



MID-TERM OUTCOME OF SURGERY FOR CHRONIC CONSTRUCTIVE PERICARDITIS: A SINGLE CENTER EXPERIENCE

Pankaj Garg¹, Amber Malhotra², Pranav Sharma³, Ketav Lakhia⁴, Komal Shah⁵, Sanjay Patel⁶

¹Assistant Professor, ²Professor, ³Associate Professor, ⁴M.Ch. resident, Department of Cardiovascular Thoracic Surgery, ⁵Research Officer, Research Department, ⁶Research Fellow, Research department, U. N. Mehta Institute of Cardiology and Research Center (Affiliated to B. J. Medical College), Civil Hospital Campus, Asarwa, Ahmedabad 380016, India.

Corresponding Author:- **Pankaj Garg**
E-mail: pnkjgarg@yahoo.com

Article Info

Received 15/03/2015
Revised 27/03/2015
Accepted 02/04/2015

Key words: Cardiac surgery, Coronary angiography, Diaphragmatic and Heart Association

ABSTRACT

We reviewed the records of 45 patients (mean age, 24.35 ± 20.46 yr; range, 21–84 yr) with a diagnosis of constrictive pericarditis who had undergone pericardiectomy from 1994 through 2006. Preoperatively, 5 of the patients (4.4%) were in New York Heart Association (NYHA) functional class I, 38 (44.4%) in class II, 39 (48.9%) in class III, and 4 (2.2%) in class IV. Pericardial calcification was detected in 14% of plain chest radiographs. Constrictive pericarditis was caused by tuberculosis in 32.6%, and chronic renal failure in 2.3%. The cause was idiopathic in 67.4% of the patients. Low-output state was the most common postoperative problem (9.8%). The mean follow-up period was 24 ± 20 months (range, 3–60 month). At three months follow up all our survived patients (n=85) were either in NYHA class I (90.6%) or in class II (9.4%). The overall mortality rate was 1.2%. One patient with tuberculosis died of sepsis early after surgery, and 1 died during follow-up probably due to arrhythmia. Our results show that pericardiectomy remains an effective procedure in the treatment of constrictive pericarditis. Tuberculosis is still an important cause of constrictive pericarditis in India, despite vaccination and use of antitubercular drugs.

INTRODUCTION

Chronic constrictive pericarditis (CCP) is an uncommon cause of heart failure. Earlier, idiopathic inflammation and tuberculosis (TB) constituted the most common causes of CCP [1]. In last two decades, radiation therapy and previous cardiac surgery have become the most important cause of CCP in the developed countries; still, TB accounts for most important cause in developing nations [2].

This retrospective study was conducted to review our single centre experience of surgery for CCP over the past 5 years, with a view to identify the important etiological factors.

PATIENTS AND METHODS

All patients who had undergone pericardiectomy at our institute from January 2010 to December 2014 were

reviewed. The study was approved by our institutional ethics committee. Retrospective review of hospital records was done for demographic and operative details of the patients, postoperative morbidity and outcome. Follow-up data was reviewed for functional class of the patients, complications and re-intervention. Perioperative death was defined as death within 30 days of the operation or during the same hospital admission.

There were 86 patients, including 50 men (58.1%), with a mean age of 24.35 ± 20.46 years (range 1–64 yr). The diagnosis of CCP was suspected on clinical presentation and transthoracic echocardiographic (TTE) study and confirmed by contrast enhanced computed tomographic (CECT) of the chest. Coronary angiography was performed in all the patients >40 years of age to rule



out coronary artery disease as per our institutional protocol. Cardiac catheterisation was done only if there was a diagnostic dilemma. During the procedure, excised pericardium and any fluid was sent for aerobic and anaerobic cultures and histopathologic studies for evaluation of the causal factors. The diagnosis of TB was confirmed on the basis of clinical findings in combination with histopathologic features, including the presence of acid-fast bacilli in Ziel-Nelson staining, typical granuloma and caseous necrosis. If required, polymerase chain reaction (PCR) test of the pericardial fluid or tissue was done for evidence of mycobacterium tuberculosis.

Eight patients were admitted with a diagnosis of TB, 2 had chronic renal failure, 5 had chronic obstructive pulmonary disease, 3 had diabetes and 5 had hypertension. The preoperative characteristics of the patients are shown in Table 1.

