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Case Report
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Pulmonary Hypertension: A Predisposing Factor to Pericardial Decompression Syndrome

Kayla Taylor JD*[1], John Sherret DO[1], Vijay Ramu[2] and Manar Jbara[1]

[1]*ETSU Quillen College of Medicine Department of Cardiology*

[2]*ETSU division of cardiology*

***Corresponding author:** Kayla Taylor JD, FNP-BC, ETSU Quillen College of Medicine Department of Cardiology, USA.

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Abstract

In this case report, we describe a 70-year-old female patient who developed pericardial decompression syndrome (PDS) after drainage of pericardial effusion. We explore whether the patient's co-morbidities, specifically pulmonary hypertension, contributed to the development of PDS and ultimately the patient's demise.

Keywords: pericardial decompression syndrome, pulmonary hypertension, pericardial fluid, pericardial window

Introduction

Pericardial decompression syndrome (PDS), which was first described in 1983 by Vandyke, is a paradoxical hemodynamic instability [1]. PDS is an unfortunate outcome that may occur after drainage of pericardial fluid to relieve symptoms, such as hypotension, of a life-threatening cardiac tamponade. The goal of drainage is to allow the heart to expand and promote cardiac output; however, if PDS occurs, it is ironically characterized by a decrease in cardiac output that ultimately leads to hypotension and tachycardia, which are the same symptoms intended to be remedied with drainage. As cardiac output gets reduced, there is also a buildup of fluid resulting in pulmonary edema and subsequent dyspnea and orthopnea. The mechanisms behind PDS are not well understood, though some postulate that ischemic, hemodynamic, or autonomic causes may be to blame [2]. Risk factors for PDS include volume of fluid removed, rate of fluid removal, method of removal (pericardial window versus needle pericardiocentesis), female gender, malignant pleural fluid, and history of radiotherapy [3]. This case report explores whether another factor, such as pulmonary hypertension, should be considered a risk factor for the development of PDS.

Case Presentation

The patient was a 70-year-old female with past medical history of persistent atrial fibrillation on warfarin prophylaxis (held on admission due to supratherapeutic INR), hypertension, hyperlipidemia, pulmonary hypertension on 2L O2 via nasal cannula, obstructive sleep apnea (unable to tolerate CPAP), end-stage renal disease (ESRD) requiring hemodialysis three times per week, hypothyroidism, and diabetes mellitus type 2. The patient had a routine appointment at the cardiology clinic for echocardiogram and stress test. While at the clinic, the cardiologist noted a large pericardial effusion (>2cm) on echocardiogram (echo) and directed the patient to the emergency department (ED). In the ED, the patient admitted worsening dyspnea on exertion for the past several weeks as well as worsening orthopnea that required her to sleep sitting up at night. Bilateral lower extremity edema had also been worsening over the past several weeks. The patient had recently had her warfarin decreased from 9mg daily to 8mg daily because of a supratherapeutic INR.

In the ED, electrocardiogram (ECG) showed atrial fibrillation with rapid ventricular rate (rate 101), QTc 451, left axis deviation, and widened QRS. INR was 4.0 and PTT 67.3. Creatinine was 3.68 and BUN 31. Chest x-ray showed cardiomegaly and bibasilar atelectasis with small effusions. Inpatient STAT echo showed moderately reduced right ventricular systolic function, severe tricuspid regurgitation, severe pulmonary hypertension, large circumferential pericardial effusion with hemodynamic changes consistent with tamponade, normal LV systolic function, and an EF 55-60%.

The patient was admitted for cardiac tamponade secondary to uremia and supratherapeutic anticoagulation vs malignancy (due to history of colon polyps and family history of mother and father having colon cancer) vs hypothyroidism.

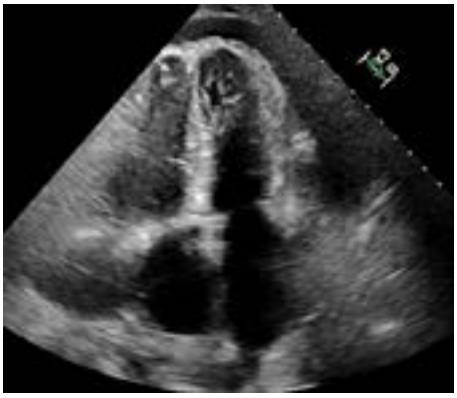
Due to the patient's supratherapeutic INR, cardiology requested a consult from cardiothoracic surgery to perform pericardial window. The patient was given vitamin K as well as 2 units

