Surgical Treatment of Chronic Constrictive Pericarditis, Is Tuberculosis Still a Common Cause?

Alireza A. Ghavidel*, Hossein Javadpour**, Mohammad-Bagher Tabatabaie, Ahmad Adambeig, Saeed Hosseini, Maziar Gholampour, Ramin Baghaie, Hassan Mirsadeghi.

Abstract

Background: Constrictive pericarditis (C.P) demonstrates a heterogeneous pattern and has different aetiologies depending on the geographic areas of reported pericarditis. Today in the western hemisphere radiation and previous cardiac surgery have become important causes of CP, but it seems that Tuberculosis is still a common cause of C.P in developing countries.

Method Material: We reviewed the records of 45 patients with mean age of 46.6 years (21-84 yr.) and the diagnosis of CP who underwent pericardiectomy between1994-2006. Preoperatively 4.5% were in New York Heart Association (NYHA) Class I, 45.5% in class II, 47.7% in class III & 2.3% in class IV. Pericardial calcification was seen in 21% of plain chest X-rays. The mean follow up period was 40+/-18 months (3-144 month).

Results: Postoperatively, only 15.6% of patients were in NYHA class III and the rest were in class I (18.2%) or II (66.2%) , (P<.001).The etiologic factors were Tuberculosis in 22.2%, chronic renal failure in 8.8%, post-sternotomy in 4.5% and malignancies in 4.5%. The cause of C.P was idiopathic in 60%. Low output state was the most common postoperative problem (22.3%). The overall mortality was 4.4%. There was one in-hospital death due to respiratory insufficiency in a tuberculosis patient and one patient died due to metastatic adenocarcinoma during follow up period.

Conclusion: We conclude that tuberculosis, despite vaccination programs and anti-tubercular medications is still an important cause of chronic CP at least in our area. Pericardiectomy is an effective treatment of chronic CP because it provides an important and durable improvement in symptoms and functional status with low mortality.

Key words: Constrictive Pericarditis, Pericardiectomy, Tuberculosis, Heart Failure

Introduction:

Constrictive pericarditis (CP) is an uncommon cause of heart failure; however its prompt diagnosis may secure a surgical cure. Approximately 15% of patients with acute pericarditis experience cardiac tamponade. Effusive pericarditis is found in five percent of cases of acute tamponade and only 1.2% of these patients end up with chronic CP. Tuberculosis (TB) is the etiologic factor of approximately 4% of patients with acute pericarditis, and seven percent of cardiac tamponade cases. According to the reports during past decade the incidence of Tuberculosis CP was 0.7% to 6 %.[1]

In the past, the most common causes of CP included idiopathic inflammation and tuberculosis. Over the past twenty years there have been reports of a change in spectrum of CP, as a result today in the western hemisphere radiation and previous cardiac surgery have become important causes of CP. [2]

We undertook this retrospective study to determine the aetiology of CP in our patient population over the past twelve years; in particular we were interested to find out if tuberculosis still continues to be as an important cause of CP.

Materials and Methods:

All patients who had undergone pericardiectomy at Shahid Rajaee Heart Centre from 1994 to 2006 were identified from our surgical database. Clinical and operative details were retrieved from the hospital notes. Follow-up was performed

Shahid Rajaee Heart Center, Cardiovascular Surgery Department, Tehran- Iran
* Corresponding author: Tel: +98 (21) 2392 2147, E-mail: aghavidel@rhc.ac.ir
** Alinasab Hospital, Cardiac Surgery Division, Tabriz-Iran

by the cardiac surgeon and cardiologist in the hospital clinic. Peri-operative death was defined as death within thirty days of the operation or during the same hospital admission.

There were 45 patients with a mean age of 46.6 +/- 14.9 years (range 21 to 84 years). There were 28 males (62.2%). The preoperative characteristic of the patients are depicted in table I.

