EDORIUM Journals

Int J Case Rep Images 2020;11:101091Z01MK2020.
www.ijcasereportsandimages.com

CASE REPORT PEER REVIEWED | OPEN ACCESS

Pericardiocentesis in cardiac tamponade: A case for "Less is More"

M Imran Khan, Wissam Al Sahali, Peter Quigley, Charles McCreery, Jonathan Dodd, Rory O'Hanlon

ABSTRACT

Introduction: Cardiac tamponade could be a life-threatening condition and immediate pericardiocentesis is the treatment of choice. Acute decompensated heart failure as a complication of pericardiocentesis is less well known. Understanding the pathophysiology of cardiac tamponade may give clues to understanding the etiology of this uncommon but life-threatening condition. Case Report: A 53-year-old female underwent emergency pericardiocentesis for acute cardiac tamponade. Baseline echocardiography demonstrated a large global pericardial effusion with normal left ventricular dimensions and systolic function, and typical features of tamponade. The clinical situation improved considerably following the removal of 1.5 liters of pericardial fluid over a period of 15 minutes. Within 24 hours she developed acute decompensated heart failure (ADHF). Echocardiography demonstrated severe biventricular systolic dysfunction while cardiac magnetic resonance imaging showed wall motion abnormalities consistent with inverted Takotsubo cardiomyopathy. On medical therapy she recovered completely after three weeks. Conclusion: Large volume pericardiocentesis may be a triggering mechanism for biventricular failure.

Keywords: Cardiac tamponade, Heart failure, Pericardiocentesis, Takotsubo cardiomyopathy

M Imran Khan[1], Wissam Al Sahali[1], Peter Quigley[2], Charles McCreery[2], Jonathan Dodd[3], Rory O'Hanlon[2]

Affiliations: [1]Cardiology Fellow, Department of Cardiology, St Vincent's University Hospital, Elm Park, Dublin 4, Ireland; [2]Consultant Cardiologist, Department of Cardiology, St Vincent's University Hospital, Elm Park, Dublin 4, Ireland; [3]Consultant Radiologist, Department of Radiology, St Vincent's University Hospital, Elm Park, Dublin 4, Ireland.

Corresponding Author: M Imran Khan, Cardiology Fellow, St Vincent's University Hospital, Elm Park, Dublin 4, Ireland; Email: sidharat1@gmail.com

Received: 16 September 2019
Accepted: 11 November 2019
Published: 20 January 2020

How to cite this article

Khan MI, Al Sahali W, Quigley P, McCreery C, Dodd J, O'Hanlon R. Pericardiocentesis in cardiac tamponade: A case for "Less is More". Int J Case Rep Images 2020;11:101091Z01MK2020.

Article ID: 101091Z01MK2020

****

doi: 10.5348/101091Z01MK2020CR

INTRODUCTION

Cardiac tamponade is a condition caused by compression of the heart due to accumulation of fluid or gas in the pericardial sac [1]. Percutaneous drainage of pericardial fluid is called pericardiocentesis [2]. With a notable exception of aortic dissection, immediate pericardiocentesis is mandatory in patients with cardiac tamponade and shock [1, 2]. Acute decompensated heart failure in the post pericardiocentesis scenario is a less well described entity. Clues to understanding this phenomenon may lie in the pathophysiology of cardiac tamponade itself. It is essential that this rare but potentially fatal complication is anticipated in patients undergoing pericardiocentesis for cardiac tamponade.

CASE REPORT

A previously healthy 53-year-old Caucasian woman presented to our emergency department with a history

of shortness of breath and abdominal pain. Her blood pressure (BP) was 125/98 mmHg and pulse rate was 114/min. Jugular venous pulse was raised.

A diagnosis of cardiac tamponade was made on the basis of echocardiographic and CT chest findings (Figures 1 and 2). Brain Natriuretic Peptide (BNP) was 49.4 pg/ml. Pericardiocentesis was performed from the subxiphoid approach and 1.5 liters of hemorrhagic pericardial effusion was aspirated immediately, and a pig tail catheter was left in situ for 24 hours. Prior to pericardiocentesis her blood pressure was 130/120 with sinus tachycardia of around 115 beats per minute.

