Case Report

Pericardial Decompression Syndrome: A Case Series and Literature Review

Chloe Thabet, MD,^a Zachary MacDonald, MD,^b Christopher Johnson, MD,^{c,d} Joel Niznick,^{c,d}
Habibat Aziz Garuba, MD,^{c,d} and Angeline Law, MD^{c,d}

^a Department of Medicine, University of Ottawa, Ottawa, Ontario, Canada

^b Department of Emergency Medicine, University of Ottawa, Ottawa, Ontario, Canada

^c Division of Cardiology, Department of Medicine, University of Ottawa, Ottawa, Ontario, Canada

^d University of Ottawa Heart Institute, University of Ottawa, Ottawa, Ontario, Canada

ABSTRACT

Cardiac tamponade is a medical emergency requiring prompt recognition and intervention to avoid potentially fatal consequences. We present a case series of ventricular dysfunction and cardiogenic shock following pericardiocentesis in 3 patients with pericardial effusions at The Ottawa Hospital between 2014 and 2020. This report highlights the need for monitoring post-pericardiocentesis and raises awareness of this phenomenon, particularly in patients with malignancy. We propose a novel pressure-monitoring protocol to guide drainage and prevent development of pericardial decompression syndrome. The novel teaching points include limiting drainage to prevent development of pericardial decompression syndrome and a protocol for intrapericardial pressure monitoring.

RÉSUMÉ

La tamponnade cardiaque est une urgence médicale qui, à défaut d'une reconnaissance et d'une intervention rapides, peut avoir des conséquences potentiellement fatales. Nous présentons une série de cas sur des dysfonctions ventriculaires et des chocs cardiogéniques survenus après une péricardiocentèse chez trois patients présentant des épanchements péricardiques traités à l'Hôpital d'Ottawa entre 2014 et 2020. Notre article souligne la nécessité de surveiller les patients au cours de la période suivant la péricardiocentèse et met en lumière le phénomène de la tamponnade cardiaque, en particulier chez les patients atteints de cancer. Nous proposons un nouveau protocole de surveillance des pressions conçu pour guider le drainage et prévenir le syndrome de décompression péricardique. Les nouveautés à enseigner comprennent la limitation du drainage afin de prévenir le syndrome de décompression péricardique et un protocole de surveillance des pressions intrapéricardiques.

Cardiac tamponade is a medical emergency resulting from excess or rapidly accumulated fluid in the pericardial space causing myocardial compression and impairment in cardiac filling and cardiac output.¹ Common causes include malignancy, acute pericarditis, post-myocardial infarction, aortic dissection, and chest trauma.¹ Emergent needle decompression and surgical pericardiostomy are the mainstays of treatment to improve cardiac hemodynamics. However, in approximately 5% of cases of percutaneously managed cardiac tamponade,² patients may experience paradoxical hemodynamic instability, also known as pericardial decompression

syndrome (PDS).³ PDS is defined as paradoxical hemodynamic instability following pericardial drainage leading to ventricular dysfunction.³ However, the exact etiology of PDS remains unclear. In this report, we describe the cases of 3 patients with PDS, and provide a review of the current literature on this topic (see Table 1 for patient characteristics).

Case 1

A 35-year-old man with a history of esophageal adenocarcinoma presented to the emergency department with a 3-week history of progressive dyspnea. He had undergone esophagectomy followed by epirubicin, cisplatin, and capecitabine chemotherapy 6 years prior. At the time, a multigated acquisition radionuclide left ventriculography scan showed a normal left ventricular ejection fraction (LVEF) of 56%. On initial assessment, he was normotensive (blood pressure [BP] 110/75 mm Hg) and tachycardic (heart rate [HR] 120 beats per minute [bpm]), with a pulsus paradoxus of 12 mm Hg and distant heart sounds. An electrocardiogram revealed

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Corresponding author: Dr Angeline Law, The Ottawa Hospital General Campus, 501 Smyth Road, Ottawa, Ontario K1H 8L6, Canada. Tel.: +1-613-737-8810; fax: +1-613-739-6727.

E-mail: alaw@toh.ca

See page 423 for disclosure information.

Novel Teaching Points

- PDS is an underrecognized and likely underreported phenomenon, owing to lack of familiarity with the condition, its variable clinical presentations, and differing terminology in the literature.
- Patients with malignancy-associated pericardial effusions seem to be at higher risk for development of PDS following pericardiocentesis.
- Current guideline-recommended pericardial drainage in 1-liter increments may still put patients at risk of developing PDS.
- We recommend pressure-monitored pericardiocentesis, using pressure transducers to minimize the risk of development of PDS.

sinus tachycardia with low voltages and electrical alternans (Fig. 1). A transthoracic echocardiogram (TTE) demonstrated a large circumferential pericardial effusion, with signs of tamponade, and normal biventricular function. Urgent echocardiographically-guided pericardiocentesis was performed via a transapical approach, draining 1500 mL of hemorrhagic fluid, with initial resolution of tachycardia and dyspnea. An additional 1000 mL was drained over the next 24 hours.

