Journal of Medical Imaging & Case Reports

https://doi.org/10.17756/micr.2020-054

Case Report

Open Access

Isolated Left Ventricular Tamponade: A Rare Cause of Cardiogenic Shock

Lance R. Ng*, Vijaya Pera, Geoffrey McCracken, Cherian Sebastian

Waikato District Health Board, Hamilton, New Zealand

***Correspondence to:**
Dr. Lance R. Ng, MBChB
Waikato District Health Board
Hamilton, New Zealand
Tel: +6421 045 0875
Fax: +647 839 8639
E-mail: lance.rab.ng@gmail.com

Received: July 16, 2020
Accepted: September 08, 2020
Published: September 08, 2020

Citation: Ng LR, Pera V, McCracken G, Sebastian C. 2020. Isolated Left Ventricular Tamponade: A Rare Cause of Cardiogenic Shock. *J Med Imaging Case Rep* 4(2): 63-66.

Published by United Scientific Group

Abstract

We describe a patient nine months post-cardiac surgery with isolated left ventricular (LV) tamponade who presented with sub-acute decompensated heart failure and subsequently developed cardiogenic shock. The pathophysiology, diagnosis and management of isolated LV tamponade are reviewed.

Keywords

Left ventricular tamponade, Regional tamponade, Cardiogenic shock, Bentall's procedure, Pericardiocentesis

List of Abbreviations

LV: Left Ventricle; RV: Right Ventricle; HR: Heart Rate; BP: Blood Pressure; JVP: Jugular Venous Pressure; LVEF: Left Ventricular Ejection Fraction; RVSP: Right Ventricular Systolic Pressure; TR: Tricuspid Regurgitation; CHF: Congestive Heart Failure; ECG: Electrocardiogram; CXR: Chest X-ray; INR: International Normalized Ratio; ALT: Alanine Transaminase; LVIDd: Left Ventricular Internal Diameter at End-Diastole; ICU: Intensive Care Unit; ESC: European Society of Cardiology

Case Report

A 73-year-old Maori gentleman presented to the emergency department after having a third episode of syncope within a week. This was preceded by three weeks of breathlessness and chest pain. At presentation, he was normotensive but cool peripherally, with a jugular venous pressure (JVP) which was elevated to the angle of the mandible, bibasal crepitations on chest auscultation and pitting oedema to the knees.

He had undergone a Bentall procedure with single-vessel coronary artery bypass graft nine months earlier. One month prior to presentation, a routine echocardiogram showed a left ventricular ejection fraction (LVEF) of 45%, moderate RV dysfunction (RV systolic pressure (RVSP) 33.7 mmHg) and moderate tricuspid regurgitation (TR) with a thin rim of pericardial effusion. His other past medical history included hypertension and dyslipidaemia.

Admission electrocardiogram (ECG) demonstrated atrial flutter with ventricular response of 75 beats per minute and long-standing anterolateral T-wave inversions (Figure 1). Chest x-ray (CXR) demonstrated cardiomegaly with no alveolar infiltrates. There was minimal change in serial high-sensitivity troponin T over 6 hours. Serum creatinine was elevated at 2.21 mg/dL compared to his baseline creatinine of 1.13 mg/dL.

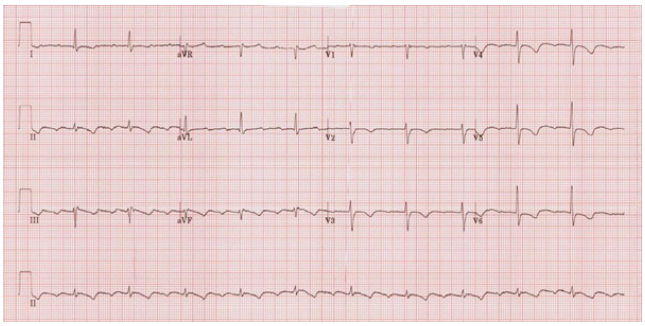

Figure 1: Admission ECG.

The patient was admitted under Internal Medicine with a diagnosis of congestive heart failure (CHF) and commenced diuresis with intravenous furosemide. Anticoagulation with dabigatran was continued.

