

BRIEF REPORTS

Outcome of Pericardiectomy for Chronic Constrictive Pericarditis

Ana M. Peset, Vicens Martí, Montserrat Cardona, José Montiel, Josep Guindo, and José M. Domínguez de Rozas

Departamento de Cardiología y Cirugía Cardíaca, Hospital de la Santa Creu i de Sant Pau, Barcelona, Spain

The aim of this study was to assess early and late outcomes following treatment of chronic constrictive pericarditis by pericardiectomy. A retrospective analysis was carried out in 31 consecutive patients who underwent surgery between 1982 and 2005. The mean follow-up period was 6.7 years. In-hospital mortality was 16% (5/31 patients). The cause of death was low cardiac output syndrome in 3 patients, septic shock in 1, and hemorrhage in the remaining patient. In 6 of the 26 surviving patients, functional class did not substantially improve and, in one patient, it worsened. The cumulative actuarial survival probability was 82% at 6 months, 82% at 1-9 years, and 64% at 10 years. In conclusion, pericardiectomy improved symptomatology in the majority of patients during late follow-up. A subgroup of patients did not experience an amelioration in clinical symptoms, probably because myocardial function did not completely recover.

Key words: *Chronic pericarditis. Pericardiectomy. Surgery. Prognosis.*

Resultados de la pericardiectomía por pericarditis constrictiva crónica

El objetivo del presente estudio fue analizar los resultados de la pericardiectomía para el tratamiento de la pericarditis constrictiva crónica. Se analizó retrospectivamente a 31 pacientes consecutivos intervenidos entre 1982 y 2005. El seguimiento medio fue de 6,7 años. La mortalidad hospitalaria fue del 16% (5/31 pacientes). La causa de la muerte fue bajo gasto en 3 pacientes, shock séptico en 1 y sangrado en otro. La clase funcional no mejoró de forma sustancial en 6 de 26 pacientes que sobrevivieron y empeoró en 1 paciente. La probabilidad acumulada de supervivencia actuarial fue del 82% a los 6 meses, el 82% a los 1-9 años y el 64% a los 10 años. En conclusión, la pericardiectomía mejoró de los síntomas a la mayoría de los pacientes en el seguimiento tardío. Un subgrupo de pacientes no experimentó alivio de los síntomas clínicos probablemente porque la función cardíaca no se restableció completamente.

Palabras clave: *Pericarditis crónica. Pericardiectomía. Cirugía. Pronóstico.*

INTRODUCTION

Chronic constrictive pericarditis is a rare disease that develops because of a chronic inflammatory process that causes fibrosis and thickening of the pericardium and leads to a loss of elasticity that limits diastolic ventricular filling. Pericardiectomy has been established as the only treatment that improves cardiac dynamics. In the present study, we analyze short- and long-term results in patients operated for chronic constrictive pericarditis.

Correspondence: Dra. Ana M Peset.
Servicio de Cardiología. Hospital de la Santa Creu i de Sant Pau.
Avda. Antoni M Claret, 167. 08025 Barcelona. España.
E-mail: apeset@sanpau.is

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METHODS

We conducted a retrospective study of all patients in our center undergoing pericardiectomy for chronic constrictive pericarditis from November 1982 thru June 2005. We included patients with clinical, echocardiographic, and hemodynamic signs of pericardial constriction in whom the diagnosis was confirmed by pathologic analysis of resection tissue. In patients with idiopathic pericardial constriction, we discounted other systemic diseases such as collagenopathies and uremia. These criteria were met by 31 consecutive patients: 17 men and 14 women aged 51 (21) years. In 1982-1990 we operated on 6 patients; in 1991-1997, 10; and in 1998-2005, 15. In all patients, pericardiectomy was performed by median sternotomy without extracorporeal circulation and with perfusion equipment at the ready. During surgery, none of the patients required venous grafts as a result of coronary heart disease. Mean hospital stay was 17 days (range 5-46;

median 13). Mean follow-up was 6.7 years (<1-22; 5.3) and included all patients.

In-hospital mortality was defined as death at ≤30 days after surgery or in-hospital. Differences between continuous variables of patients who survived and those who died at ≤6 months were determined using the nonparametric Mann-Whitney U test. Qualitative variables were analyzed using χ^2 . Cumulative survival probability was determined with Kaplan-Meier survival curves. Results are expressed as percentages and mean (SD) and as median when the distribution of variables was not normal. Values of $P<.05$ were considered significant.

