

## Pericardiectomy: Prompt Surgical Management of Constrictive Pericarditis

Kamran Y. Inamdar, MS, Maimaiti Aikebaier, MD, Lijunhong, MS, Abudunaibi, MD PhD, Abudureheman Mulati, MD

Cardiovascular Surgery Department, The First Affiliated Hospital of Xinjiang Medical University, Urumchi, Xin Jiang, China

### ABSTRACT

**Background:** Constrictive pericarditis is a slow progressive fibrosis of the pericardium leading to a variety of symptoms and signs over time. The disease poses a diagnostic challenge; restrictive cardiomyopathy and other syndromes associated with right-sided pressure abnormalities share similar symptoms and clinical findings. Pericardiectomy is considered the treatment of choice for constrictive pericarditis. Here we studied the effects of total radical pericardiectomy on hemodynamics in 37 patients diagnosed with constrictive pericarditis.

**Methods:** Between 2005 and 2012 thirty-seven patients, 31 males and 6 females, age range 15 to 69 years, underwent total pericardiectomy for constrictive pericarditis. Diagnosis was made on the basis of clinical, pathological and diagnostic modalities—ECG, x-rays, magnetic resonance imaging, computed tomography and echocardiogram. The surgical approach was median sternotomy and surgery was conducted without cardiopulmonary bypass.

**Results:** Postoperative outcomes showed overall improvement in the majority of patients. Hemodynamics—stroke volume, cardiac output, ejection fraction, central venous pressure—were all measurably improved postoperatively. There was no postoperative mortality.

**Conclusion:** Radical pericardiectomy is a demonstrably useful procedure for correction of hemodynamic abnormalities and improvement of overall heart function in symptomatic patients with constrictive pericarditis.

### INTRODUCTION

Constrictive pericarditis has historically posed challenges in medicine and remains a diagnostic challenge to this day. The pericardium is a fibrous sac surrounding the heart and mediastinal great vessels. The pericardial fluid between the visceral and parietal pericardium minimizes friction and energy loss during cardiac motion. Severe thickening and fibrosis or calcification of the pericardial sac causing obliteration of the pericardial space with absent or normal volume of pericardial fluid leads to constrictive pericarditis [Shabetai 1981; Brockington

1990; Spodick 1997]. The most severe form of this process is Coeur de Stein, in which the entire visceral surface of the heart is covered with an armor-like calcification. When this calcified pericardium adheres to the heart and great vessels it causes marked limitation in diastolic filling of the ventricles, leading to decreased stroke volume and cardiac output, increased right ventricular diastolic and right atrial pressures and increased CVP, ranging from 10-30 mmHg. Left untreated, this can result in hepatomegaly, ascites and peripheral edema, slowly progressing to right heart failure. Cardiac tamponade, heart failure, or in severe cases, death can result without surgical management. Here we studied the effects of total radical pericardiectomy on hemodynamics in 37 patients diagnosed with constrictive pericarditis.

### PATIENTS AND METHODS

Thirty-seven patients (31 males and 6 females, age range 15 to 69 years) who had undergone pericardiectomy for constrictive pericarditis between 2005 and 2012 were included in the study. Tuberculosis was the etiological factor in 19 (51.35%) patients; 18 (48.64%) of the patients had idiopathic constrictive pericarditis. Two of the 19 patients with tuberculosis had calcified pericardium. Diagnosis was made on the basis of clinical, pathological and diagnostic modalities—ECG, x-rays, magnetic resonance imaging, computed tomography and echocardiogram. Pericardial biopsies were done in patients with pleural effusion, which confirmed the diagnosis of tuberculosis in 3 patients. Patients with tuberculosis underwent drug therapy for 6 months before surgery. A median sternotomy was performed in all patients due to the likelihood of dense pericardial adhesions to the myocardium. All cases were done without cardiopulmonary bypass (off pump).

### Surgical Procedure

The procedures were performed under general anesthesia with tracheal intubation, and pressure measurements taken through radial artery and superior vena cava catheters. A median sternotomy was preferred over left anterior incision for maximal exposure of the heart; in order for the constricting pericardium to be removed from all surfaces of the ventricle, the heart needed to be freely mobile within the operative field. With a small round cutter minus “-” or plus “+” shaped incisions were made around the left heart avascular zone, where the pericardium was the most thickened and fibrous, and the visceral pericardium was incised to expose the myocardium. Lidocaine soaked gauze was used to alleviate

Received May 26, 2013; received in revised form September 16, 2014; accepted October 5, 2014.