Table I. *Preoperative Characteristics of patients*

	No (%)
Age (years)	Mean: 46.6+/-14 (Range: 21-84)
Male: Female	28 : 17
Clinical Presentation (Main Symptom):	
DOE*	26 (59.1)
DOE + Leg Edema	2 (4.5)
DOE + Ascitis	4 (9.3)
DOE + Ascitis + Edema	6 (13.5)
DOE + Generalized Edema	2 (4.5)
DOE + Chronic Cough	1 (2.3)
Atypical Chest Pain	2 (4.5)
Syncope	1 (2.3)
Medical History:	
Pulmonary Tuberculosis	6 (13.5)
Chronic Renal Failure	4 (9. 3)
COPD**	1 (2.3)
AVR***	1 (2.3)
MVR****	1 (2.3)
Exposure to Chemical bombs	1 (2.3)
Clinical Findings:	
Ascitis	3 (6.7)
Peripheral edema	4 (9.3)
Ascitis + Peripheral edama	7 (16.5)
Ascitis + Hepatpomegaly	12 (27.9)
Pulsus paradoxus	2 (4.7)
Pericardial rub	4 (9.3)
High JVP	4 (9.3)
Normal physical findings	7 (16.3)
Chest X-Ray Findings:	
Cardiomegaly	24 (51.2)
Pericardial calcification	9 (20.9)
Pleural effusion	4 (9.3)
Normal	8 (18.6)
Cardiac Rhythm:	
NSR #	21 (46.6)
AF ##	19 (42.2)
LBBB ###	3 (6.7)
LAH####	2 (4.5)

*Dyspnea on exertion **Chronic obstructive pulmonary disease***Aortic Valve Replacement ****Mitral Valv Replacement #Normal Sinus Rhythm ##Atrial Fibrillation ###Left Bundle Branch Block ####Left Anterior Hemi-block

Dyspnea with or without peripheral edema or ascitis was the most common complaint. Of note, six patients (13.5%) were admitted with the diagnosis of tuberculosis, four had chronic renal failure, two had history of open cardiac surgery and one patient had a history of exposure to chemical weapons. Only few patients had pericardial rub, high jugular venous pressure or pulsus paradoxus in physical examination. Seven patients had normal physical findings. Nineteen patients (42.2%) had atrial fibrillation at the time of surgery. Pericardial calcification was seen in 19 patients (20.9%) and eight cases had no remarkable abnormality in plane chest X-ray. The patients were followed for 3 to 144 months (Median: 41 months). Three and six months follow up was completed for 100% and 86.7% of patients respectively.

Hemodynamic and echocardiographic characteristics of the patients are shown in tables II & III.

Table II. *Hemodynamic and echocardiographic characteristics of the patients*

Pressure (mmHg)	Mean (Range)
LVEDP*	18.7 +/- 7.1 (11 – 26)
RVEDP**	17.2 +/- 4.4 (5 – 23)
Right Atrium	19.8 +/- 5.3 (18 – 28)
Right Ventricle	41.2 +/-10 (25 – 70)
Left Ventricle	122 +/- 21 (80 – 190)
Aorta	126 +/- 20 (90 – 180)
Pulmonary Artery	38 +/- 9.3 (14 -50)

*Left Ventricular End Diastolic Pressure ** Right Ventricular End Diastolic Pressure

Table III: *Findings of Echocardiographic Study*

Parameter		**No. of Cases**	**(%)**
Mild PE*		17	(34.8)
Moderate to Sever PE		4	(8.9)
Massive PE (Tamponade)		1	(2.3)
Thickened Pericardium		43	(95.6)
Sever MR**, Mild TR***		1	(2.3)
Sever TR		1	(2.3)
Mild to Moderate TR		3	(6.8)
Sever MS#		1	(2.3)
Sever AS## Moderate AI###		1	(2.3)
Preoperative LVEF*#	Mean: 47%+/-6.6	(Ranged: 30-60%)	
PAP*#*	Mean: 38 +/-9.2mmHg	(Ranged 14-50)	

*Pericardial Effusion, **Mitral Regurgitation, ***Tricuspid Regurgitation, #Mitral Stenosis, ##Aortic Stenosis, ###Aortic Insufficiency, *#Left Ventricular Ejection Fraction, *#*Pulmonary Artery Pressure.

