Case Report

Transient acute left ventricular dysfunction post-pericardiocentesis for cardiac tamponade

Chong Yu Edwin Sng (MD)*, Choong Hou Koh (MD), Aileen Mae Lamarda (MD), Swee Yaw Tan (MD)
National Heart Centre, Singapore

A R T I C L E   I N F O

Article history:
Received 16 February 2015
Received in revised form 24 May 2015
Accepted 15 June 2015

Keywords:
Pericardiocentesis
Left ventricular dysfunction
Left ventricular failure
Pulmonary edema
Cardiac tamponade

A B S T R A C T

A rare but serious complication of pericardiocentesis is the development of transient left ventricular dysfunction. In this report, we present a case of a 65-year-old male patient with cardiac tamponade who suffered from acute left ventricular heart failure post-pericardiocentesis.

Learning objective: Acute left ventricular dysfunction is a rare but serious complication of pericardiocentesis. However, there is lack of existing guidelines on pericardial fluid drainage and monitoring post-pericardiocentesis. To minimize the risk of the development of acute left ventricular dysfunction, the authors propose that every pericardiocentesis should be guided by predetermined parameters, e.g. rate of drainage, while taking into account each patient’s physical profile.

© 2015 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Introduction

Acute left ventricular heart failure is an uncommon but serious complication of pericardiocentesis. However, its exact mechanism remains unknown. In this report, we present a case of a 65-year-old male patient with hemopericardium and cardiac tamponade who developed acute left ventricular heart failure after pericardiocentesis.

Case report

A 65-year-old male patient was admitted to our institution with a 1-month history of intermittent lower chest discomfort and dyspnea that was worse on exertion. His past medical history was unremarkable except for mild gastritis, diverticulosis, and a gradual loss of weight of 4 kg over the past 2 months. He was a chronic smoker of 50 pack years but did not have diabetes, hypertension, or dyslipidemia.

On physical examination, blood pressure, heart rate, and respiratory rate were within normal ranges. Cardiovascular examination was unremarkable. Significantly, the patient did not have muffled heart sounds or raised jugular venous pulsations. Electrocardiogram showed sinus rhythm with low voltage QRS complexes. Chest X-ray (Fig. 1A) revealed an enlarged cardiac silhouette with mild pulmonary venous congestion. Routine blood tests were largely unremarkable except serum sodium of 128 mmol/L and hemoglobin of 11.5 g/dl. Serial cardiac enzymes were within normal ranges.

Transthoracic echocardiogram (Fig. 1B) demonstrated a large circumferential pericardial effusion with right atrial diastolic collapse. The echocardiographic measurements of the pericardial effusion were posterior to left ventricle = 2.1 cm; adjacent to left ventricle apex = 2.8 cm; anterior to right ventricle = 2.8 cm; adjacent to right ventricle = 1.7 cm. The inferior vena cava was plethoric with diminished respiratory variation. Left ventricular systolic function was preserved, and there was no observable regional wall motion abnormality. The ejection fraction was estimated at 60–65%. Uncomplicated therapeutic pericardiocentesis under fluoroscopic guidance was performed and 700 ml of hemopericardium was drained. A pericardial pigtail catheter was left to drain the remaining pericardial fluid.

Immediately post-pericardiocentesis, blood pressure was 136/82 mmHg, compared to 120/60 mmHg pre-procedure. Eight hours post-pericardiocentesis, the patient developed hypotension with a blood pressure of 80/60 mmHg, pulse rate of 85 beats per minute, respiratory rate of 21 breaths per minute and SpO2 100% on 2 L/min of intranasal oxygen. He was resuscitated with intravenous crystalloids and started on intravenous dopamine infusion. Electrocardiogram (Fig. 2C) that was repeated showed sinus rhythm with new-onset hyperacute T waves in the anterolateral leads, compared to baseline (Fig. 1C). Serum cardiac...
enzymes were performed and showed a creatine kinase-MB level of 40.1 μg/L (14.0 μg/L pre-procedure) and a troponin T level of 0.48 U/L (0.01 U/L pre-procedure). Repeat renal panel did not show any renal impairment. Liver panel and brain natriuretic peptide were not repeated. Repeat chest X-ray (Fig. 2A) showed airspace shadowing in bilateral lung bases.

