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CASE REPORT

ADVANCED

CLINICAL CASE SERIES

Pericardial Decompression Syndrome After Drainage of Chronic Pericardial Effusions

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ABSTRACT

Pericardial decompression syndrome (PDS) is a potentially fatal disorder of left ventricular function that sometimes occurs after drainage of a pericardial effusion for cardiac tamponade. Patients at risk for PDS are difficult to identify. Here, we report 2 cases where PDS developed after drainage of effusions that had been present for years, suggesting that patients with chronic effusions are at higher risk for PDS. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:1515–1521) © 2022 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/).

Pericardial decompression syndrome (PDS) is a life-threatening complication of pericardiocentesis for cardiac tamponade.[1,2] The main clinical manifestations are pulmonary edema, hemodynamic instability, and cardiogenic shock.[3,4] The onset of PDS is usually delayed, occurring several hours after successful pericardiocentesis following a period of initial improvement. PDS is a diagnosis of exclusion; a similar clinical picture may occur with recurrent tamponade, laceration or perforation of the right ventricle (RV) with hemopericardium or exsanguination, tension pneumothorax, myocardial infarction, pulmonary embolism, or stress cardiomyopathy. The mainstays of treatment are respiratory support; fluid management; and, when necessary, support with intravenous inotropic agents or mechanical circulatory assist devices. Patients requiring higher levels of support have a higher mortality (Table 1). There is no specific treatment. The incidence of PDS is estimated to be 5% to 10%, and the mortality based on case reports is about 30%.[3-5] To prevent PDS, some authorities recommend initially removing the minimum volume of pericardial fluid required to relieve tamponade, followed by prolonged slow drainage until the output is <30 mL/d.[6] Fatal cases of PDS have been associated with drainage

LEARNING OBJECTIVES

- To increase awareness of pericardial decompression syndrome (PDS) and its association with chronic pericardial effusions.
- To understand the differential diagnosis and treatment of PDS.
- To understand theories of the pathogenesis of PDS.

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ABBREVIATIONS AND ACRONYMS

CXR = chest x-ray

ED = emergency department

LV = left ventricular

LVEF = left ventricular ejection fraction

PDS = pericardial decompression syndrome

RA = right atrium

RV = right ventricle

TTE = transthoracic echocardiogram

by surgical pericardiostomy rather than by percutaneous pericardiocentesis.[3] If the patient survives, there are no known long-term sequelae.

The pathogenesis of PDS is poorly understood, and patients at risk for PDS are difficult to identify. Here we describe 2 cases of PDS that were remarkable because they occurred in patients with chronic pericardial effusions. Our attention was drawn to these cases because they occurred within 6 months of each other. Our observations suggest that patients with chronic effusions are at higher risk for PDS.

CASE SERIES

PATIENT 1. An 86-year-old man with a chronic pericardial effusion presented to the emergency department (ED) complaining of cough and dyspnea for 2 weeks. His effusion had been first noted 45 months previously on a routine transthoracic echocardiogram (TTE). At the time, it was 1.6 cm in width, there was right atrial (RA) diastolic collapse but no other evidence of tamponade, and the left ventricular (LV) ejection fraction (LVEF) and valves were normal (**Figures 1A and 1D,** Video 1). In the ED, a chest x-ray (CXR) showed an enlarged cardiac silhouette, pulmonary vascular congestion, and bilateral pleural effusions (**Figure 1G**). A TTE showed that the pericardial effusion had increased to 3 cm in width (**Figures 1B and 1E,** Video 2). The estimated RA pressure was 15 mm Hg and there was RA diastolic collapse with respiratory variation in the mitral inflow and aortic outflow. His LVEF had decreased to 33%. At pericardiocentesis, the opening pericardial pressure was 8 mm Hg, and 970 mL of serous fluid was drained. The pericardial fluid was a transudate without malignant cells. A postprocedural TTE showed no residual effusion (**Figures 1C and 1F**). He initially reported marked improvement in his dyspnea, but 3 hours later he developed chest pain, tachycardia (120 beats/min), hypotension (85/50 mm Hg), and hypoxia (oxygen saturation: 80%-85%). A CXR showed worsening pulmonary edema (**Figure 1H**), and an electrocardiogram showed no ischemic changes. A portable bedside echocardiogram and an echocardiogram the next morning (Video 3) showed no reaccumulation of pericardial fluid. He was treated with supplemental oxygen, intravenous fluids, and intravenous norepinephrine. His oxygen saturation and blood pressure recovered, and the next day gentle diuresis was instituted. Serial measurements of the serum troponin remained normal. Three days later, left and right heart catheterization showed moderately severe coronary artery disease and normal hemodynamics (RA pressure: 9 mm Hg; pulmonary capillary wedge pressure: 10 mm Hg; cardiac index: 3.0 L/min/m^2). His coronary artery disease was treated by multivessel percutaneous intervention and he was discharged on the 11th hospital day.

