American Journal of Case Reports

ISSN 1941-5923
© Am J Case Rep, 2015; 16: 245-4249
DOI: 10.12659/AJCR.893209

Received: 2014.12.03
Accepted: 2015.01.06
Published: 2015.04.26

Pericarditis-Induced Hyponatremia after Cardiac Electronic Implantable Device (CEID) Procedures

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEFG 1,2 Elnaz Rakhshan
ABCDEFG 1 Seyed Abbas Mirabbasi
ABCDEFG 1,3 Bahar Khalighi
ABCDEFG 1,2 Koroush Khalighi

1 Easton Cardiovascular Associates, Cardiovascular Institute, Easton, PA, USA.
2 Department of Medicine, Easton Hospital, Drexel University College of Medicine, Easton, PA, U.S.A.
3 School of Pharmacy, Temple University, Philadelphia, PA, U.S.A.

The manuscript was presented at: International Academy of Cardiology, 19th World Congress on Heart Disease, Annual Scientific Sessions 2014: July 25–28; Boston, MA, U.S.A.

Corresponding Author: Koroush Khalighi, e-mail: koroushkhalighi@gmail.com

Conflict of interest: None declared

Case series

Patient: Female, 87 • Female, 83

Final Diagnosis: Hyponatremia induced by pericardial effusion

Symptoms: Shortness of breath

Medication: Colchicine

Clinical Procedure: Cardiac Electronic Implantable Device (CEID)

Specialty: Cardiology • Cardiac Electrophysiology

Objective: Unusual clinical course

Background: Pericardial effusion along with pleural effusion is one of the rare complications of permanent pacemaker placement. Although extremely uncommon, it is more prevalent in elderly patients and may be complicated with hyponatremia.

Case Report: We observed development of hyponatremia in association with pericardial effusion and pleural effusion, within one month after pacemaker placement in two women with BMI of <20.

Case 1: An 87-year-old woman underwent implantation of a transvenous AV sequential pacemaker because of severe bradycardia and complete heart block. Three weeks later, she complained of progressive left-sided rib cage pain and poor oral intake. Her echocardiography showed a moderately large amount of pericardial effusion, but no evidence of tamponade. She also had hyponatremia (Na=119 mEq/dl). Extensive work-up suggested hyponatremia presumably due to SIADH, caused by pericardial/pleural effusion.

Case 2: An 83-year-old woman with history of severe sick sinus syndrome required a transvenous Av sequential pacemaker 3 weeks before. She then presented with generalized weakness, fatigue, and poor oral intake of over one week. There was a small-moderate pericardial effusion echocardiographically, and her serum sodium was 116 mEq/dl.

Conclusions: Although extremely uncommon, pericarditis can develop following transvenous pacemaker insertion, which may result in hyponatremia, likely due to SIADH. The most common scenario is an elderly, petite woman with low BMI (<20), usually after using a helical screw/active fixation pacing leads, several weeks post-implant. Early recognition and therapy can significantly improve outcome and morbidity.

MeSH Keywords: Hyponatremia • Inappropriate ADH Syndrome • Pacemaker, Artificial • Pericardial Effusion

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/893209

Background

Hyponatremia (serum sodium concentration <135 mEq/L) is a common electrolyte abnormality observed in daily practice. It is commonly asymptomatic but can cause a wide range of symptoms such as lethargy and coma, depending on its severity or acuity [1]. Although insertion of a cardiac electronic implantable device (CEID) such as a permanent pacemaker is usually a benign procedure with extremely low complication rates, they may rarely be associates with serious mechanical complications such as myocardial perforation, tamponade, or even death [2]. The development of pericardial effusion after a CEID procedure is uncommon and is felt to be immune-mediated. An extremely rare scenario where hyponatremia is associated with pleural and pericardial effusion is also reported [3].

We describe development of pericardial effusion within one month after CEID placement in two patients, who both presented with acute hyponatremia, but due to two different mechanisms.

