

Constrictive Pericarditis Presenting as Unexplained Dyspnea With Recurrent Pleural Effusion

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Introduction

There are myriad causes of unexplained dyspnea and pleural effusion. When a patient presents with these symptoms, an underlying pulmonary pathology is the commonly suggested etiology. We present a patient with unexplained dyspnea and recurrent right-side pleural effusion who was evaluated with thorough physical examination and definitive imaging modalities, including right and left heart catheterization. The diagnosis was constrictive pericarditis. It is important to consider cardiac diseases, including constrictive pericarditis, in addition to underlying lung pathology, in patients with unexplained dyspnea and recurrent pleural effusion.

Case Report

A 54-year-old previously healthy man presented for evaluation of an undiagnosed pleural effusion. He initially presented to his primary physician 1 year prior, with a 1-week history of right-side “chest pain and shortness of breath.” Extensive in-patient work-up revealed mild troponin elevation and normal electrocardiogram. Transthoracic echocardiogram was normal, and chest computed tomogram for pleural effusion protocol was negative and showed no evidence of pleural or parenchymal lung disease. No etiology was identified to explain his presenting complaint, and he was discharged home.

He next presented 2 months later with dyspnea due to atrial flutter, and was treated with chemical rhythm restoration, followed by atrioventricular nodal ablation and pacemaker placement. This resolved his dyspnea, but a month

later he returned again with dyspnea and pedal edema, which responded to diuretics. Six months after his initial presentation he was seen with dyspnea again, and a repeat chest radiograph revealed a new pleural effusion that occupied greater than half of the right hemithorax. The fluid from a diagnostic and therapeutic thoracentesis was classified by the treating physician as an exudate with no known etiology. Diagnostic video-assisted thoracoscopic surgery and pleurodesis found no pleural abnormalities, and random pleural biopsies and cultures performed during video-assisted thoracoscopic surgery were not diagnostic. Though the pleurodesis did prevent recurrence of the pleural effusion, it did not resolve the patient’s dyspnea, and he was referred to our pulmonary clinic for further work-up.

Physical examination revealed a mildly obese patient who was dyspneic at rest. He was afebrile (36.5°C), with

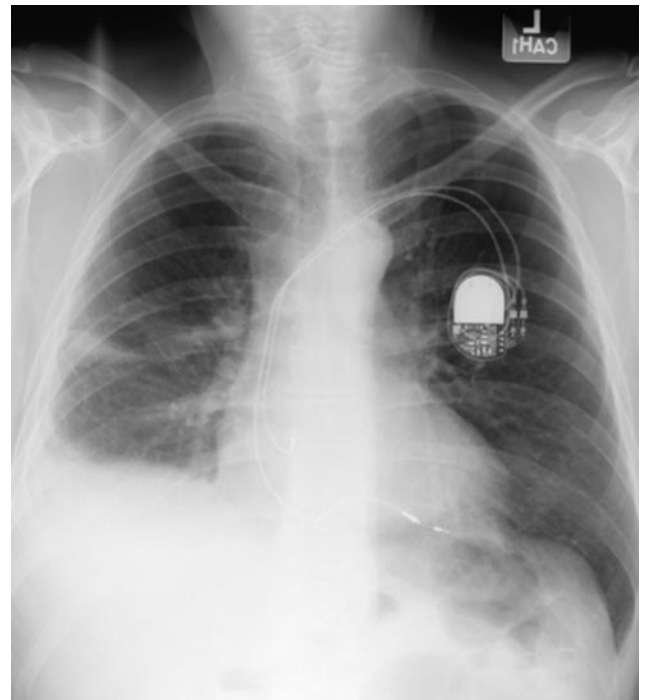


Fig. 1. Chest radiograph shows mild effusion or pleural thickening on the right, with loculated fluid in the minor fissure.

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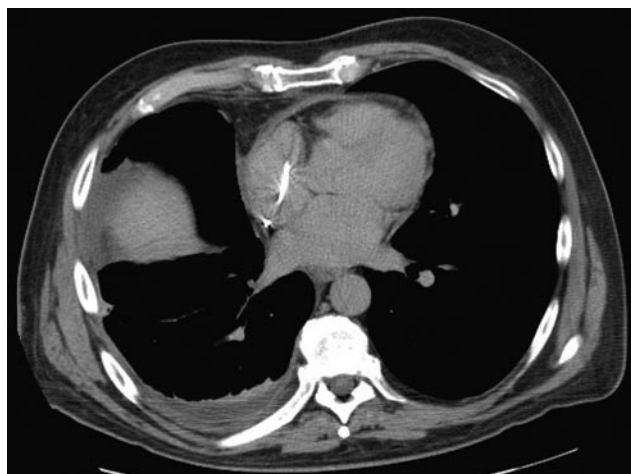


Fig. 2. Computed tomogram shows slight thickening of the pericardium and pleural thickening on the right side.

a blood pressure of 107/76 mm Hg and heart rate of 79 beats/min. Cardiovascular examination demonstrated regular rhythm without murmur or rub. Jugular venous pulsations were elevated and carotid upstrokes were brisk. Chest examination showed healing of the video-assisted thoracoscopic surgery puncture sites on the right chest, and absent breath sounds and dullness to percussion at the right base. His abdomen was remarkable for a small fluid wave and shifting dullness. His extremities were unremarkable except for pedal edema.

