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CASE REPORT

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Cardiac tamponade in association with anorexia nervosa: A case report and review of the literature

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Abstract

Anorexia nervosa (AN) is a complex psychiatric disorder that can have devastating cardiovascular complications. Its lesser-known association with pericardial effusion has been recently described in the literature. We present the case of a 45 year-old female who presented with a recurrent small bowel obstruction requiring lysis of adhesions and who was found to have a large pericardial effusion that progressed to cardiac tamponade necessitating surgical intervention. The patient had a body mass index of 14.8 kg/m^2 (i.e. 71% of ideal body weight) and a long-standing history of food aversion, extreme exercise habits, and weight obsession consistent with AN. To the best of our knowledge, this is the first case of AN-associated cardiac tamponade in the United States, and the first requiring surgical intervention. In conclusion, with this and current data regarding AN-associated pericardial effusions, we recommend a low threshold for performing pre-operative echocardiography for those in whom AN is suspected. (Cardiol J 2012; 19, 6: 635–638)

Key words: anorexia, pericardial effusion

Introduction

Anorexia nervosa (AN) is a serious and complex disorder characterized by fear of weight gain, caloric restriction, and a distorted self image that can have devastating health consequences, with cardiac complications constituting one of the major causes of death [1]. It is widely accepted that cardiac manifestations are commonly seen with this disorder, including electrocardiographic abnormalities, orthostatic hypotension, and poor myocardial contractility [1]. The lesser-known association with pericardial effusion has been increasingly described in the literature. We present a case of AN-associated pericardial effusion progressing to cardiac tamponade in a young female with no previous cardiac history.

Case report

A 45 year-old female presented to the emergency department with a recurrent small bowel obstruction requiring lysis of adhesions and exploratory laparotomy. While undergoing induction of anesthesia with propofol, the patient became acutely hypotensive and bradycardiac, with associated non-sustained ventricular tachycardia. An electrocardiogram (ECG) revealed transient inferior

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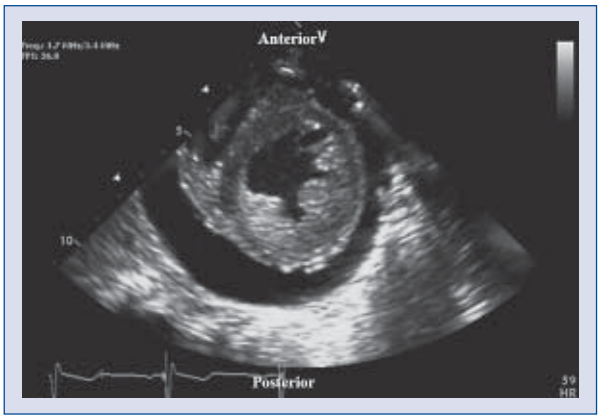

Figure 1. Parasternal short axis view of effusion; large, 2.5 cm in circumference, predominantly posterior pericardial effusion.

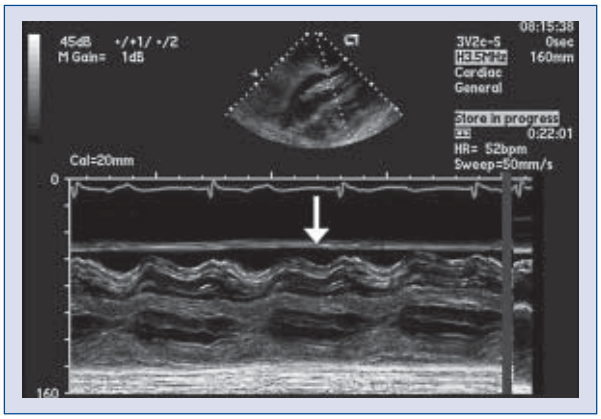

Figure 2. Two-dimensional (2D) subcostal view of the right ventricle (RV); subcostal M-mode of RV with 2D inset demonstrating late diastolic collapse of the RV free wall (arrow).

ST-depressions which resolved after initial fluid resuscitation, with subsequent cardiac enzyme elevations (peak TnT = 0.22 ng/mL, normal < 0.1), suggestive of demand-related (type 2) myocardial infarction (MI). A transthoracic echocardiogram (TTE) was performed, which revealed a normal ejection fraction (EF = 55–60%) with normal regional wall motion, and an incidental, large (2.5 cm in circumference), predominantly posterior pericardial effusion with no initial echocardiographic evidence of tamponade (Fig. 1).

Over the next 24 hours, she became progressively hypotensive with narrowing pulse pressures, decreased urine output, and transient desaturations necessitating 2L oxygen via nasal cannula. Physical exam revealed jugular venous distention to 15 cm H_2O, and a pulsus paradoxus of 10 mm Hg. Repeat ECG demonstrated diffuse low voltage without electrical alternans. Follow-up TTE showed a persistent pericardial effusion with new, subtle late diastolic collapse of the right ventricular free wall (Fig. 2), enhanced ventricular interdependence with tricuspid and mitral inflow velocities of 26% and 18%, respectively, and an increased inferior vena cava diameter (2.3 cm in M-mode) with decreased inspiratory collapse (< 50%), consistent with evolving tamponade.

Due to the predominantly posterior location of the effusion, she was taken to the operating room for a pericardial window. Pericardial fluid and tissue samples were sent for analysis.