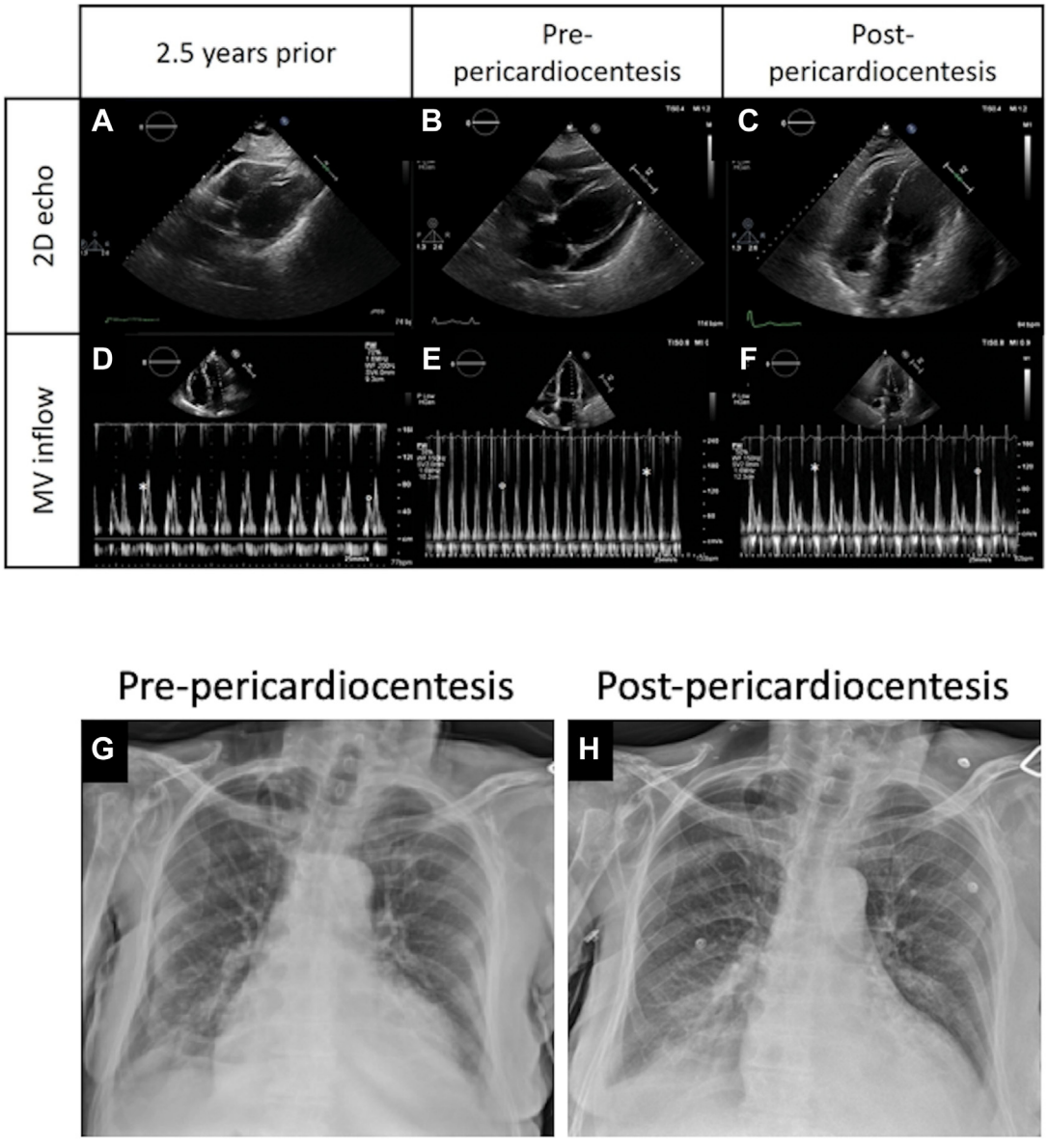
TABLE 1 Treatment and Outcome of Pericardial Decompression Syndrome