Case Report

Case 1

An 87-year-old Caucasian woman who underwent a transvenous AV sequential pacemaker presented to our emergency room with acute intermittent left-sided rib cage pain, associated with three-day history of nausea and vomiting, three weeks after CEID implantation. At the time of admission her blood pressure was 104/54 mmHg with a heart rate of 74 BPM and an oral temperature of 99.4°F. Her oxygen saturation was 96% at room air. She was 60 inches tall and weighed 125 pounds, with a BMI of <19. Although she complained of pleuritic chest wall pain, she was not in any other acute distress. Her physical examination was otherwise unremarkable except for mild bibasilar decreased breath sounds and crackles. Her cardiovascular exam showed a regular rate and rhythm with no thrill or rubs. She had RUQ and LLQ tenderness with deep inspirations and trace ankle edema. Twelve-lead electrocardiograms showed a ventricular paced rhythm. Her chest X-ray was significant for a moderate-sized bibasilar pleural effusion and mild pulmonary venous prominence (Figure 1). Her CT-scan of the chest and abdomen showed moderate bilateral pleural effusion with overlying atelectasis. Her laboratory data was significant for severe hyponatremia (Table 1). Her serum cortisol was normal. Her echocardiogram showed a well-preserved non-dilated left ventricular with an estimated ejection fraction of 65–70%. There was a moderate amount of circumferential pericardial effusion with no evidence of tamponade. The estimated right ventricular systolic pressure was 40 mmHg. Based on her clinical presentation and laboratory data, she was treated for hypovolemic hyponatremia, resulting in gradual improvement of her serum sodium level. Due to her fragile state and since she was hemodynamically stable, medical treatment started and pericardiocentesis was not performed.

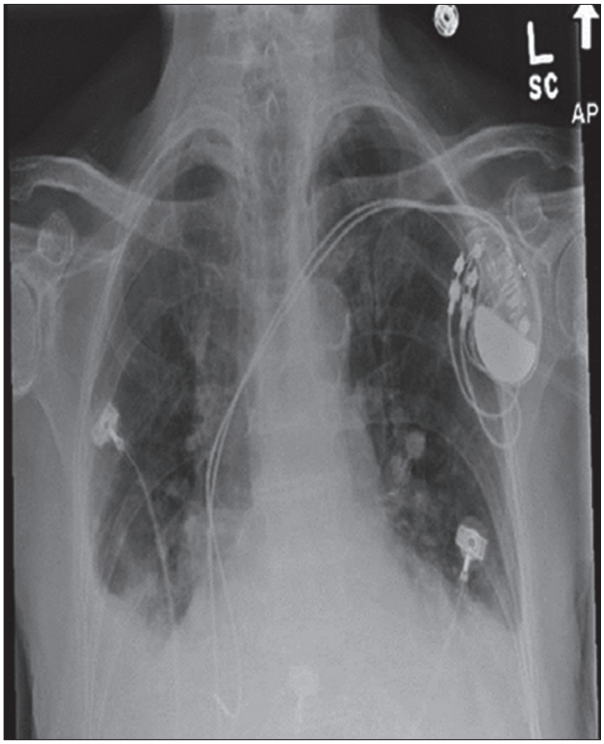

Figure 1. Chest X-ray of patient 1 three weeks after pacemaker implantation.

A bedside thoracentesis showed 450 ml of straw-colored fluid, which was removed. The pleural fluid glucose was 112 mg/dl, protein level of 2.7, LDH of 98(U/L), WBC 207 per cubic millimeter, with 34% neutrophil. Her serum total protein was 5 g/dl, suggesting an exudative fluid.

She was treated with a 5-day course of NSAID (Naproxen) followed with 14 days of colchicine. Echocardiogram two weeks after initiation of treatment showed complete resolution of the pericardial effusion.

Case 2

An 83-year-old Caucasian woman who underwent a transvenous AV sequential pacemaker implantation presented to the emergency room with fatigue, nausea, and poor oral intake of over one week, four weeks after implantation. At presentation the blood pressure was 134/73 mmHg, with heart rate of 66 BPM, respiratory rate 18/minute, temperature 97.9°F and oxygen saturation 99% at room air. She was 59 inches tall and weighed 105 pounds, with a BMI of <19. Her physical examination was otherwise unremarkable. She had bilateral air entry with bilateral decreased breath sounds. There were scattered bibasilar rhonchi.

Table 1. Demographic characteristic, laboratory analysis, indication for pacing and pacing mode of 2 patients with pericardial effusion.

	Case 1	Case 2
Age	87	83
Gender	Female	Female
Serum sodium (mEq/L)	119	116
Serum potassium (mEq/L)	5	4.6
Urea (mg/dL)	19	6
Creatinine (mg/dL)	0.6	0.55
TSH	0.83	2.77
Serum chloride (mEq/L)	96	93
BNP (pg/mL)	120	90
Serum uric acid	4.4	5.5
AST	40	37
ALT	49	44
Alk phos	152	51
Pleural fluid	Exudative	Exudative
Urine sodium (mEq/L)	<10	53
Urine potassium (mEq/L)	31.9	24.7
Urine chloride (mEq/L)	16	75
Urine Creatinine (mg/dL)	–	10.2
Urine Osmolality (mOsm/kg)	393	467
Fe NA	–	0.02
BMI	<20	<20
Pacing mode	Transvenous AV sequential pacemaker	Transvenous AV sequential pacemaker
Indication for pacemaker	Severe bradycardia and complete heart block	Sick sinus syndrome

Fe NA – fractional excretion of sodium; BMP – B-type natriuretic peptide; BMI – body mass index; Alk phos – alkalin phosphatase; TSH – thyroid stimulating hormone; AST – aspartate aminotransferase; ALT – alanine transaminase.

