CASE REPORT

Cardiac tamponade causing severe reversible hyponatraemia

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SUMMARY

Severe hyponatraemia in setting of cardiac tamponade is very rare and only few case reports have been reported so far. This case report highlights pericardial tamponade as a rare but easily treatable cause of severe hyponatraemia. Pertinent literature is also reviewed. A 70-year-old woman presented to the emergency department with altered mental status. She was tachycardic and hypotensive with cardiomegaly on a chest X-ray. Serum sodium was severely low at 109 mmol/L and was identified as the likely cause for her abnormal mentation. She was also in acute renal failure with serum creatinine of 4.1 mg/dL. A transthoracic echocardiogram was done that showed a large pericardial effusion with evidence of tamponade physiology. She underwent emergent pericardiocentesis with rapid improvement in clinical picture and blood pressure. Her serum sodium level rapidly improved and was normal in 48 hours.

BACKGROUND

Severe hyponatraemia is a potentially life-threatening condition. Most common causes are related to medication use, hormonal imbalances (like syndrome of inappropriate antidiuretic hormone, Addison's disease, etc), kidney or liver disorders and severe cardiac failure. Pericardial effusion with tamponade has been very rarely described as an aetiology for hyponatraemia.^{1–6} Hyponatraemia may be asymptomatic or may present as a myriad of symptoms including nausea, malaise, lethargy, decreased level of consciousness, headache, seizures and coma.⁶ In this report, we describe development of severe hyponatraemia in a patient with a large pericardial effusion with cardiac tamponade and its rapid resolution following pericardiocentesis. Pericardiocentesis is life-saving in these situations by preventing side effects of severe hyponatraemia. This case report also explains the pathophysiology of hyponatraemia in setting of cardiac tamponade.

CASE PRESENTATION

A 70-year-old woman presented to the emergency room (ER) with progressive worsening of mental status for 2 days. More recently, she had also been stumbling and falling. Her medical history included diabetes mellitus, coronary artery disease, hypertension and chronic kidney disease stage III. Two weeks prior to current presentation, she had been

admitted for atypical chest pain. At that time, a nuclear cardiac stress test was normal. She also had a computed CT scan of the chest (with iodinated contrast) at that time which did not show any pulmonary embolism, aortic dissection and pericardial effusion. In the ER, she was hypotensive (87/61 mm Hg), tachycardic (107 beats/min) and hypoxic (pulse oxygen saturation was 84%). She was confused without any localising neurological deficits. There was no peripheral edema although there was jugular venous distention. Her cardiac sounds were muffled and lung auscultation revealed slightly diminished air entry at the bases.

INVESTIGATIONS

Chest X-ray showed mild cardiomegaly with mild pulmonary congestion. A non-contrast CT head was unremarkable. Laboratory evaluation showed serum sodium level of 109 mmol/L (baseline 137 mmol/L), non-anion gap metabolic acidosis on arterial blood gas analysis (pH: 7.32/pCO₂: 32/bicarbonate: 17.4/pO₂: 74). Serum chemistry showed anion gap of 9, serum creatinine of 4.09 mg/dL (baseline 1.6) and brain natriuretic peptide (BNP) of 361 pg/mL (0–100 pg/mL). Other significant laboratory findings include normal thyroid stimulating hormone, normal fasting morning serum cortisol, serum osmolality of 257 mOsm/kg (280–307 mOsm/kg), urine osmolality: 134 mOsm/kg (50–1400 mOsm/kg), urine sodium: 23 mmol/L, FeUrea: 10.6% (<35% suggests prerenal aetiology). She was on no medications that are known to cause hyponatraemia. A bedside transthoracic echocardiogram showed normal left ventricular systolic function and normal valvular function. There was a large circumferential pericardial effusion with grey scale and Doppler evidence of cardiac tamponade physiology (figure 1).

TREATMENT

Initially, she was given 2 L bolus of normal saline intravenously without much change in her haemodynamics. She underwent emergent pericardiocentesis and a total of 750 mL of haemorrhagic pericardial fluid was removed. A drain was left in place but was removed in 24 hours, as there was no further drainage.

OUTCOME AND FOLLOW-UP

The blood pressure improved quickly and heart rate also normalised after the pericardiocentesis. Serum sodium and creatinine returned to baseline within

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Figure 1 (A) Transthoracic echocardiogram. Blue arrow showing RA collapse due to tamponade. (B) Transthoracic echocardiogram demonstrating the variable filling of LV pressure during MV inflow evaluation. LV, left ventricle; RA, right atrium; MV: mitral valve.