Correspondence: Dr. Kamran Y. Inamdar, 387, A/2 Bhawani Peth, Chudaman Talim, Sunaina Apt, Inamdar Clinic, Pune, 411042, Maharashtra, India; 00-91-901-1243524.

## Paired Sample t-test: Pre-operative and post-operative clinical standards.

Clinical Standards	Pre-operative (x ± s)	Post-operative (x ± s)	t	P
CVP	18.97 ± 5.525	11.86 ± 3.845	10.577	.000
SV	51.75 ± 18.967	58.19 ± 14.655	-2.054	.047
CO	4.87 ± 1.879	05.295 ± 1.655	-1.305	.200
EF	59.98 ± 5.111	62.12 ± 5.487	-1.964	.057
HR	101.62 ± 18.343	88.00 ± 12.717	7.437	.000
R wave (mm)	0.84 ± 0.377	01.10 ± 0.417	-6.383	.000
R. atrium	40.59 ± 8.497	38.95 ± 6.519	1.655	.107
R. ventricle	20.08 ± 4.271	20.46 ± 2.641	-0.611	.545
RVOT	28.19 ± 4.415	26.76 ± 3.328	2.489	.018
LVDD	42.35 ± 5.308	44.19 ± 4.630	-2.637	.012
LVSD	28.46 ± 3.790	30.16 ± 3.051	-2.977	.005
Aortic sinus	29.38 ± 4.316	31.35 ± 4.008	-4.440	.000

$P < .05$  is statistically significant. CVP indicates central venous pressure; SV, stroke volume; CO, cardiac output; EF, ejection fraction; HR, heart rate; RVOT, right ventricular outflow tract; LVDD, left ventricular diastolic diameter; LVSD, left ventricular systolic diameter; (x ± s), (mean ± standard deviation).

the irritability of the myocardium when separating the pericardium. It was difficult to find the exact anatomic layer, but on incising the visceral pericardium the heart muscle was visualized with each beat. Areas with a distribution of coronary vessels were carefully incised. The apex was freed along the interface, followed by the right ventricular outflow tract and great vessels. The pericardium was then removed anteriorly from the pulmonary vein on the right to pulmonary vein on the left. Both phrenic nerves were identified, mobilized and protected. In cases where the epicardium was thickened and calcified this was often tedious, difficult to remove and resulted in diffuse bleeding. Dissociated pericardium or interrupted mattress sutures were used to stop the bleeding. In patients with ascites, the diaphragm was incised near the xiphoid and the extra fluid suctioned to avoid heart failure and lung edema. Intracardiac pressures were measured before and after the pericardiectomy.

## STATISTICAL ANALYSIS

Statistical analysis was performed using SPSS 17.0 software. Descriptive statistics were presented as mean ± standard deviation. Comparison of operative effects was performed by t-test. The tests were considered statistically significant at  $P \leq .05$ .

## RESULTS

Mean age was 36 years, with a range of 15-69 years. There were 31 (84%) males and 6 (16%) females. In 19 (51%) of

37 patients tuberculosis was the etiological factor. The cause of pericardial constriction in 18 (49%) patients was undetermined. Follow-up was between 6-12 months, with a mean follow-up of 9 months. Patients were classified using the New York Heart Association (NYHA) functional and therapeutic classification I-IV. Twenty-four patients in class III preoperatively reverted to classes I and II after surgery. Nine patients in class II preoperatively reverted to class I post-operatively, and 4 patients in class IV reverted to class II after surgery. Data analysis (Table) showed the mean ejection fraction (EF) increased from 60 to 62 post-operatively; stroke volume (SV) increased from 52 to 58 with a concomitant increase in cardiac output (CO), from 4.9 to 5.3 post-operatively. Heart rate (HR) decreased from 102 to 88 post-operatively. Central venous pressure (CVP) decreased from 19 to 12 post-operatively. Echocardiography demonstrated improvement of cardiac function after surgery. There was no postoperative mortality. Four patients in the study were lost to follow-up.

