sodium >30 mmol/L), the potential underlying causes include syndrome of inappropriate antidiuresis (SIAD), hypothyroidism, adrenal insufficiency, and chronic kidney disease (CKD). In this patient, SIAD was ruled out by the abnormal cortisol and thyroid function test results and CKD by the normal urea and creatinine levels. The patient's normal blood glucose and low serum osmolality ruled out hyperglycaemia as a possible cause whilst normal total protein and triglyceride levels combined with low serum osmolality, made pseudohyponatraemia unlikely.

Endocrine disorders such as adrenal insufficiency and hypothyroidism are uncommon causes of hyponatraemia but are important to identify as they typically respond well to hormone replacement therapy [4]. Nevertheless, it has been reported that they are under investigated in patients with euvolemic hyponatraemia [5]. A study demonstrated that thyroid function was evaluated in only 69% of cases, and adrenal function was assessed in just 29% of the study population, despite patients having medical histories and clinical presentations suggestive of these conditions [5]. In this patient, hypothyroidism was thought to be the most likely cause given his prior history. However, while hypothyroidism has been associated with the development of hyponatraemia, the connection is relatively weak and typically occurs in cases of severe hypothyroidism and myxoedema [5]. In cases of mild to moderate hypothyroidism, it's essential to evaluate other potential contributors to hyponatraemia [5]. Factors such as medications, infections, and adrenal insufficiency should be considered in the diagnostic process.

Hyponatraemia in hypothyroidism results from impaired free water excretion due to elevated levels of antidiuretic hormone (ADH), which occurs in response to decreased cardiac output seen in hypothyroidism [4]. The increased urine sodium levels which are seen in SIAD have also been demonstrated in some patients with hypothyroidism [4]. Additionally, impaired renal function has also been suggested to have a role in the development of hypothyroidism-associated hyponatraemia, although in this case, it was unlikely given the normal eGFR.

Hyponatraemia may occur in both primary andor secondary adrenal insufficiency [4]. Primary adrenal insufficiency was considered a possible cause in this patient based on the low serum cortisol levels (<50 nmol/L), but other typical clinical features of primary adrenal insufficiency such as postural hypotension, hypovolaemia, and hyperkalaemia were not demonstrated in this patient. Conversely, his blood pressure was elevated on presentation in ED which could be in response to pain. In the ward, his blood pressure was stable despite the anti-hypertensive being withdrawn. Recognition of a central cause of adrenal insufficiency (stemming from disorders of the hypothalamus or pituitary) requires a high index of suspicion due to the subtle nature of its clinical manifestations [4]. Symptoms of hypopituitarism such as fatigue and lethargy, especially in the elderly, are often put down as attributed to aging, which results in hypopituitarism being underdiagnosed [4]. Measurement of plasma ACTH would help identify a central cause of adrenal insufficiency in this patient, in which ACTH is expected to be low, as opposed to primary adrenal insufficiency, where ACTH would be elevated Unfortunately, in this patient's case, the ACTH specimen was rejected by the laboratory due to improper collection and handling. The use of an incorrect blood tube, delayed processing, or storage at room temperature can lead to ACTH degradation and result in falsely low levels. Despite the absence of overt clinical signs of adrenal insufficiency, the patient's low morning serum cortisol level warrants further evaluation with a short Synacthen test, which remains the gold standard for confirming the diagnosis of adrenal insufficiency [4,5]. Measurement of other pituitary hormones should be measured in cases of suspected hypopituitarism.

In this patient, the aetiology of the patient's hypocortisolism remained unknown. The patient denied taking medications including over-the-counter drugs

or traditional herbs. Prolonged usage of steroid is a common cause of adrenal suppression [5]

In a patient with hypothyroidism, the initiation of levothyroxine without identification of concomitant adrenal insufficiency may worsen the adrenal insufficiency [54]. This occurs because thyroid hormones can trigger an acute adrenal crisis by accelerating the metabolism and clearance of glucocorticoids [4,5]. In this patient, oral hydrocortisone was commenced prior to administration of oral thyroxine.

Additionally, hypothyroidism and primary adrenal insufficiency may coexist in a condition called polyglandular autoimmune syndrome type II, a rare HLA-linked condition which may also present with hyponatraemia [5]. However, this was unlikely in our patient as the diagnosis typically requires the presence of at least two of the following primary adrenal insufficiency, autoimmune thyroid disease (Graves' disease or hypothyroidism), and type 1 diabetes mellitus (T1DM) [6]. It is also associated with other autoimmune conditions such as coeliac disease, alopecia, and vitiligo, none of which are present in this patient. In addition, this condition is more common in females and typically affects young to middle-aged adults, although it can present at almost any age [6].

## CONCLUSION

This case highlights the potential for atypical presentations of severe hyponatraemia. It is crucial to recognize that a patient may have multiple underlying causes of hyponatraemia, and investigations for both hypothyroidism and hypocortisolism should be included in the diagnostic work-up.

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