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Case Report

Prolonged hyponatremia due to hypopituitarism in a patient with non-ST-elevation myocardial infarction

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ABSTRACT

A 58-year-old man was admitted for non-ST-elevation myocardial infarction. A medicated stent was used for severe coronary artery stenosis. However, consciousness level progressively deteriorated after angioplasty. Computed tomography showed no brain lesion but laboratory tests showed hyponatremia (serum sodium: 113 meq./l) and urine analysis showed syndrome of inappropriate antidiuretic hormone secretion (SIADH). SIADH was first suspected to be drug-induced by enalapril. However, hyponatremia persisted even after withdrawal of enalapril and required oral sodium intake. Hormone assays indicated secondary adrenal insufficiency, which was caused by adrenocorticotropic hormone (ACTH) deficiency. Furthermore, in addition to ACTH deficiency, adult growth hormone deficiency was diagnosed following tests. Treatment with hydrocortisone relieved hyponatremia and re-institution of enalapril did not reduce serum sodium concentration. The final diagnosis was hyponatremia caused by hypopituitarism. <**Learning objective:** Secondary adrenal insufficiency with subsequent hypopituitarism should be suspected in cases with sudden-onset and prolonged hyponatremia in acute illness. Furthermore, the management of hypopituitarism should include assessment of growth hormone release to exclude growth hormone deficiency.>

Introduction

Various disease states are associated with hyponatremia [1,2]. In acute coronary syndrome (ACS), one study reported the presence of hyponatremia on admission in 20 (10.5%) of 235 patients with myocardial infarction, and demonstrated that hyponatremia in the early phase of ACS was a predictor of negative clinical outcome [3].

ACS results in marked activation of the renin–angiotensin system and increased catecholamine production. These factors promote renal vasoconstriction leading to diminished glomerular filtration rate and subsequent delivery of tubular fluid to the diluting segment of the nephron, further contributing to a reduction in renal water excretion. Consequently, patients with ACS are predisposed to hyponatremia, especially in the presence of marked neurohormonal activation [4]. We describe a patient who presented with non-ST-elevation myocardial infarction (NSTEMI), developed disturbances of consciousness following angioplasty, and was found to have hyponatremia associated with adrenal insufficiency and hypopituitarism.

Case report

A 58-year-old Japanese man with no past medical history visited our hospital due to exacerbating chest pain, which appeared 6 months earlier. He had visited a general practitioner 1 week before, and the electrocardiogram (ECG) and chest X-ray taken at that visit showed no significant findings. The coronary risk factors included dyslipidemia and smoking, but he was free of other risk factors including hypertension, diabetes mellitus, and familial history. His height, weight, and body mass index were

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176 cm, 79.1 kg, and 25.54. He did not show any disturbance of growth in childhood. He did not take any medications.

On admission, the ECG showed inverted T wave in V_{1-4} (Fig. 1), and blood tests showed leukocyte count of 7000 mm^{-3}, serum sodium of 137 meq./l, potassium of 4.4 meq./l, creatine kinase (CK) of 223 U/l, CK-MB 16 U/l, and high-sensitive cardiac troponin T of 0.78 ng/mL. The clinical diagnosis was NSTEMI. He was treated with infusion of isosorbide dinitrate, heparin, oral aspirin (100 mg/day), carvedilol (2.5 mg/day), rosuvastatin calcium (2.5 mg/day), and enalapril (2.5 mg/day).

On day 4, plasma sodium concentration decreased to 123 meq./l with low plasma osmolality of 237 mOs/l, high urinary osmolality of 673 mOs/l, and high urinary sodium concentration of 250 meq./l. At that point, we suspected drug-induced syndrome of inappropriate antidiuretic hormone secretion (SIADH) and started the infusion of normal saline for correction of hyponatremia. However, the chest symptom exacerbated and ECG showed ST-segment elevation on day 5 despite adequate infusion therapy. Percutaneous coronary intervention (PCI) was urgently performed; coronary angiography showed 90% narrowing of the left anterior descending artery, segment 7 (Fig. 2). A drug-eluting stent was implanted successfully during the same PCI. Infusion of normal saline was continued during the PCI. However, marked disorientation and mental confusion developed after the PCI. An urgent computed tomography showed no brain lesion. Based on a further decrease in serum sodium concentration to 113 meq./l, worsening hyponatremia was suspected as the cause of the disorder of consciousness. We started the correction of sodium concentration using 3% sodium chloride and stopped the prescription of enalapril, which was thought to be the causative drug. Besides, he also showed low concentration of cortisol (6.1 μg/dL, at the day before PCI), we infused 100 mg hydrocortisone for 3 days. Serum sodium concentration rose to 118 meq./l the following day, together with recovery of mental status. Based on the improvement, the infusion was replaced with normal saline. Although serum sodium concentration continued to rise to 132 meq./l, the patient was placed on oral sodium chloride intake to maintain serum sodium concentration (Fig. 3).

