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MINI-FOCUS ISSUE: PROCEDURAL COMPLICATIONS: PART 2

INTERMEDIATE

CASE REPORT: CLINICAL CASE

Severe Right Ventricular Failure Following Pericardiocentesis

A Case Report of Pericardial Decompression Syndrome

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ABSTRACT

Pericardial decompression syndrome, a rare but potentially fatal complication following pericardiocentesis, is defined as paradoxical hemodynamic deterioration. The exact pathophysiology is unknown, but it is likely that several mechanisms involving hemodynamic, ischemic, and autonomic imbalance play a role. There is no specific treatment; however, early supportive interventions should be implemented. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:58–63) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 73-year old woman presented to the hospital with 5 days of worsening dyspnea and fatigue. Examination was noticeable for tachycardia of 110 beats/min, blood pressure of 90/50 mm Hg, jugular venous distention, and the presence of pulsus paradoxus. A computed tomography scan showed multiple hepatic and pulmonary lesions, enlarged abdominal lymph nodes, and pleural and pericardial effusions concerning for advanced metastatic cancer of unknown primary origin (Figures 1A and 1B). She had elevated serum tumor markers, including carbohydrate antigen 19-9, alpha-fetoprotein, and carcinoembryonic antigen. An echocardiogram showed a large pericardial effusion with right ventricular (RV) diastolic collapse and 25% to 30% respiratory variation in Doppler mitral inflow concerning for cardiac tamponade (Figures 2A and 2B, Video 1).

She was taken to the catheterization laboratory for emergency needle pericardiocentesis. Approximately 750 ml of sanguineous fluid was drained, and the patient was transferred to the intensive care unit with the drain in place. She experienced brief improvement in her symptoms, and the drain was removed 1 day later. On day 2, however, hypotension and worsening tachycardia developed, with cold extremities and jugular venous distention on examination.

LEARNING OBJECTIVES

- To recognize PDS as a rare but potentially fatal complication following needle or surgical pericardiocentesis.
- To understand the possible mechanisms involved in paradoxical hemodynamic deterioration following pericardiocentesis.
- To recognize the importance of close clinical monitoring and implementation of early supportive interventions.

PAST MEDICAL HISTORY

Her medical history included hypertension.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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DIFFERENTIAL DIAGNOSIS

The differential diagnosis included coronary artery or cardiac chamber puncture, hemothorax, massive pulmonary emboli, and pericardial decompression syndrome (PDS).

INVESTIGATIONS

Laboratory test results showed metabolic acidosis with elevated lactic acid. Repeat echocardiograms showed massive RV dilation and dysfunction and persistent ventricular interdependence (**Figures 3A and 3B** and **4A to 4D**, Videos 2, 3, and 4). An electrocardiogram showed no changes suggestive of myocardial ischemia. Hemodynamic values from bedside right-sided heart catheterization using a Swan-Ganz catheter showed right atrial pressure of 30 mm Hg, RV diastolic pressure of 31 mm Hg, pulmonary artery pressure of 48/29 mm Hg, and pulmonary capillary wedge pressure of 31 mm Hg. These findings were all consistent with RV failure and elevated pulmonary capillary wedge pressure likely secondary to ventricular interdependence from RV enlargement. The cardiac index was 1.7 l/min/m^2. Computed tomography angiography demonstrated a small, subsegmental pulmonary embolus in a right lower segmental branch. The pulmonary embolism was not believed to be large enough to explain the degree of RV compromise.

MANAGEMENT

Inotropic support was initiated with dobutamine. She remained stable over the next 48 h. Subsequently, her condition acutely deteriorated, with refractory hypotension, hypoxemia, and multiorgan failure despite pharmacological inotropic support. An echocardiogram showed persistent RV enlargement with abnormal septal motion.