Surgical Technique:

The primary surgical strategy (goal) was total pericardiectomy, including resection of anterior pericardium between the two phrenic nerves, basal aspect of the pericardium over the diaphragm, posterior part of the pericardium lying on the left and right ventricles, and also pericardium over the great arteries and both atria. Meanwhile, pericardial resection beyond the phrenic nerves was performed in patients who had thick, dense and constrictive pericardium over the pulmonary veins. In these patients the phrenic nerves were saved as a pediculated tissue. However left phrenic nerve was sacrificed in two patient because of severe calcification and adhesion of the nerve overlying pericardium In addition we tried to decorticate the constrictive white, fibrotic and thickened layer of epicaridum over the ventricles. In some patients because of inadequate exposure (in two patients

with thoracotomy approach), high risk of coronary artery or myocardial damage and sever bleeding we had to carry out partial pericardiectomy. In such cases the pericardium over the right atrium or superior and inferior vena cava were left intact. In two patients we could only resect the pericardium in patches and some islands of epicardium and pericardium were left intact.

In three patients who needed additional valve replacement, anterior wall of pericardium was resected before cannulation and resection of the remaining pericardium and epicardium was carried out on cardiopulmonary bypass (CPB). Except for the two patients who underwent partial pericardiectomy via left thoracotomy based on surgeon's preference, all others were approached via median sternotomy. The primary intention was pericardiectomy without CPB but we had to constitute CPB because of concomitant cardiac procedure (3 patients), hemodynamic instability during manipulation , severe surgical bleeding or to get better exposure.

Statistical Analysis:

All continuous variables were expressed as mean +/- standard deviation and categorical variables as percentages. Chi square and student t tests were performed as appropriate. A P-value of less than 0.05 was considered statistically significant.

Results: The overall mortality rate in this series was 4.5% (2/45). The only early mortality case was a 38 year old man who had presented in NYHA Functional Class III and had complete destruction of his lung due to pulmonary tuberculosis (2.2%). He died in 24th postoperative day because of pulmonary insufficiency. The late mortality rate was 2.3% in our study. This mortality case had presented in NYHA class II and the surgical pathology indicated that the pericardium was involved with a metastatic adenocarcinoma. Finally he died 7 months later as a result of hepatic insufficiency due to metastatic adenocarcinoma of colon.

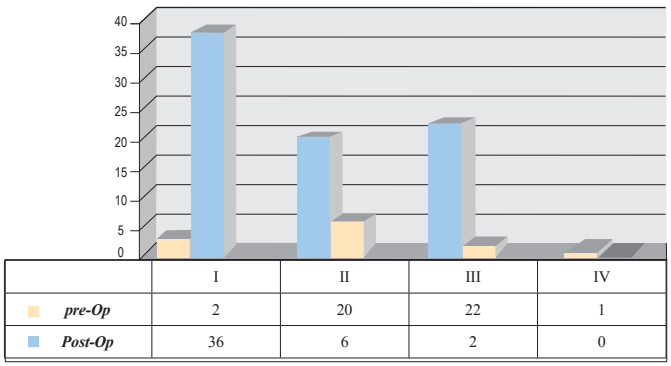
The surgical approach included sternotomy in 43 (95.4%) and thoracotomy in 2 patients.. In addition to pericardiectomy one patient underwent aortic valve replacement, one had mitral valve replacement and another patient had his tricuspid valve replaced. Interestingly 22 patients (48.9%) had constrictive pericarditis associated with pericardial effusion (4 with massive effusions). The most common early postoperative complication was low output syndrome (22.3%). Table IV shows the early post-pericardiectomy complications.

Table IV: *Early Post-operative Complications*

Complication	No. (%)
Low-output Syndrome	10 (22.3)
Surgical bleeding	1 (2.3)
Pleural effusion	4 (9.2)
Acute Renal Failure	1 (2.3)
Respiratory Failure	1 (2.3)
Long-term Intubation	2 (4.5)
Hepatic Failure	None
Wound Problems	None
Re-Pericardiectomy	None

The functional status of patients improved after pericardiectomy and while half the patients were in New York Heart Association (NYHA) class III and IV prior to the operation, only 2 patients (4.5%) were in NYHA class III post-operatively with the rest of the patients being in class I (82%) or II (13.5%) (Fig.I).