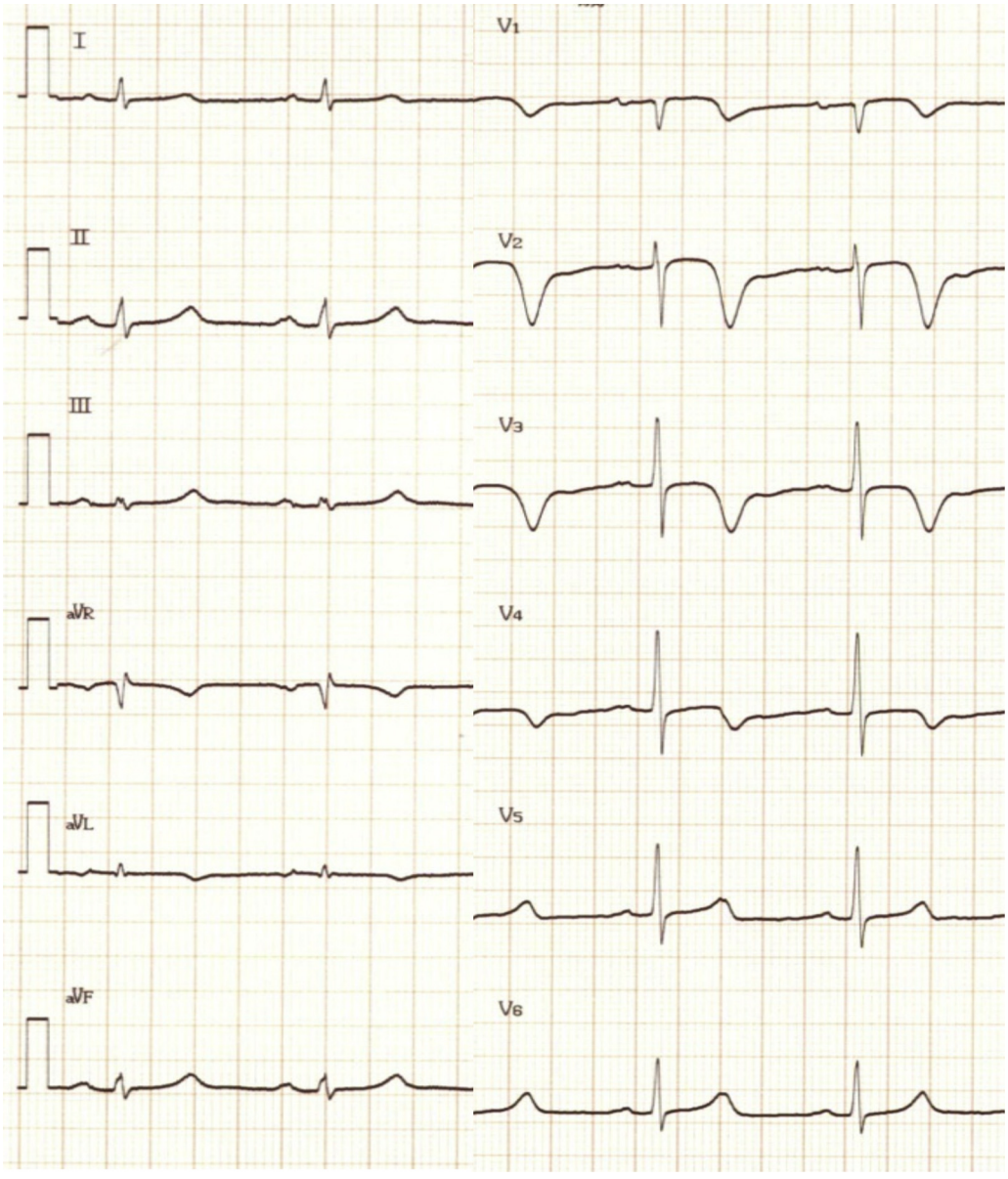

Fig. 1. Electrocardiogram recorded on admission showed inverted T waves in V_{1-4}.