48 hours (figure 2). Pericardial fluid analysis was negative for any infectious, autoimmune or malignant aetiology. The aetiology for the pericardial effusion was thought to be idiopathic. Though the spontaneous sodium correction was quite rapid, she did not suffer from any side effects of rapid sodium correction.

DISCUSSION

Hyponatraemia related to pericardial effusion is uncommon but has previously been described. Severe hyponatraemia from

pericardial disease is extremely rare with only a few case reports published so far.¹⁻⁶

Severity of hyponatraemia in cardiac tamponade has been linked to the underlying aetiology. Chang *et al* suggested a higher incidence of severe hyponatraemia in malignant cases as compared with non-malignant cases.⁶ They proposed that this was related to significant pericardial fibrosis, alteration of atrial transmural pressure and atrial natriuretic peptide (ANP) secretion secondary to active cancer cells.⁶ Jong *et al* studied 48 patients with cardiac tamponade of whom 65% of patients had hyponatraemia. All cases had mild to moderate hyponatraemia (>120 mmol/L).⁷ Among these, 35% cases were of malignant pericardial effusion and were strongly associated with hyponatraemia and showed significant improvement following pericardiocentesis.⁷ However, in non-malignant cases they did not find any significant improvement in serum sodium levels postpericardiocentesis.⁷ In contrast, Chang *et al* showed significant improvement in hyponatraemia in both malignant and benign cases of cardiac tamponade. Although, rapid correction of hyponatraemia is generally considered dangerous, all cases discussed by Chang *et al* and the case reported by us did not suffer any significant neurological complications. Kim *et al* have suggested NT-pro BNP as a marker of severity of disease in cardiac tamponade.⁸ In our case too, there was three times elevation in BNP levels thereby suggesting increased severity.

Possible aetiologies of hyponatraemia in cardiac tamponade have been so far linked to syndrome of inappropriate antidiuretic hormone (SIADH) or ANP-related natriuresis (especially in patients with cancer) in few case reports but definitive physiological mechanism is still undetermined.⁶ Low-flow state is seen in many cardiac conditions with cardiogenic shock including cardiac tamponade can also contribute to hyponatraemia. This leads to increase in ADH hormone due to baroreceptor stimulation thereby causing hyponatraemia. Laboratory findings suggests SIADH as cause of hyponatraemia in our case too.

Earlier, it was thought that increase in atrial pressure can cause rise in ANP, but now we know that it was atrial stretch that cause increase in ANP.⁴ In cardiac tamponade, there

Figure 2 The trend of serum sodium and creatinine levels before and after the pericardiocentesis. Day 0 is the day of pericardiocentesis and is represented by ★.

Table 1 Published case reports for cardiac tamponade and associated hyponatraemia along with management and outcome

Author	Age/sex	Serum Na level (mmol/L)	Aetiology of pericardial effusion	Treatment given	Outcome
Present case (2017)	70/F	109	Uraemic pericarditis	Pericardiocentesis	Complete resolution
Rakhshan <i>et al</i> ¹	87/F	119	Postoperative pericardial effusion after the pacemaker placement (complete heart block)	Naproxen for 5 days followed by Colchicine for 14 days	Complete resolution
Rakhshan <i>et al</i> ¹	83/F	116	Postoperative pericardial effusion after the pacemaker placement (severe sick sinus syndrome)	Colchicine for 14 days	Complete resolution
Weekes <i>et al</i> ²	49/M	95	Idiopathic pericardial effusion	Pericardiocentesis, oral fluid restriction, hypertonic saline infusion	Complete resolution
Shafique <i>et al</i> ³	59/M	104	Idiopathic pericardial effusion	Fluid restriction, pericardiocentesis	Complete resolution
Mouallem <i>et al</i> ⁴	58/F	N/F	Malignant pericardial effusion secondary to adenocarcinoma of lung	Pericardiocentesis	Complete resolution
Groves <i>et al</i> ⁵	29/M	117	Spontaneous haemorrhagic pericardial effusion in a patient on warfarin	Warfarin was stopped, FFP and vitamin K was transfused followed by pericardiocentesis	Complete resolution
Chang <i>et al</i> ⁶	Two cases	<120	Malignant pericardial effusion	Pericardiocentesis	Complete resolution

F, female; M, male; FFP, fresh frozen plasma; N/F, not found.

is increase in intra-atrial pressure but no atrial distension; hence, ANP is not elevated. It is postulated that cancer leads to pericardial fibrosis leading to increase in ANP.⁶ Improvement in hyponatraemia after the pericardiocentesis can be explained by following factors: (1) improvement in systolic blood pressure which leads to decrease in ADH production and hence increased diuresis and (2) increase in atrial stretch after the pericardial fluid drainage leads to increase in ANP and hence causes further diuresis; this leads to improvement in hyponatraemia. Mancini *et al* also mentioned that during their experiment on dogs, the ANP levels rose dramatically after the pericardiocentesis.⁹ Very low-serum sodium levels as seen in our case suggests need of exploring further additional unidentified causes for better explanation of severe hyponatraemia.