DISCUSSION

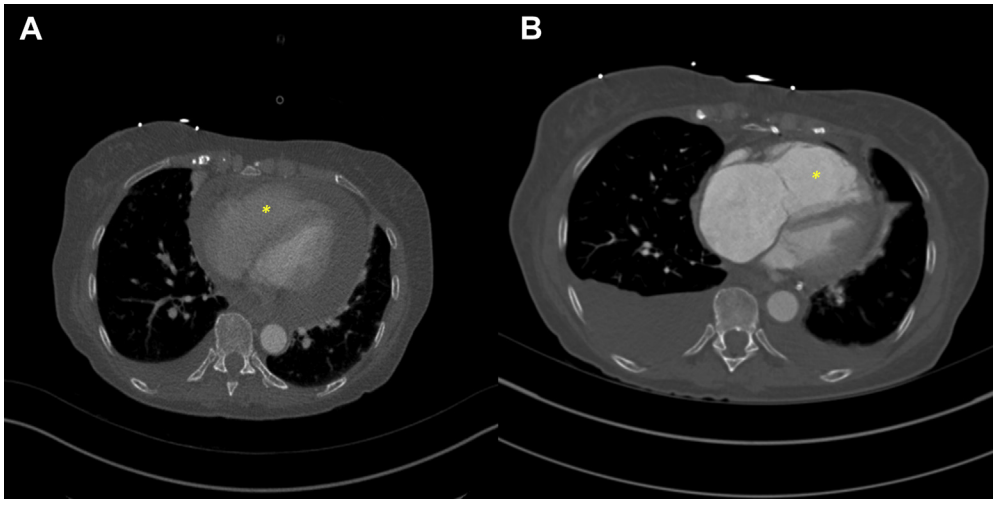
Pericardiocentesis is a lifesaving therapeutic procedure for patients presenting with cardiac tamponade. It is relatively safe; however, physicians must be aware of potential post-procedural complications. The risk of complications ranges from 4% to 10%, with the most common being arrhythmias, coronary artery or cardiac chamber puncture, hemothorax, pneumothorax, and pneumopericardium (1). Our patient illustrates a case of PDS, a rare but potentially fatal complication after pericardiocentesis. It is defined as worsening of hemodynamics after uncomplicated pericardial drainage in patients with pericardial effusions and cardiac tamponade when hemodynamic values are expected to improve. Other names used in the past for this clinical entity include "low cardiac output syndrome" and "paradoxical hemodynamic instability" (2,3). Since the first

