Severe heart failure with moderately impaired left ventricular function and pericardial calcification after acute myocardial infarction

Clinical Case Portal

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Case Report

We describe the case of a patient with a post-infarction mixed constrictive-restrictive cardiomyopathy evaluated with transthoracic echocardiography and cardiac catheterization.

Patient history prior to current observation :67-year-old, caucasian male referred to our Institution on July 2006 for acute myocardial infarction. His previous medical history was remarkable for unstable angina treated with CABG (vein graft on proximal LAD) in 1988; onset of fatigue, exertional dyspnoea and recurring pleural effusion since 2004 (with no inducible myocardial ischemia at a myocardial scintigraphy), hyperuricemia and polycythemia.

Clinical findings on admission, evolution and outcome :

On admission the patient was symptomatic for chest pain and diaphoresis. ECG (fig. 1), laboratory tests and coronary angiography (fig. 2, fig. 3) confirmed an acute myocardial infarction in a patient with severe trivessel disease and vein graft occlusion. During the recovery phase he developed congestive heart failure (NYHA class IV) and intolerance to beta-blockers and ACE-inhibitors for marked hypotension.

Physical examination: Heart Rate = 115/min, regular, Blood Pressure = 90/60 mmHg, 2/6 ejective basal murmur, hypophonesis and vescicular murmur reduction at right basal pulmonary field, moderate peripheral oedema, jugular veins' distention and hepatomegaly.

Transthoracic echocardiography (fig. 4, fig. 5, fig. 6, fig. 7) showed apical aneurysm with layered mural thrombus (2,5 x 1,6 cm), thinning and diskinesia of the apical and medium septal segments, "septal bounce" (diagnostic for constriction) and global systolic dysfunction (ejection fraction = 40%). Pericardium appeared dense, immobile and thickened at postero-inferior level. Atria, inferior vena cava and hepatic veins were dilated. Doppler study during inspiration (fig. 8), showed reduced mitral flow (reduced E-wave velocity > 25%), augmented tricuspid flow (> 40%), reduced aortic velocity (< 14%) and augmented pulmonic velocity (< 16%). Hepatic vein PW-Doppler showed S > D wave velocity (fig. 9). Color M-Mode (fig. 9) showed slow intraventricular filling flow propagation. Tissue-Doppler recorded from the lateral mitral anulus (fig. 9) demonstrated low early myocardial (Em< 8 cm/sec).

Thoracic CT showed pericardial calcification which was more evident at postero-inferior level, no pericardial effusion and right postero-basal pleural effusion extended to medial level.

Cardiac catheterization revealed dip-and-plateau sign. A difference greater than 5 mm Hg between left and right ventricular end-diastolic pressure and a right ventricular systolic pressure = 61 mmHg were recorded.

Improvement of clinical conditions occurred after introduction of high dose diuretic therapy. Pericardiectomy was excluded from treatment options because of the high operative risk and the uncertain benefit obtainable from the procedure. The patient was discharged in II-III NYHA class, in therapy with low-dose beta-blockers, diuretics and oral anticoagulant therapy.

Conclusion

In this patient we can't obtain a definite diagnosis of constrictive or restrictive cardiomyopathy because of the presence of criteria for both pathophysiological patterns (1). We concluded for a mixed constrictive-restrictive cardiomyopathy. In this kind of patient the 10-year survival rate is worse than that of patients with pure constriction (50% vs 69%, respectively) (2). Furthermore, restrictive physiology and symptoms may persist even after successful surgical pericardiectomy owing to intrinsic abnormalities of ventricular compliance (3). In addition, LV systolic dysfunction is an independent predictor of reduced survival rate after pericardiectomy (4). Therefore, for the uncertain clinical benefits obtainable from pericardiectomy and the high surgical risk, intervention was not considered.

References

- 1. Kambiz Y et al. Differentiating constrictive pericarditis from restrictive cardiomyopathy. Rev Cardiovasc Med 2005;6(2):61-71.
- 2. Yamada H et al. Clinical features of mixed physiology of constriction and restriction: echocardiographic characteristics and clinical outcome. Eur J Echocardiography (2006). Article in press.
- 3. Goldstein JA. Cardiac tamponade, constrictive pericarditis and restrictive cardiomyopathy. Curr Probl Cardiol 2004;29:503-67.

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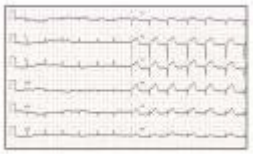
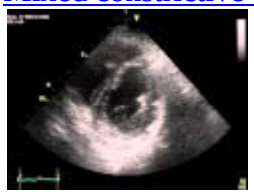
Fig. 1 :
Mixed constrictive-restrictive cardiomyopathy_ECG



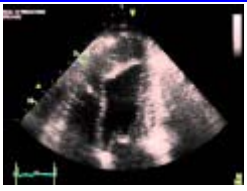
Video 1 :
Mixed constrictive-restrictive cardiomyopathy_Coronary angiography (right caudal oblique projection)

Video 2 :
Mixed constrictive-restrictive cardiomyopathy_Coronary angiography (left anterior oblique projection)

Video 3 :
Mixed constrictive-restrictive cardiomyopathy_Transthoracic parasternal midventricular short-axis view

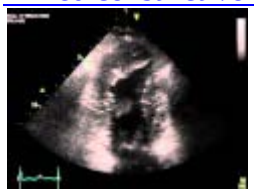


Video 4 :
Mixed constrictive-restrictive cardiomyopathy_Transthoracic apical four-chamber view



Video 5 :

Mixed constrictive-restrictive cardiomyopathy_Transthoracic apical two-chamber view



Video 6 :
Mixed constrictive-restrictive cardiomyopathy_Transthoracic apical three-chamber view

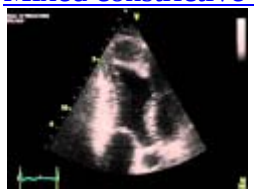


Fig. 2 :
Mixed constrictive-restrictive cardiomyopathy_Transvalvular Doppler flow

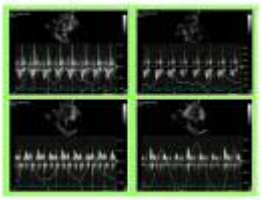


Fig. 3 :
Mixed constrictive-restrictive cardiomyopathy_Color M-Mode and Tissue Doppler Imaging

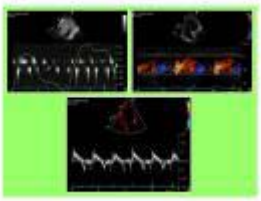


Fig. 4 :
Mixed constrictive-restrictive cardiomyopathy_Cardiac catheterism