The pertinent laboratory results were hemoglobin 12 g/dL, leukocyte count 8.2×10^3 cells/ μ L, and erythrocyte sedimentation rate 37 mm/h. Chest radiograph (Fig. 1) revealed a small right-side effusion and loculated fluid in the minor fissure. A repeat diagnostic thoracentesis was

attempted under ultrasound guidance, but the ultrasound revealed possible pleural thickening, with no pockets of fluid collection that could be tapped, so thoracentesis was not performed. A chest computed tomogram confirmed the finding of a small right-side pleural effusion and loculated fluid in the minor fissure, and also showed post-video-assisted-thoracoscopic-surgery and pleurodesis changes, including pleural thickening and slight thickening of the pericardium (Fig. 2). The minimal pleural fluid collection was unlikely to give rise to his current debilitating dyspnea, so our further evaluation had a cardiac focus. Transthoracic echocardiogram revealed mild bi-atrial enlargement, mild thickening of the pericardium, no pericardial effusion, and normal size and function of the left ventricle. Doppler echocardiogram revealed increased flow reversal during expiration in the hepatic veins, increased respiratory variation in the mitral inflow velocity, and inferior vena cava dilation with minimal collapse, which suggest constrictive pericarditis. Diagnostic right and left heart catheterization (Table 1) confirmed constrictive pericarditis. We believe our patient had pleuropericarditis that lead to constrictive pericarditis. He underwent pericardectomy, and at surgery an abnormal fibrinous-appearing and diffuse thickening of the pericardium of about 6 mm was noted. Immediately following the pericardectomy there was a drop in the right atrial pressure, from 23 mm Hg to 12 mm Hg. His postoperative course was uneventful. At 2 month follow-up the patient had complete resolution of his dyspnea and pedal edema.

Discussion

Pleural effusion occurs in 44–50% of patients with constrictive pericarditis.^{1,2} Tomaselli and co-workers³ retro-

Table 1. Hemodynamic Data

Time	Site	Systolic Pressure (mm Hg)	Diastolic Pressure (mm Hg)	End-Diastolic Pressure (mm Hg)*	Mean Pressure (mm Hg)	A Wave (mm Hg)	V Wave (mm Hg)	Maximum dP/dT (mm Hg/s)	Heart Rate (beats/min)
09:14	RA	ND	ND	ND	21	26	24	ND	77
09:15	RV	36	6	22	ND	ND	ND	768	78
09:40	PA	36	20	ND	27	ND	ND	ND	77
09:43	LV	116	4	22	ND	ND	ND	1,488	78
09:43	PCWP	ND	ND	ND	20	24	22	ND	78
09:51	Mean LV	109	10	21	ND	ND	ND	1,056	79
09:51	Mean PCWP	ND	ND	ND	20	23	22	ND	79

* Note the equalization of the end-diastolic pressure.
 dP/dT = derivative of pressure over time (rate of pressure change)
 RA = right atrium
 ND = no data available
 RV = right ventricle
 PA = pulmonary artery
 LV = left ventricle
 PCWP = pulmonary capillary wedge pressure