	Total Patients	Died	% Mortality
Conservative therapy			
Observation only	2	0	0
Intravenous fluids	3	0	0
Diuretics	18	0	0
Guideline directed medical therapy for heart failure	14	0	0
Conservative therapy only	12	0	0
Interventional therapy			
Intravenous pressor agents	30	6	20
Mechanical ventilation	15	3	33
Mechanical circulatory assistance	3	0	0
Pericardial window[a]	4	0	0
Total	45	7	13

Values are n or %. Data were collected from the cases listed in **Table 2.**
[a]All 4 patients had initially undergone percutaneous pericardiocentesis.

PATIENT 2. A 71-year-old man with immunoglobulin A nephropathy, cirrhosis, diabetes, and a chronic pericardial effusion presented to the ED complaining of dyspnea on exertion, orthopnea, cough, and peripheral edema for 2 weeks. He had undergone kidney and liver transplantation 10 years previously. His effusion had been discovered 20 months before on a routine TTE (**Figures 2A and 2D,** Video 4). At the time it was 2.0-2.5 cm in width, there was no evidence of tamponade, and the LVEF and valves were normal. In the ED, a CXR showed an enlarged cardiac silhouette and pulmonary edema (**Figure 2G**). A TTE showed that the effusion had increased to 3.5 cm in width and that the LVEF had decreased to 45% (**Figures 2B and 2E,** Video 5). The inferior vena cava was engorged with an estimated RA pressure of 15 mm Hg, and there was RV diastolic collapse and variation in the LV stroke volume of >25%. At pericardiocentesis, the opening pericardial pressure was 10 mm Hg, and 1,100 mL of serous fluid was drained. A postprocedural TTE showed complete drainage of the effusion (**Figures 2C and 2F**). The pericardial fluid was a transudate without malignant cells. He initially did well, but 8 hours after the procedure he complained of chest pain and increasing respiratory distress. He was tachycardic (120 beats/min) and hypotensive (94/60 mm Hg from a baseline of

FIGURE 1 **Echocardiographic and Chest X-Ray Findings From Patient 1**



(Top) Subxiphoid views of the pericardial effusion and mitral inflow velocity at 6 months before, at the time of, and immediately after pericardiocentesis. The asterisk indicates maximum mitral inflow velocity, and the open circle indicates minimum mitral inflow velocity. **(A and B)** Growth of the effusion with time. **(B, C, E, F)** Complete drainage of the effusion and resolution of the variation in mitral inflow after pericardiocentesis. **(Bottom)** Chest x-rays taken before pericardiocentesis and at the time signs and symptoms of PDS developed. **(G, H)** Smaller cardiac silhouette and slightly worse pulmonary edema after pericardiocentesis. 2D = 2-dimensional; MV = mitral valve.

150/73 mm Hg), but his oxygen saturation was maintained at 90% to 95%. A CXR showed worsening pulmonary edema with new bilateral pleural effusions (**Figure 2H**). A portable bedside echocardiogram and an echocardiogram the next morning (Video 6) showed no reaccumulation of pericardial fluid. He was treated with supplemental oxygen and intravenous fluids. His blood pressure recovered, and the next day diuresis with intravenous furosemide was instituted. An echocardiogram showed that his LVEF had returned to 58%. He was discharged on the 11th hospital day.