Transthoracic echocardiogram was repeated at 15 h (Fig. 2B) after the first echocardiogram showed reduced left ventricular systolic function with an ejection fraction estimated at 35–40% and new-onset akinesia of the mid to apical anterior, apical lateral, and midanteroseptal regions. There was minimal residual pericardial effusion and no evidence of myocardial perforation. Cardiogenic shock secondary to acute myocardial infarction was considered. Emergent coronary angiography performed on the same day showed normal coronary arteries and dyskinesia of the anterolateral wall on ventriculogram.

Just 4 days after the acute left ventricular dysfunction, a repeat transthoracic echocardiogram (Fig. 3A) showed normalization of left ventricular systolic function to 61% with no regional wall motion abnormality and a small residual circumferential pericardial effusion. Repeat electrocardiogram (Fig. 3B) at this point showed persistent inverted T waves in the anterolateral leads. Cytology and immunohistochemistry staining of the pericardial fluid were consistent with that of a metastatic adenocarcinoma, suggestive of an upper gastrointestinal or pancreatobiliary primary.

Discussion

Case series demonstrated that malignancy accounted for between 13% and 23% of etiologies of pericardial effusion [1]. Cardiac tamponade occurred in approximately 60% of those with
malignant pericarditis, tuberculous or purulent pericarditis [2].

Pericardiocentesis is a life-saving intervention in patients with cardiac tamponade, and provides a mean to alleviate the symptoms by relieving the elevated intrapericardial pressure and improving hemodynamic status. At the same time, analysis of the pericardial fluid gives clues to the underlying etiology. Establishment of a definitive diagnosis guides subsequent management.

Infrequently, complications may arise from pericardiocentesis. Large observational studies of real time image-guided (typically echocardiographic guidance) pericardiocentesis report a major complication rate of less than 2% [3,4]. The most serious mechanical complications of pericardiocentesis are myocardial puncture or laceration, vascular injury (coronary, intercostal, internal mammary, or intraabdominal), pneumothorax, air embolism, and arrhythmia. Rarely, the needle may enter the peritoneal cavity, injuring intraabdominal organs such as the liver or causing visceral perforation. Vasovagal response to pericardial decompression occurs in up to 25% of patients and may be hemodynamically significant [5]. Successful pericardial decompression may also induce hemodynamic derangements such as acute left ventricular dysfunction, pulmonary edema, and cardiogenic shock that are unrelated to anatomic injury [6–9]. Finally, pericardiocentesis may fail to relieve the tamponade, especially when the pericardial effusion is loculated or rapidly re-accumulating.

Acute left ventricular dysfunction is an uncommon complication of pericardiocentesis and its exact pathophysiology is still being speculated. It is hypothesized that the development of acute left ventricular dysfunction is a result of interventricular volume mismatch after sudden depression of the cardiac tamponade [6]. Extrinsic compression of the right heart by the pericardial effusion results in a decrease in right ventricular filling and consequently, stroke volume. The cardiovascular system compensates for this drop in cardiac output by increasing the basal heart rate or increasing the preload through neurohumoral mechanisms. When large volume pericardiocentesis is performed over a relatively short time, the cardiac tamponade, which has resulted in an

---

**Fig. 2.**

(A) Post-pericardiocentesis chest X-ray showing bibasal airspace shadowing. (B) Left – parasternal long-axis view. Thinned out septum during systole; right – short-axis view at the mid ventricle. Akinetic anteroseptum. (C) Post-pericardiocentesis electrocardiogram. Sinus rhythm with hyperacute T waves.
interventricular tension relationship (i.e. the right ventricle having a higher pressure/volume milieu compared to the left ventricle), is released. An acute imbalance between the right ventricular end-diastolic volume and the left ventricular end-diastolic volume ensues. The right ventricular end-diastolic volume now overloads the left ventricle, resulting in increased left ventricular wall stress and consequently, increased left ventricular end-diastolic pressure. Ultimately, acute left ventricle dysfunction develops.

