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Pericardial effusion in the setting of Takosubo Cardiomyopathy

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Introduction

Takosubo cardiomyopathy (TC), also known as stress cardiomyopathy, is a syndrome characterized by cardiac dyskinesis with symptoms that mimic those of myocardial infarction, but present in the absence of obstructive coronary artery disease. While its pathogenesis is not completely understood, TC is believed to be triggered by acute illness and/or emotional stress [1]. It is theorized that this syndrome's inflammatory features extend to the pericardium; however little data exists regarding the incidence of pericardial effusion in the setting of TC [2].

Objective

To illustrate an incident of TC with concurrent pericardial effusion – the association of which remains questionable.

Case description

A 76 year-old woman, with a past medical history of diverticulitis, rheumatoid arthritis, hypertension and hyperlipidemia, presented with fecal urination secondary to rectovaginal fistula. While awaiting preoperative clearance for a diverting colostomy, she developed non-radiating, pressure-like chest pain localized substernally.

Physical exam revealed slightly muffled heart sounds and trace bilateral lower extremity edema. Initial troponin level measured 0.03 ng/dl, but increased to 2.39 ng/dl six hours later. CPK, CKMB, and BNP were not assessed.

Electrocardiography demonstrated normal sinus rhythm with a heart rate of 75 beats per minute, a bifascicular block, inverted T waves in the inferolateral leads, and mild (<1 mm.) ST segment elevations in the inferolateral leads. An electrocardiogram done one week prior also displayed a bifascicular block, but did not elucidate any ST segment or T wave abnormalities.

Cardiac catheterization revealed only non-obstructive coronary artery disease. The LAD and the LCx arteries displayed 30-50% occlusions. The patient was given Isosorbidemononitrate, with a complete resolution of symptoms thereafter.

Echocardiogram displayed a mild left ventricular dilatation with an ejection fraction of 20%, and severe hypokinesis of the mid and basal myocardial segments with preserved function of the basal segments – a wall pattern consistent with TC. Additionally, there was a small to medium sized pericardial effusion visible anterior and inferior to the right ventricle. These findings were vastly different from those of an echocardiogram performed just 6 days prior, which demonstrated a left ventricular ejection fraction of 58% with normal size and function of the ventricle.

The patient remained stable and asymptomatic for 5 days; after which a repeat echocardiogram was performed still showing hypokinesis of the mid left ventricle, but now demonstrating an ejection fraction of 35-40% and a decreased size in the pericardial effusion. Furthermore, she was discharged with instructions to follow-up regarding her surgery after six to eight weeks, in order to allow for complete resolution of her cardiomyopathy.

Discussion

Literature regarding pericardial effusion in the setting of TC is sparse. However, several reports describe concomitant cardiac tamponade and stress cardiomyopathy. Management largely includes supportive care and the short-term use of beta blockers, ACE inhibitors, and aspirin. Most cases completely resolve within one to four weeks, but reported in-hospital mortality rates have ranged from zero to eight percent. This data does not account for effusions, which can cause further complications and an increased mortality [3]. Additionally, the emergence of cardiac magnetic resonance imaging as tool for diagnosing myocardial inflammation has led to a correlation between TC and pericardial effusion. Eitel *et al.* found that over half of all Takosubo cases studied presented with pericardial effusions, but the power of this study was low as only twenty-six cases were assessed [4]. Furthering the inflammation-based theory between TC and effusion, endomyocardial biopsy specimens have been shown to possess mononuclear inflammatory areas of fibrotic responses - indicating a potential process whereby myocardial inflammation extends to the overlying pericardium, causing effusion [1]. As such, we believe this is an area that would benefit from further clinical and basic science research.

References

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