It was a hemorrhagic pericardial effusion. Consistent with an exudate. Malignant cytology analysis was negative. Acid fast stain and gram stain was negative. The cause of pericardial effusion remained unknown.

Within 24 hours the patient developed acute breathlessness and circulatory shock. Echocardiography showed severely reduced left and right ventricular systolic function and no recurrence of pericardial effusion (Figures 3 and 4).

A repeat BNP had increased to 914 pg/ml. After stabilisation and treatment for ADHF, the patient underwent a cardiac MRI which confirmed a dilated left ventricle with severe hypokinesis of the left ventricle (LV) and right ventricle (RV) myocardial segments at mid ventricular level with preserved contraction of the basal and apical myocardial segments and an ejection fraction of 29% (Figures 5 and 6). Delayed enhancement images did not show any evidence of myocarditis, acute myocardial infarction or scar.

Over a period of three weeks, the patient's clinical situation improved with optimal medical therapy. Echocardiography, pre discharge, had returned to normal (Figures 7 and 8). The etiology of her pericardial effusion remained unknown.

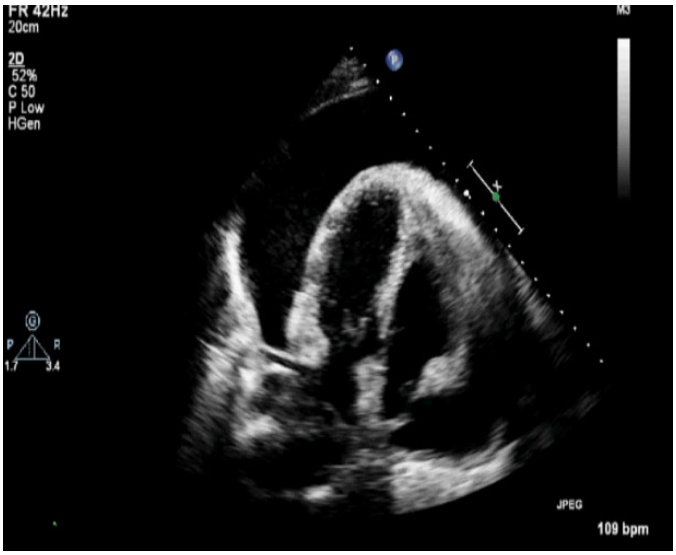

Figure 1: Massive pericardial effusion (Apical view).

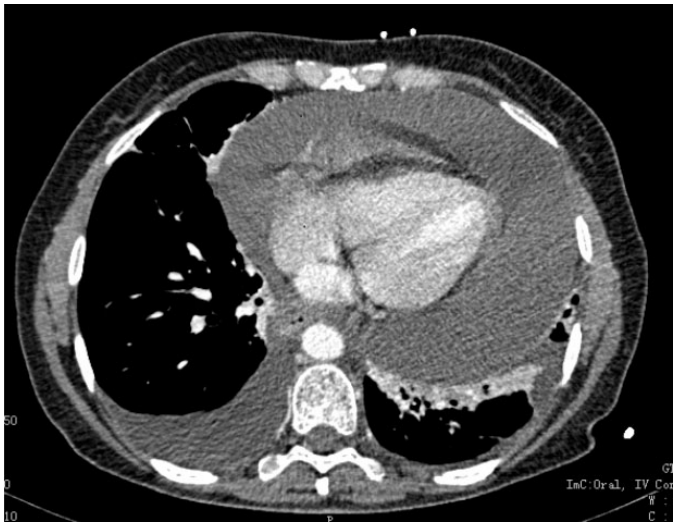

Figure 2: CT chest showing massive circumferential pericardial effusion.