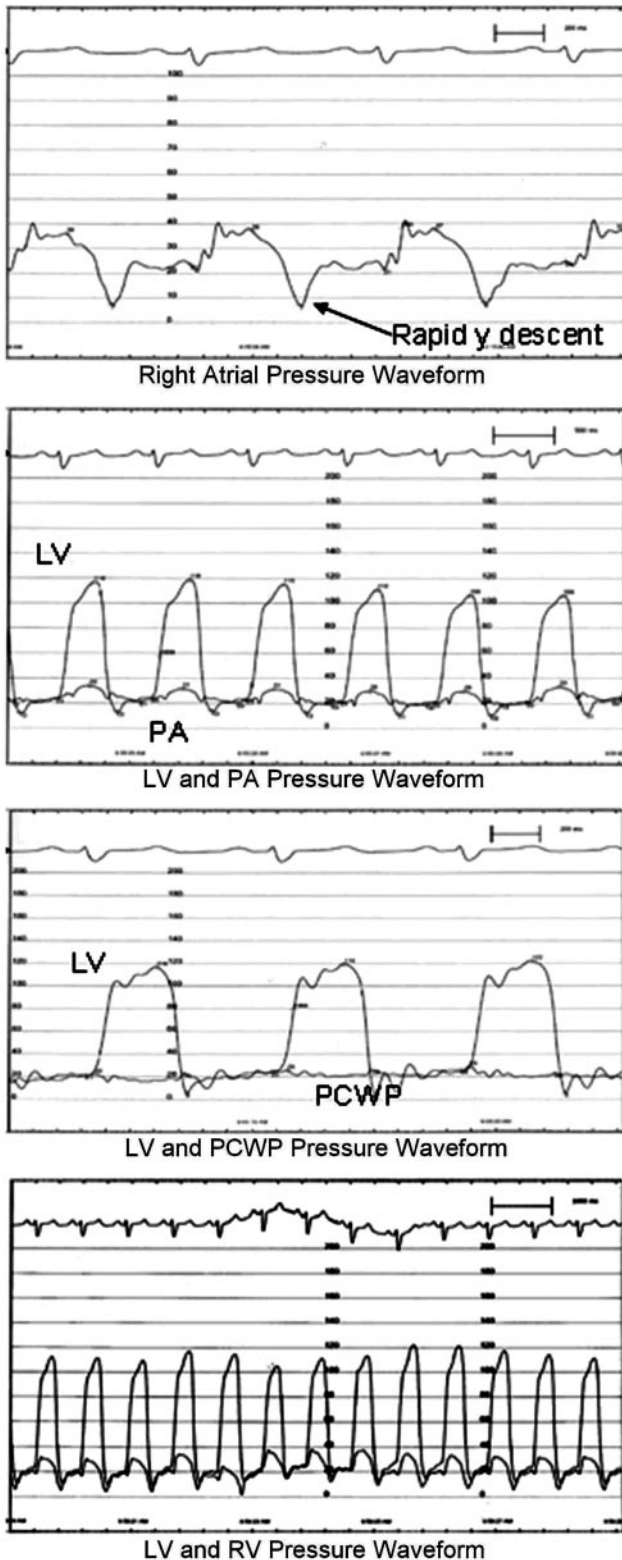


Fig. 3. Pressure waveforms show equalization of diastolic pressures. LV = left ventricle. PA = pulmonary artery. PCWP = pulmonary capillary wedge pressure.

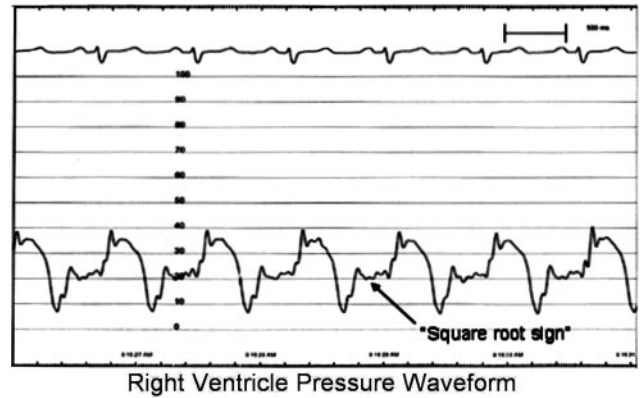


Fig. 4. "Square root" sign on a right-ventricle pressure waveform.

spectively analyzed 30 patients who presented with constrictive pericarditis, and found that 60% (18 patients) had pleural effusion. Bilateral and symmetrical effusions were found in 12 patients, and the remaining 6 had unilateral pleural fluid: 3 had right-side effusion and 3 had left-side effusion. There are several postulated mechanisms for pleural effusion in patients with constrictive pericarditis. Transudative effusion may occur due to diastolic dysfunction of the left ventricle, with resultant elevated intravascular hydrostatic pressure. That same mechanism may cause systemic venous congestion and ascites, which may result in transudative pleural effusion through diaphragmatic defects that allow fluid tracking into the pleural cavity. Exudative pleural effusions in the setting of pericarditis are primarily inflammatory in etiology, and may result from viral infections, tuberculosis, post-cardiac-injury syndrome, radiotherapy, or carcinoma.

The most common cause of constrictive pericarditis used to be tuberculosis infection. Currently, common causes of pericardial constriction are cardiac surgery, mediastinal irradiation, post-viral pericarditis, and idiopathic etiologies.⁴ Since our patient denied exposure to tuberculosis or asbestos, and had no previous cardiac intervention or therapeutic radiation that might have put him at risk, we suspect he had viral pleuropericarditis that lead to constrictive pericarditis. The subsequent atrial flutter may have developed from enlarged atrial size or myocardial irritation.