RESULTS

The etiology of pericarditis was idiopathic in 15 (48%) patients, tuberculous in 9 (29%), following radiotherapy in 3 (9%), neoplasia in 2 (6%), and following heart surgery in 2 (6%). Figure 1 shows the distribution of causes of chronic constrictive pericarditis in 1982-1990, 1991-1997, and 1998-2005. Postsurgical constriction presented clinically at ≥14 years after operating for valvular heart disease and presentation following radiotherapy was at ≥6 years of treatment for Hodgkin's disease.

Mean evolution of symptoms was 17 (1-120; 5) months. Table 1 presents the variables evaluated. On classifying patients by preoperative New York Heart Association (NYHA) functional class, we found 9 (29%) were in class I; 21 (67%), in II-III; and 1 (3%), in IV.

In-hospital mortality was 16% (5/31 patients). Mortality was distributed between the 3 periods as follows: 0 of 6 patients operated in 1982-1990, 3 of 10 in 1991-1997, and 2 of 15 in 1998-2005. Cause of death was low cardiac

output due to right ventricular failure with elevated ventricular filling pressure in 3 patients; septic shock in the context of heart failure in 1; and hemorrhage due to atrial tear, hypocoaguability, and hypovolemic shock in 1. Of 14 patients in preoperative NYHA functional class III-IV, 3 (21.4%) died of heart failure; of 17 patients in functional class I-II, 1 died of heart failure, and 1 of hemorrhage (2/17 patients; 11.7%). We found no relation between clinical, echocardiographic, or hemodynamic variables, time of evolution of symptoms, and mortality. Presence of radiological calcification was not associated with increased mortality: 6 patients with calcification survived and 2 died of heart failure.

Figure 2 shows pre- and postoperative functional class of 26 survivors. Functional class did not improve substantially in 6 patients despite surgery and progressively worsened in 1 patient who had undergone radiotherapy.

During follow-up, 4 patients died: 1 of late heart failure (following irradiation), 2 of neoplasia (1 patient with Hodgkin's lymphoma due to pleural mesothelioma and 1 of metastatic adenocarcinoma). The remaining patient died of stroke. Cumulative actuarial survival probability was 82% at 6 months, 82% at 1-9 years, and 64% at 10 years (Figure 3).

DISCUSSION

In the past, the most frequent etiology of chronic constrictive pericarditis was tuberculosis and in some countries, it is the cause of half of the pericardiectomies.¹ In certain centers, pericarditis following mediastinal irradiation can be the cause of surgery in one third of

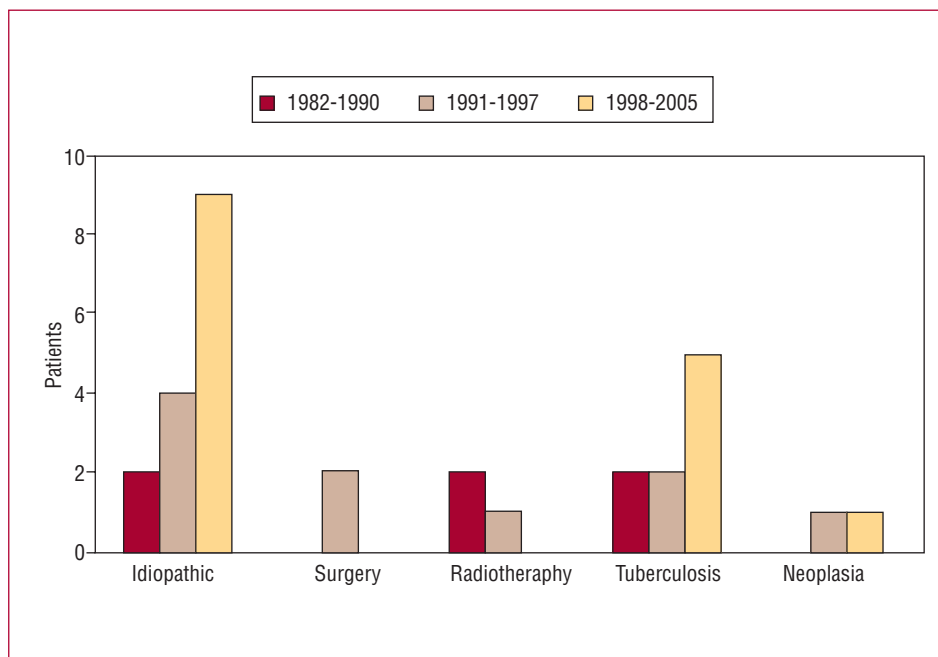


Figure 1. Temporal distribution of the cause of chronic constrictive pericarditis in patients who underwent pericardiectomy.