A second proposed mechanism is myocardial stunning due to oxygen supply-demand mismatch across the left ventricular and right ventricular myocardium. An acute increase in myocardial wall stress due to the sudden increase in stretch of the cardiac chambers, secondary to the increase of venous return at high filling pressures, is combined with a relatively negative pressured environment in the pericardial cavity immediately after large volume evacuation of pericardial fluid. This abrupt increase in wall stress can result in significant cellular injury and tissue dysfunction.

A coronary angiogram was performed emergently to exclude any significant obstructive coronary artery disease and demonstrated normal coronary arteries. While variant angina or coronary vasospasm was a possible differential, a provocation test with acetylcholine was not performed during coronary angiogram as the clinical presentation of the patient was unlike that of variant angina, in which patients typically experience repeated episodes of transient chest pain accompanied by ST elevations that return to baseline when the symptoms resolve. In the case of our patient, there was no complaint of chest pain and the T wave inversions in the electrocardiogram persisted.

Takotsubo cardiomyopathy was considered as a possible mechanism for the acute left ventricular dysfunction. However, the clinical course did not follow that of Takotsubo cardiomyopathy, in which typical clinical and ejection fraction recovery takes weeks to months. In this case, the ejection fraction recovered in 4 days. In addition, hyperacute T waves and atypical echocardiogram findings were not suggestive of Takotsubo cardiomyopathy, where one would expect ST elevations and apical ballooning on echocardiogram. Hyperacute T waves are a manifestation of repolarization abnormalities which can be due to electrolyte imbalances (hyperkalemia) or myocardial injury. The hyperacute T waves demonstrated may represent transient myocardial strain or injury patterns secondary to the sympathetic surge and acute left ventricular dysfunction.

A key factor leading to acute left ventricular failure is the rate of pericardial fluid drainage. Hence, the authors propose that pericardial drainage be guided by pre-determined parameters to minimize the development of acute left ventricular dysfunction. In patients with chronic pericardial effusion complicated by cardiac tamponade, it is prudent to decompress the pericardium gradually until tamponade physiology resolves [6]. There are no definite guidelines in the literature either for the quantity of fluid that may be safely removed or for the rate of fluid drainage. In acute cardiac tamponade, intrapericardial pressure generally drops rapidly and hemodynamic improvement ensues after aspiration of the first 50–200 ml of fluid. Thereafter, the pericardial fluid should be left to drain passively at a controlled rate per day to prevent massive fluid shifts, which predispose to left ventricular dysfunction. The controlled rate of pericardial fluid drainage allows
adaptive changes in coronary flow, myocardial mechanics, and wall stress to occur. The European Society of Cardiology Guidelines on the Diagnosis and Management of Pericardial Diseases advise that the pericardial effusion be drained in steps of less than 1 L at a time to avoid acute right ventricular dilatation (“sudden decompression syndrome”) [10].

In addition, the authors postulate that the patient’s physical profile may also affect the risk of developing left ventricular dysfunction after pericardiocentesis, specifically, body surface area (BSA). It is conceivable that an individual with a larger BSA can tolerate a higher rate of drainage of pericardial effusion. As Asians generally have a smaller BSA compared to Caucasians, rate of drainage of pericardial effusion would conceivably be lower. A therapeutic drainage volume index, using the BSA as a denominator, may be useful to guide the amount of fluid drained. In the above-described case study, an initial amount of 700 ml of pericardial effusion was drained from the patient over 6–8 h. In retrospect, a gentler approach should have been undertaken, especially since the patient’s BSA was only 1.84 m². Onset of hemodynamic derangement secondary to left ventricular dysfunction in the case studies ranges from almost immediate post-pericardiocentesis to several hours post-procedure. Hence, the authors recommend that intensive monitoring of clinical parameters be extended in all post-pericardiocentesis patients to at least 24 h so that any signs of acute left ventricular dysfunction can be detected early and treatment instituted without delay.

Conclusion

Pericardiocentesis is a critical intervention in patients with cardiac tamponade. A rare complication from large volume pericardial drainage is the development of acute left ventricular dysfunction which may mimic mechanical or ischemic insults. To minimize such a complication, the authors propose that pericardial drainage be guided by pre-determined parameters and that close monitoring of patients be extended to at least 24 h.

Conflict of interest

The authors declare that there is no conflict of interest with any organization or person regarding the material discussed in this manuscript.

References