

Short Communications

Pulmonary Venous Flow in Constrictive Pericarditis

OZ M. SHAPIRA, M.D., GILBERT P. CONNELLEY, M.D.,* GABRIEL S. ALDEA, M.D., RICHARD J. SHEMIN, M.D.

The Departments of Cardiothoracic Surgery and *Anesthesia, Boston University Medical Center, Boston, Massachusetts, USA

Summary: A 35-year-old white female patient presented with radiation-induced constrictive pericarditis. Intraoperative transesophageal echocardiography revealed a uniphasic diastolic flow pattern. This pulmonary flow pattern reverted to a normal biphasic systolic/diastolic profile immediately following radical pericardiectomy. This abnormal flow pattern was previously described as typical for restrictive cardiomyopathy. However, in this case it is most likely due to a marked decrease in left atrial compliance induced by the thick fibrous scar encasing the atria.

Key words: pulmonary venous flow, transesophageal Doppler echocardiography, constrictive pericarditis

Introduction

Pulmonary venous flow patterns as assessed by two-dimensional Doppler echocardiography has recently emerged as a useful diagnostic adjunct in a variety of cardiac disorders.¹ Characteristic flow patterns have been described for constrictive pericarditis and restrictive cardiomyopathy in an effort to distinguish between these clinically similar syndromes.^{2,3}

We describe a patient with radiation-induced constrictive pericarditis in which a uniphasic diastolic pulmonary flow was recorded by intraoperative transesophageal echocardiography (TEE). This pulmonary venous flow profile reverted to a normal flow pattern immediately following radical pericardiectomy.

Case Report

A 35-year-old white woman presented with increasing shortness of breath, fatigue, and edema. Ten years prior to her admission she underwent thymectomy with mediastinal irradiation and adjuvant chemotherapy including adriamycin for T-cell lymphoma of the thymus. A complete remission was achieved. Five years later she returned with an effusive pericarditis. This was treated with nonsteroidal anti-inflammatory drugs and diuretics with good response. She remained asymptomatic until 1 year prior to her present admission, when she began to experience increasing shortness of breath, fatigue, weight loss, ascites, and peripheral edema. A diagnosis of constrictive pericarditis with chemotherapy-induced cardiomyopathy was made and treatment with digitalis and diuretics was begun. However, because of progression of symptoms she was referred for surgery. Physical examination was remarkable for cachexia, a heart rate of 112 beats/min, blood pressure 90/50 mmHg with 25 mmHg pulsus paradoxus, and notable jugular venous congestion with a positive Kussmaul sign. The heart sounds were distant with 2/6 apical systolic murmur. The liver was palpated 2 cm below the costal margin with no ascites. Mild ankle-pitting edema was observed. Plain films of the chest revealed minimal cardiomegaly. Electrocardiogram revealed sinus tachycardia, left ventricular (LV) hypertrophy, and left anterior fascicular block.

Laboratory data were unremarkable. Preoperative transthoracic echocardiogram (TTE) showed an LV ejection fraction of 31%, a very thickened pericardium, and +1 mitral regurgitation (MR) with a small central jet. Right atrial or right ventricular collapse were not documented (Table I).

At surgery the pericardium was found to be very thickened and densely adhered to the heart and the great vessels, particularly in the upper mediastinum. Both atria, but more so on the left side, were extensively encased in a dense solid fibrous scar, severely limiting wall distensibility. Because of the extent of the disease the procedure was performed under cardiopulmonary bypass. The epicardial-pericardial plane was identified and, using sharp dissection, a radical pericardiectomy was performed. This included excision of the anterior, posterior, and diaphragmatic pericardium, leaving only a 1" strip around each phrenic nerve.