TABLE 1. Variables in 31 Patients With Chronic Constrictive Pericarditis^a

Preoperative clinical variables	
Age, y	51 (21) (15-81)
Gender (men)	9 (61%)
Hepatomegaly	21 (68%)
Jugular venous distension	2 (68%)
Ascites	9 (29%)
Peripheral edema	9 (29%)
Time of evolution of symptoms, mo	15 (31) (1-120)
Functional class I/II-III/IV	9/21/1
Atrial fibrillation	10 (32%)
Pericardial calcification	8 (26%)
Postoperative clinical variables	
Time of intubation, h	3 (5) (1-26)
Hospital stay, d	17 (13) (5-46)
Mean follow-up, y	6 (5) (1-22)
Biochemical variables in blood	
Sodium, mmol/L	136.8 (4.2) (127-141)
Hemoglobin, g/L	126.4 (23.3) (87-170)
AST, U/L	34.3 (30.9) (11-148)
Bilirubin, μ mol/L	25 (23) (6-92)
Albumin, g/L	36.6 (5.9) (27-47)
Urea, mmol/L	8.4 (5.1) (2-24)
Creatinine, μ mol/L	94.4 (27.6) (54-172)
Echocardiographic variables	
End-diastolic left ventricular diameter, mm	41 (6) (35-56)
End-systolic left ventricular diameter, mm	27 (6) (19-42)
Left ventricular ejection fraction, %	66 (14) (57-82)
Interventricular septal thickness, mm	9 (2) (6-12)
End-diastolic right ventricular diameter, mm	23 (3) (18-27)
Hemodynamic variables	
Pulmonary capillary wedge pressure, mm Hg	19 (4) (12-21)
Systolic pulmonary arterial pressure, mm Hg	30(6) (23-50)
Mean right atrial pressure, mm Hg	17 (2) (17-20)
Cardiac output L/min	4.3 (1.5) (2.1-5.0)
Systolic arterial pressure, mm Hg	118 (18) (95-148)
Diastolic arterial pressure, mm Hg	70 (12) (50-97)
Heart rate, beats/min	(87 (14) (60-120)

^aResults of qualitative variables are expressed in absolute values and percentages and results of continuous variables, in mean (SD) and minimum-maximum range.

patients.² In our center, the most frequent cause was idiopathic. Chronic constrictive pericarditis after heart surgery is rare and appears years after the intervention. The incidence of this complication is in the order of 0.025%-0.3%.³ In the present series, we found only 2 patients with constriction following mitral valve surgery.

In-hospital mortality was 16%. In other series, mortality is between 5.3% and 15%.¹ The principal cause of mortality in the immediate postoperative period was low cardiac output syndrome with right ventricular failure. This caused 4 of 5 deaths in our study. McCaughan et al³ found that 28% of patients presented low output syndrome following pericardiectomy, independently of the extension of pericardial resection, and that this has been related to ventricular dysfunction associated with cardiac dilatation and myocardial atrophy.^{3,4} After

pericardiectomy, most patients' symptoms improve although clinical response can be slow and take months. In late follow-up (mean 21 months), cardiac Doppler shows diastolic function is normal in only 40% of patients in the immediate postoperative period and that 43% present constrictive-restrictive diastolic pattern.⁵

In our series, the number of patients studied was small as chronic constrictive pericarditis is relatively rare and this may explain why we cannot identify factors with a bad prognosis. In-hospital mortality has been associated with advanced age, the duration of symptoms, functional class, presence of atrial fibrillation, left ventricular dysfunction, kidney failure, hyponatremia, hyperbilirubinemia, and elevated right atrium pressure.⁶⁻⁸ In terms of etiology, pericardiectomy following mediastinal irradiation entails the worst prognosis with a late survival rate of <50%. In