Fig. I: *Post-operative improvement of functional status based on New YorkHeart Association Classification*



Chronic non-specific inflammatory changes were the most common histopathologic findings (33/45). Eight patients had characteristic histopathologic features of tuberculosis (17.8%), one patient had pericardial involvement with Non-Hodgkin lymphoma and one case had pericardial metastatic adenocarcinoma. Interestingly two patients had normal histologic findings. Additional microbiologic examinations including PCR in two other patients indicated the presence of tuberculosis, so the overall tuberculosis CP was 22.2% (10/45) in this series. Table V shows the final diagnosis in our patients which indicate that the tuberculosis is the most known cause of CP in this patient population

TableV. *Final diagnosis in patients with constrictive pericarditis*

Final Diagnosis	Number of patients (%)
Idiopathic	27 (60)
Tuberculosis	10 (22.2)
Chronic Renal Failure	4 (8.9)
Post-pericardiotomy	2 (4.5)
Metastatic Adenocarcinoma of Colon	1 (2.2)
Non-Hodgkins Lymphoma	1 (2.2)

Discussion:

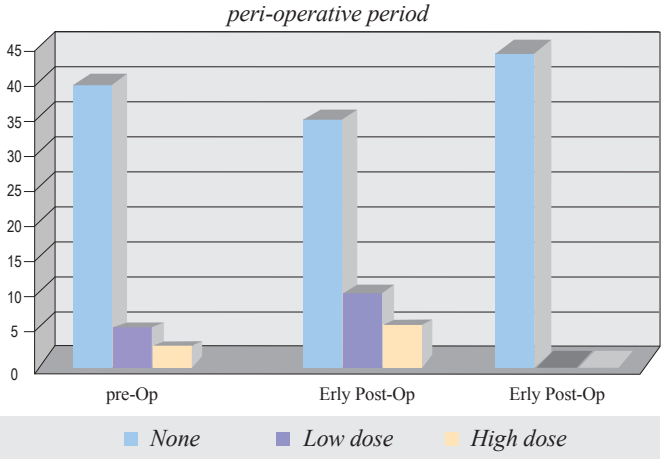
Constrictive pericarditis is an uncommon cause of a very common condition i.e. heart failure. It is defined as chronic fibrous thickening of the wall of the pericardial sac that results in abnormal diastolic filling. [1,2]The course of the disease is usually slow and the symptoms are non-specific and therefore, in many cases the symptoms may be present for twelve months or longer before a diagnosis is made.[2]

The diagnosis of CP could be a challenging process and patients with heart failure but preserved left ventricular function should be considered for this diagnosis. Although the right and left ventricular diastolic pressures are equalised in this disease, symptoms of right heart failure dominate. Traditionally the hallmark of CP has been the presence of pericardial thickening of more than 3 mm , however this sign has limited sensitivity. [2,3]

Pericardiectomy is the accepted treatment for constrictive pericarditis. Although hemodynamic results after surgery have been variable from complete recovery to not being effective at all. [3-8] These variable results may be due to different approaches to the surgical procedure and because of incomplete pericardial resection.Seni and colleagues in their study which had a long term follow up period showed that in approximately 40 % of patients some degree of left ventricular diastolic dysfunction remain even after total pericardiectomy.[3,25] They also suggested that this abnormal ventricular compliance may be due to myocardial changes in addition to incomplete pericardiectomies In a report, Dr. DeValeria and his colleagues found myocardial atrophy and fibrosis in autopsy of patients with CP that may indicate some degrees of restrictive abnormality of ventricle after pericardiectomy in these patients[6] Low output syndrome was the most common problem during early postoperative phase. As shown in figure II,

ten patients needed moderate doses of inotropic agents and three required higher doses, while in the preoperative period only 5 patients needed inotropic support, and in none of the survivors inotropic agents had to be used during the late phase of hospitalization.

Fig. II: *Comparison of Inotropic agents use during peri-operative period*



Although these changes appear important, however, they were not statistically significant (P= 0.068). In addition, we did not find any correlation between the use of inotropic agents and using CPB (P=0.08). Although this low-output state after pericardiectomy may be due to incomplete pericardiectomy , but the above mentioned changes and also post operative transient interstitial edema may have some role to play .Bozbuga and co-workers suggested that the low output syndrome can also be caused by changes in cardiac architecture. Long period of myocardial compression may contribute to remodelling of the ventricles and to greater involvement of the myocardium in patients who have undergone long periods of symptomatic pericardial constriction[9] In a study carried out by Omoto and colleagues postoperative low output state had improved gradually in majority of patients.[10]

The majority of our patients were operated through a median sternotomy incision which allows excellent access with a better possibility of complete resection. We used cardiopulmonary bypass (CPB) in 9 patients. The primary reasons for CPB use was sever hemodynamic instability during heart manipulation in 4 patients, concomitant valve replacement in three and sever iatrogenic mediastinal bleeding in two other patients. Although our preference was pericardiectomy without using CPB, but we believe that complete total pericardiectomy (if possible) using CPB is more advantageous than partial decortication of

pericardium without CPB. Thoracotomy may be preferable in patients with purulent pericarditis where sternotomy might result in greater possibility of wound infection and sternal dehiscence.[11]In our study we used the thoracotomy approach for only two patients. One of them had history of mitral valve replacement and in one other patient we avoided sternotomy approach due to cosmetic considerations.