Dyspnoea on exertion (DOE) was the most common presenting symptom. Most of the patients had raised jugular venous pressure while pulsus paradoxus was present in only 5 patients. Four patients had anasarca with tamponade. Four patients had atrial fibrillation and 59 patients had pleural effusion at the time of presentation. Twelve patients had pericardial calcification on plain chest radiography. The diaphragmatic, apical, and left borders of the heart were the most common sites of calcification. Two of the 12 patients had egg shell type of pericardial calcification. As a protocol, CECT chest was performed in all the patients to confirm the diagnosis of CCP, to look for pericardial thickness, calcification and necrosis and to assess the condition of both the lungs. Average pericardial thickness was 7-8 mm with maximum thickness of 15mm in 5 patients. In four patients, preoperative CECT was not done as these patients were operated in emergency with the diagnosis of recurrent massive pericardial effusion with tamponade and intraoperatively, thick visceral pericardium was seen encasing the heart. The preoperative TTE and CECT findings of the patients are presented in Tables 2.

There was one early postoperative death. The surviving patients were followed every 3-6 monthly for 3 to 57 months (median, 30 months). Six month follow-up was completed for 82 (96.5%) of the patients.

Surgical Technique

All the patients were operated by midline sternotomy. In all the patients, our preference was surgery without cardiopulmonary bypass (CPB). The primary surgical goal was total pericardiectomy i.e. resection of the anterior pericardium between the two phrenic nerves, part of pericardium over the diaphragm and the pericardium over the great arteries and both atria. Whenever, pericardial resection was performed beyond the phrenic nerves, phrenic nerve was saved as a pediculated tissue. None of the patient required sacrifice of the phrenic nerve. Attempts were always made to decorticate the white, fibrotic and thickened layer of the epicardium over the ventricles. Five

patients could undergo only partial pericardiectomy due to high risk of coronary artery or myocardial damage, or severe bleeding. In 3 patients, the pericardium could be resected only in patches due to heavy calcification penetrating the myocardium at places; therefore, some islands of epicardium and pericardium were left intact with multiple turtle shell incisions.

Five patients were operated on CPB due to concomitant procedure (ostium secundum atrial septal defect (OS-ASD) closure in 1 patient and coronary artery bypass grafting in one patient), marked hemodynamic instability during manipulation (1 patient), and surgical bleeding from inferior vena cava (IVC) (2 patients).

Statistical Analysis

All of the continuous variables are expressed as mean \pm SD and the categorical variables as percentages. The χ^2 and Student t tests were performed as appropriate. The Wilcoxon signed rank test was used to compare the New York Heart Association (NYHA) functional classes of patients preoperatively and postoperatively. A P value <0.05 was considered statistically significant.

RESULTS

The overall mortality rate in our series was 2.3% (2/86). One early death (1.2%) was that of a 62 year old patient, who presented in NYHA class IV and had sepsis at the time of presentation. He died on 5th postoperative day due to multi-organ failure. There was 1 late death (1.2%) three months after surgery. Patient had sudden death at home probably due to arrhythmia.

The surgical approach was through midline sternotomy in all the patients. CPB was performed in only 5 patients (5.8%). Severe hemodynamic instability in 1 patient (1.2%), concomitant cardiac procedure in 2 patients (2.3%), and iatrogenic IVC injury in 2 patients (2.3%) were the primary reasons for CPB use. An interesting finding was that 4 patients (4.6%) in our series presented with massive pericardial effusion with tamponade and anasarca. All four patients were operated in emergency due to recurrent massive pericardial effusion with tamponade despite pericardial tapping. Intraoperatively, there was large straw coloured pericardial collection. Parietal pericardium was of normal thickness while 3-4mm thick non-calcified visceral pericardium was encasing the heart. Five of the operated patients were in pediatric age group (2 patients 1 year old, 1 patient 3 years old, 1 patient 5 years old and 1 patient was 10 years old). Four of these patients had history of treatment for staphylococcal purulent pericarditis.

Total 20 (23%) patients developed early postoperative complication (Table 3). The most common complication was low-output syndrome (17 patients). The mean volume of postoperative mediastinal bleeding was 120 ± 150 mL (range, 50–475 mL). None of the patient was re-explored. The mean number of postoperative blood transfusions was 1.2 ± 0.7 units (range, 0–3 units) while mean no of fresh frozen plasma transfusion were 3.0 ± 1.6 .



Table 3 shows the early post-pericardiectomy complications.

The functional status of the surviving patients improved after pericardiectomy. Eighty one (94.2%) patients were in NYHA class II - IV before the operation, but 3 months postoperatively, all 85 surviving patients were either in class I (90.6%) or II (9.4%). Most common histopathological finding was chronic nonspecific

inflammation (70/86; 81.4%). In these 70 patients, PCR confirmed the TB in 12 tissue specimens or fluid. Sixteen patients (18.6%) had histopathologic features suggestive of tuberculosis. Therefore, TB was the most common known cause of CCP in our patient population with total of 32.6% patients.

