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Case Report

Impending cardiac tamponade caused by salt supplement in a hyponatremic patient with chronic kidney disease

Abstract

We documented a hyponatremic patient who developed imminent cardiac tamponade upon oral salt supplement. A 72-year-old diabetic woman had hemorrhagic stroke; pericardial effusion; and chronic kidney disease, stage IV. She developed hyponatremia (serum sodium level, 125 mmol/L), compatible with the syndrome of inappropriate antidiuretic hormone, and received oral salt supplement 9 g/d for 4 days. Shortness of breathing and increasing heart rate ensued, and the echocardiography found accumulation of pericardial effusion with signs of impending cardiac tamponade. Pig-tail drainage through pericardiocentesis was done, and the vital signs were stabilized. We found the production of pericardial effusion increased from 100 to 220 mL/d after oral salt supplement at 3 g/d was reassumed. We discuss the relationship between serum sodium levels, the dose of salt supplement and the accumulation of pericardial effusion.

A 72-year-old woman was hospitalized for rehabilitating right hemiparesis caused by multiple intracranial hemorrhage episodes that occurred 4 months before this admission. She had diabetes mellitus and hypertension for which she was taking gliclazide, sitagliptin, bisoprolol, amlodipine, and irbesartan. She also had chronic kidney disease, stage IV. Pericardial effusion was noticed 4 months before this admission, without etiology found after intensive investigation. Two months later, follow-up echocardiography found left ventricular ejection fraction 67%, left ventricle end-diastolic diameter 39.7 mm, and the thickness of the pericardial effusion 2.1 cm (Fig. 1A and 1A1). Colchicine was prescribed. She did not have shortness of breathing.

Upon admission, her blood pressure was 131/70 mm Hg, pulse rate was 100 beats per minute, respiratory rate was 18 breaths per minute, and body temperature was 36.2°C. There was no jugular venous engorgement. Her heart rhythm was regular, without murmurs, rubs, or gallops. No lower leg pitting edema was found. Blood chemistry tests revealed the following results: serum sodium 125 mmol/L, blood urea nitrogen 45 mg/dL, and serum creatinine 1.9 mg/dL.

For hyponatremia, oral salt supplement 3 g 3 times a day had been given for 4 days. From days 3 to 4 at hospitalization, her heart rate was increasing to 120 beats per minute. On day 4, her serum sodium level increased to 152 mmol/L. Blood urea nitrogen became 67 mg/dL, and serum creatinine level was 2.7 mg/dL. On day 6, pulsus paradoxus was demonstrated. Echocardiography found compression sign on right atrium, without compression sign on right ventricle; left ventricular ejection fraction was 67%, and left ventricle end-diastolic diameter was 38.9 mm. The thickness of the pericardial effusion was 3.2 cm (Fig. 1C and 1C1).

Pericardiocentesis with pig-tail open drainage was performed on day 6, and 680-mL serosanguinous effusion was drained. Thereafter, the amount of pig-tail drain was averaged 80 to 100 mL/d. Her heart rate decreased gradually over the next 5 days, down to 80 to 90 beats per minute. Blood urea nitrogen was 41 mg/dL, and serum creatinine level was 1.6 mg/dL on day 13 (7 days after pericardiocentesis). Hyponatremia was detected again on day 12 (serum Na level, 128 mmol/L), and oral salt supplement 1 g 3 times per day was given for 1 day. Her serum sodium level returned to reference range (142 mmol/L) on day 14. Twice amount of pericardial effusion drain (220 mL/d) was recorded on day 15. She received pericardiopleural window creation on day 26. Microscopic examination of the pericardium specimen did not reveal evidence of malignancy or tuberculosis. High urine osmolality (244 mOsm/kg) and urine sodium content (30 mmol/L) were compatible with the syndrome of inappropriate antidiuretic hormone, likely secondary to the central nervous system lesions.

To our best knowledge, this is the first report in the literature describing the response of pericardial effusion upon salt supplement. Our patient had experienced 2 episodes of rapid production of the pericardial effusion in timely manner after oral salt supplement, which made it difficult to refute salt loading as the etiology. However,