ABBREVIATIONS AND ACRONYMS

PSD = pericardial decompression syndrome

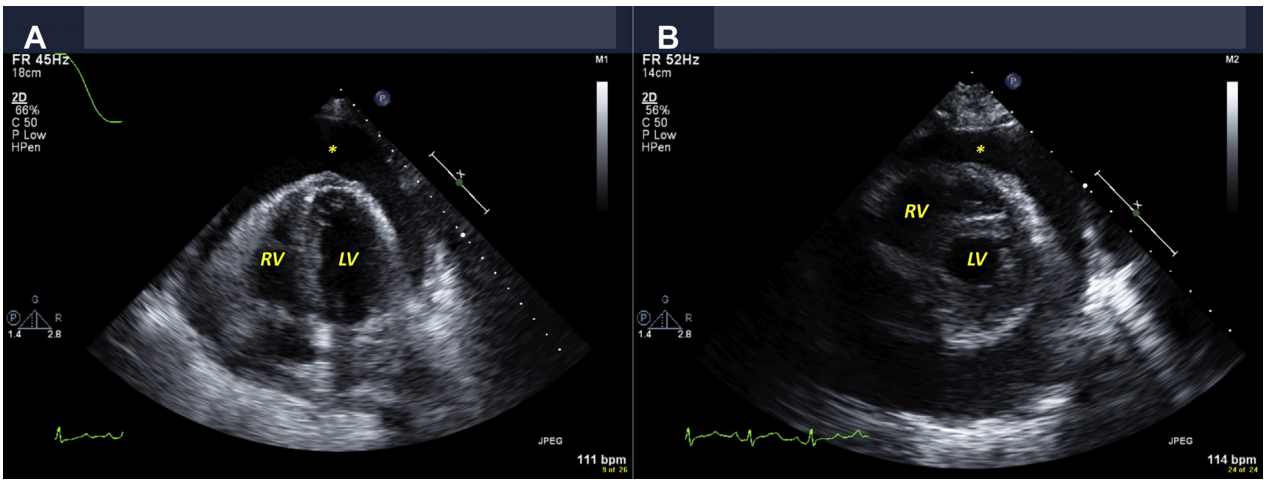
RV = right ventricular

FIGURE 1 Computed Tomography



(A) Large pericardial effusion **(asterisk). (B)** Scan after pericardiocentesis showing significant right ventricular enlargement **(asterisk).**

FIGURE 2 2-Dimensional Echocardiography



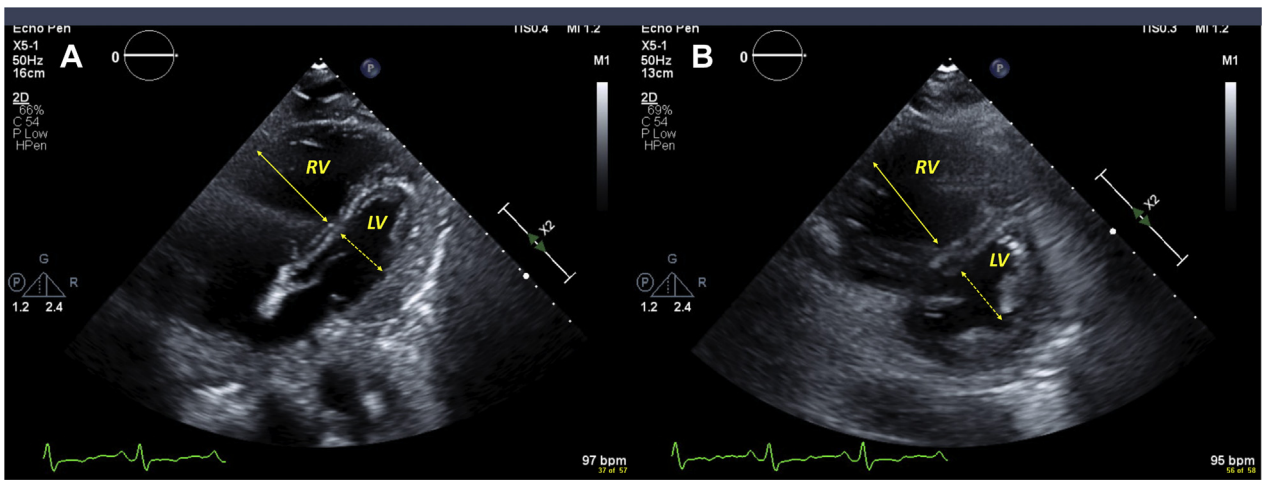
(A) Apical 4-chamber and **(B)** short-axis views revealing massive pericardial effusion **(asterisks).** LV = left ventricle; RV = right ventricle.

description by Vandyke et al. in 1983 (4), many other reports have allowed wider recognition of this complication among clinicians (**Table 1**).

The exact incidence of PDS is not precisely known given the wide variability in occurrence rates in different small case series, but it is estimated to be approximately 5%. Clinical factors associated with an increased risk of PDS include a history of malignant disease or radiation therapy, pre-existing cardiomyopathy with decreased systolic function, and connective tissue disorders. In a study, surgical pericardiocentesis was the only variable associated with increased mortality when compared with needle pericardiocentesis in patients with PDS (5).