Within the next 24 hours, the patient developed cardiogenic shock with hypotension, hypoxemia, and renal failure. Computed tomography of the chest showed no air space disease, pulmonary embolism, or procedural complication. A repeat echocardiogram showed new-onset severe biventricular failure (LVEF < 25%) despite resolution of the pericardial effusion. He required inotropic support with dobutamine, diuresis, and introduction of captopril, carvedilol, and digoxin, which led to significant clinical improvement. A multigated acquisition radionuclide left ventriculography scan 7 days later revealed a mildly dilated left ventricle (LV) with recovered systolic function (LVEF 53%) and normal right ventricle (RV) size and function. Pericardial fluid cytology confirmed malignancy, and staging imaging revealed new brain metastases. He was discharged for treatment of recurrent esophageal cancer. A follow-up echocardiogram 3 months later showed normal biventricular function.

Case 2

A 35-year-old woman with a history of non-small cell lung adenocarcinoma, previously treated with cisplatin and

vinorelbine, presented to the emergency department with chest pain, dyspnea, and orthopnea. She had been seen 3 weeks prior with acute pericarditis. On initial assessment, she was normotensive (BP 136/82 mm Hg) and tachycardic (HR 110 bpm), with distended neck veins. An electrocardiogram revealed sinus tachycardia. A TTE demonstrated a large pericardial effusion with right atrial diastolic compression suggestive of tamponade. A computed tomography pulmonary angiogram confirmed the large pericardial effusion and revealed bilateral pleural effusions and findings suggestive of lymphangitic carcinomatosis. Echocardiographically-guided pericardiocentesis was performed, with drainage of approximately 500 mL of hemorrhagic pericardial fluid. Cytologic analysis demonstrated recurrent adenocarcinoma. A bedside echocardiogram was done pre-and post-pericardiocentesis, confirming normal LV systolic function at both those times.

In the hours following pericardiocentesis, she developed progressive hypotension despite volume resuscitation and inotropic support. A repeat bedside echocardiogram revealed mildly reduced LV systolic function (LVEF 45%-50%), severe RV dilatation, severe RV dysfunction, and severe tricuspid regurgitation. Despite maximal inotropic support, she suffered a cardiac arrest that was unresponsive to resuscitation efforts.

Case 3

A 62-year-old woman with no past medical history presented to the emergency department with a 4-week history of progressive dyspnea, back pain, generalized weakness, dysphagia, and weight loss. On presentation, she was hypotensive (BP 100/85 mm Hg) and tachycardic (HR 110 bpm), with distended neck veins and palpable pulsus paradoxus. An electrocardiogram revealed sinus tachycardia and low QRS voltages. A TTE demonstrated a large circumferential pericardial effusion with echocardiographic signs of tamponade, bilateral pleural effusions, and ascites. Urgent echocardiographically-guided pericardiocentesis was performed via a transapical approach, draining 450 mL of hemorrhagic fluid, with rapid improvement in her HR (70 bpm).

Despite improvement in HR, hypotension worsened immediately following pericardiocentesis (BP 80/60 mm Hg). TTEs were repeated at 10 and 30 minutes post-pericardiocentesis and revealed worsening LV systolic dysfunction and global hypokinesis. In addition, her RV was noted to be dilated with severe RV free wall hypokinesis and dysfunction. A computed tomography scan of the thorax, abdomen, and pelvis revealed appropriate pericardial drain placement, but also a large locally invasive left upper-lobe lung

Table 1. Summary of patient characteristics

Patient	Age, y	Sex	Pre-pericardiocentesis					Post-pericardiocentesis					
			BP, mm Hg	HR, bpm	PP	Size	Cause	Volume, mL	BP	HR	Failure	Inotrope	Outcome
1	35	M	110/75	120	+	L	Malignancy	1500, (1000)	n/a	n/a	RV/LV	+	Survival
2	35	F	136/82	110	n/a	L	Malignancy	500	91/76	n/a	RV/LV	+	Death
3	62	F	100/85	110	+	L	Malignancy	450 (400)	80/60	70	RV	+	Death

Volume indicates volume (volume over following 24 h).

BP; blood pressure; bpm, beats per minute; F; female; HR; heart rate; L, large; LV; left ventricle; n/a, not available from chart review; M; male; PP; pulsus paradoxus; RV; right ventricle; +, presence.

Electrocardiogram showcasing the classic electrocardiographic signs of cardiac tamponade: sinus tachycardia, low voltage QRS complexes and electrical alternans.