Table 1. Preoperative Characteristics of the 86 Patients

Variable	No (%)
Mean age (years)	24.35±20.46
Range	1-64
Male	50(58.1%)
Symptom at presentation	
DOE	8 (9.3)
DOE with pedal edema	36 (41.8)
DOE with ascites	10 (11.6)
DOE with ascites and pedal edema	28 (32.6)
DOE with anasarca	4 (4.7)
NYHA Class	
I	5 (5.8)
II	38 (44.2)
III	39 (45.3)
IV	4 (4.7)
Co-morbidities	
Pulmonary kochs	8 (9.3)
Chronic renal failure	2 (2.3)
Chronic obstructive pulmonary disease	5 (5.8)
Diabetes	3 (3.5)
Hypertension	5 (5.8)
Coronary artery disease	1 (1.2)
Clinical findings	
Ascites	38 (44.2)
Pedal edema	64 (74.4)
Anasarca	4 (4.7)
Pulssus paradoxus	5 (5.8)
Raised jugular venous pulse	65 (75.6)
Pericardial rub	12 (14)
Chest radiograph findings	
Calcification	12 (14)
Pleural effusion	59 (68.6)
Cardiomegaly	8 (9.3)
Electrocardiogram findings	
Sinus rhythm	80 (93)
Atrial fibrillation	4 (4.7)
Bundle branch block pattern	2 (2.3)

DOE- dyspnoea on exertion, NYHA- New York Heart Association

Table 2. Pre-operative echocardiographic and contrast enhanced computed tomographic (CECT) findings

Variable	No (%)
TTE findings	
Mild to moderate pericardial effusion	8 (9.3)
Massive pericardial effusion	4 (4.7)



Thickened pericardium	63 (73.2)
Mild tricuspid regurgitation	5 (5.8)
Mean preoperative LVEF(%) (range)	51 ± 5.5 (40-65)
Mean pulmonary artery pressure (mmHg) (range)	40 ± 5.8 (30-55)
OS-ASD	2 (2.3)
Mild mitral regurgitation	7 (8.1)
CECT findings	
Pericardial thickening >15mm	5 (5.8)
Pericardial calcification	32 (37.2)
Pleural effusion	68 (79)
Enlarged mediastinal/ tracheobronchial lymphnodes	38 (44.2)
Pulmonary fibrosis	16 (18.6)
Pericardial effusion	18 (21)

TTE- transthoracic echocardiography, LVEF- left ventricle ejection fraction, OS-ASD- ostium secundum atrial septal defect, CECT- contrast enhanced computed tomography.

Table 3. Post-operative Complication

Complications	No (%)
Low output syndrome	17 (19.8)
Pleural effusion	4 (4.6)
Acute renal failure	3 (3.5)
Respiratory failure	2 (2.3)
Sepsis	1 (1.2)
Prolonged intubation	1 (1.2)
Wound complications	1 (1.2)

DISCUSSION

Chronic constrictive pericarditis defined as the chronic fibrous thickening of the pericardium leads to abnormal diastolic filling. Mostly, the pericardial thickening is more than 3mm by the time patient becomes symptomatic. The course of disease is usually slow and symptoms are nonspecific; consequently, in many cases the symptoms may be present for more than 12 months before the diagnosis is made.^[2] The diagnosis of CCP can be challenging but, it should always be kept as a differential in any patient presenting with heart failure with preserved ventricular function. Although, diastolic pressures of both the ventricles are equalized, predominant symptoms are of right heart failure.^[2,3]

The overall incidence of pericardial calcification detected on chest radiographs range between 5% and 27%^[4] but, it may be as high as 44% in patients with tubercular pericarditis.^[5] In our series, 14% patients had pericardial calcification while in patients with TB pericarditis,

calcification was seen in 30%. In our study, 75.5% patients had elevated jugular venous pressure in contrast to 10% study by ghavidel et al.^[6] This contrast might be due difference in reporting in their series as suggested by authors.