of fresh frozen plasma prior to the procedure the next day. The patient developed worsening hemodynamics with introduction of anesthesia which required urgent intervention. A total of 550 cc of serosanguinous fluid was removed. Pleural spaces did not have significant effusion at that time. A drain was placed. A central line and arterial line were also placed. The patient was started on vasopressors (norepinephrine and phenylephrine) post-operatively and transferred to the ICU. She was extubated later that night but remained on pressors due to continued hypotension. She was monitored on Vigileo. Amiodarone IV was started to assist with heart rate control given atrial fibrillation with rapid ventricular rate.

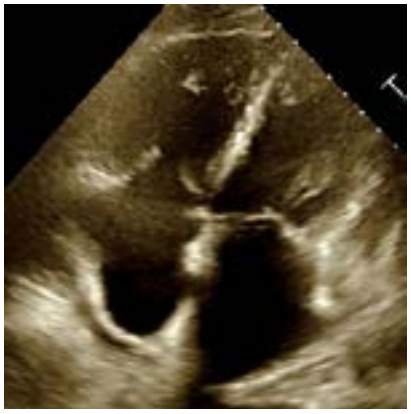
On post-op day 3, the patient's condition had somewhat improved, and vasopressors were weaned. She remained on amiodarone. The drain was discontinued on post-op day 3 with a total output of approximately 200cc. Pericardial fluid results were unremarkable. Post-operative echo showed EF 50-55%, decreased left ventricular cavity size, moderately dilated right atrium, and severely enlarged right ventricle with moderately to severely reduced right ventricular systolic function. Hemodialysis was resumed.

After removal of 2L of ultrafiltrate, the patient developed hypotension with systolic BP in the 70s-80s. Norepinephrine was restarted. Cardiology performed right heart catheterization and found wedge pressure 27mmHg, pulmonary artery pressure 63/47 mmHg (mean 55mmHg), right ventricle pressure 66/21 mmHg, right atrium pressure 29 mmHg, Cardiac Output (Fick) 6.5L/min, and Cardiac Index (Fick) 3.2L/min/m^2. Thus, the right heart catheterization showed evidence of pre- and post-capillary pulmonary hypertension. Cardiology recommended milrinone, a phosphodiesterase 3 inhibitor, to decrease pulmonary vascular resistance. Unfortunately, the patient's hypotension worsened, and this medication had to be stopped. During her time on milrinone, norepinephrine also needed to be increased from 4mcg to 20mcg. Inhaled epoprostenol was also attempted for approximately eight hours in an attempt to improve pulmonary hypertension but this was also unsuccessful and discontinued.

Another echo was performed showing EF 40-45% (which was newly mild to moderately decreased), mildly reduced right ventricular systolic function, severely enlarged right ventricle, small circumferential pericardial effusion, and large pleural effusion in the left lateral region. The patient remained fluid overloaded with anuria, so continuous renal replacement therapy was started. The patient ultimately could not be weaned from vasopressors and her condition worsened. She was transitioned to comfort care and passed with family by her bedside.



Pre-Operative Echocardiogram showing pericardial effusion compressing right ventricle



Post-Operative Echocardiogram showing dilated right ventricle that is impinging on left ventricle

Discussion

Three hypotheses have been offered to describe the occurrence of PDS: ischemic, hemodynamic, and autonomic [2]. The ischemic hypothesis identifies decreased coronary perfusion due to compression from increased pericardial fluid as the cause of myocardial stunning that ultimately leads to diastolic dysfunction [2]. The hemodynamic hypothesis postulates that the pericardial fluid develops into a counterforce against the exterior of the right ventricle, preventing increased venous return from dilating the right ventricle. Once the pericardial fluid is removed, the right ventricle expands significantly due to increased venous return, resulting in right heart failure. The right ventricle dilation prevents cardiac myocytes from effectively pumping blood to the left ventricle, ultimately causing interventricular dependence as well as impingement on the left ventricle that leads to decreased left ventricular output [2]. Finally, the autonomic hypothesis suggests that pericardial effusions stimulate sympathetic responses to keep cardiac output elevated [2]. When fluid is drained, the sympathetic stimulus is removed, leading to new systolic dysfunction or unmasking of underlying ventricular dysfunction [2].