Address for reprints:

Gabriel S. Aldea, M.D.
Department of Cardiothoracic Surgery
Boston University Medical Center
88 East Newton Street
Boston, MA 02118, USA

Received: January 19, 1994

Accepted with revision: April 14, 1994

TABLE I Pre- and postpericardiectomy TTE and TEE data^a

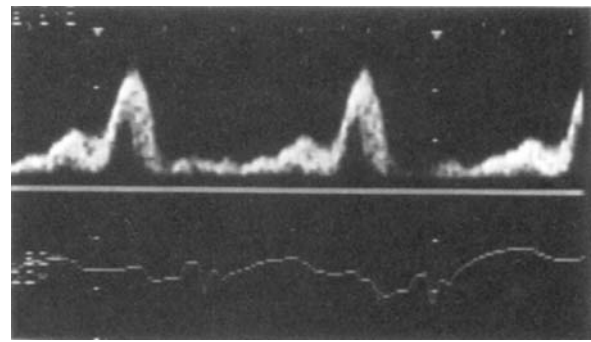
	Prepericardiectomy		Postpericardiectomy	
	TTE	TEE	TTE	TEE
Right atrial diameter (cm)	2.7	3.6	2.8	3.4
Right ventricular end-diastolic diameter (cm)	1.2	1.3	1.6	1.7
Left atrial diameter (cm)	2.8	3.6	2.8	3.3
Left ventricular end-diastolic diameter (cm)	5.2	5.0	5.3	4.9
Left ventricular end-systolic diameter (cm)	4.3	4.0	3.7	3.5
Left ventricular fractional shortening	17%	25%	30%	28.6%
Left ventricular ejection fraction	31%	40%	30%	40%
Grade of mitral regurgitation	+1	+1	+1	+1
Pulmonary venous flow velocity (m/s)				
x		0.20		0.67
y		0.53		0.64

^a The TEE recordings were performed intraoperatively pre- and postpericardiectomy. The TTE recordings were performed one month prior to the procedure and on the third postoperative day.

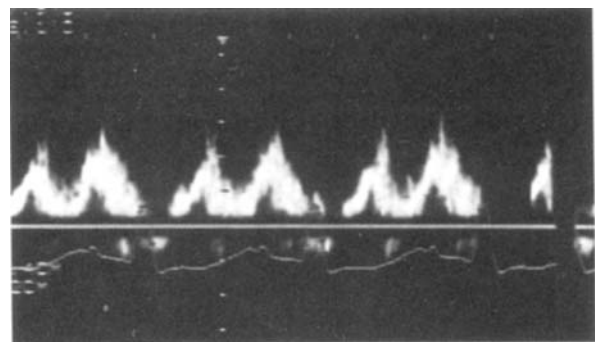
Intraoperative pre- and postpericardiectomy pulmonary venous flow measurements were obtained by sampling the left upper pulmonary vein, about 2 cm distal to its entrance into the left atrium. These TEE recordings, as well as the intraoperative hemodynamics changes and perioperative TTE data are depicted in Tables I and II and Figure 1. A significant drop in central venous, pulmonary artery, and pulmonary capillary wedge pressures associated with marked increase in cardiac output were noted. Initial uniphasic diastolic pulmonary venous flow pattern reverted to a normal systolic/diastolic biphasic waveform. There was no change in global LV ejection fraction, fractional shortening, MR grade, or jet direction and chamber sizes.

TABLE II Intraoperative pre- and postpericardiectomy hemodynamics

	Heart rate (beats/min)	Blood pressure (mmHg)	Central venous pressure (mmHg)	Pulmonary artery pressure (mmHg)	Pulmonary wedge pressure (mmHg)	Cardiac index (l/min/m ²)	Pulmonary venous flow velocity (m/s)	
							x	y
Pre	92	100/60	13	45/19	17	2.1	0.20	0.53
Post	120	110/50	3	36/15	10	3.0	0.67	0.64



(A)



(B)

FIG. 1 Pulmonary venous flow velocity recordings pre (A) and post (B) radical pericardiectomy in a patient with constrictive pericarditis. Initial uniphasic flow in diastole reverted to a normal systolic/diastolic biphasic waveform.