The clinical and physiologic features of constrictive pericarditis are due to restricted cardiac filling in diastole, which reduces diastolic compliance and decreases cardiac output. Presenting symptoms in constrictive pericarditis are similar to right-side congestive heart failure with increasing leg edema, dyspnea, fatigue, weight-gain, and ascites. Physical signs include a raised jugular venous pressure, which increases further during inspiration (Kussmaul sign).

Cardiac filling occurs in 2 stages; the first occurs during ventricular systole, followed by a transient drop in intrapericardial pressure (results in the "x" descent on the jug-

ular venous pressure waveform), and the second occurs at diastole, when the tricuspid valve opens. Constrictive pericarditis is characterized by rapid filling in early diastole, followed by diastasis during the later part of diastole. This leads to a rapid and prominent “y” descent (Fig. 3) due to early, rapid, resistance-free filling of the right ventricle and a “square root sign” on the right-ventricle pressure waveform (Fig. 4). During inspiration the right-ventricle systolic pressure increases, which shifts the interventricular septum toward the left ventricle, which decreases the left-ventricle cavity, the left-ventricle systolic pressure, and the systemic arterial pressure. This process is reversed during expiration: the interventricular septum shifts toward the right ventricle, which decreases right-ventricle systolic pressure and stroke volume and increases left-ventricular systolic pressure and stroke volume. This explains the changes observed in constrictive pericarditis, including the pulsus paradox, echocardiographic Doppler velocities in the hepatic veins (as explained below), tricuspid and mitral inflow velocities, and discordant changes in right-ventricle and left-ventricle systolic pressure. During diastole, pressure is equal in all the chambers of the heart (see Fig. 3), as seen in our patient. Other conditions that can cause equalization of diastolic pressures are cardiac tamponade and large atrial septal defect or single common atrium. The other physical sign of constrictive pericarditis is a pericardial knock,⁵ which is a diastolic sound that occurs 0.06–0.10 seconds after the aortic component of the second heart sound, usually before the third heart sound.

Other tests have characteristic findings for constrictive pericarditis. Echocardiography shows characteristic respiratory variation in Doppler flow velocities, including an increase in the tricuspid E velocity on inspiration (which is also present in the hepatic veins), an increase of > 25% in the mitral E velocity, and a decrease in the tricuspid and hepatic venous diastolic flow velocity on expiration.⁶ Com-

puted tomography or cardiac magnetic resonance imaging can help determine pericardial thickness and assess for pericardial calcification.⁷ Chest radiograph may show pericardial calcification in 50% of cases, but is not required for diagnosis.⁸ Similarly, 18% of patients may have normal pericardial thickness (≤ 2 mm). Our patient had pericardial thickness but no evidence of calcification.

As in our patient, the diagnosis of constrictive pericarditis can be difficult. Unexplained pleural effusion, especially in conjunction with a raised jugular venous pressure, Kussmaul sign, leg edema, and ascites, should alert the physician to the possibility of constrictive pericarditis. Our patient illustrates the importance of a thorough physical examination to help narrow the differential diagnosis, which can then be substantiated by definitive imaging modalities.

REFERENCES

1. Wychulis AR, Connolly DC, McGoan DC. Surgical treatment of pericarditis. *J Thorac Cardiovasc Surg* 1971;62(4):608-617.
2. Bertog SC, Thambidorai SK, Parakh K, Schoenhagen P, Ozduran V, Houghtaling PL, et al. Constrictive pericarditis etiology and cause-specific survival after pericardiectomy. *J Am Coll Cardiol* 2004; 43(8):1445-1452.
3. Tomaselli G, Gamsu S, Stulbarg MS. Constrictive pericarditis presenting as pleural effusion of unknown origin. *Arch Intern Med* 1989;149(1):201-203.
4. Talreja DR, Edwards WD, Danielson GK, Schaff HV, Tajik AJ, Tazelaar HD, et al. Constrictive pericarditis in 26 patients with histologically normal pericardial thickness. *Circulation* 2003;108(15): 1852-1857.
5. Tyberg TI, Goodyer AV, Langou RA. Genesis of pericardial knock in constrictive pericarditis. *Am J Cardiol* 1980;46(4):570-575.
6. Oh JK, Hatle LK, Seward JB, Danielson GK, Schaff HV, Reeder GS, Tajik AJ. Diagnostic role of Doppler echocardiography in constrictive pericarditis. *J Am Coll Cardiol* 1994;23(1):154-162.
7. Wang ZJ, Reddy GP, Gotway MB, Yeh BM, Hetts SW, Higgins CB. CT and MR imaging of pericardial disease. *Radiographics* 2003; 23(Spec No):S167-S180.
8. Sadikot RT, Fredi JL, Light RW. A 43-year-old man with a large recurrent right-sided pleural effusion. Diagnosis: constrictive pericarditis. *Chest* 2000;117(4):1191-1194.