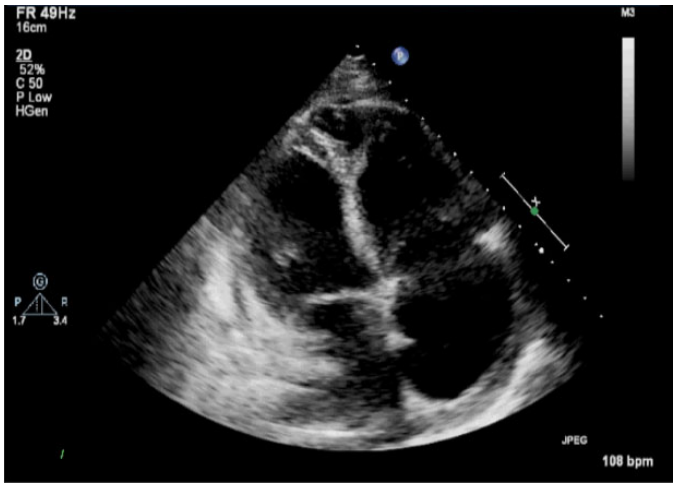

Figure 3: Post pericardiocentesis biventricular dilatation (Apical view).

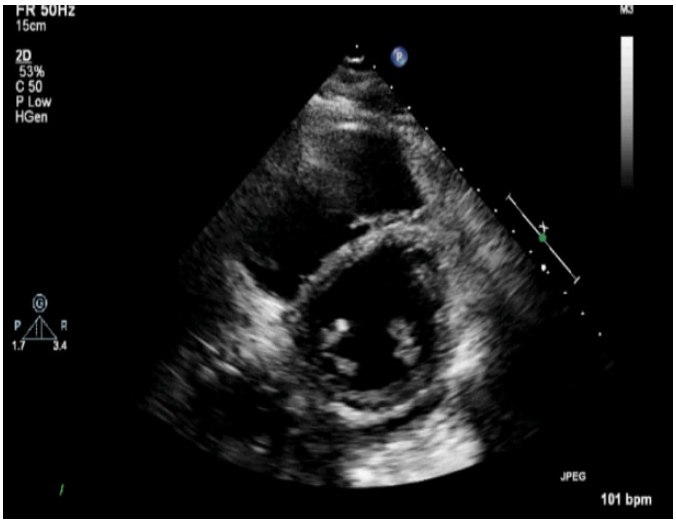

Figure 4: Post pericardiocentesis biventricular dilatation (Parasternal short axis view).

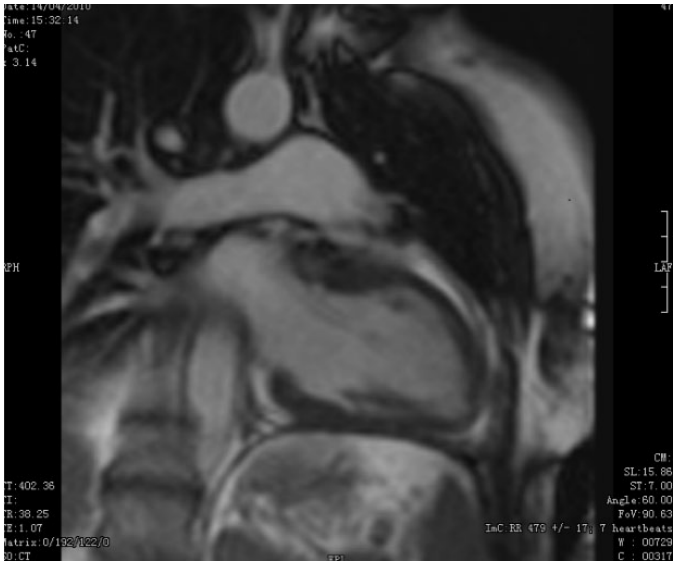

Figure 5: Cardiac MR showing dilated left ventricle.