## DISCUSSION

Early descriptions of the pericardium date back to Hippocrates (460 to 377 BC) [Spodick 1970]. Jean Riolan (1649) suggested treating pericarditis with trephination—"removing bone in circular section" of the sternum. The first successful pericardiectomy was performed by Romero in 1819. The pathophysiology of constrictive pericarditis was further elucidated by Cheever in 1842. Pericardial resection for constrictive pericarditis was proposed by Weill (1895) and Delorme (1898), with pericardiectomy ultimately performed by Rehn in 1913) and Ferdinand Sauerbruch in 1925. Early surgical treatment of constrictive pericarditis in the United States was reported by Beck (1930), Churchill (1936), and Blalock (1937). Radical pericardiectomy, including excision of thickened epicardium when necessary, was advocated by Holman in 1955 [Abdel-Halim 2007].

The most common etiology of constrictive pericarditis in Western countries is idiopathic, with prior cardiac operation and mediastinal irradiation also common [Bertog 2004]. Today tuberculosis is the leading cause of constrictive pericarditis in developing countries. One report from India showed tuberculosis was the cause for 40% of patients with constrictive pericarditis [Tiruvoipati 2003]. On routine cardiac operations, closure of the pericardium can induce some degree of immediate pericardial constriction and decrease cardiac index but has little real chance of causing late constrictive pericarditis [Rao 1999]. Eighty percent of patients after heart surgery show pericardial effusion on echocardiography in the first 3 weeks, and 75% have some chance of developing tamponade [Hoit 2007]. It is preferable to avoid closure of the pericardium in patients with ventricular dysfunction, risk of tamponade, or older age, except when there is a chance of reoperation [Rao 1999].

Pericardial thickening with minimal pericardial fluid on echocardiography, CT or MRI is present in 85% of patients after operation [Goldstein 2004]. Pericardial thickening supports the diagnosis of constrictive pericarditis, but the pericardium may not be thickened in 15% to 18% of patients with

constriction. Diagnosis of constrictive pericarditis requires demonstration of right heart hemodynamics typical of constriction. The findings on right heart catheterization include decreased cardiac output, equalization of right and left diastolic pressures, and the characteristic square-root sign, with a steep “y” descent in the right and left ventricular diastolic pressure tracings [Shabetai 1995]. Detection of these findings can be augmented by a 500 ml volume challenge at the time of right heart catheterization.

Treatment for pericardial constriction is pericardiectomy [Tuna 1990; Ling 1999]. Pericardiectomy should be done before the onset of class IV symptoms to lower cardiac output and minimize postoperative mortality [McCaughan 1985]. Roughly 20% of patients with constriction have at least moderate tricuspid regurgitation, which in turn is associated with worse 5-year survival (47% vs. 87%). Significant tricuspid regurgitation in patients undergoing pericardiectomy should be a consideration for tricuspid surgery [Gongora 2008]. Rarely, the underlying cause may also require treatment in cases such as tuberculous pericarditis [Cinar 2006]. Long-term survival in selected patients without myocardial involvement can approach that of the general population [McCaughan 1985; Chowdhury 2006; Cinar 2006].

Older age, poor renal function, abnormal left ventricular systolic function, high pulmonary artery systolic pressure, low serum sodium, worsening NYHA classification, and radiation therapy as the cause of constrictive pericarditis all influence the prognosis. Pericardial calcification does not influence survival after pericardiectomy. As with many diseases that once were predominantly infectious in origin, the clinical spectrum of constrictive pericarditis has changed. Approximately 9% of patients with acute pericarditis from any cause go on to develop constrictive physiology. The true frequency is dependent on the incidence of the specific causes of pericarditis, but given that acute pericarditis is clinically diagnosed in only 1 in 1000 hospital admissions, the frequency of a diagnosis of constrictive pericarditis must be less than 1 in 10000 admissions. In the developing world, infectious etiologies remain more prominent—tuberculosis has the highest total incidence. Some studies report a male to female ratio of 3:1. No racial predilection exists for this disorder. Development of new technologies and devices such as video assisted thoracoscopy may help improve diagnostic and therapeutic options with pericardial disease in the future [Clare 2007]. The best prognosis after pericardiectomy is seen in patients with idiopathic constrictive pericarditis (88% survival at 7 years), followed by constriction due to cardiac surgery (66% survival at 7 years); the worst prognosis occurs in patients with post radiation constrictive pericarditis (27% survival at 7 years).