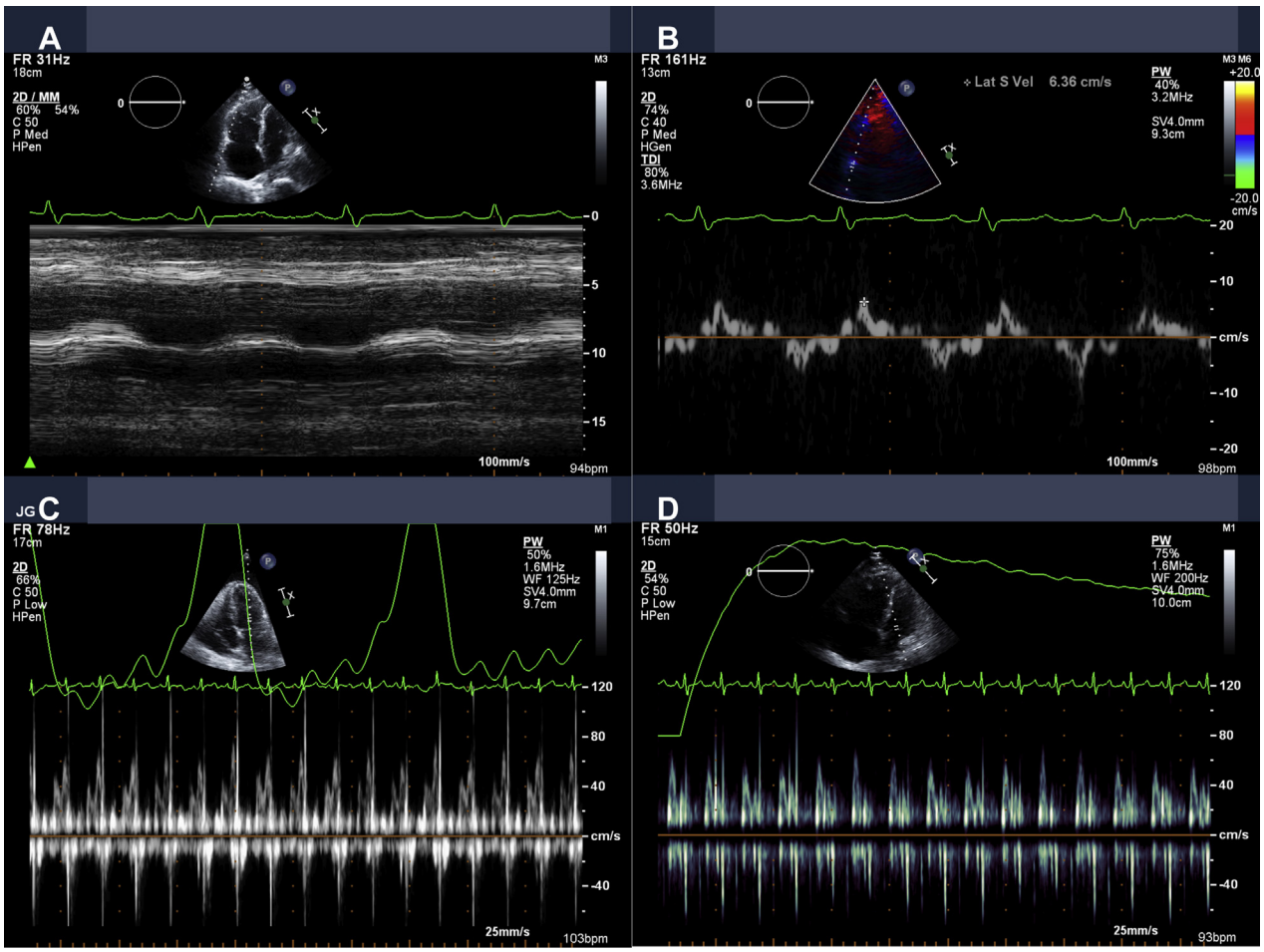
The onset of PDS after pericardial drainage is also variable. It can occur immediately after a brief initial improvement in a patient's hemodynamics or within a couple of days, and it most commonly manifests as

FIGURE 3 2-Dimensional Echocardiography After Pericardiocentesis



(A) Apical 4-chamber and **(B)** short-axis views showing massive enlargement of the right ventricle (RV). Note the diameter of the right ventricle **(solid arrow)** in comparison with that of the left ventricle (LV) **(dashed arrow).**