Here, the patient admitted to having had bouts of food avoidance throughout her life, with extreme exercise habits that included daily 10-mile runs. On admission, she had a body mass index (BMI) of 14.8 kg/m^2 (71% of her ideal body weight), and chart review was notable for weights consistently below the DSM-IV cutoff for AN, defined as less than 85% of ideal body weight. Laboratory analysis revealed a history of mild hyponatremia (mean = 130 mmol/L), hypoalbuminemia (1.7 g/dL), and low prealbumin (12.3 mg/dL). Her pericardial fluid analysis and other labs were otherwise unremarkable, including a negative work-up for infectious or inflammatory causes with normal thyroid and renal function. Due to the patient's low to intermediate pre-test probability of having obstructive coronary artery disease (CAD), and the suspected demand nature of her MI, cardiac computed tomographic angiography was performed. This revealed no evidence of CAD. The patient recovered uneventfully from her pericardial window and is currently undergoing treatment for AN.

Discussion

We describe a rare case of cardiac tamponade as a complication of AN with significant hemodynamic compromise in the perioperative setting. Only one previous report has described AN-associated tamponade requiring pericardiocentesis [2]. A recent case series described three patients with AN-associated pericardial effusions, two of whom underwent elective pericardiocentesis to prevent tamponade [3]. To the best of our knowledge, this is the first described case of AN-associated cardiac tamponade in the United States, and the first requiring surgical intervention.

While tamponade is a very unusual complication of AN, pericardial effusions appear to be more common than previously recognized (Table 1). The association was first noted in 1983 in a series of four

Table 1. Anorexia-associated pericardial effusions: Literature to date.

	Silvennan [4]	Von Walter**	Silvetti [5]	Frolich [9]	Inagaki [10]	Ramacciotti [6]	Polli [2]	Cho [3]	Docx [7]
Year	1983	1996	1998	2001	2003	2003	2006	2008	2010
Journal	Ped Cardiol	Monatszeitschrift Kinderheilkunde	G Ital Cardiol	Eur Child Adolesc Psychiatry	Int J Eat Disord	Eat Weight disord	Int J Eat Disord	Korean J Pediatr	Eating Disorders
N	4	81	23	65	1	15	1	3	128
Mean age	19.5 ± 3.3	*	14.7 ± 2	15.2 ± 2.1	*	*	*	14.2 ± 0.9	14.4 ± 1.5
% with effusions	*	19.8	61	15.4	*	71.4	*	*	22.2
Mean BMI with effusion	10.8 ± 1.0	*	***	13.5 ± 1.2	142	*	11.8	11.7 ± 1.8	13.6 ± 1.1
Mean BMI without effusion	*	*	*	14.1	*	*	*	*	15.8 ± 1.6
Mean % weight loss with effusion	46.5 ± 6.2	*	*	*	43.7	*	23.0	31.0 ± 3.7	25.0 ± 9.0
Concern for tamponade?	No	No	No	No	No	No	Yes	No	No

*Not applicable or not available; **Referenced in Frolich et al. [9]; ***While specific numbers are not listed here, this study reported clinically silent pericardial effusion that was inversely related to body mass index (BMI)

AN patients with pericardial effusions [4]. One study of echocardiographic findings in 23 asymptomatic AN patients found a mild to moderate pericardial effusion in 61% of patients; the presence of this finding, as well as a reduction in left ventricular thickness and mass, appeared to be inversely related to BMI, caloric intake, free T3 levels, and sodium concentrations [5]. Another study comparing echocardiography in a sample of 15 AN patients compared to ten constitutionally thin controls of comparable age and activity levels found clinically silent pericardial effusions in 71.4% of the patients *vs* 10% of the controls ($p < 0.05$), suggesting that this finding could be suggestive of early cardiovascular involvement with the disease [6]. While these two articles reported clinically silent pericardial effusions in the majority of AN patients, other studies have found AN-associated effusions in only 15–22% of patients [7].

The pathophysiology behind the development of pericardial effusions with AN remains unclear. Malnutrition itself may promote effusions. Indeed, children with severe protein malnutrition in sub-Saharan Africa frequently manifest pericardial effusions in the absence of co-infection with TB or HIV [8]. In these patients, most pericardial effusions resolved with nutritional support only. Conversely, one study of AN patients with pericardial effusions noted a correlation between normalization of body mass and resolution of effusions, yet in those cases there did not appear to be a significant protein deficiency [9].

In the largest study to date, Docx et al. [7] performed echocardiograms in 128 AN patients and found that 22% of patients had clinically silent effusions that significantly resolved with three months of re-feeding. Specific risk factors included BMI ≤ ≤ 13.5 kg/m, weight loss ≥ 25% and IGF-1 level ≤ ≤ 100 ng/mL. They concluded that the presence of pericardial effusions are potentially due to the reduction in pericardial fat and myocardial muscle wasting, associated with more severe forms of starvation, and that the presence of pericardial effusions may be an indicator of AN disease severity [7].

Conclusions

Pericardial effusions are commonly seen in AN, and may progress to cardiac tamponade in rare cases. In view of the literature to date, and the wide prevalence of reported AN-associated pericardial effusions, it is reasonable to consider preoperative echocardiography for patients in whom AN is suspected.

Disclosures

The views expressed in this paper reflect the opinions of the authors only and not the official policy of the United States Army, United States Navy, Uniformed Services University, or the Department of Defense. This is a U.S. Government work. There are no restrictions on its use. There are no conflicts of interest, financial disclosures, grant support or writing assistance.

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