On day 4, the patient was noted to be persistently breathless with oxygen saturations of 93% (on room air) and a further rise in creatinine to 3.08 mg/dL. Repeat CXR demonstrated pulmonary congestion (Figure 2) and intravenous furosemide was continued.

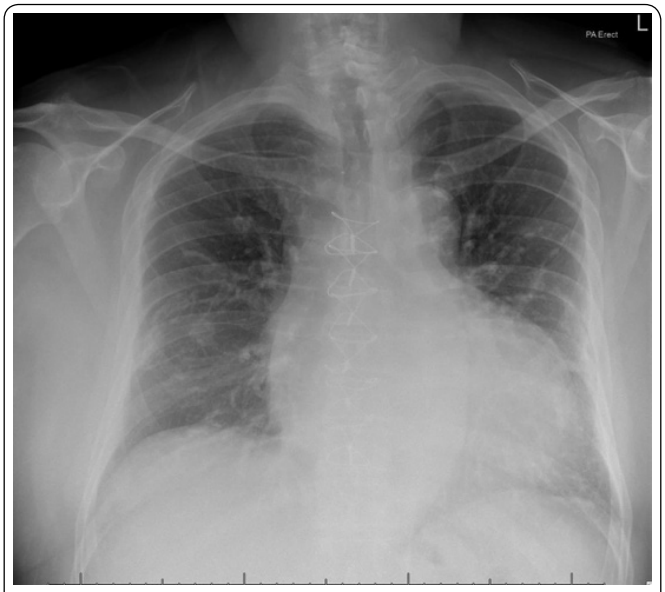

Figure 2: CXR (Day 4).

The next day, the patient developed hypotension (BP 87/54 mmHg) and hypoxia (oxygen saturations 95% on 2 L/min via nasal prongs) with a heart rate (HR) of 60 bpm (beta-blocked). There was now objective evidence of multiorgan failure (creatinine 5.09 mg/dL, international normalized ratio (INR) 5.0, alanine transaminase (ALT) 612 U/L, serum lactate 4.6 mmol/L). An urgent after-hours cardiology review was requested.

An echocardiogram showed a 3.7 cm pericardial effusion localized posteriorly, around the LV lateral wall. There was no effusion seen around the RV and there was no evidence of RV collapse. A very small LV cavity was demonstrated, compressed between the dilated RV and the loculated pericardial effusion (LV internal diameter at diastole (LVIDd) 3.4 cm). The LVEF was 60% with stroke volume of 56.6 ml (Figures 3-5). RV systolic function was severely reduced. Free-flowing TR was noted. Right atrial pressure was > 15 mmHg. An RVSP value was not obtained due to severe TR and RV dysfunction.

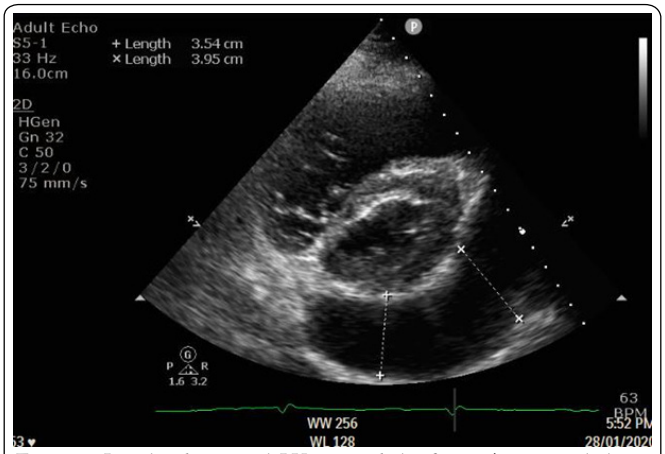

Figure 3: Loculated, regional LV pericardial effusion (parasternal short-axis view).

