CASE REPORT
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Acute Severe Symptomatic Hyponatremia Following Coronary Angiography

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ABSTRACT

Hyponatremia is a relatively common electrolyte disorder. Although severe acute hyponatremia following coronary angiography is rare, potentially lethal neurologic manifestations may result. We describe a patient with severe, symptomatic hyponatremia, an unusual complication of coronary angiography. Lack of familiarity with contrast media-related hyponatremia caused a delay in diagnosis and therapy in our case. The diagnosis of acute hyponatremia should be considered in any patient who develops behavioral or neurologic manifestations following coronary angiography. Prompt diagnosis and treatment is essential to avoid permanent neurologic damage or death. **(Korean Circ J 2011;41:552-554)**

KEY WORDS: Hyponatremia; Coronary angiography.

Introduction

Hyponatremia is a relatively common electrolyte disorder, most often reflecting water imbalance rather than sodium imbalance. Although most patients are asymptomatic and do not require treatment, hyponatremia sometimes results in serious complications. Aggressive or inappropriate therapy, however, can be more harmful than the condition itself, thus the clinician should be familiar with the diagnosis and management of various forms of hyponatremia. The etiology of most cases of hyponatremia can be deduced from the history, physical examination, and basic laboratory tests. Importantly, the intravascular administration of contrast medium may induce hyponatremia. Theoretically, contrast medium, such as glucose or mannitol, osmotically pull intracellular water into the extracellular space, which dilutes all the extracellular fluid electrolytes, thus resulting in hyponatraemia, but such a process is extremely rare. We describe a patient with severe, symptomatic hyponatremia, an unusual complication of coronary angiography.

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Case

An 84-year-old female was admitted for elective coronary angiography due to chest pain. Her height and body weight were 135 cm and 42.7 kg, respectively. She was healthy except for hypertension which was treated with amolodipine besilate (5 mg qd) and hydrochlorothiazide (12.5 mg qd). The blood creatinine was 0.9 mg/dL, the blood urea nitrogen (BUN) was 15.4 mg/dL, the glucose was 105 mg/dL, the sodium was 142 mmol/L, and the potassium was 3.8 mmol/L. Coronary angiography was performed from the left radial artery under local anesthesia. One hundred mL of Visipaque 320 (Iodixanol) contrast media was used. The coronary angiography showed minimal luminal narrowing of the mid-left anterior descending artery and was completed without complication. Twelve hours later, however, the patient had severe nausea and vomiting. Sixteen hours following coronary angiography, she was markedly confused, and was in a stuporous state with recurrent generalized tonic-clonic-type seizures. The neurologic examination was without focality. A brain MRI and electroencephalography were normal (Fig. 1). The diagnosis of hyponatremia was not entertained until blood was drawn and

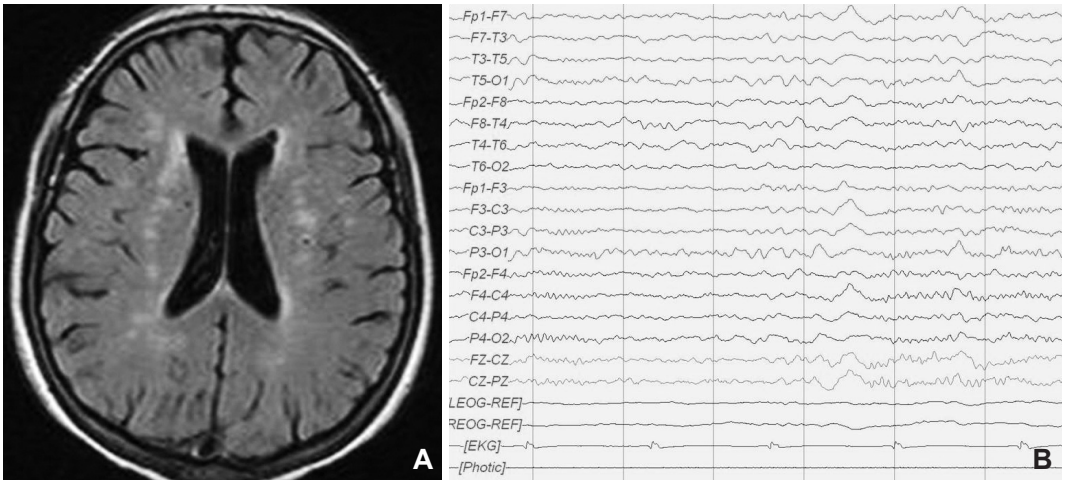

Fig. 1. An acute infarction was not noted on the brain MRI (A) and an electroencephalography was normal (B).