Figure 1. Electrocardiogram results of a 35-year-old woman with a history of esophageal carcinoma and large-volume pericardial effusion. Displayed are the classic electrocardiographic signs of cardiac tamponade: sinus tachycardia, low-voltage QRS complexes, and electrical alternans.

mass with pulmonary and adrenal metastases, and a right lower-lobe pulmonary embolism. Pericardial cytology conducted after the patient's death was consistent with malignancy.

She was transferred to the intensive care unit and required escalating doses of vasopressors, inotropes, and corticosteroids. Despite intensive care, she developed refractory hypotension, hypoxia, and multiorgan hypoperfusion. She was transitioned to comfort care and passed away within 24 hours of the performed pericardiocentesis.

Discussion

We present 3 cases of PDS following large-volume pericardiocentesis in malignancy-associated cardiac tamponade.

Pathophysiology

Although the etiology of PDS is unknown, numerous hypotheses have been suggested. The hemodynamic hypothesis suggests that hemodynamic changes occur as a result of interventricular dependence following drainage of pericardial fluid.² In this case, removal of pericardial fluid, initially compressing the right heart, allows for increased venous return, causing rapid right-chamber expansion, leading to decreased LV filling and subsequently cardiac output.² Pericardial decompression also leads to increased pulmonary venous return, which with compensatory increased systemic vascular resistance can precipitate HF.² In contrast, the ischemic hypothesis suggests that external compression of the pericardium by accumulated fluid may lead to reduced coronary artery perfusion. Following decompression, transient myocardial stunning may occur, causing transient ventricular dysfunction.² Additional hypotheses regarding autonomic and

sympathetic mismatch have also been suggested as a potential mechanism of PDS. In this case, tamponade may act as an inciting trigger for catecholamine release. After drainage and subsequent resolution of tamponade physiology, the sudden reduction in catecholamine release may lead to systolic dysfunction and PDS.² This pathophysiology may share similarities with stress-induced cardiomyopathy, with a certain overlap between these 2 syndromes, which can be difficult to differentiate.²

The true incidence of PDS is unknown, but it is estimated to be around 5% following subxiphoid pericardiocentesis.² However, this estimate is likely an underestimate, owing to lack of familiarity with PDS, leading to underreporting, variable clinical presentations, and differing terminology throughout the literature.⁴

Risk factors

Literature is scarce on the risk and predictive factors of development of PDS. A recent review of PDS identified 5 characteristics associated with the development of PDS following drainage: malignancy-related effusions, history of radiotherapy, female sex, volume of pericardial fluid drained, and the rate of pericardial drainage.³ In this review, 59 cases of PDS were identified between 1983 and 2019.³ Among these, malignancy was the most common cause of pericardial effusion, occurring in 38.7% of cases, followed by idiopathic effusions.³ This distribution is similar across the literature.^{3,5,6} Sabzi and Faraji also reported malignancy as an independent predictor of PDS, alongside a history of radiotherapy.⁶ Therefore, a reasonable inference is that malignancy-driven pericardial effusions have a higher propensity for development of PDS than non-malignancy-associated effusions, although the underlying pathophysiology remains unknown.

At our centre, all cases were associated with malignancy. In addition, Amro et al. and Sabzi and Faraji identified a higher proportion of PDS among women than men^{3,6}; however, our sample is too small for comparison. In a separate study, drainage volume was identified as an independent risk factor for development of PDS, with a mean drainage volume of 647 ± 217 mL in patients who developed PDS, compared with 495 ± 231 mL in those who did not ($P = 0.003$).⁵ We identified similar results, with a mean drainage volume of 1266 ± 887 mL, suggesting that higher volumes drained are more likely to result in PDS. Further reports have shown a significant difference in development of PDS in patients undergoing pericardiostomy vs pericardiocentesis, with higher rates for pericardiostomy (5% vs 3%, $P = 0.036$),⁷ and some reports of higher PDS mortality with pericardiostomy.⁷

Novel pericardial drainage protocol

Despite advances in research, few evidence-based guidelines exist advising on safe pericardial drainage techniques. Following a case series of 3 patients with RV dysfunction following pericardiocentesis, the European Society of Cardiology recommended performing staged pericardial drainage, limiting drainage to increments of 1 liter to avoid PDS.⁸ As seen from 2 of our cases, this volume may still be far too much. A smaller volume may be sufficient to reverse tamponade physiology and allow for adequate ventricular filling. We propose the use of a pressure transducer attached to the intrapericardial catheter to guide the volume of pericardial drainage. In this way, pericardial fluid can be withdrawn serially until the intrapericardial pressure falls below 10 mm Hg. Such a protocol was developed by expert consensus and is being piloted at our centre (see [Supplemental Appendix S1](#)). To our knowledge, our centre is the first to trial noninvasive pressure monitoring during pericardiocentesis with the aim of reducing the risk of development of PDS.

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Disclosures

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Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Open* at <https://www.cjopen.ca/> and at <https://doi.org/10.1016/j.cjco.2021.11.011>.