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Generalized Seizure Due To Acute Hyperosmolar Hyponatremıa Followıng Coronary Angıography: A Lesson Learned From A Case Report

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Abstract

Hyponatremia is a common electrolyte disorder, but symptomatic hyponatremia following coronary angiography is rare. Although patients with hyponatremia are generally asymptomatic, neurological manifestations may result in lethal complications.

Here we presented a 69-year-old female, admitted for elective coronary angiography due to exertional chest pain. Seven hours later after coronary angiography, however, the patient had a headache, temporary mental confusion and her speech was slurring. A brain magnetic resonance imaging (MRI) test, arterial blood gas analysis, and the neurological examination were carried out.

The neurological examination showed no focality and brain diffusion MRI was normal. Arterial blood gas analysis revealed sodium of 110 mmol/L and potassium of2.8 mmol/L. The patient was treated with hypertonic saline (3% NaCl) and vasopressin antagonist tolvaptan 15 mg at first and isotonic saline (0,09 NaCl) with a gradual normalization of electrolytes.

Diagnosis of acute hyperosmolar hyponatremia should be considered in patients with developing mental or behavioral abnormalities following coronary angiography.

Keywords: Coronary angiography, hyponatremia, seizure

Introduction

Hyponatremia is a common electrolyte abnormality in hospitalized patients. However, following coronary angiography, it is an uncommon finding. The etiology of most cases of hyponatremia can be deduced from the history, physical examination, and basic laboratory tests. Theoretically, contrast medium, like mannitol, may pull osmotically intracellular water into the extracellular space, which dilutes all the extracellular fluid electrolytes, thus resulting in hyponatremia. However, such process is extremely rare. Here, we describe a patient with acute severe symptomatic hyponatremia, a rarely seen complication of coronary angiography.

Case Illustration

A 69-year-old female was admitted for elective coronary angiography due to exertional chest pain. Her height, body weight, and body mass index were 166 cm, 67 kg and 24.3 kg/m^2 respectively. She had hypertension and a history of coronary stenting for left anterior descending artery (LAD) one year ago. She was under treatment with losartan potassium/hydrochlorothiazide (100/12.5 mg/d), clopidogrel (75 mg/d), metoprolol (25 mg/d) and rosuvastatin (10 mg/d). The blood creatinine was 0.71 mg/dL, the blood urea nitrogen (BUN) was 21 mg/dL, the sodium was 132 mmol/L, and the potassium was 3.8 mmol/L.

Coronary angiography was performed from the left radial artery under local anesthesia, and showed subtotal osteal narrowing of second diagonal osteal artery originating from stented mid LAD. After balloon kissing to bifurcation, lesion stent was placed and completed without complications. One hundred and twenty millilitre of iohexol contrast media was used. Seven hours later, however, the patient had a headache and temporary mental confusion. Her speech was slurring. The neurological examination was without focality and brain diffusion MRI was normal. Her mental situation was normal thereafter. However, fourteen hours following coronary angiography, she was markedly confused and developed a generalized tonic-type seizure which was relieved after

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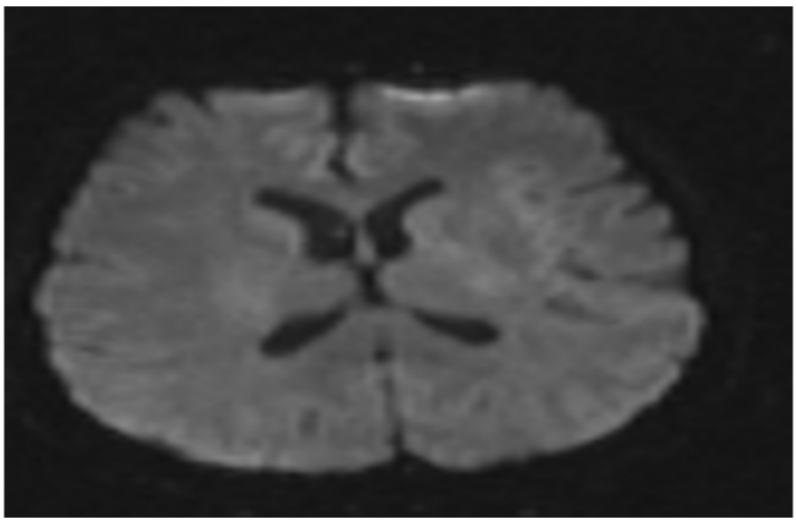

Figure Brain MRI Showed No Acute Infarction

intravenous diazepam. Again, the neurological examination was without focality. A repeat diffusion MRI (Figure 1) was also normal. However, arterial blood gas analysis revealed sodium of 110 mmol/L and potassium of 2.8 mmol/L. With the suspicion of laboratory error, a repeat arterial blood specimen was withdrawn from the femoral artery, but the results were similar. Venous biochemistry confirmed that arterial gas analysis, creatinine (0.6 mg/dL) and BUN (7.3 mg/dL) were normal. (Na:115mmol/L,K:3,0mmol/L,magnesium of 1,34 mmol/L and calcium was7,8 mg/dL). She also had sufficient amount of urination (>2000 ml). Therefore we excluded contrast-induced nephropathy. For the differential diagnosis of hypo-osmolar hyponatremia, we measured the serum-free T4, thyroid stimulating hormone and serum cortisollevels, which were normal.

The patient was treated with hypertonic saline (3% NaCl) at a rate of 2 mL/hr and vasopressin antagonist tolvaptan 15 mg at first and isotonic (0,09 NaCl). The patient mental status returned to normal 12 hours later and the serum and urine sodium levels returned to normal 36 hours later. After having her placed to general room from the intensive care unit, she was followed up for 3 days and then discharged. Thiazide was stopped and calcium antagonist and angiotensin receptor blocker was prescribed.

Discussions

Sodium is an essential electrolyte that maintains the extracellular fluid volume and osmotic equilibrium.[1-9] The plasma sodium concentration is maintained in the normal range by water and sodium intake, and the renin-angiotensin system.[10,11] Patients with hyponatremia are generally asymptomatic. However, it may sometimes be associated with increased mortality and morbidity[12-17], especially when it occurs rapidly, as in our case.

There may be several mechanisms for hyponatremia. Fluid homeostasis impaired with aging and the risk of hyponatremia among elderly people is compounded by chronic diseases and long-term medication use. Women are more affected than men as a result of the smaller fluid volume and sex-related hormonal factors. Thiazides are known to induce mild hyponatremia. Our patient had been taking thiazide for control of blood pressure. Her previous sodium levels were ranged between 127-134 mmol/L. It is a routine and advised procedure to increase the amount of oral fluid intake or iv infusion of serum saline for prevention of kidney injury after contrast exposure which may contribute to dilution and decrease in osmolality of intravascular fluid.

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 in adult patients. As far as we searched, there are five cases reported a developing of severe symptomatic hyponatremia following coronary angiography or intervention[18-20], of which they have used iohexol or urografin. Interestingly,

the case we presented had coronary angiography and intervention with the same pharmacologic contrast material content with same density (iohexol 350mg/100mL) one year before without complication,however a different company trademark. Previous contrast exposure was not mentioned in other reported cases. Because of unfamiliarity with contrast media-related hypo-osmolality and hyponatremia, we have delayed in diagnosis and treatment in our case. First, we suspected anxiety and cerebrovascular complications. An earlier biochemical or arterial blood gas analysis for serum electrolytes would lead us for a prompt diagnosis and treatment.

In conclusion, the diagnosis of acute hyperosmolar hyponatremia should be considered in patients developing mental or behavioral abnormalities following coronary angiography.

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