Her cardiovascular examination was remarkable for an irregularly irregular rhythm with normal heart sounds. There was 1–2/6 holo-systolic murmur in the apex with radiation to axilla and 1–2/6 systolic ejection murmur over the left sternal border. There was no heave, thrill, or rub. Her 12-lead electrocardiogram showed atrial fibrillation with controlled ventricular response and a ventricular paced rhythm on demand. Her chest X-ray was significant for bilateral pleural effusions with enlargement of her cardiac silhouette, suspicious for pericardial effusion. Her echocardiogram showed a large pericardial effusion with no evidence for cardiac tamponade. She had a well preserved systolic function with an estimated ejection fraction of 65–70%. There was mild left ventricular hypertrophy. The aortic valve leaflets were thickened but without significant restriction in their motion. Her estimated right ventricular systolic pressure was 35–40 mmHg. There was evidence for mild-moderate aortic valve stenosis, mild mitral regurgitation, and tricuspid regurgitation. Her chest CT-scan showed a large right pleural effusion and a large pericardial effusion (Figure 2). Her laboratory data was significant for severe hyponatremia and increased BNP and urine sodium suggesting SIADH (Table 1). Her serum cortisol was normal. She had a transvenous AV-sequential pacemaker three weeks prior to admission for sick sinus syndrome and symptomatic bradycardia.

After right thoracentesis, 400 cc of clear fluid was removed, which had LDH level of 87 U/L, with a protein level of 3.1 g/dl,

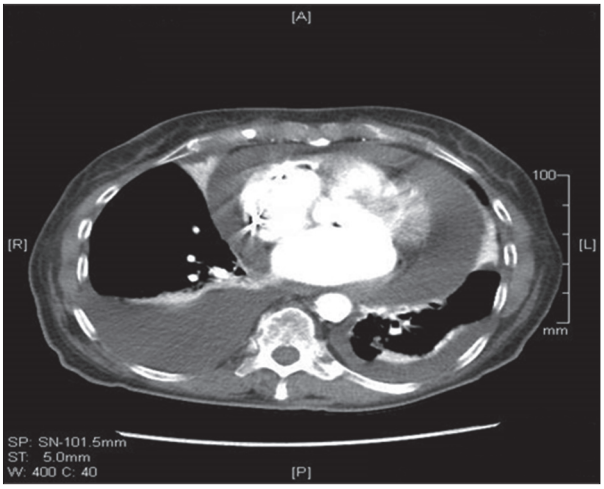

Figure 2. CT scan of patient 2 showed pericardial and pleural effusion four weeks after pacemaker implantation.

and WBC count of 309 per cubic millimeter, with 35% neutrophil count. Analysis was consistent with exudative pleural effusion.

Based on her clinical presentation and laboratory data, she was treated for SIADH, resulting in gradual improvement of her serum sodium level. She was also treated with a two-week course of colchicine, resulting in complete resolution of her pericardial pleural effusion and symptoms.

Discussion

Pericardial Effusion after Permanent Pacemaker Implantation: Although extremely uncommon, post-operative pericarditis may occur following transvenous CEID insertion. The true incidence of pericardial effusion is not known, but is reported to be 2% [4], the majority of which have sub-clinical manifestation.

Mahapatra et al. reported that in the Mayo Clinic's experience, 1.2% of patients developed pericardial effusion and symptoms consistent with perforation after permanent pacemaker implantation [2].

The exact mechanism of pericardial effusion after CEID implantation is unknown. Based on prior studies, an inflammatory process may play a major role in the development of pericardial effusion. Furthermore, the inflammatory process resulting from the active helical screw leads apparatus may have caused direct irritation of the pericardium without an actual lead perforation or dislodgment [5–7].

As also seen in our patients, development of pericardial effusion after CEID is more prevalent in an "elderly female patient presenting a few weeks post-operatively". It is commonly associated with helical screw/active fixation pacing leads and is in association with a low BMI (<20) in an elderly woman [2]. This phenomenon is an inflammatory process and usually has an exudative nature, which responds well to non-steroidal anti-inflammatory drugs (NSAIDS), colchicine, and in some cases, prednisone therapy [5].