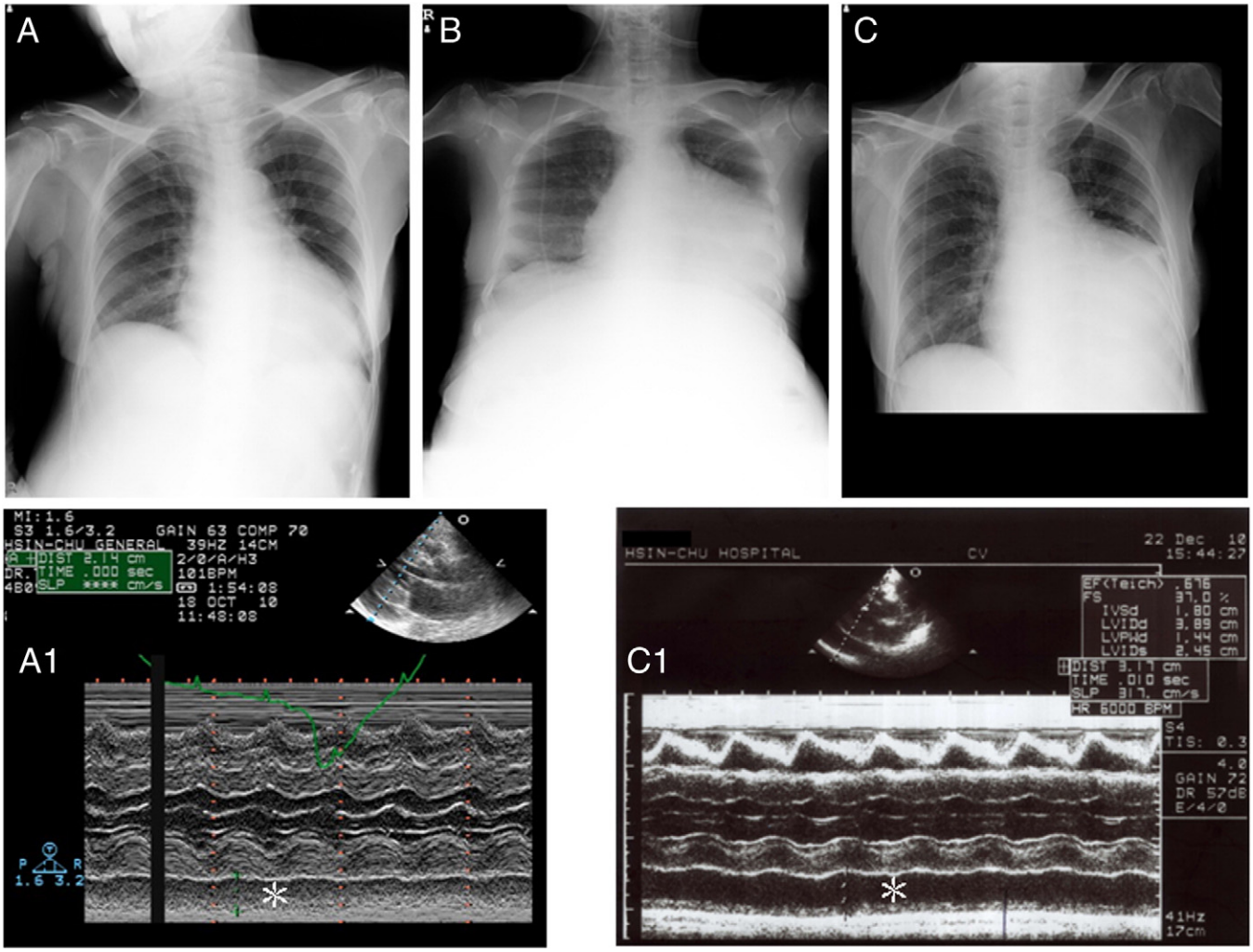

Fig. 1 Chest x-ray films (A-C) and echocardiography (A1 and C1) showing pericardial effusion. A and A1, Four months before this admission. The thickness of the pericardial effusion was 2.1 cm (asterisk in A1). B, Chest x-ray at admission. C and C1, Day 6 on admission, right before pericardiocentesis was performed. The thickness of the pericardial effusion was 3.2 cm (asterisk in C1).

one may argue that the chest x-ray at admission already indicated massive pericardial effusion, and therefore, salt supplement might have not worsened the pericardial effusion. We believe that this is not the case. The patient developed shortness of breathing and racing heart rate after salt supplement, and pericardiocentesis relieved both signs. Meanwhile, evidences including the presence of pulsus paradoxus and the compression sign of the right atrium detected by echocardiography strongly suggested impending cardiac tamponade.

Clinical implication of our observation is multifold. First, review of the patient's history and the chest roentgenography for possible presence of pericardial effusion is advised before initiation of salt supplement. Second, elevation of serum sodium levels to either over upper limit or within reference range could accelerate the production of pericardial effusion. Third, even a seemingly naïve amount of salt can cause cardiac tamponade unexpectedly.

In our case, at the episode of impending cardiac tamponade, the serum sodium level rose 32 mmol/L in 4 days; later when the rapid effusion production occurred again, the sodium level rose 14 mmol/L in 2 days. If giving sodium is medically necessary in patients with pericardial effusion, we suggest that the rate be set to make the serum sodium elevation at most 7 to 8 mmol/L per day, slower than that recommended (10-12 mmol/L for the first 24 hours) by a recent consensus expert panel [1]. However, the applicability may be limited by the possibility that the production rate of pericardial effusion may vary widely by different etiologies. In an interventional study, Luft et al [2] gave 8 healthy male volunteers sodium supplement in oral form and in intravenous form. They found that left ventricle end-diastolic diameter, measured by echocardiography, increased significantly without the presence of pericardial fluid. The study implied that healthy pericardium is not susceptible to produce effusion by salt supplement.

In summary, imminent cardiac tamponade could develop when exogenous salt was given in the presence of preexistent pericardial effusion.

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