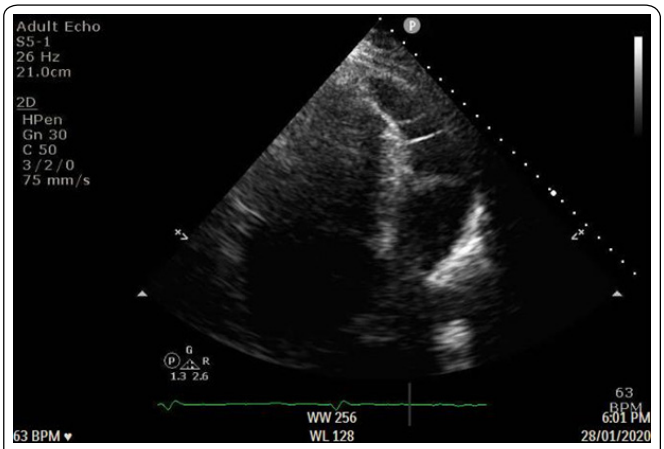

Figure 4: Evidence of septations and loculation (apical four-chamber view).

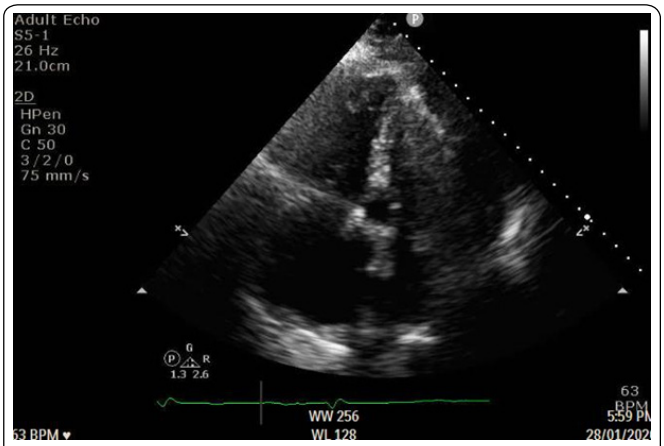

Figure 5: Tamponade of the LV and a severely dilated RV (apical four-chamber view).

The case was discussed with the interventional cardiologist who identified that the location of the effusion was not easily accessible percutaneously and the risk of bleeding was prohibitive given the concurrent high INR and dabigatran therapy. The on-call cardiothoracic surgeon was then consulted. The surgeon felt that it was unclear whether

the pericardial effusion was causing tamponade given the lack of classical RV collapse and the subacute presentation. There was also concern about the significant perioperative risk given the severe RV dysfunction in addition to the aforementioned bleeding risk. Therefore, the surgical team decided that surgery could be deferred until the next day. A CT scan of the chest was requested in the interim to better delineate the pericardial effusion and help guide an intervention.

The patient was moved to the coronary care unit and commenced on inotropic support with intravenous dopamine but had a cardiac arrest (pulseless electrical activity) three hours after this. Return of spontaneous circulation was initially achieved after 9 minutes of cardiopulmonary resuscitation.

An emergency bedside pericardiocentesis under echocardiographic guidance was undertaken by the on-call intensivist and 100 ml of blood-stained fluid was aspirated. The patient's BP markedly improved to 120/60 mmHg post-pericardiocentesis and the patient was transferred to the intensive care unit (ICU), intubated and ventilated. An urgent CT of the chest with contrast was performed demonstrating a hyperdense pericardial effusion pooled posterior to the LV with a maximal depth of 3 cm. There was no obvious arterial extravasation of contrast to suggest active bleeding. It was also noted there was marked RV dilatation and reflux of contrast into the inferior vena cava, consistent with RV strain.

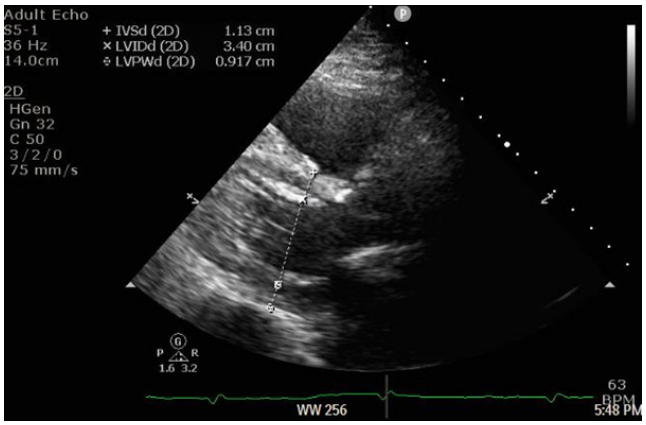

Figure 6: Pre-drainage echocardiogram with reduced LVIDd (parasternal long-axis view).

