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Original Research Article

Surgery for Constrictive Pericarditis: A Single Center Experience

Angui Li[1], Feng Lin[1], Fugui Ruan[1], Xiaolin Sun[1], Jiangbin Sun[1], Tianci Qian[1], Jianfei Song[2], Zhenzong Du[2], Haiyong Wang[1]*

[1]Department of Cardiovascular Surgery, Affiliated Hospital of Guilin Medical University, Guilin 541001, China
[2]Department of Cardiothoracic Surgery, the Second Affiliated Hospital of Guilin Medical University, Guilin 541199, China

***Corresponding author**
Haiyong Wang
Email: docwanghy@foxmail.com

Abstract: Constrictive pericarditis is a relatively rare disease that can result in chronic fibrous thickening of the pericardium. We performed this study to summarize the clinical experience of a series of patients with constrictive pericarditis underwent pericardiectomy. We analyzed the clinical features of 30 constrictive pericarditis patients who underwent surgery from January 2010 to June 2015 at our institution. Of the patients, 24 (80%) had tuberculous constrictive pericarditis, 6 (20%) had idiopathic pericarditis. All patients underwent partial or complete pericardiectomy by midline sternotomy. There was no in-hospital mortality rate. There were no case of recurrent constrictive pericarditis after discharge. The 1-year survival rate was 100%. There were 3 cases of perioperative drug induced liver injury. After first discharge, 2 patients were rehospitalized for heart failure. Prompt treatment of constrictive pericarditis are crucial to reduce mortality and morbidity. Pericardiectomy should be performed early after diagnosis. After surgery, inotropes, diuretics and nutrition supply are also critical to improve the prognosis.
Keywords: Surgery, Constrictive Pericarditis, Pericardium

INTRODUCTION

Constrictive pericarditis is chronic inflammation of the pericardium with thickening, scarring, and muscle contracture. Constrictive pericarditis is an uncommon cause of heart failure. Constrictive pericarditis results from inflammation and fibrosis of the pericardium, ultimately leading to heart failure by impaired diastolic ventricular filling [1]. The definitive treatment for constrictive pericarditis is pericardial stripping. Prompt treatment of constrictive pericarditis is necessary to reduce morbidity and mortality. This retrospective study was conducted to examine the clinical features of patients who have undergone pericardial resection for constrictive pericarditis.

PATIENTS AND METHODS

Clinical Features

From January 2010, through June 2015, all patients whose pericardial specimens surgically excised at affiliated hospital of Guilin Medical University were reviewed under a protocol approved by the Institutional Review Board.

The diagnosis of constrictive pericarditis was made on the basis of clinical, diagnostic, surgical and pathological criteria. Before surgery, we integrated information from clinical features and diagnostic tools: echocardiography, computed tomography (CT) or magnetic resonance imaging (MRI) to select patients for pericardiectomy. Surgical and pathological findings were subsequently reviewed to confirm the preoperative diagnosis.

Medical records were reviewed independently by two reviewer. The clinical information recorded included gender, age, relevant symptoms, physical examination findings and preoperative images, including echocardiography, CT and MRI.

Surgical Procedure

A midline sternotomy was performed in all cases. The 27 patients underwent radical pericardiectomy. Radical pericardiectomy included removal of the anterior pericardium between the phrenic nerves on the right and left, from the level of the great arteries to the diaphragmatic surface, lateral pericardium posterior to the left phrenic nerve, inferior pericardium along the diaphragmatic surface and

posterior pericardium to the atrioventricular junction sections.The remaining 3 patients had dense diaphragmatic adhere tissue that prevented complete resection of this portion of the pericardium. We did not remove these inaccessible areas, but the pericardial tissue between the two phrenic nerves was resected, and a portion of the diaphragmatic pericardium was left behind. Cardiopulmonary bypass was not used during our surgery. Operative findings included markedly thicken, adhesions and calcification of pericardium.