Our patient had acute on chronic kidney injury based on elevation of serum creatinine compared with her baseline. The most likely cause of this was hypotension and venous congestion secondary to cardiac tamponade leading to decreased blood supply to the kidneys (prerenal). This is further supported by rapid improvement in kidney function after pericardiocentesis. Patient did not require any

haemodialysis as her kidney function improved significantly after pericardiocentesis.

We have summarised all published case reports as of the present to best of our knowledge which have shown improvement in hyponatraemia following pericardiocentesis (table 1). This case further consolidates the impressive reversibility of hyponatraemia following pericardiocentesis which is graphically depicted in the picture (figure 2).

The approximate mortality with serum sodium level of <110 mmol/L is very high 8%.¹⁰ Hence with prompt treatment, we were able to restore the haemodynamic instability and electrolyte imbalance, thereby preventing the mortality in our case. This case report emphasises that tamponade should be considered in the differential diagnosis of unexplained hyponatraemia. Prompt cardiac evaluation and emergent pericardiocentesis can be life-saving in such cases.

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REFERENCES

- 1 Rakhshan E, Mirabbasi SA, Khalighi B, *et al*. Pericarditis-induced hyponatremia after cardiac electronic implantable device (CEID) procedures. *Am J Case Rep* 2015;16:245–9.
- 2 Weekes MP, Reddi BA, Wharton S, *et al*. Hyponatraemia associated with cardiac tamponade and chronic fluid excess. *BMJ Case Rep* 2010. doi: 10.1136/bcr.07.2009.2113. [Epub ahead of print: 9 Jul 2010].
- 3 Shafique R, Sarwar S, Wall BM, *et al*. Reversible hyponatremia related to pericardial tamponade. *Am J Kidney Dis* 2007;50:336–41.
- 4 Mouallem M, Wolf I, Mindlin G, *et al*. Pericardial tamponade-associated hyponatremia. *Am J Med Sci* 2003;325:51–2.

Learning points

- Severe hyponatraemia is a potentially life-threatening condition. Most common causes are related to medication use, hormonal imbalances (eg, syndrome of inappropriate antidiuretic hormone, Addison's disease, etc), kidney or liver disorders and severe cardiac failure.
- Hyponatraemia in setting of cardiac tamponade is rare. The excess release of antidiuretic hormone due to decreased cardiac output in cardiac tamponade is the most commonly postulated mechanism causing hyponatraemia in cardiac tamponade.
- Cardiac tamponade should be considered in the differential diagnosis of unexplained hyponatraemia. Prompt cardiac evaluation and emergent pericardiocentesis can be life-saving in such cases.

- 5 Groves PH, Shah AM, Hutchison SJ. Hyponatraemia secondary to an inappropriately high release of antidiuretic hormone in cardiac tamponade. *Br Heart J* 1990;64:206–7.
- 6 Chang FK, Lee YC, Chiu CH. Hyponatremia in patients with symptomatic pericardial effusion. *J Chin Med Assoc* 2012;75:509–12.
- 7 Jong BH, Wei CC, Shyu KG. Improved hyponatremia after pericardial drainage in patients suffering from cardiac tamponade. *BMC Cardiovasc Disord* 2016;16:135.
- 8 Kim SJ, Shin ES, Lee SG. N-terminal pro-B-type natriuretic peptide as a marker of disease severity in patients with pericardial effusions. *Korean J Intern Med* 2008;23:78–86.
- 9 Mancini GB, McGillem MJ, Bates ER, *et al*. Hormonal responses to cardiac tamponade: inhibition of release of atrial natriuretic factor despite elevation of atrial pressures. *Circulation* 1987;76:884–90.
- 10 Sterns RH. Severe symptomatic hyponatremia: treatment and outcome. A study of 64 cases. *Ann Intern Med* 1987;107:656–64.

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