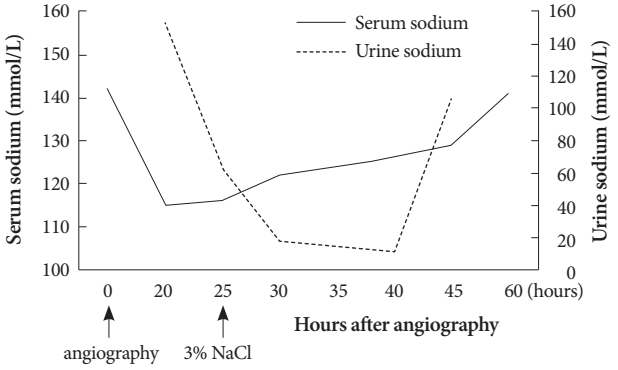

Fig. 2. The serum and urine sodium levels returned to normal values within 48 hours with a full clinical recovery.

showed a sodium of 115 mmol/L, a potassium of 3.4 mmol/L, a glucose of 205 mg/dL, a creatinine of 0.6 mg/dL, a BUN of 7.3 mg/dL, a uric acid of 3.1 mg/dL, and an osmolarity of 241 mosm/L. A laboratory error was suspected until a second blood sample confirmed the initial results. With a differential diagnosis of hypo-osmolar hyponatremia, we measured the serum-free T4 and thyroid stimulating hormone, which were normal. A rapid Adenocorticotropic hormone stimulation test was performed and the results were within the normal range. The patient was treated with hypertonic saline (3% NaCl) at a rate of 2 mL/hr. She improved slowly over the following 26 hours. The serum and urine sodium levels returned to normal within 48 hours, together with a full clinical recovery (Fig. 2).

Discussion

Sodium is an electrolyte which is essential in maintaining the extracellular fluid volume and regulating the blood pressure and osmotic equilibrium.[1)2)] Although the plasma sodium concentration is maintained in the normal range by water and sodium intake, and the renin-angiotension system,[3)] hyponatremia is a relatively common electrolyte disorder. Most patients with hyponatremia are asymptomatic, but sometimes hyponatremia is associated with increased mortality, morbidity, and hospital stay,[4)] especially when hyponatremia occurs over a short period of time (<48 hours), as in our case.

Hyponatremia following invasive procedures is triggered by multiple causes. The release of antidiuretic hormone, the use of narcotics, and a large intake of water are known precipitating factors.[5)] Women are more affected than men as a result of the smaller fluid volume and sex-related hormonal factors. Normal ageing impairs fluid homeostasis. The risk of hyponatraemia among elderly people is compounded by chronic diseases and long-term medication use. Thiazides are known to induce mild hyponatraemia and have been related to the rapid onset of serious post-operative complications.[6)] Our patient had been taking thiazide for control of blood pressure. The mechanisms leading to acute hyponatremia in our patient were complex, as described above. Osmotic diuresis secondary to contrast medium may be an additional factor contributing to hyponatremia. Theoretically, a contrast medium might also be a cause of hyponatremia. However, symptomatic hyponatremia following coronary angiography is rare, especially in a patient without advanced kidney disease. Until now, only several cases of contrast-induced hyponatremia have been reported. Arieff[7)] reported one patient who developed severe symptomatic hyponatremia following angioplasty of the right coronary artery.[7)] Boulos et al.[8)] also reported three patients who experienced severe hyponatremia after coronary angiography. In our case, although the creatinine level was within the normal range, the patient's renal function was slightly decreased for her age (glomerular filtration rate, 43.4 mL/min, as calculated by the Cockcroft-Gault formulation). Considering the patient's body weight, the total amount of contrast medium used was relatively high. A high amount of hypertonic solute infused into her extracellular fluid and her homeostatic mechanisms could not compensate, which

resulted in hyponatremia.

The therapeutic approach to the hyponatremic patient is determined more by the presence or absence of symptoms than by the absolute level of serum sodium. Correction of hyponatremia should be of sufficient pace and magnitude to reverse the manifestations of hypotonicity, but not so rapid and large as to pose a risk for the development of osmotic demyelination. Inappropriate therapy of symptomatic hyponatremia can be more harmful than no correction. Although severe acute hyponatremia following coronary angiography is rare, potentially lethal neurologic manifestations may result. Lack of familiarity with contrast media-related hyponatremia caused a delay in diagnosis and therapy in our case. The diagnosis of acute hyponatremia should be considered in any patient who develops behavioral or neurologic manifestations following coronary angiography. In such cases, laboratory tests for electrolytes and osmolarity should be performed immediately. Prompt diagnosis and treatment is essential to avoid permanent neurologic damage or death.

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