Pericardiectomy is the accepted treatment for CCP. Various studies have shown variable hemodynamic outcome of surgery ranging from no benefit to complete recovery [3, 7-11]. The variation in results may be due to

difference in surgical procedure or incomplete pericardial resection. Senni et al [3] reported the persistence of some degree of left ventricular diastolic dysfunction in approximately 40% of the patients during long term follow up, even after total pericardiectomy. The authors proposed that this might be due to abnormal ventricular compliance after incomplete pericardiectomy. De Valeria et al [9] found myocardial atrophy and fibrosis at autopsy in CCP patients. They suggested that this might be responsible for some degree of restrictive abnormality of the ventricles after pericardiectomy.

Low-output syndrome during early postoperative period was the most common problem in our series. This low-output state after pericardiectomy might have been due to incomplete pericardiectomy, postoperative transient interstitial edema or changes in cardiac architecture after pericardiectomy as suggested by Bozbuga et al [5]. The probable pathophysiology is that long periods of myocardial compression may lead to the remodeling of the ventricles and weakening of the myocardium especially in patients with longstanding symptomatic pericardial constriction. However, this low output state gradually improves in most of the patients as shown by Omoto and colleagues [12].

All our patients were operated on through median sternotomy, which allows excellent access and a better possibility of complete resection. CPB was used in 5 patients only as our preferred technique was pericardiectomy without CPB. However, we believe that



total pericardiectomy using CPB is better than partial resection of the pericardium.

Our results showed considerable improvement in NYHA functional status after pericardiectomy. All survivors in our series were in NYHA class I or II postoperatively (P <0.001). No patients had significant postoperative mitral regurgitation on TTE. This is contrast to results of Buckingham et al [13]. In their study, they reported significant mitral insufficiency after pericardiectomy as a result of papillary muscle elongation.

The overall and in-hospital mortality rates in the present study were 1.2% and 1.2%, respectively. Previously reported data showed in-hospital mortality rates ranging from 4.9% to 16%. [9, 13, 14, 16–19] The known predictors for post-pericardiectomy death include advanced age, atrial fibrillation, concomitant severe TR, postoperative inotropic support, high pulmonary artery pressure, radiation history, renal failure, low left ventricular ejection fraction, and incomplete pericardectomy. This contrast in our results may be because most of our patients did not have high risk factors. [5, 14 -16]

Tubercular pericarditis is found in approximately 1% of all autopsied cases of TB and in 1% to 2% of patients with pulmonary TB [5, 20]. Tuberculosis has been reported to account for 0.7-6.1% cases of CCP in developed countries [20, 21, 2]. In contrast, 38% to 83% of the cases of constrictive pericarditis are still caused by TB in developing countries [9, 14, 22]. In our series, TB accounted for 32.8% cases suggesting that TB continues to be a frequent cause of constrictive pericarditis in our country. Tubercular pericarditis has 3 clinical presentations:

pericardial effusion, constrictive pericarditis, and a combination of the two. The clinical features of TB pericarditis are highly variable, ranging from an absence of symptoms to severe constriction, and the diagnosis is frequently missed on cursory clinical examination [20].

The prevalence of idiopathic constrictive pericarditis was 67.2% in our series. In our series, there was no case of CCP due radiation therapy, malignancy or post-pericardiectomy syndrome. This was either short duration of our study or these patients may have died before the symptoms of constrictive pericarditis became apparent or due to the under diagnosis of post-cardiotomy constrictive pericarditis.

Limitations of our study are its retrospective design, small number of patients, and its relatively short duration of follow-up period. Therefore, our findings may have shortcomings and may not be conclusive.

CONCLUSION

Our results show that pericardiectomy is an effective procedure in the treatment of constrictive pericarditis in that it yields low mortality rates and excellent functional outcomes. Another finding of significance is that, despite vaccination and use of anti-TB drugs, TB is still an important cause of constrictive pericarditis in our country.

ACKNOWLEDGMENT

The authors acknowledge Ms. Himani Pandya, Mr. Pratik Shah and Mr. Ramanand Sharma for their contribution in preparation of manuscript.

