Redistribution of pericardial effusion during respiration simulating the echocardiographic features of cardiac tamponade

Raymond Maung M. Khin Hou, Angel I. Martin, Emmanuel A. Bassily, G. Joseph Coffman, Maqsood A. Siddique, David M. Whitaker

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Introduction: The aim of the study was to identify the significance of pericardial effusion and physiology of cardiac tamponade.

Case Report: We used cardiac imaging with echocardiography and cardiac computed tomography (CT) in a patient with pericardial effusion. Two-dimensional transthoracic echocardiography was used to identify the significance of pericardial effusion and 64 multi-slice, ECG retrospectively gated cardiac CT was used to confirm the physiology of cardiac tamponade. On follow-up of serial echocardiograms, there was a development of pericardial effusion and possible signs of cardiac tamponade. To confirm the significance of these findings, a cardiac CT scan was performed which showed redistribution of the pericardial effusion with respiration causing transient echocardiographic features mimicking cardiac tamponade.

Conclusion: The cardiac CT scan is helpful in cases of pericardial effusion with equivocal echocardiographic features of cardiac tamponade. In asymptomatic patients, small to moderate effusions can be followed with serial echocardiograms to evaluate progression and signs of hemodynamic compromise. Although some echocardiographic features may mimic signs of cardiac tamponade, cardiac CT scan can be used to rule out hemodynamic compromise.
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Keywords: Cardiac computed tomography, Cardiac tamponade, Echocardiography, Pericardial effusion

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INTRODUCTION

Pericardial effusion is normally located along the pericardial space of the inferior wall of the heart when standing and posterior wall when supine. As the pericardial effusion increases, it becomes more circumferentially distributed. Though the pericardial sac may be compliant when accommodating a small to moderate effusion, distribution changes can be seen with normal respiration.

The management of pericardial effusions depends on the size, etiology, rate of progression, as well as signs and symptoms suggesting the clinical presence of cardiac tamponade. Asymptomatic small to moderate effusions can be followed with serial echocardiograms to evaluate progression and early signs of hemodynamic compromise. However, some echocardiographic features may mimic the early appearance of cardiac tamponade or pericardial constriction. Further testing with a different imaging modality such as, cardiac computed tomography scan, is useful to evaluate for the presence of hemodynamic compromise. This has a great impact on clinical decision-making regarding the timing and urgency of pericardiocentesis or other surgical interventions.

CASE REPORT

A 67-year-old male presented for preoperative evaluation of cataract surgery. His medical history included squamous cell carcinoma of the left lung with mediastinal involvement, diagnosed two years prior requiring partial resection, chemotherapy, and radiation. On presentation, the patient had a functional capacity of more than four metabolic equivalents without chest pain, dyspnea, or any other complaints. Physical examination revealed no evidence of jugular venous distention, Kussmaul sign or pulsus paradoxus. Lung fields were clear to auscultation. Heart sounds were normal without murmurs, rubs or gallops. The electrocardiogram (ECG) showed normal sinus rhythm, no evidence of low voltage or electrical alternans. Transthoracic echocardiogram showed normal left ventricular systolic function and a small pericardial effusion (inferoposterior effusion; <10 mm pericardial space thickness) without echocardiographic evidence of cardiac tamponade. The patient remained asymptomatic, however follow-up echocardiogram three months later revealed moderate size inferoposterior pericardial effusion (10–20 mm thickness), which was larger than the previous study. There was possible early systolic inversion of the right atrium (RA) as shown in Figure 1 and early diastolic collapse of the right ventricle (RV) during inspiration as shown in Figure 2b. These findings were concerning for early cardiac tamponade. A cardiac CT was subsequently done to further evaluate this significant change in pathophysiology of the pericardial effusion. The cardiac CT images obtained during inspiration and expiration, showed no pericardial thickening, calcification, constriction or cardiac tamponade physiology. However, marked redistribution of the pericardial fluid was noted with each respiratory cycle.

Clinically, the patient continued to remain asymptomatic. Due to concerns for malignant pericardial effusion, he was monitored with periodic physical examinations as well as follow-up echocardiograms. For three months, no changes in the pericardial effusion were noted. Four months later, the patient developed pleuritic chest pain with dyspnea. The pericardial effusion had grown in size (>20 mm thickness) as confirmed by echocardiogram with Doppler evidence of hemodynamic significance. As a result, he underwent a right anterior thoracotomy, creation of pericardial window and drainage of 400 mL of pericardial fluid. He remained asymptomatic postoperatively with only a trace residual amount of pericardial fluid noted.