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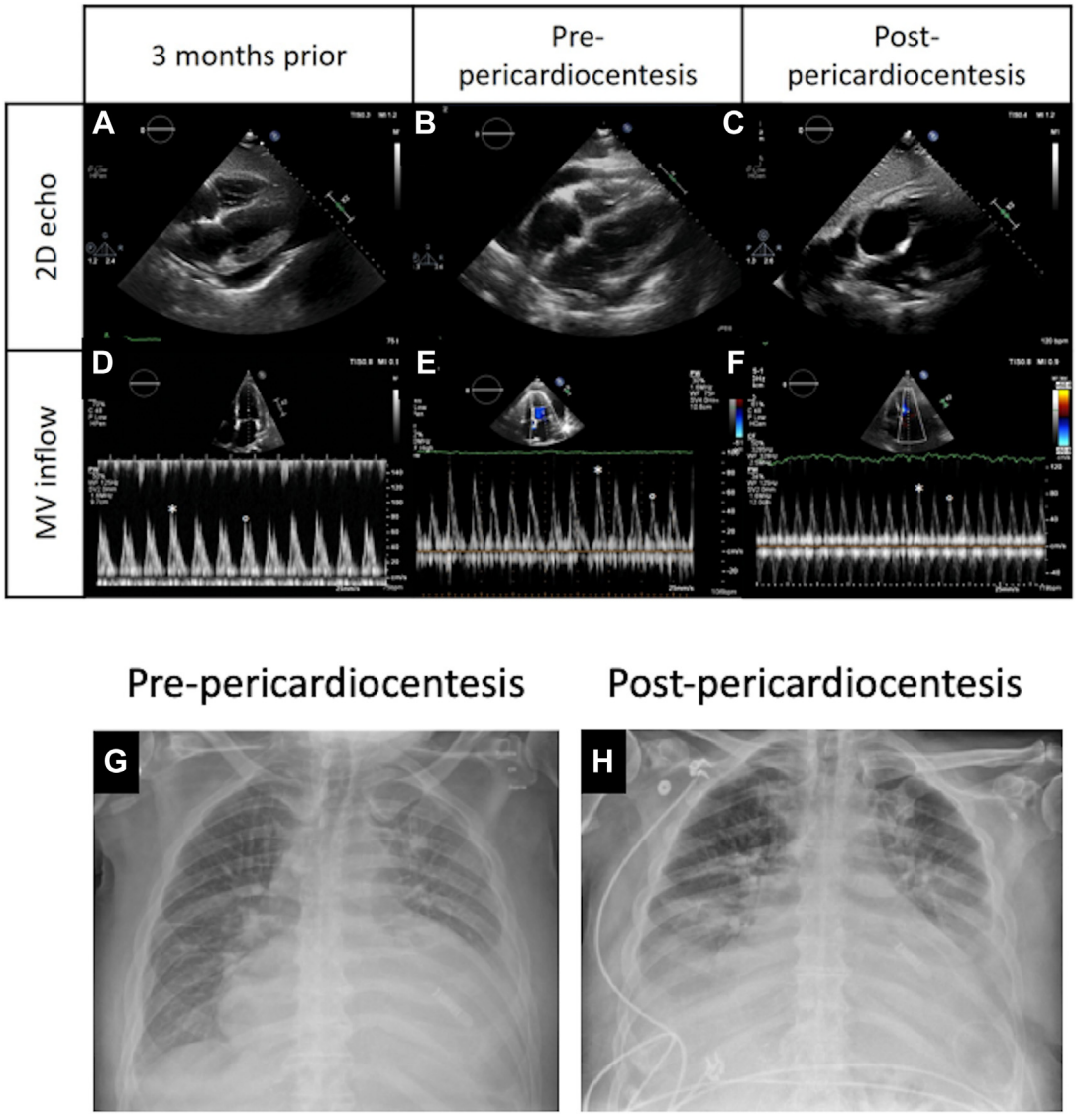
FIGURE 2 **Echocardiographic and Chest X-Ray Findings From Patient 2**



(Top) Subxiphoid views of the pericardial effusion and mitral inflow velocity at 3 months before, at the time of, and immediately after pericardiocentesis. (Images of the effusion 20 months before presentation were not available.) The asterisk indicates maximum mitral inflow velocity, and the open circle indicates minimum mitral inflow velocity. **(A, B)** Growth of the effusion with time. **(B, C, E, F)** Complete drainage of the effusion with resolution of the variation in mitral inflow after pericardiocentesis. **(Bottom)** Chest x-rays taken before pericardiocentesis and at the time signs and symptoms of PDS developed. **(G, H)** Smaller cardiac silhouette, worsening pulmonary edema, and a new large right-sided pleural effusion after pericardiocentesis. 2D = 2-dimensional; MV = mitral valve.