FIGURE 4 **2-Dimensional Echocardiography After Pericardiocentesis**



(A) Decreased tricuspid annular plane systolic excursion of 6 mm on M-mode imaging and **(B)** tricuspid annular S′ velocity of 6 cm/s on tissue Doppler imaging consistent with right ventricular dysfunction. Pulsed-wave Doppler revealed >25% respiratory mitral flow variation consistent with ventricular interdependence **(C)** before and **(D)** after pericardiocentesis.

acute congestive heart failure, often with pulmonary edema. In up to one-third of cases, PDS may manifest with the development of cardiogenic shock.

The exact pathophysiology of PDS is not well understood, but several mechanisms involving hemodynamic, ischemic, and autonomic imbalance have been suggested. It is believed that right-sided chamber expansion resulting from increased venous return after removal of the compressing pericardial fluid can affect left ventricular filling and the effective cardiac output. Simultaneously, the net increase in pulmonary venous return with greater systemic vascular resistance can cause a pre-load-afterload mismatch and result in congestive heart failure. Additional contributing factors may be myocardial ischemia and stunning caused by impaired coronary artery perfusion because it is known that increased intrapericardial pressure affects maximal hyperemic coronary flow (6). Finally, the acute withdrawal of sympathetic stimulus after removal of effusion has been hypothesized to provoke autonomic imbalance. This imbalance is theorized to occur either because of an unmasking of pre-existing myocardial dysfunction that was not apparent in the hyperadrenergic state with increased circulating catecholamines (7) or because of induction of new myocardial dysfunction secondary to overwhelming autonomic stress through a mechanism similar to that of stress-induced cardiomyopathy. In fact, some investigators have suggested that stress-induced cardiomyopathy and PDS

TABLE 1 Summary of Published Case Reports of Pericardial Decompression Syndrome

Authors (Ref. #)	Journal	Year of Publication	Age (yrs)	Sex	Drain Method	Drained Fluid (ml)	Symptom Onset	Outcome
Cerrud-Rodriguez et al.	J Am Coll Cardiol Case Rep	2020	70	Male	P	2,060	Minutes	Improved
Rao et al.	Cureus	2020	84	Female	S	1,200	2 days	Died
Ricarte et al.(9)	Crit Care Med	2020	69	Male	P	900	Immediate	Improved
Prabhakar et al.	World J Cardiol	2019	58	Female	P	2,200	1 h	Improved
Chung et al.	J Cardiothorac Vasc Anesth	2019	41	Female	S	250	1 h	Improved
Albeyoglu et al.	Int J Surg Case Rep	2016	43	Female	S	1,000	Hours	Improved
Fozing et al.	BMJ Case Rep	2016	44	Male	P	2,760	3 h	Improved
Basmaji et al.	Int J Cardiol	2015	54	Female	P	460	Minutes	Improved
Pradhan et al. (5)	Eur Heart J Acute Cardiovasc Care	2015	41	Male	P	550	30 min	Improved
Ayoub et al. (8)	Cardiovasc Ultrasound	2015	62	Male	P	1,800	9 h	Improved
Lim et al.	BMJ Case Rep	2011	44	Female	S	1,000	9 h	Died
Versaci et al.	J Cardiovasc Med	2010	78	Female	P	1,000	24 h	Improved
Moreno Flores et al.	Rev Esp Cardiol	2009	80	Male	P	1,200	48 h	Improved
Karamichalis et al.	Ann Thorac Surg	2009	19	Female	S	1,600	30 min	Died
Sunderji et al.	BMJ Case Rep	2009	56	Male	P	1,500	24 h	Improved
Sharaf et al.	Can J Cardiol	2008	55	Female	P	600	6 h	Improved
Sevimil et al.	Turk Kardiyol Dern Ars	2008	42	Female	P	500	24 h	Improved
Bernal et al.	Can J Cardiol	2007	45	Female	P	500	6 h	Improved
Dosios et al. (2)	Angiology	2007	66	Female	S	500	Hours	Died
Ligero C et al.	Eur J Heart Fail	2006	41	Female	P	1,000	3 h	Improved
Geffroy et al.	Can J Anaesth	2004	53	Male	S	1,500	30 min	Died
Dosios et al.	Chest	2003	37	Female	S	700	3 h	Died
			67	Female	S	900	5 h	Died
			31	Male	S	450	3 h	Died
			69	Female	S	650	7 h	Improved
			70	Male	S	1,000	6 h	Died
Chamoun et al.	Clin Cardiol	2003	36	Female	P	1,070	12 h	Improved
Sunday et al.	Ann Thorac Surg	1999	60	Female	S	700	Immediate	Died
Thrush	J Cardioth Vasc Anesth	1998	58	Female	S	600	15 min	Improved
Anguera et al.	Int J Cardiol	1997	68	Female	P	800	Immediate	Improved
Uemura et al.	Jpn Circ J	1995	18	Male	P	450	20 min	Improved
Braverman et al.	Ann Intern Med	1994	27	Female	S	1,000	Immediate	Improved
Hamaya et al.	Anesth Analg	1993	16	Female	P	700	Immediate	Improved
Wolfe et al. (7)	Ann Intern Med	1993	46	Female	P	650	12 h	Improved
			50	Female	P	650	Immediate	Improved
Voller et al.	Z Kardiol	1993	22	Female	P	700	Immediate	Improved
Downey et al.	Crit Care Med	1991	50	Male	P	1,500	4 h	Improved
Glasser et al.	Chest	1998	33	Male	S	2,100	Immediate	Improved
Barniek et al.	Cardiologia	1987	36	Female	P	560	60 min	Improved
Shenoy et al.	Chest	1984	57	Male	S	1,000	Immediate	Improved
Vandyke et al. (4)	N Engl J Med	1983	42	Male	Both	680	Immediate	Improved