In our study more than 51% of patients had tuberculosis, which was the etiological factor leading to slowly progressive constrictive pericarditis. Data analysis showed that EF, SV and cardiac output increased postoperatively, indicating considerable improvement in cardiac function. Tachycardia resolved, with a drop in HR from 101 to 88, with mean HR within normal limits. The initially high CVP, secondary to the constrictive pressure of the pericardium over the right heart, decreased from 19 to 12 post-operatively. Right atrial

and right ventricular pressures showed no significant change, however, which led us to conclude that the myocardial fibers were stretched or hypertrophied due to excess work done against the slowly progressive constricted pericardium over time. Comparison of the pre- and post-operative pericardiectomy results showed that, in spite of the differences in some values, overall the condition of the patients showed significant improvement.

## CONCLUSION

All patients in our study showed correction of hemodynamic abnormalities and improvement in functional capacity after the surgery. Intensive peri-operative monitoring and management reduced the incidence of morbidity and mortality. Radical pericardiectomy is a useful and efficacious procedure for symptomatic patients with constrictive pericarditis.

## REFERENCES

- Abdel-Halim RE, Elfaqih SR. 2007. Pericardial pathology 900 years ago. A study and translations from an Arabic medical textbook. *Saudi Med J* 28:323-5.
- Bertog SC, Thambidorai SK, Parakh K, et al. 2004. Constrictive Pericarditis: etiology and cause-specific survival after pericardiectomy. *J Am Coll Cardiol* 43:1445-52.
- Brockington GM, Zebede J, Pandian NG. 1990. Constrictive pericarditis. *Cardiol Clin* 8:645-61.
- Chowdhury UK, Subramaniam GK, Kumar AS, et al. 2006. Pericardiectomy for constrictive pericarditis: a clinical echocardiographic and hemodynamic evaluation of two surgical techniques. *Ann Thorac Surg* 81:522-29.
- Cinar B, Enc Y, Goksel O, et al. 2006. Chronic constrictive tuberculous pericarditis: risk factors and outcome of pericardiectomy. *Int J Tuberc Lung Dis* 10:701-06.
- Clare GC, Troughton RW. 2007. Management of constrictive pericarditis in the 21st century. *Curr Treat Options Cardiovasc Med* 9:436-42.
- Goldstein JA. 2004. Cardiac Tamponade, constrictive pericarditis, and restrictive cardiomyopathy. *Curr Prob Cardiol* 29:503-67.
- Gongora E, Dearani JA, Orszulak TA, et al. 2008. Tricuspid Regurgitation in patients undergoing pericardiectomy for constrictive pericarditis. *Ann Thorac Surg* 85:163-70.
- Hoit BD. 2007. Pericardial disease and pericardial tamponade. *Crit Care Med* 35S:355-64.
- Ling LH, Oh JK, Schaff HV, et al. 1999. Constrictive pericarditis in the modern era: evolving clinical spectrum and impact on outcome after pericardiectomy. *Circulation* 100:1380-86.
- McCaughan BC, Schaff HV, Piehler JM, et al. 1985. Early and late results of pericardiectomy for constrictive pericarditis. *J Thorac and Cardiovasc Surg* 89:340-50.
- Rao V, Komeda M, Weisel RD, et al. 1999. Should the pericardium be closed routinely after heart operations? *Ann Thorac Surg* 67:484-88.
- Shabetai R. Constrictive Pericarditis. In: Shabetai R, ed. *The Pericardium*. New York: Grune & Stratton; 1981:211.
- Shabetai R. Pericardial Disease: etiology, pathophysiology, clinical

recognition, and treatment. In: New York NY: Churchill Livingstone; 1995:1024-35.

Spodick DH. 1970. Medical History of the pericardium. The hairy hearts of hoary heroes. *Am J Cardiol* 26:447-54.

Spodick DH. *The Pericardium*. New York: Marcel Dekker; 1997.

Tiruvoipati R, Naik R D, Loubani M, Billa G. 2003. Surgical approach for pericardiectomy: a comparative study between median sternotomy and left anterolateral thoracotomy. *Interact Cardiovasc Thorac Surg* 2:322-6.

Tuna IC, Danielson GK. 1990. Surgical management of pericardial diseases. *Cardiol Clin* 8:683-96.