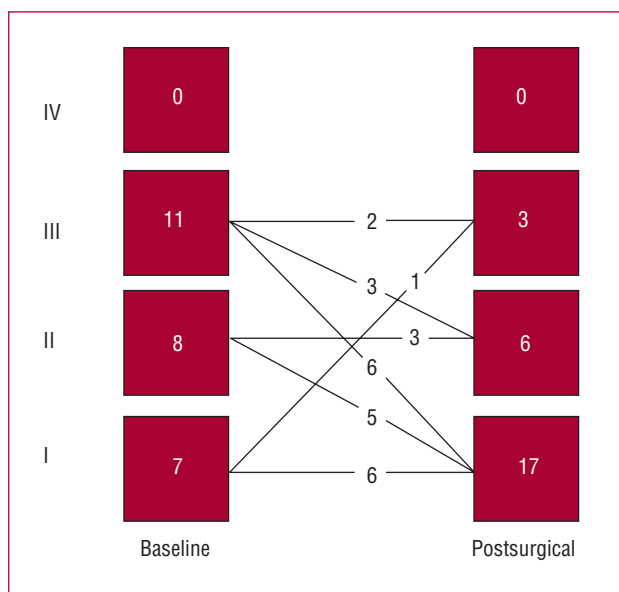


Figure 2. Functional class before and after pericardiectomy for chronic constrictive pericarditis.

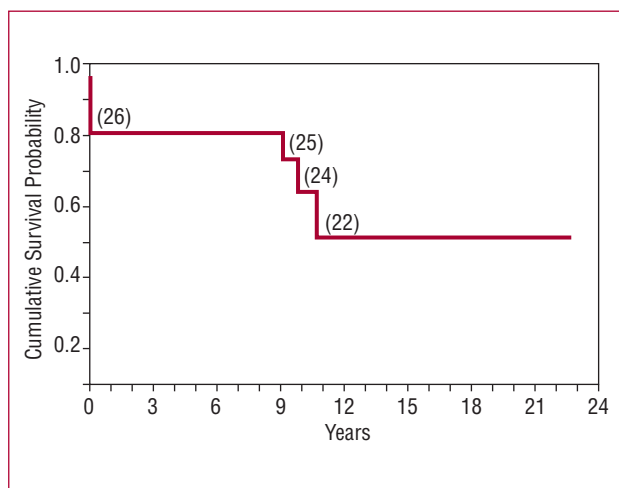


Figure 3. Cumulative survival probability of patients undergoing pericardiectomy for chronic constrictive pericarditis. The number of patients at risk in each period appears in parentheses.

our study, the only patient who died of late heart failure had had Hodgkin’s lymphoma irradiated 10 years previously. Bashi et al¹ found a reduction in mortality from 16% to 11% over 32 years that they attributed to improved perioperative care. In our series, mortality was not related with the periods analyzed.

Computerized tomography and magnetic resonance are diagnostic techniques that are especially efficient at determining the extent of constriction and evaluating the presence of calcification and the degree of fibrosis or

atrophy of the adjacent myocardium.⁹ We should remember that severe chronic constrictive pericarditis can produce minimal thickening of the pericardium whereas substantial thickening of the pericardium can cause no constriction.

In terms of the surgical approach, McCaughan et al³ reported postsurgical results showing no differences between left anterolateral thoracotomy and median sternotomy. Although the former permits good exposure and frees the left ventricle, we prefer median sternotomy because it enables us to resection all the anterolateral parietal pericardium between both phrenic nerves, facilitates the excision or epicardiolysis of the visceral pericardium in the thinner walls with less right atrium and venas cavae pressure, and permits us to explore the pleural cavities. In the areas in which epicardial resection is not possible because it is adherent or due to bleeding, we make linear cuts or extract strips to permit diastolic dilatation of the cavities.

To conclude, pericardiectomy improves or alleviates symptoms in most patients with chronic constrictive pericarditis in late follow-up. The appearance of postsurgical low output syndrome is the principal cause of the relatively high in-hospital mortality. During follow-up, a subgroup of patients presents no obvious clinical improvement due to completely recovery of diastolic cardiac function, perhaps because of adjacent fibrotic myocardial alterations. Identification of patients with fibrosis and/or myocardial atrophy can help reduce morbidity and mortality.

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