In our patients there has been a great improvement in functional status following pericardiectomy with the majority of patients being in NYHA class I or II post-operatively (P<0.001). Except for the patient who died in early postoperative period due to respiratory failure and two other patients, other survivors achieved better functional status. One patient who had presented in FC III remained in the same functional status after partial pericardiectomy. In the other case who was in FC III shortly after pericardiectomy, postoperative echocardiographic study revealed moderate to severe tricuspid regurgitation (TR). There was no evidence of TR in preoperative studies. This patient got better with medication in mid-term follow up and he did not need to undergo further intervention. Most probably this tricuspid regurgitation related to underestimation of existed TR in preoperative TTE. Although Johnson an co-workers showed that the postop-pericardiectomy TR can be observed as a result of right ventricular dilatation,[12] however underestimation of TR severity may be due to decreased regurgitant tricuspid flow in a high pressure right atrial chamber and pressure equalization state of CP. [13]

Buckingham and colleagues using TEE suggested that mitral insufficiency may be seen after pericardiectomy as a result of papillary muscle elongation.[14] In this series we did not encounter significant postoperative mitral regurgitation.

The overall incidence of pericardial calcification seen in the chest X-Ray has been reported to be between 5% and 27% in two different studies.[15,16] Buzbuga and co-workers found pericardial calcification in 44% of Tuberculosis CP[9]. The incidence of this radiographic finding in our series was 20.9% in all patients and 30% in tuberculosis C.P. Statistically there was no correlation between pericardial calcification and tuberculosis (P=0.07)

The overall mortality rate in present study was 4.4% and in-hospital mortality was 2.2%(1/45). Reported data shows that the in-hospital mortality rate ranged from 4.9% to16%.[6,13,17,18,19,20]

The mortality rate after pericardiectomy in patients with tuberculosis CP ranges from 3% to 16%..[21] Although the known predictors for post-pericardiotomy mortality include advanced age, atrial fibrillation, concomitant sever tricuspid regurgitation, post-op inotropic support, high pulmonary artery pressure, radiation history, renal failure, low left ventricular ejection fraction and incomplete pericardiectomy, we did not find any risk factor for early mortality by univariate or multivariate analysis. This may be due to small number of patients in our study. [9,13,17,18]

Constrictive pericarditis demonstrates a heterogeneous pattern and has different aetiologies depending on the geographic areas reported. Recent reports from developed countries have indicated that 50% of cases are due to prior pericarditis, cardiac surgery and radiation therapy, while nearly 30% of cases are idiopathic [2].

Tuberculosis pericarditis is found in approximately 1% of all autopsied cases of TB and 1% to 2% of patients with pulmonary TB. [9,21] Tuberculosis was reported as a responsible cause for approximately 4% of acute pericarditis, 7% of cardiac tamponade and 6% of patients with CP.[22] Active tuberculosis was reported to be present in 6.1% of CP by McCoulgan et.al, and was even rarer in a report from the Mayo clinic(0.7%) [2,23]. In developing countries however 38% to 83% of CPs are still caused by tuberculosis. [9,18,24]

Pericardial involvement in TB patients usually occurred as a result of lymphatic extension of Mycobacterium tuberculosis or hematogenous spreading of primary TB infection.[21] TB pericarditis has three clinical presentations, pericardial effusion, constrictive pericarditis and a combination of the two. Clinical features of TB pericarditis is highly variable ranging from asymptomatic to severe constriction and the diagnosis is frequently missed on cursory clinical examination.[21] In our series we made the diagnosis of tuberculosis in 22.2% of our patients, this means despite vaccination programs and anti-tubercular medications this disease still remains an important cause of constrictive pericarditis. Although one third of our tuberculosis patients were Afghan immigrants, the incidence of 17% for tuberculosis C.P in native Iranian people(7/41) indicate the TB still to be a common cause

of C.P in our country and probably in other developing countries tuberculosis remains an epidemiologic challenge yet. We believe that the prevalence of tuberculosis CP may be higher because of underdiagnosis of TB infection or overlapping of symptoms between CP and pulmonary TB. Even in developed countries it seems that due to association of TB and HIV infection tuberculosis CP during next decade will not remain a rare disease.