Hyponatremia after Pericardial Effusion: The mechanism of hyponatremia due to pericardial effusion is not clear. Several animal studies showed the circulating ADH level was elevated after increased atrial pressure in dogs with experimental cardiac tamponade [8]. Also, increased an ADH level has been seen in patients after pericardial effusion [9]. However, the serum sodium levels were paradoxically decreased after water restriction; this finding is against the diagnosis of inappropriate secretion of ADH [10]. Atrial natriuretic factor (*ANF*) secretion from atrial myocardiocytes after atrial distension and not due to atrial pressure elevation may be responsible for hyponatremia after cardiac tamponade [8]. Decreased glomerulus filtration rate (GFR) due to cardiac tamponade can cause hyponatremia. In these types of cases, fluid hydration can correct hyponatremia, as seen in case one. However the exact mechanism of pericardial effusion induced hyponatremia is still unknown [3].

Pericardial effusion could cause hyponatremia in two ways: An increased atrial pressure resulting in ADH secretion that leads to SIADH, or an increased atrial distension resulting in atrial natriuretic factor release from atrial myocardiocytes [3,4]. Poor nutritional status could also be a contributing factor.

The mechanism by which cardiac tamponade causes hyponatremia is not clear.

There was no evidence for hyponatremia or pleural and pericardial effusions before CEID implantation in either of our cases. We believe that hyponatremia developed due to pericardial effusion and especially pleural effusion production, which developed acutely after helical screw pacemaker CEID implantation.

Referring to Table 1, the laboratory data are not compatible with hypoaldosteronism, hypothyroidism, or liver failure. Our patients did not take thiazide diuretics. Since there was no history of hyponatremia in either patient prior to device implantation, and since there was no further evidence for hyponatremia observed in either patient after over one-year follow-up, we concluded that the only likely etiology for development of hyponatremia was acute pericardial and/or pleural effusion. Once the pericardial effusion resolved clinically, radiographically (CXR and CT scan), and echocardiographically, no further hyponatremia was observed despite over 12-month follow-up.

In our first patient, based on the laboratory data and patient symptoms, the hyponatremia could be due to a combination of poor oral intake and increased atrial distension secondary

to pericardial effusion that causes atrial natriuretic factor (ANF) secretion [3,10]. In our second patient, the laboratory studies were suggestive of a SIADH mechanism as an etiology for hyponatremia. Generally, SIADH is one of the main mechanisms for hyponatremia-induced pericardial effusion [9].

Both of our patients were treated with colchicine for 10–14 days, and repeat echocardiogram after treatment showed resolution of pericardial effusion. Unlike a large hemodynamically significant pericardial effusion, pericardiocentesis may not be required with small-to-moderate pericardial effusion that responds to conventional medical therapy. Early detection and prompt diagnosis can prevent complications.

Conclusions

Although extremely uncommon, we observed hyponatremia caused in association with pericardial effusion and pericardial effusion, induced after ICED implantation with active fixation pacemaker leads.

In our patients, hyponatremia pleural effusion and pericardial effusion resolved after therapy with colchicine. A more aggressive therapy such as pericardiocentesis may be necessary if tamponade or acute clinical symptoms of early tamponade develop, signifying the importance of clinical judgment in determination the timing for invasive therapy, especially in patients at high risk and with multiple co-morbidities.

Although the mechanism for hyponatremia development may be different, pericardial effusion should be considered in the differential diagnosis of hyponatremia in patients with recent CEID placement.

Disclosures

The authors declare that there is no conflict of interest.

References:

1. Lien YH, Shapiro JI: Hyponatremia: clinical diagnosis and management. Am J Med, 2007; 120: 653–58
2. Mahapatra S, Bybee KA, Bunch TJ et al: Incidence and predictors of cardiac perforation after permanent pacemaker placement. Heart Rhythm, 2005; 2: 907–11
3. Chang FK, Lee YC, Chiu CH: Hyponatremia in patients with symptomatic pericardial effusion. J Chin Med Assoc, 2012; 75: 509–12
4. Levy Y, Shovman O, Granit C et al: Pericarditis following permanent pacemaker insertion. Isr Med Assoc J, 2004; 6: 599–602
5. Cevik C, Wilborn T, Corona R et al: Post-cardiac injury syndrome following transvenous pacemaker insertion: A case report and review of the literature. Heart Lung Circ, 2009; 18: 379–83
6. Aizawa K, Kaneko Y, Yamagishi T et al: Oozing from the pericardium as an etiology of cardiac tamponade associated with screw-in atrial leads. Pacing Clin Electrophysiol, 2001; 24: 381–83
7. Khalighi K, Aung TT, Elmi F: The role of prophylaxis topical antibiotics in cardiac device implantation. Pacing Clin Electrophysiol, 2014; 37: 304–11
8. 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
9. 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
10. Mouallem M, Wolf I, Mindlin G, Farfel Z: Pericardial tamponade-associated hyponatremia. Am J Med Sci, 2003; 325: 51–52