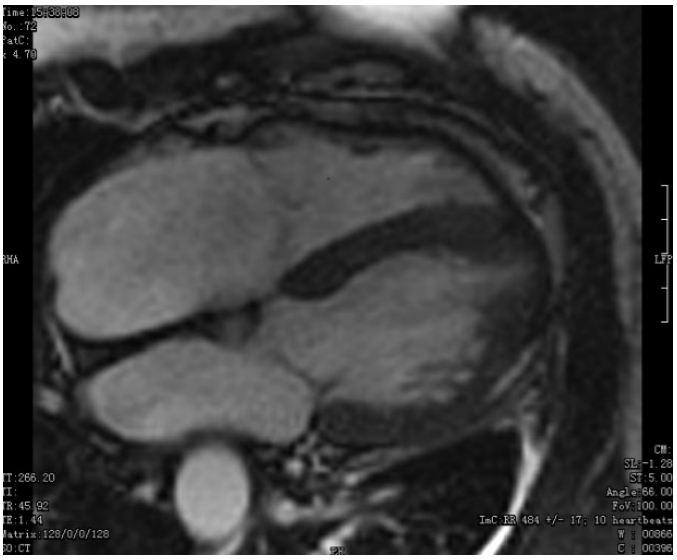

Figure 6: Cardiac MR showing mid ventricular hypokinesia with apical sparing.

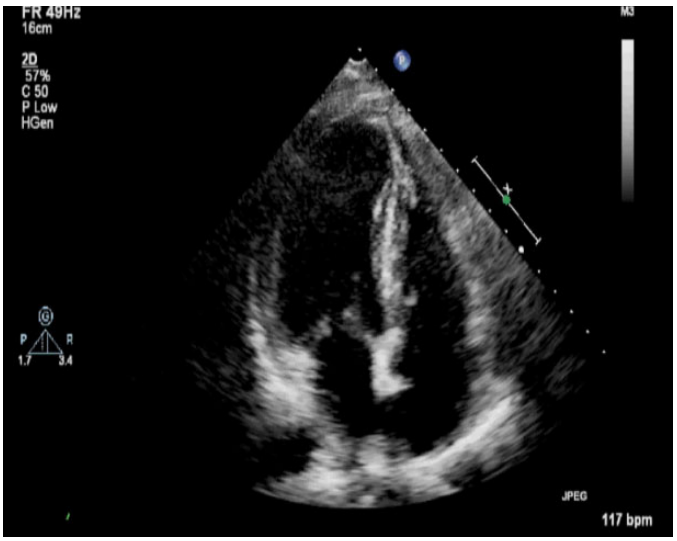

Figure 7: Normalization of biventricular systolic function (Apical view).

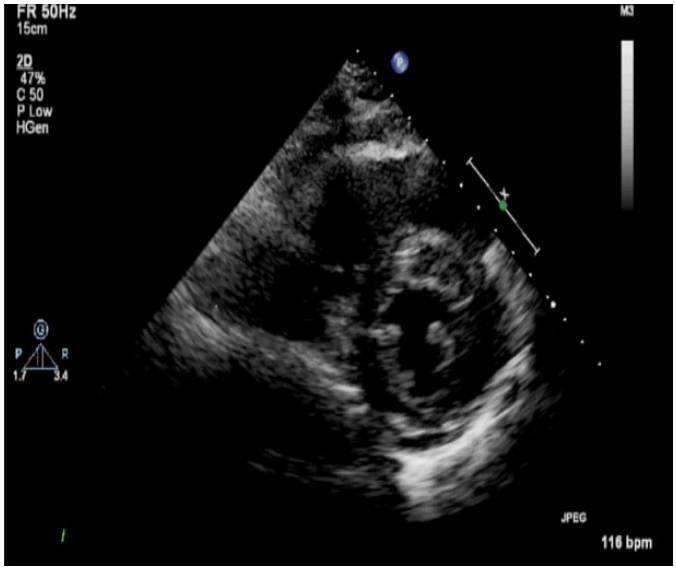

Figure 8: Normalization of biventricular systolic function (Parasternal short axis view).

DISCUSSION

Echocardiographically guided pericardiocentesis is generally safe with low incidence of major complications [3, 4]. Transient post pericardiocentesis heart failure or post pericardiocentesis "low cardiac output syndrome" in patients with no previous history of cardiac decompensation is a rare phenomenon [5]. This as yet poorly explained phenomenon can present as right ventricular (RV) and/or left ventricular (LV) failure. Persistent low blood pressure after pericardial decompression requiring presser support has also been described with normal biventricular systolic function [6].