An emergency CT-guided pericardial drain insertion was then undertaken by the interventional radiologist. A follow-up echocardiogram showed improvement in LV function and size (LVIDd 4.6 cm) (Figures 7 and 8) and no residual pericardial effusion (Figure 9).

The patient remained in ICU thereafter and was unable to be weaned off ventilator and inotropic support and eventually passed away on day 14 of his admission.

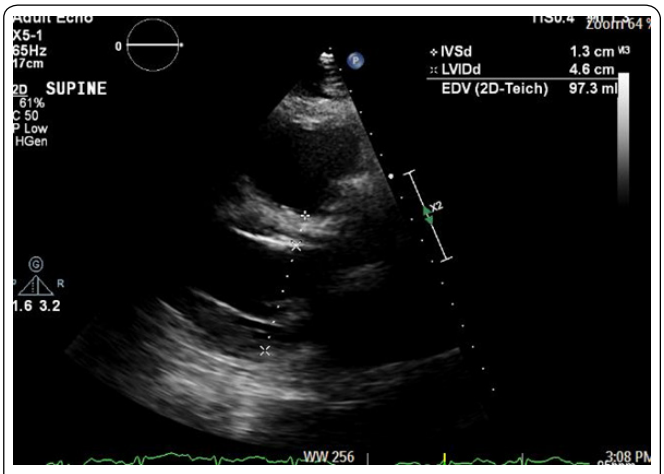

Figure 7: Post-drainage echocardiogram with improved LVIDd (parasternal long-axis view).

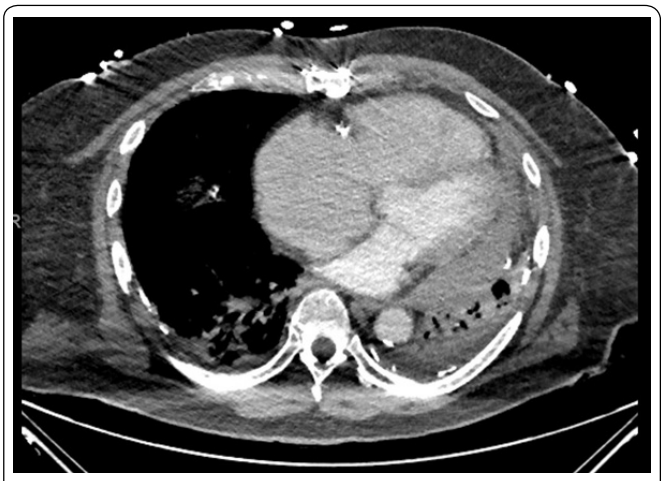

Figure 8: CT chest demonstrating the loculated, regional LV pericardial effusion.

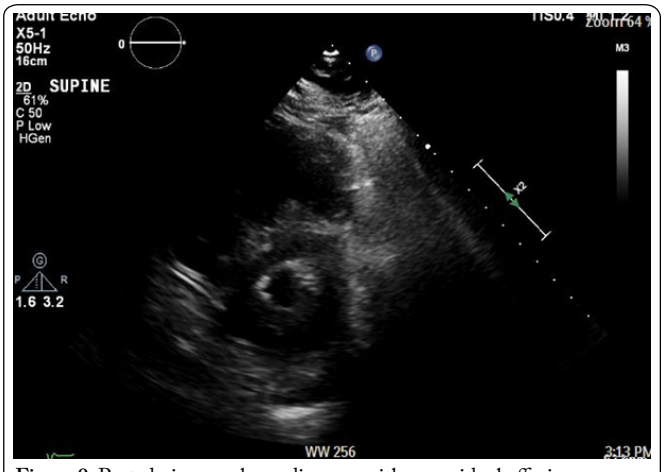

Figure 9: Post-drainage echocardiogram with no residual effusion.