DISCUSSION

During inspiration, the pulling of the diaphragmatic parietal pericardium makes the inferior pericardial space adjacent to the RV larger, while the heart remains in the same position due to its superior attachments to the diaphragmatic base (Figure 3a, lower panel). This allows for the pericardial fluid from other parts of the heart to shift, or redistribute to the inferior space (Figure 3a, upper panel, adjacent to the RV wall). The reverse of this process occurs during expiration as the inferior space becomes smaller, thus allowing the fluid to shift back to other parts of the pericardial space (Figure 3b, upper and lower panels). In addition, during inspiration, the increase in diastolic filling of the RV also leads to more fluid accumulating in this inferior space [1]. During this process, the intra-cardiac pressure of the RV becomes the lowest, temporarily leading to RV collapse (Figure 2b).

Studies have been done in normal subjects without pericardial effusion and have demonstrated an increase in RV diastolic filling and decrease in left ventricle (LV) diastolic filling during inspiration. Reciprocal effects occur during expiration, referred to as ventricular interdependence [1]. The net effect of these changes on pericardial fluid during respiration is neutral in the normal population [1]. However, the ventricular interdependence is more pronounced in cases of cardiac tamponade or pericardial constriction where the intra-pericardial pressure exceeds the intra-cardiac pressures [2]. Abnormal inspiratory increase of right ventricular dimensions and abnormal inspiratory decrease of left ventricular dimensions are suggestive features of cardiac tamponade [3].

In our case, there were no significant changes in RV and LV end diastolic dimensions during inspiration and expiration (Figure 4). Similarly, non-significant changes were seen in LV volumes and LV systolic functions with respiration. This suggests that neither RV and LV
diastolic fillings nor systolic functions are affected by respiration. Therefore, changes in ventricular volumes during respiration play a minor role in redistribution of pericardial effusion. Acute and rapid accumulation of a small amount of pericardial fluid can cause a sudden increase in intra-pericardial pressure causing cardiac tamponade. Chronic accumulation of a pericardial effusion does not result in cardiac tamponade until the pericardial effusion is large enough to increase intra-pericardial pressure beyond the pericardium’s compliance. Collapse of the lower pressure chambers (RA and RV) occurs first when the intra-pericardial pressure exceeds the intra-cardiac pressures. The RA inversion is not a very specific sign for cardiac tamponade and observed in approximately 18% of patients with pericardial effusion without clinical cardiac tamponade (82% specificity) [4]. During late diastole or early systolic phase of the cardiac cycle, the intra-cardiac pressure of the RA is the lowest and its thin wall is most vulnerable to invagination. This effect is most prominent in the supine position when posterior redistribution of the effusion accumulates around the RA. Narrowing or compression of the RV in diastole during expiration is strongly associated with cardiac tamponade [5, 6]. The echocardiogram of our patient showed RV collapse during inspiration but no RV collapse during expiration. This phenomenon is due to the inferior redistribution of pericardial fluid during inspiration as demonstrated by cardiac CT scan. These mimicking features of RA and RV collapse were observed in our patient's simulated cardiac tamponade physiology. Other characteristic two dimensional echocardiographic signs such as inferior vena cava plethora with blunted response to respiration (dilated inferior vena cava with lack of inspiratory

Figure 1: Long-axis (apical 4-chamber) echocardiographic view of the heart in early systole during normal respiration. Short duration (< 1/3 of cardiac cycle) of RA inversion (white arrow) in early systole (indicated by the ECG) without significant pericardial effusion was seen in this view.

Figure 2: Subcostal long-axis (4-chamber) echocardiographic view of the heart with pericardial effusion during early diastole of cardiac cycle (indicated by the ECG) with expiration (Figure 2a) and inspiration (Figure 2b) on the same study. Moderate amount of fluid measuring 12 mm (line in Figure 2a) at tricuspid annulus, adjacent to the inferior wall of RV with normal RV excursion during expiration and apparent large pericardial effusion measuring 31 mm (line in Figure 2b) at tricuspid annulus with RV collapse (white short arrow) during inspiration. The long white arrows indicate tissues inferior to the heart in Figure 2a and the larger size of pericardial effusion and the collapse of RV wall in Figure 2b.