Anguera et al. [7] have described a case of acute right ventricular dilatation after emergent pericardiocentesis in a 68-year-old woman with cardiac tamponade with resolution after 10 days of supportive care. Geffroy et al. [8] described a fatal case of prolonged RV failure along with persistent right to left shunt through a patent foramen ovale following emergent pericardiotomy for malignant cardiac tamponade in a 53-year-old patient.

Ischaki et al. [9] have described a case of 25-year-old man with tuberculous pericarditis. Post pericardiocentesis the patient required inotropic support and assisted ventilation for five days. Left ventricular ejection fraction (LVEF) was 25%. At discharge from intensive care the LVEF had improved to 45%. Similar cases of initial hemodynamic improvement post pericardial decompression followed by rapid deterioration have been described by others. All patients improved with supportive treatment [10–15]. Some common features are readily identifiable in all the aforementioned case reports. All had evidence of cardiac tamponade. All patients underwent emergent pericardial decompression either surgical or catheter based. All patients had complete evacuation of their pericardial sacs.

An interesting case of severe apical and septal hypokinesia with apical thrombus formation despite stepwise pericardiocentesis (500 ml each time) was described by Sevimli et al. [16] in a 42-year-old female patient. Left ventricular function improved over 10 days with anti-heart failure treatment. Rapid removal of large volume third spaced fluid is well known to cause hemodynamic derangement in the setting of thoracentesis [17, 18] and paracentesis. Interestingly the so called persistent low cardiac output syndrome (PLCOS) is more common after surgical pericardiotomy. Surgical decompression of massive pericardial effusion results in more rapid and complete evacuation of pericardial cavity. Thus, Dosios et al. [5] in their series showed that out of 104 patients who underwent surgical subxiphoid drainage of pericardial cavity, five patients (4.8%) developed PLCOS, four of whom died.

Wagner et al. [6] in their retrospective review of 174 cancer patients with pericardial effusion who underwent surgical drainage, reported an 11% incidence of paradoxical hemodynamic instability (PHI) (defined as unexpected vasopressor dependent hypotension in the immediate postoperative period requiring admission to the intensive care unit). The presence of cardiac tamponade and higher volume of pericardial fluid drained were two factors strongly associated with development of PHI. Interestingly majority of patients who developed PHI had normal biventricular function on echocardiograms performed during period of hemodynamic instability.

The mechanism/s of this potentially fatal complication of pericardiocentesis is/are unknown. However, many investigators have made some interesting speculations.

A similar syndrome of low cardiac output and cardiac dilatation has been described in 28% of patients who undergo pericardiectomy for constrictive pericarditis [13]. Downy et al. [19] have compared a rapid decompression of pericardial sac to functional pericardiectomy. Pericardial sac provides an external constraint that prevents over-distension of the cardiac chambers. A gradually accumulating pericardial fluid would compress the heart and expand the pericardial capacity. The resulting low cardiac output would trigger compensatory mechanisms including tachycardia and expansion of the intravascular volume. After rapid removal of pericardial fluid, increased venous return at high filling pressures could lead to rapid increase in wall stress leading to dilatation of the thin walled and more compliant right ventricle. Moreover, rapid decompression of the pericardial sac would allow insufficient time for the pericardial sac to contract and therefore permit overexpansion of the right ventricle resulting in right ventricular systolic dysfunction.

While the above mentioned sequence of events might explain right ventricular dilatation, the pathogenesis of acute pulmonary edema, needs further elucidation. Manyari et al. [20] using ECG-gated blood pool cardiac scintigraphy have shown that after pericardiocentesis there is greater increase in right ventricular stroke volume which is not matched by a comparable increase in left ventricular stroke volume. Such a disproportionate increase in RV output could result in flooding of the pulmonary vasculature [18].

Acute transient dilatation of a previously normal ventricle as a result of increased venous return is difficult to explain in the case of left ventricle. Moreover, such a mechanism does not explain the occurrence of cardiac failure in patients who undergo gradual or staged decompression of their pericardial effusions [15]. Researchers have therefore looked elsewhere to explain this phenomenon.