Discussion

Cardiac tamponade is characterized by the accumulation of pericardial fluid under pressure. The presentation can be acute or subacute, depending on the time taken for the pericardial fluid to accumulate. As a result, the pathognomic triad of hypotension, muffled heart sounds and elevated JVP (Beck's Triad) may not be apparent in those with a more subacute presentation. As in our case, the patient may be asymptomatic for a significant period of time but then develop manifestations (dyspnoea, peripheral oedema, chest pain) once intrapericardial pressure reaches a critical value [1].

In classic cardiac tamponade, all cardiac chambers are compressed by the increased pericardial pressure. The intrapericardial volume then becomes fixed, venous return becomes compromised and eventually, the intrapericardial pressure exceeds the pressure of an intracardiac chamber (typically lower-pressure right-sided chambers) thus causing the classical "RV collapse" seen on echocardiography [2]. Additionally, an ECG may demonstrate low QRS voltages and electrical alternans (a specific, but low sensitivity ECG finding in cardiac tamponade) [3].

Conversely, isolated LV tamponade is a relatively uncommon phenomenon given its thick, muscular wall and is typically seen in the post-cardiac surgery setting [4]. In our case, there was evidence of a persistent pericardial effusion prior to presentation which, in addition to post-operative anticoagulation, is a recognized risk factors for subacute cardiac tamponade [5, 6]. Chuttani et al. assessed 18 echocardiograms of patients with post-operative cardiac tamponade and demonstrated that while only 4 out of 18 had evidence of RA or RV collapse, all 18 had LV diastolic collapse. Additionally, they found that 15 out of 18 effusions were loculated – which was also the case with our patient [7].

With this in mind, our patient's atypical presentation – devoid of the aforementioned conventional findings associated with classic tamponade – and the subsequent delays in diagnosis and timely intervention, are entirely conceivable.

Furthermore, the option of a conventional percutaneous pericardial drain was thought to be associated with prohibitive risk because of the loculated, postero-lateral location of the pericardial fluid and the concurrent coagulopathy. The European Society of Cardiology (ESC) guidelines on the diagnosis and management of pericardial diseases suggest that in such cases, a surgical approach may be preferable [2].

Conclusion

Although uncommon, clinicians should have a high index of suspicion for isolated LV tamponade in the post-cardiac surgery setting given its insidious and non-specific presentation. Early multi-specialty assessment is required to establish and provide the lowest-risk therapeutic intervention for the patient.

Conflict of Interest

Authors declare no conflict of interest.

References

1. Reddy PS, Curtiss EI, O'Toole JD, Shaver JA. 1978. Cardiac tamponade: hemodynamic observations in man. *Circulation* 58(2): 265-272. https://doi.org/10.1161/01.CIR.58.2.265
2. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, et al. 2015. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and management of pericardial diseases of the European Society of Cardiology (ESC) endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 36(42): 2921-2964. https://doi.org/10.1093/eurheartj/ehv318
3. Spodick DH. 2003. Acute cardiac tamponade. *N Engl J Med* 349(7): 684-690. https://doi.org/10.1056/nejmra022643
4. Fusman B, Schwinger ME, Charney R, Ausubel K, Cohen MV. 1991. Isolated collapse of left-sided heart chambers in cardiac tamponade: demonstration by two-dimensional echocardiography. *Am Heart J* 121(2 Pt 1): 613-616. https://doi.org/10.1016/0002-8703(91)90741-y
5. You SC, Shim CY, Hong GR, Kim D, Cho IJ, et al. 2016. Incidence, predictors, and clinical outcomes of postoperative cardiac tamponade in patients undergoing heart valve surgery. *PLoS One* 11(11): e0165754. https://doi.org/10.1371/journal.pone.0165754
6. Leiva EH, Carreño M, Bucheli FR, Bonfanti AC, Umaña JP, et al. 2018. Factors associated with delayed cardiac tamponade after cardiac surgery. *Ann Card Anaesth* 21(2): 158-166. https://doi.org/10.4103/aca.aca_147_17
7. Chuttani K, Pandian NG, Mohanty PK, Rosenfield K, Schwartz SL, et al. 1991. Left ventricular diastolic collapse. An echocardiographic sign of regional cardiac tamponade. *Circulation* 83(6): 1999-2006. https://doi.org/10.1161/01.CIR.83.6.1999