Discussion

The normal flow in pulmonary veins is pulsatile with biphasic systolic (x) and diastolic (y) peaks. The mechanisms underlying this flow pattern are complex and involve multiple factors including left atrial contraction and relaxation, function of the mitral valve apparatus, and LV function and compliance.¹

Rajagopalan *et al.* demonstrated an inverse relationship between the phases of pulmonary venous flow and left atrial pressure, and suggested a "suctioning effect" from the left atrium and left ventricle on the pulmonary veins as the primary mechanism for the biphasic pulsatile flow.⁴

The changes in pulmonary venous flow pattern in constrictive pericarditis and restrictive cardiomyopathy were studied, using TEE, by Schiavone *et al.* in a small series of patients.^{2,3} Constrictive pericarditis was characterized by greater systolic flow during inspiration and nearly equal systolic and diastolic flows during expiration. In contrast, in restrictive cardiomyopathy the flow was greater in diastole and was not affected by respiration.^{2,3} They attributed their results in constrictive pericarditis to the fact that the left atrium is isolated from the chest by the thickened pericardium. Thus, during inspiration the pulmonary venous pressure decreases more than the left atrial pressure, decreasing the pulmonary venous to left atrial pressure gradient, resulting in a decreased forward flow.² In restrictive cardiomyopathy, the thickening of the left atrial wall causes a marked decrease in left atrial compliance resulting in a predominantly diastolic flow.³

In our patient, pulmonary flow was uniphasic in diastole, was not affected by respiration, and reverted to a normal biphasic pattern following pericardiectomy. Several factors may explain these conflicting results. It is possible that varied degrees of pericardial scarring may affect left atrial compliance differently. In cases of severe pericardial scarring, the decrease in left atrial compliance may be similar to that observed in restrictive disorders and result in predominantly diastolic flow.

Our patient was studied intraoperatively with the chest open, whereas the patients studied by Schiavone *et al.* were breathing spontaneously. Orihashi *et al.* demonstrated that mechanical ventilation may decrease systolic flow during inspiration because of elevated airway pressure, and diastolic flow can be increased at the end of expiration.⁵ Although an open chest may alter the respiratory effects on pulmonary venous to left atrial pressure gradient, the change in pattern post pericardiectomy suggests that this is not a predominant factor.

Significant mitral regurgitation, or even a small eccentric MR jet, can affect pulmonary venous flow pattern.¹ In this case, the MR grade was trivial, with a small central jet, and it did not change postoperatively. Thus, it is unlikely to be a major factor.

Conclusion

A uniphasic diastolic pulmonary venous flow was documented by TEE in constrictive pericarditis. This pattern was previously described as typical for restrictive cardiomyopathy. Thus, the value of this added diagnostic modality to differentiate between these clinical syndromes remains limited. Further studies are necessary to better characterize the mechanisms underlying normal and pathologic pulmonary venous flow patterns.

References

1. Klein AL, Tajik AJ: Doppler assessment of pulmonary venous flow in healthy subjects and in patients with heart disease. *J Am Soc Echocardiogr* 4, 379–392 (1991)
2. Schiavone WA, Calafiore PA, Currie PJ, Lytle BW: Doppler echocardiographic demonstration of pulmonary venous flow velocity in three patients with constrictive pericarditis before and after pericardiectomy. *Am J Cardiol* 63, 145–147 (1989)
3. Schiavone WA, Calafiore PA, Salcedo EE: Transesophageal Doppler echocardiographic demonstration of pulmonary venous flow velocity in restrictive cardiomyopathy and constrictive pericarditis. *Am J Cardiol* 63, 1286–1288 (1989)
4. Rajagopalan B, Friend JA, Stallard T, Lee de J: Blood flow in the pulmonary veins: (I) Studies in dog and in man. *Cardiovasc Res* 13, 667–676 (1979)
5. Orihashi K, Goldiner PL, Oka Y: Intraoperative assessment of pulmonary vein flow. *Echocardiography* 7, 261–271 (1990)