Figure 3: Cardiac CT demonstrates redistribution of pericardial effusion in diastole during inspiratory and expiratory breath holding. Antero-lateral pericardial effusion (at the level of RV free wall on axial views in top panel) measures 8.1 mm during inspiration (Figure 3a, upper panel) and 13.4 mm during expiration (Figure 3b, upper panel). Inferior pericardial effusion (adjacent to the RV and LV inferior wall on coronal views in bottom panel) measuring 14.1 mm during inspiration (Figure 3a, lower panel) and 7.25 mm during expiration (Figure 3b, lower panel). RV diastolic diameters show 43.1 mm during inspiration (Figure 3a, upper panel) and 41.0 mm during expiration (Figure 3b, upper panel).
collapse) [7] and abnormal ventricular septal motion [8] are useful in supporting cardiac tamponade, but often seen in patients with congestive heart failure and with constrictive pericarditis. Doppler echocardiographic evidence of abnormal inspiratory increase of blood flow velocity through the tricuspid valve and decrease of mitral valve flow velocity may be too sensitive to indicate hemodynamically significant cardiac tamponade [9]. These abnormal flow velocity findings are non-specific, and seen in obstructive airway disease, pulmonary embolism, acute cardiac dilatation, pleural effusion, constrictive pericarditis, or in right ventricular infarction. Moreover, these signs may be exaggerated in dehydration or hypovolemic states where intra-cardiac pressures are relatively lower than intra-pericardial pressure. On the other hand, the signs of RA or RV diastolic collapse may be blunted, masked or absent in conditions where right sided intra-cardiac pressures are chronically elevated, with decreased compliance, or hypertrophy of these chambers.

The diagnosis of cardiac tamponade by echocardiographic features alone is not sufficient in supporting hemodynamic significance in some cases where cardiac CT scan may be useful [10]. In addition, constrictive pericarditis can complicate chronic pericardial effusion, causing an effusive constrictive pericarditis. This can pose a diagnostic dilemma and multimodality imaging may play a role in diagnosing these complex diseases [11, 12].

CONCLUSION

Small and moderate sized pericardial effusions are usually asymptomatic and can be managed conservatively with follow-up echocardiograms to monitor their progression. The smaller pericardial effusions may resolve. However they may become larger, leading to cardiac tamponade requiring pericardial drainage, window or pericardiectomy. When echocardiographic features are inconclusive or equivocal for hemodynamic significance or compromise, advanced cardiac imaging with cardiac CT or magnetic resonance imaging may be useful. Advances in ECG gated cardiac CT technique allows clinicians, to not only evaluate pericardial pathology, but also cardiac function and dynamic changes in pericardial effusion during respiration. Our case provides insight into this area of research thus prompting further investigation which can allow these imaging modalities to serve as new diagnostic tools for clinical decision making. In summary, non-invasive cardiac imaging can provide the diagnosis, the early detection of hemodynamic significance, and the assistance in timing for pericardiocentesis or surgery of pericardial effusion in asymptomatic patients.

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Author Contributions

Raymond Maung M. Khin Hou – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
Angel I. Martin – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
Emmanuel A. Bassily – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published
G. Joseph Coffman – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Figure 4: Cardiac CT (4-chamber long-axis views in top panel and mid-LV short-axis view in bottom panel) shows RV and LV measurements in end diastole during inspiratory and expiratory breath hold. The RV end diastolic diameter measures 43.0 mm in maximal diameter and 36.2 mm in mid cavity during inspiration (Figure 4a, upper panel) and 41.2 mm in maximal diameter and 35.1 mm in mid cavity during expiration (Figure 4b, upper panel). The LV end diastolic diameters measure 52.0 mm in mid cavity during inspiration (Figure 4a, lower panel) and 51.0 mm in mid cavity during expiration (Figure 4b, lower panel).
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David M. Whitaker – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest
The authors declare no conflict of interest. All funding sources as supporting this case report have been provided through the James A. Haley Veteran Affairs hospital.

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