The mechanisms leading to PDS are not well understood, but Prabhakar speculated that underlying ventricular dysfunction may contribute to PDS [1]. Cerrud-Rodriguez et al. noted that the only factor associated with increased mortality is surgical pericardiostomy compared to needle pericardiocentesis [3]. Thabet et al. found draining larger volumes is a risk factor for development of PDS and noted that the European Society of Cardiology recommends staged pericardiocentesis with no more than 1 liter removed in a single episode to avoid PDS [4]. However, the amount of fluid removed is not necessarily always a risk factor because Chung et al reported an episode of PDS within 10 minutes of only 250 cc of clear fluid drained via pericardial window

procedure [5]. Chung et al. noted that transesophageal echocardiography (TEE) showed a severely enlarged and hypokinetic right ventricle [5]. Based on their literature review, Chung et al. focused on rapid expansion of the right ventricle as the important factor in developing PDS rather than the amount of volume removed [5]. Literature review by Thabet et al. found that female sex, malignancy-related effusions, and history of radiotherapy also seem to be identified with the development of PDS in addition to volume and rate of fluid drained [4].

A review of current literature and case reports regarding PDS uncovered similarities in comorbidities with our patient. Abdelmalek et al. reported on a patient with ESRD requiring hemodialysis who had 400cc of serosanguinous fluid removed via pericardial window [6]. He also had pericardial drain placed [6]. Several hours post-operative, the patient developed PDS and emergent TEE showed EF 30-34% with moderate global myocardial wall hypokinesis and significantly reduced right ventricular systolic function [6]. Supportive care with vasopressors ultimately lead to the patient's recovery after three days [6]. Abdelmalek et al. hypothesized that their patient developed biventricular failure and subsequent cardiogenic shock due to significantly increased venous return after pericardial window and increased afterload due to sympathetic overdrive [6]. In the case reported by Rao et al., their patient (an 84-year-old female) had a history of severe pulmonary hypertension (PA systolic pressure of 76 mmHg) and pericardial effusion of undetermined etiology [7]. She had 1.2 liters of serous fluid removed via pericardial window and experienced post-operative cardiogenic shock and severe hypoxic respiratory failure [7]. TEE confirmed severe right atrial dilation and severe right ventricle systolic failure [7]. She was ultimately transitioned to comfort care and passed after a few days [7].

After review of current literature and case reports and consideration of the present case, there may be a correlation between PDS and pulmonary hypertension that leads to worse outcomes for patients requiring pericardial effusion. Our patient had been diagnosed with pulmonary hypertension (which was confirmed with right heart catheterization) that was likely due to OSA as well as obesity. The patient's OSA went untreated because the patient was unable to tolerate a CPAP. Pericardial drainage decreased compression of the right ventricle to allow for increased venous return and expansion; however, with the patient's pulmonary hypertension, her right ventricle was unable to overcome the pulmonary arterial pressure enough to effectively pump blood to the left ventricle. The patient's right ventricle kept expanding creating hemodynamic compromise of the left ventricle (as is seen on her final inpatient echo showing an EF of 40-45%). Although the patient's situation was compounded by other comorbidities like ESRD requiring hemodialysis, the survival of Abdelmalek et al.'s patient with supportive care does call into question whether pulmonary hypertension creates a higher risk of PDS. Regardless of whether pulmonary hypertension alone increases risk, the combination of pulmonary hypertension with atrial fibrillation, OSA, and ESRD creates a scenario in which the patient can become easily fluid overloaded, creating an even worse clinical outcome when PDS develops.

Conclusions

PDS is a sequalae of hypotension, tachycardia, and pulmonary edema that can occur after pericardial fluid drainage which is a tool utilized to relieve those very symptoms being caused by cardiac tamponade. The cause of PDS has not yet been determined, but myocardial stunning from ischemia of compression of coronary arteries, increased venous return that overexpands the right ventricle, and removal of sympathetic stimulus that leads to systolic dysfunction are currently the leading hypotheses. Although some risk factors for PDS have been identified, there is still much the medical community does not know about what mechanisms precipitate PDS. The method of drainage (pericardial window vs needle pericardiocentesis), volume of drainage, and rate of drainage have been found to be contributing factors to development of PDS; however, this case seems to purport that co-morbidities may influence risk of PDS, specifically pulmonary hypertension. In the future, we hope this case report can inform physicians of the possible risks for PDS, recognize signs and symptoms of PDS, and help physicians manage the care of their patient needing pericardial drainage.

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