The prevalence of CP with idiopathic aetiology was 57.6% and there is no significant difference between the reported rate of 46% from Cleveland Clinic or 46.6% that has been reported from Spain but interestingly the prevalence of CP after cardiotomy or following chest radiation therapy was significantly lower than these two centers (table VI). This difference may indicate that our patients with malignant tumours needing curative or palliative radiation therapy have not achieved similar outcome and survival, so probably these patients died before symptoms of CP became apparent. Also it is likely that more patients remain un-operated due to under-diagnosis of post-cardiotomy CP or perceived high surgical risk of re-operations.

Table VI: *Prevalence of etiologic factor for constrictive pericarditis in three different countries*

Etiology	Cleveland Clinic*	Barcelona	Recent Series
Idiopathic	46%	46.6%	57.6%
Postcardiotomy	37%	6.7%	4.4%
Radiation	9	13.4%	0
Malignancy	0	26.7%	4.4%
Tuberculosis	0	6.7%	22.3%
End-stage Renal disease	0	0	9.1%

* Bertog SC., J Am Coll Cardiol. 2004 21;43:1445-52
** Jamue SS., N Eng J Med. 2004;350:469-75

One of our patients had a history of exposure to the chemical weapons during the Iran-Iraq war. His histopathologic study revealed only non-specific chronic inflammatory findings. In review of literature we could not find any distinct relationship between chemical bomb exposure and CP.

We acknowledge the limitations of this study due to a retrospective design and relatively short follow-up. In conclusion pericardiectomy remains an effective procedure in treating constrictive pericarditis with low mortality and excellent functional outcome. Despite intensive vaccination and use of anti-tuberculous drugs in our country TB still remains an important cause of constrictive pericarditis in our area.

References:

1. *Sagristà-Sauleda J, Angel J, Sánchez A, Permanyer-Miralda G, Soler-Soler J. Effusive-constrictive pericarditis. N Engl J Med. 2004 Jan 29;350(5):469-75*

2. *Ling LH, Oh JK, Schaff HV, Danielson GK, Mahoney DW, Seward JB, Tajik AJ. Constrictive pericarditis in the modern era:Evolving clinical spectrum and impact on outcome after pericardiectomy. Circulation 1999;100:1380-1386.*

3. *Senni M, Redfield MM, Ling LH, et al. Left ventricular systolic and diastolic function after pericardiectomy in patients with constrictive pericarditis. J Am Coll Cardiol 1999; 33:1182– 8.*

4. *Talreja DR, Edwards WD, Danielson GK, Schaff HV, Tajik AJ, Tazelaar HD, Breen JF, Oh JK Constrictive pericarditis in 26 patients with histologically normal pericardial thickness. Circulation 2003;108:1852-7*

5. *Harrison EC, Crawford DW, Lan FYK. Sequential left ventricular function before and after pericardiectomy for constrictive pericarditis. Am J Cardiol 1970;26:319 –23.*

6. *DeValeria PA, Baumgartner WA, Casale AS, Greene PS, Cameron DE, Gardner TJ, Gott VL, Watkins L Jr, Reitz BA. Current indications, risks, and outcome after pericardiectomy.Ann Thorac Surg. 1991 Aug;52(2):219-24.*

7. *Culliford AT, Lipton M, Spencer FC. Operation for chronic constrictive pericarditis: do the surgical approach and degree of pericardial resection influence the outcome significantly? Ann Thorac Surg 1980;29:146 –52.*

8. *Astudillo R, Ivert T. Late results after pericardiectomy for constrictive pericarditis via left thoracotomy. Scand J Thorac Cardiovasc Surg 1989;23:115–9.*

9. *Bozbuga N, Erentug V, Eren E, Erdogan HB, Kirali K, Antal A, Akinci E, Yakut C. Pericardiectomy for chronic constrictive tuberculous pericarditis. Tex Heart Inst J 2003;30:180 –5.*