REFERENCES

1. Sagrista-Sauleda J, Angel J, Sanchez A, Permanyer-Miralda G, Soler-Soler J. (2004). Effusive-constrictive pericarditis. *N Engl J Med*, 350(5), 469-75.
2. Ling LH, Oh JK, Schaff HV, Danielson GK, Mahoney DW, Seward JB, Tajik AJ. (1999). Constrictive pericarditis in the modern era: evolving clinical spectrum and impact on outcome after pericardiectomy. *Circulation*, 100(13), 1380-6.
3. Senni M, Redfield MM, Ling LH, Danielson GK, Tajik AJ, Oh JK. (1999). Left ventricular systolic and diastolic function after pericardiectomy in patients with constrictive pericarditis: Doppler echocardiographic findings and correlation with clinical status. *J Am Coll Cardiol*, 33(5), 1182-8.
4. Ling LH, Oh JK, Breen JF, Schaff HV, Danielson GK, Mahoney DW, et al. (2000). Calcific constrictive pericarditis. *Ann Intern Med*, 132(6), 444-50.
5. Bozbuga N, Erentug V, Eren E, Erdogan HB, Kirali K, Antal A, et al. (2003). Pericardiectomy for chronic constrictive tuberculous pericarditis: risks and predictors of survival. *Tex Heart Inst J*, 30(3), 180-5.
6. Alireza A. Ghavidel, Maziar Gholampour MD Majid Kyavar MD, FACC Yalda Mirmesdagh MD Mohammad-Bagher Tabatabaie MD. (2012). Constrictive Pericarditis Treated by Surgery. *Tex Heart Inst J*, 39(2), 199-205
7. Talreja DR, Edwards WD, Danielson GK, Schaff HV, Tajik AJ, Tazelaar HD, et al. (2003). Constrictive pericarditis in 26 patients with histologically normal pericardial thickness. *Circulation*, 108(15), 1852-7.
8. Harrison EC, Crawford DW, Lau FY. (1970). Sequential left ventricular function studies before and after pericardiectomy for constrictive pericarditis. Delayed resolution of residual restriction. *Am J Cardiol*, 26(3), 319-23.
9. DeValeria PA, Baumgartner WA, Casale AS, Greene PS, Cameron DE, Gardner TJ, et al. (1991). Current indications, risks, and outcome after pericardiectomy. *Ann Thorac Surg*, 52(2), 219-24.
10. Culliford AT, Lipton M, Spencer FC. (1980). Operation for chronic constrictive pericarditis: do the surgical approach and degree of pericardial resection influence the outcome significantly. *Ann Thorac Surg*, 29(2), 146-52.
11. Astudillo R, Ivert T. (1989). Late results after pericardectomy for constrictive pericarditis via left thoracotomy. *Scand J Thorac Cardiovasc Surg*, 23(2), 115-9.



12. Omoto T, Minami K, Varvaras D, Bothig D, Korfer R. (2001). Radical pericardiectomy for chronic constrictive pericarditis. *Asian Cardiovasc Thorac Ann*, 9(4), 286-90.
13. Buckingham RE Jr, Furnary AP, Weaver MT, Floten HS, Davis RF. (1994). Mitral insufficiency after pericardiectomy for constrictive pericarditis. *Ann Thorac Surg*, 58(4), 1171-4.
14. Johnson TL, Bauman WB, Josephson RA. (1993). Worsening tricuspid regurgitation following pericardiectomy for constrictive pericarditis. *Chest*, 104(1), 79-81.
15. Gongora E, Dearani JA, Orszulak TA, Schaff HV, Li Z, Sundt TM. (2008). Tricuspid regurgitation in patients undergoing pericardiectomy for constrictive pericarditis. *Ann Thorac Surg*, 85(1), 163-71.
16. Bertog SC, Thambidorai SK, Parakh K, Schoenhagen P, Ozduran V, Houghtaling PL, et al. (2004). Constrictive pericarditis: etiology and cause-specific survival after pericardiectomy. *J Am Coll Cardiol*, 43(8), 1445-52.
17. Cinar B, Enc Y, Goksel O, Cimen S, Ketenci B, Teskin O, et al. (2006). Chronic constrictive tuberculous pericarditis: risk factors and outcome of pericardiectomy. *Int J Tuberc Lung Dis*, 10(6), 701-6.
18. Peset AM, Marti V, Cardona M, Montiel J, Guindo J, Dominguez de Rozas JM. (2007). Outcome of pericardiectomy for chronic constrictive pericarditis. *Rev Esp Cardiol*, 60(10), 1097-101.
19. Mayosi BM, Burgess LJ, Doubell AF. (2005). Tuberculous pericarditis. *Circulation*, 112(23), 3608-16.
20. McCaughan BC, Schaff HV, Piehler JM, Danielson GK, Orszulak TA, Puga FJ, et al. (1985). Early and late results of pericardiectomy for constrictive pericarditis. *J Thorac Cardiovasc Surg*, 89(3), 340-50.
21. Fowler NO. (1991). Tuberculous pericarditis. *JAMA*, 266(1), 99- 103.
22. Raffa H, Mosieri J. (1990). Constrictive pericarditis in Saudi Arabia. *East Afr Med J*, 67(9), 609-13.