Skalidis et al. [21] in their pioneering study analysed the effects of increasing pericardial pressure on blood flow in a non-diseased left anterior descending artery of a man who underwent pericardiocentesis for malignant pericardial effusion. There was a marked progressive decline in coronary blood flow with increase in pericardial pressure. Reduction in coronary blood flow coupled with a short diastolic period of the coronary cycle during cardiac tamponade may cause myocardial stunning leading to heart failure. It has been suggested [4] that myocardial systolic dysfunction may already be present during cardiac tamponade but may be masked due to small chamber sizes by virtue of external compression. Indeed Braverman and Sundaresan [22] have described a patient whose left ventricular systolic function was grossly impaired at the time of hemodynamically significant pericardial effusion. Left ventricular function normalised after pericardiocentesis!

Cardiac MRI in our patient revealed mid-ventricular hypokinesia along with apical sparing. This pattern of wall motion abnormalities has been reported in patients with pheochromocytoma [23, 24] and has been called “inverted Takotsubo cardiomyopathy” [25]. It is widely believed that stress induced catecholamine surge leads to myocardial toxicity of Takotsubo cardiomyopathy [26]. It is possible that the intense adrenergic response leading to tachycardia and severe hypertension prior to pericardial drainage in our patient may have caused an inverted Takotsubo cardiomyopathy. A strikingly similar pattern of hypokinesia with apical sparing occurring after emergent pericardiocentesis has been reported by Bernal et al. [14].

CONCLUSION

In conclusion, the exact pathophysiology of post pericardiocentesis low cardiac output syndrome remains elusive. All components and variable patterns of this enigmatic syndrome may not be explainable based on a single unifying aetiology. We recommend that with the first evidence of cardiac tamponade prompt pericardiocentesis is carried out so that the deleterious consequences of cardiac tamponade such as coronary arterial compromise or the consequences of adverse compensatory responses to low cardiac output are aborted. While being aware of the possibility of hypotension and heart failure even

with slow decompression, we believe that enough circumstantial evidence exists to recommend partial initial decompression of the pericardial sac followed by gradual removal of the pericardial fluid with the help of an indwelling intrapericardial catheter. This would allow resolution of tamponade physiology and prevent abrupt fluctuations in venous return otherwise associated with more rapid decompression of the pericardial sac. If however, heart failure or circulatory shock does develop despite the above mentioned measures, these patients should be aggressively treated in the intensive care unit because despite their initial apparently dismal prognosis, such patients are fully curable with no long term sequelae.