10. *Tadashi Omoto, Kazutomo Minami, Dimitrios Varvaras, Dietmer Bothig, Reiner Korfer.Radical Pericardiectomy for Chronic Constrictive Pericarditis Asian Cardiovasc Thorac Ann, Dec 2001; 9: 286 - 290.*

11. *Ujjwal K. Chowdhury, Diplomate NB, Ganapathy K. Subramaniam, A. Sampath Kumar, , Balram Airan, Rajvir Singh, Sachin Talwar, Sandeep Seth, DM, Pankaj K. Mishra, Kizakke K. Pradeep, MS, Siddhartha Sathia, and Panangipalli Venugopal, Pericardiectomy for Constrictive Pericarditis: A Clinical, Echocardiographic, and Hemodynamic Evaluation of Two Surgical Techniques Ann Thorac Surg 2006;81:522–30*

12. *Johnson TL, Bauman WB, Josephson RA.Worsening tricuspid regurgitation following pericardiectomy for constrictive pericarditis. Chest. 1993 Jul;104(1):79-81.*

13. *Góngora E, Dearani JA, Orszulak TA, Schaff HV, Li Z, Sundt TM 3rd. Tricuspid regurgitation in patients undergoing pericardiectomy for constrictive pericarditis.*
Ann Thorac Surg. 2008 Jan;85(1):163-70; discussion 170-1

14. *Buckingham RE Jr, Furnary AP, Weaver MT, Floten HS, Davis RF.Mitral insufficiency after pericardiectomy for constrictive pericarditis. Ann Thorac Surg. 1994 Oct;58(4):1171-4*

15. *Ling LH, Oh JK, Breen JF, Schaff HV, Danielson GK, Mahoney DW, Seward JB, Tajik AJ. Calcific constrictive pericarditis: is it still with us? Ann Intern Med. 2000 Mar 21;132(6):444-50.*

16. *Cameron J, Oesterle SN, Baldwin JC, Hancock EW.The etiologic spectrum of constrictive pericarditis. Am Heart J. 1987 Feb;113(2 Pt 1):354-60.*

17. *Bertog SC, Thambidorai SK, Parakh K, Schoenhagen P, Ozduran V, Houghtaling PL, Lytle BW, Blackstone EH, Lauer MS, Klein AL.Constrictive pericarditis: etiology and cause-specific survival after pericardiectomy.J Am Coll Cardiol. 2004 Apr 21;43(8):1445-52.*

18. *Chowdhury UK, Subramaniam GK, Kumar AS, Airan B, Singh R, Talwar S, Seth S, Mishra PK, Pradeep KK, Sathia S, Venugopal P.Pericardiectomy for constrictive pericarditis: a clinical, echocardiographic, and hemodynamic evaluation of two surgical techniques.Ann Thorac Surg. 2006 Feb;81(2):522-9.*

19.*Cinar B, Enç Y, Göksel O, Cimen S, Ketenci B, Teskin O, Kutlu H, Eren E.Chronic constrictive tuberculous pericarditis: risk factors and outcome of pericardiectomy.Int J Tuberc Lung Dis. 2006 Jun;10(6):701-6. Review*

20. *Peset AM, Martí V, Cardona M, Montiel J, Guindo J, Domínguez de Rozas JM. Outcome of pericardiectomy for chronic constrictive pericarditis Rev Esp Cardiol. 2007 Oct;60(10):1097-101.*

21. *Mayosi BM, Burgess LJ, Doubell AF. Tuberculous pericarditis. Circulation. 2005 Dec 6;112(23):3608-16.*

22. *Fowler NO.Tuberculous pericarditis.JAMA. 1991 Jul 3;266(1):99-103*

23. *McCaughan BC, Schaff HV, Piehler JM, Danielson GK, Orszulak TA, Puga FJ, Pluth JR, Connolly DC, McGoon DC. Early and late results of pericardiectomy for constrictive pericarditis. J Thorac Cardiovasc Surg.1985;89:340 –350.*

24. *Raffa H, Mosier J. Constrictive pericarditis in Saudi Arabia. East Africa Med J 1990;67:609 –13.*

25. *Senni M, Redfield MM, Ling LH, Danielson GK, Tajik AJ, Oh JK.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. 1999 Apr;33(5):1182-8.*