P = Percutaneous pericardiocentesis; S = surgical pericardiostomy.

may not be distinct entities but rather may belong to the same spectrum of disease, given the overlapping clinical presentation seen in some cases (8). Our case, however, lacks typical features of stress-induced cardiomyopathy: global RV involvement as opposed to apical ballooning with preserved basal function, no chest pain, and no subsequent improvement of myocardial function.

There is no specific treatment for PDS other than supportive care. The exact mortality rate is not well known, but it has been estimated to be approximately 30% on the basis of case reports. Although ventricular dysfunction is transient and is expected to recover in survivors of PDS, patients require advanced support measures such as aggressive heart failure therapy, inotropic medications, and the use of mechanical circulatory support (9). In cases of profound shock, the ideal type of mechanical support depends on the degree of myocardial dysfunction as well as the pattern of

ventricular involvement, whether left ventricular, RV, or biventricular dysfunction is noted.

Currently there are no proven measures known to prevent PDS. Despite the recommendation in the European Society of Cardiology 2015 guidelines (1) to drain fluid in <1-liter steps to avoid acute RV dilatation, PDS may occur with drainage volumes <500 ml. A reasonable strategy is as follows: remove only enough fluid to alleviate tamponade physiology at first (this can be achieved under hemodynamic and echocardiographic guidance if available); and then slowly remove the remaining fluid by leaving the pericardial drain in place, especially in patients with cancer-related effusions or impressive chamber collapse (10).

FOLLOW-UP

While preparing for emergency endotracheal intubation, she had pulseless electrical activity cardiac arrest. Despite resuscitative efforts she remained pulseless, and she was declared dead. Post-mortem examination was declined by the family.

CONCLUSIONS

This uncommon case of PDS highlights the high morbidity and mortality associated with this complication, the possibility of instituting preventive strategies in high-risk cases, and the importance of prompt recognition of PDS, as well as close clinical monitoring and aggressive supportive care.

AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS cardiogenic shock, cardiomyopathy, pericardial tamponade, right ventricular dysfunction

APPENDIX For supplemental videos, please see the online version of this article.