DISCUSSION

PDS is a potentially fatal consequence of pericardial drainage characterized by hemodynamic instability, pulmonary edema, and cardiogenic shock. Clinical studies of the condition are difficult to conduct because of its rarity. Our knowledge of the pathophysiology is entirely based on case reports. The most consistent objective finding in PDS is a transient drop in the LVEF. The LVEF is typically normal while tamponade is present, declines precipitously at the time when PDS develops, and recovers within 2 weeks (Table 2).

Here, we report 2 cases of PDS after drainage of a chronic pericardial effusion that was known to have been present for years. The cases suggest that

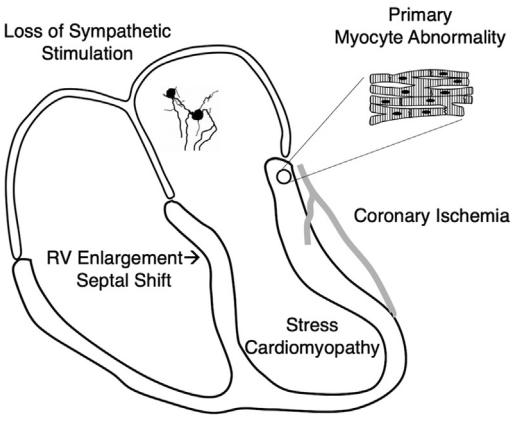
TABLE 2 Clinical Features of PDS

First Author	Etiology	Age of Effusion	Symptom Duration	Tamponade LVEF, %	PDS LVEF, %	Convalescent LVEF, %
Vandyke et al, 1983[1]	Malignant	ND	ND	ND	67	ND
Downey, 1991[7]	Post-MVA	≤3 wk	3 wk	ND	ND	ND
Wolfe, 1993[8]	Malignant	5 mo	2 wk	82	30	73
Wolfe, 1993[8]	Malignant	ND	2 wk	75	25	70
Hamaya, 1993[9]	Radiation	32 mo	ND	ND	ND	ND
Braverman, 1994[10]	Idiopathic	ND	3 wk	20	25	Normal
Uemura, 1995[11]	Myocarditis	ND	3 wk	27	Sustained dysfunction	70
Thrush, 1998[12]	Malignant	Recurrent	ND	75	35	70
Neelkanden, 1996[13]	Infectious	ND	ND	ND	ND	ND
Neelkanden, 1996[13]	ND	"Chronic"	ND	ND	ND	ND
Anguera, 1997[14]	Malignant	ND	1 mo	ND	Slightly reduced	Normal
Sunday, 1999[15]	Malignant	ND	3 d	65	30	ND (died)
Chamoun, 2003[16]	Postoperative MVR	≤2 mo	1 wk	Normal	Severe LV dysfunction	Normal
Chamoun, 2003[16]	Malignant	ND	3 wk	Preserved	Severe LV dysfunction	Normal
Geffroy, 2004[17]	Malignant?	ND	ND	ND	Normal	ND (died)
Ligero, 2006[18]	Malignant	ND	>10 d	75	25	Recovered
Bernal, 2007[19]	Idiopathic	ND	ND	60-65	30	Normal
Dosios, 2007[20]	Idiopathic	ND	10 d	Good	25	ND (died)
Sevimli, 2008[21]	Tuberculous	ND	3 mo	Normal	20	Normal
Sharaf, 2008[22]	Malignant	ND	1 wk	ND	ND	ND
Karamichalis, 2009[23]	Post-MVA	≤8 wk	ND	ND	ND	ND
Moreno-Flores, 2009[24]	Idiopathic	2 y	1 wk	>60	13	64
Sundrji, 2009[25]	Idiopathic	ND	ND	Good	Severe LV dysfunction	Near normal
Lim, 2011[26]	Hypothyroid	ND	4 mo	73	46	ND
Al Banna, 2011[27]	Tuberculous	ND	ND	62	10-15	60
Philippakis, 2013[28]	Idiopathic	ND	Days	Good	20	35
Weijers, 2013[29]	Malignant	ND	ND	Hyperdynamic	Poor	Normal
Sng, 2015[30]	Malignant	ND	1 mo	60-65	35-40	61
Pradhan et al, 2015[3]	ND	ND	ND	50	Severe LV dysfunction	Normal
Ayoub et al, 2015[31]	ND	ND	10 d	Normal	Severe LV dysfunction	Normal
Koerner, 2015[32]	Postoperative MVR	≤3 mo	ND	<50	<20	ND
Versaci, 2015[33]	Postoperative MVR	≤3 mo	ND	>60	28	Normal
Han, 2016[34]	ND	ND	ND	ND	Hyperdynamic	ND
Albeyoglu, 2016[35]	Postoperative MVR	≤3 wk	3 d	50	24	50
Takeuchi, 2016[36]	Post-SCT	1-3 mo	ND	34	ND	ND
Moon, 2017[37]	ND	ND	7 d	64	29	Recovered
Klimis et al, 2018[38]	Inflammatory	ND	2 mo	Normal	Mildly impaired	Normal
Prabhakar and Goyal, 2019[4]	ND	ND	2 wk	60-65	50	Normal
Cerrud-Rodriguez et al, 2020[39]	Idiopathic	ND	2 wk	Preserved	30	50%
Villaneuva, 2020[40]	ND	ND	2 mo	ND	38	Improved
Ricarte Bratti, 2020[41]	Postoperative MVR	≤6 wk	12 h	Normal	15	Increased
Curiale, 2021[42]	Malignant	ND	2 wk	Normal	30	Restored
Abdelmalek, 2021[43]	Uremia	ND	ND	50-54	30-34	50-54
This study, case 1	Idiopathic	45 mo	2 wk	33	Unchanged	43
This study, case 2	Idiopathic	20 mo	2 wk	45	Unchanged	58