REFERENCES

1. Ristić AD, Imazio M, Adler Y, et al. Triage strategy for urgent management of cardiac tamponade: A position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J 2014;35(34):2279–84.
2. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. Eur Heart J 2001;22(18):1642–81.
3. Tsang TS, Enriquez-Sarana M, Freeman WK, et al. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: Clinical profile, practice patterns, and outcomes spanning 21 years. Mayo Clin Proc 2002;77(5):429–36.
4. Gibbs CR, Watson RD, Singh SP, Lip GY. Management of pericardial effusion by drainage: A survey of 10 years' experience in a city centre general hospital serving a multiracial population. Postgrad Med J 2000;76(902):809–13.
5. Dosios T, Steanidis A, Chatziantoniou C, Sgouropoulou S. Thorough clinical investigation of low cardiac output syndrome after subxiphoid pericardiostomy. Angiology 2007;58(4):483–6.
6. Wagner PL, McAleer E, Stillwell E, et al. Pericardial effusions in the cancer population: Prognostic factors after pericardial window and the impact of paradoxical hemodynamic instability. J Thorac Cardiovasc Surg 2011;141(1):34–8.
7. Anguera I, Paré C, Perez-Villa F. Severe right ventricular dysfunction following pericardiocentesis for cardiac tamponade. Int J Cardiol 1997;59(2):212–4.
8. Geffroy A, Beloeil H, Bouvier E, Chaumeil A, Albaladejo P, Marty J. Prolonged right ventricular failure after relief of cardiac tamponade. Can J Anasth 2004;51:(5):482–5.
9. Ishcki E, Vasileiadis I, Koroneos A, et al. Acute heart failure following decompression of tuberculosis induced pericardial tamponade. Respiratory Medicine CME 2008;1(2):87–9.
10. Wolfe MW, Edelman ER. Transient systolic dysfunction after relief of cardiac tamponade. Ann Intern Med 1993;119(1):42–4.
11. Ligero C, Leta R, Bayes-Genis A. Transient biventricular dysfunction following pericardiocentesis. Eur J Heart Fail 2006;8(1):102–4.
12. Sunday R, Robinson LA, Bosek V. Low cardiac output complicating pericardiectomy for pericardial tamponade. Ann Thorac Surg 1999;67(1):228–31.
13. Shenoy MM, Dhar S, Gittin R, Sinha AK, Sabado M. Pulmonary edema following pericardiotomy for cardiac tamponade. Chest 1984;86(4):647–8.
14. Bernal JM, Pradhan J, Li T, Tchokonte R, Afonso L. Acute pulmonary edema following pericardiocentesis for cardiac tamponade. Can J Cardiol 2007;23(14):1155–6.
15. Chamoun A, Cenz R, Mager A, et al. Acute left ventricular failure after large volume pericardiocentesis. Clin Cardiol 2003:26(12):588–90.
16. Sevimli S, Arslan S, Gündoğdu F, Senocak H. Development of left ventricular apical akinesis and thrombus during pericardiocentesis for pericardial tamponade. Turk Kardiyol Dern Ars 2008;36(5):388–41.
17. Feller-Kopman D, Berkowitz D, Boiselle P, Ernst A. Large-volume thoracentesis and the risk of reexpansion pulmonary edema. Ann Thorac Surg 2007;84(5):1656–61.
18. Echevarria C, Twomey D, Dunning J, Chanda B. Does re-expansion pulmonary edema exist? Interact Cardiovasc Thorac Surg 2008;7(3):485–9.
19. Downey RJ, Bessler M, Weissman C. Acute pulmonary edema following pericardiocentesis for chronic cardiac tamponade secondary to trauma. Crit Care Med 1991;19(10):1323–5.
20. Manyari DE, Kostuk WJ, Purves P. Effect of pericardiocentesis on right and left ventricular function and volumes in pericardial effusion. Am J Cardiol 1983;52(1):159–62.
21. Skalidis EI, Kochiadakis GE, Chrysostomakis SI, Igoumenidis NE, Manios EG, Vardas PE. Effect of pericardial pressure on human coronary circulation. Chest 2000;117(3):910–2.
22. Braverman AC, Sundaresan S. Cardiac tamponade and severe ventricular dysfunction. Ann Intern Med 1994;120(5):442.
23. Kim S, Yu A, Fillippone LA, Kolansky DM, Raina A. Inverted-Takotsubo pattern cardiomyopathy secondary to pheochromocytoma: A clinical case and literature review. Clin Cardiol 2010;33(4):200–5.
24. Di Valentino M, Balestra GM, Christ M, Raineri I, Oertli D, Zellweger MJ. Inverted Takotsubo cardiomyopathy due to pheochromocytoma. Eur Heart J 2008;29(6):830.
25. Van de Walle SO, Gevaert SA, Gheeraert PJ, De Pauw M, Gillebert TC. Transient stress-induced cardiomyopathy with an "inverted takotsubo" contractile pattern. Mayo Clin Proc 2006;81(11):1499–502.
26. Bybee KA, Prasad A. Stress-related cardiomyopathy syndromes. Circulation 2008;118(4):397–409.

****

Author Contributions

M Imran Khan – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of

the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Wissam Al Sahali – Conception of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Peter Quigley – Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Charles McCreery – Design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Jonathan Dodd – Acquisition of data, Analysis of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Rory O'Hanlon – Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission
The corresponding author is the guarantor of submission.

Source of Support
None.

Consent Statement
Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.