LV = left ventricle; LVEF = left ventricular ejection fraction; MVA = motor vehicle accident; MVR = mitral valve replacement; ND = no data; PDS = pericardial decompression syndrome; SCT = stem cell transplant.

patients with a chronic pericardial effusion are at higher risk for PDS and that gradual accumulation of pericardial fluid may be a necessary requirement for PDS to develop. To our knowledge there has never been a reported case of PDS after relief of acute cardiac tamponade, for example after drainage of an iatrogenic hemopericardium from a procedural complication. In previous case reports the etiology of the effusion commonly suggested that it had accumulated gradually (eg, a malignant effusion), and the

FIGURE 3 Postulated Mechanisms of Pericardial Decompression Syndrome



See text for details. RV = right ventricle.

onset of symptoms was subacute (Table 2). In some instances the effusion was known to have been present for years. Two cases of PDS have been reported where postoperative effusions were drained 3 weeks to 3 months after the initial surgery. These may represent the minimum age of an effusion that can cause PDS.

We speculate that during tamponade there is a reversible physiologic decrease in LV contractility when filling is chronically impaired, such that the LV cannot handle the reimposition of a normal volume load from the RV once tamponade is relieved. This physiologic change likely occurs in all patients with tamponade to some extent, but in patients who develop PDS it is more pronounced. We speculate that the length of time that the pericardial effusion has been present is one factor that determines the degree to which LV contractility is affected. Because LV filling is poor during tamponade, the decrease in contractility may not be reflected as a decrease in the ejection fraction. Slow drainage of the pericardial fluid may prevent PDS by allowing more time for contractility to recover.[6]

Several theories have been advanced to explain why LV function is decreased in cases of PDS, and the pathogenesis may be different in different patients (Figure 3). One possibility is that there is some change intrinsic to the cardiac myocytes themselves that decreases their contractility when LV filling has been chronically impaired. Myocardial ischemia is another possibility; increased pericardial pressure may reduce blood flow to the extent that LV dysfunction can develop. Sudden withdrawal of exaggerated sympathetic activity after pericardiocentesis may lead to hemodynamic deterioration.[1] Some cases of PDS exhibit apical ballooning of the LV cavity, the hallmark of stress cardiomyopathy, but this is an uncommon finding.[31,39] Relief of tamponade may cause increased RV filling and RV dilatation that impinges on the LV cavity and inhibits LV filling. Septal shift has been observed by echocardiography in some cases of PDS, but this is also an infrequent feature.[5,38,44] Finally, although abnormalities of systolic function are most evident, abnormalities of diastolic function may also contribute to the pathogenesis of PDS.

CONCLUSIONS

Currently the best defense against PDS is an awareness that the condition exists so that it may be recognized and treated promptly. Patients with chronic pericardial effusions may be at higher risk for PDS and could benefit from increased vigilance.

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KEY WORDS cardiac tamponade, pericardial decompression syndrome, pericardial effusion, pericardiocentesis, pulmonary edema, systolic heart failure

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