RESULTS

Of the 30 patients, 24 patients (80%) had tuberculosis constrictive pericarditis and 6 (20%) had idiopathic constrictive pericarditis. When classifying patients using the New York Heart Association functional class, 18 (60%) were in class II, 6 (20%) were in class III, and 6 (20 %) were in class IV. The median duration of symptoms was 5 months (range 1 to 60 months). The most common symptoms were exertional dyspnea (63%), shortness of breath (70%), lower limb edema (66%), abdominal distension (56%) and palpitation (36%). On physical examination, the most common clinical signs were increased cardiac dullness (66%), raised jugular vein distension (66%), distant heart sounds (60%), hepatomegaly (50%). Laboratory tests showed increases in serum total and direct bilirubin, alkaline phosphatase, C-reactive protein and erythrocyte sedimentation rate. Among the 24 patients with tuberculosis constrictive pericarditis, not all patients had pulmonary infection (7 patients still had tuberculosis pleuritis and 2 patients had a definite past history of pulmonary tuberculosis).Of these patients, the most prominent symptoms and signs were lower leg edema (70%), raised jugular vein distension (66%), exertional dyspnea (63%) and distant heart sounds (66%). On the electrocardiograms, nonspecific T wave changes were detected in 16 patients (53%), low-voltage was observed in 9 (30%) patients, ST segment depression was seen in 6 (18%) patients, and atrial arrhythmias or fibrillations were found in 5 (16%) patients. Preoperative imags revealed a thickened pericardium in 28(30%), calcification of pericardium in 12(30%), cardiomegaly in 27(90%), pleural effusion in 14(46%). The chest radiograms revealed changes suggestive of pulmonary tuberculosis in 10 (33%) patients.

The median duration of mechanical ventilation was 9.2± 3.3 hours. In 12 patients, blood transfusions were given during or after surgery. The average volume of perioperative transfusion was 1,180± 820mL. The median intensive care unit stay was 2.3± 1.6 days. The median hospital stay was 22.8± 4.7 days (range 7 to 32 days). There was no in-hospital mortality. The mean duration of the operation was 150± 68 minutes. Pathological changes, which included fibrosis, inflammation, calcification, caseous necrosis, and hyaline plaque formation, occurred in many but not all patients. All of the bacteria cultures were negative. There were several postsurgical complications in the surviving patients, which included pleural effusion in 10 patients, low output syndrome in 3 patients, drug-induced liver injury in 3 patients. One patient had recurrence of heart failure before discharge. For 27 patients in whom tuberculosis constrictive pericarditis was diagnosed, they all received antituberculosis agents (isoniazid, rifampicin and ethambutol daily for 6 months).

Among 30 patients, 24 (80%) presented an uneventful stable clinical condition without reoperation. After first discharge, 2 patients were rehospitalized for heart failure, at 2 and 4 weeks of follow-up. Of the 30 included patients, no patient died during follow-up after pericardiectomy. The 1-year overall survival was 100%.

DISCUSSION

Constrictive pericarditis is characterized by the encasement of the heart by rigid and pericardial tissue, leading to heart failure by impairing diastolic cardiac filling [1, 2]. Constrictive pericarditis is usually a long-term consequence of acute or chronic forms of pericarditis, trauma, cardiac surgery, thoracic radiation therapy, malignant neoplasm, acute viral pericarditis, rheumatoid arthritis, systemic lupus erythematosus, and can occur in patients with chronic renal failure who require chronic dialysis. But, in some patients the cause of constrictive pericarditis is unknown and in these patients an asymptomatic or forgotten episode of idiopathic or viral pericarditis could be the starting process. In those patients, clinical diagnosis of chronic constrictive pericarditis is not easy. Especially in patients with no history of acute pericarditis who present to the medical clinic with physical and functional signs of right congestive heart failure, have long posed a diagnostic challenge to the clinician.

Historically, the most frequent etiology of constrictive pericarditis was tuberculosis in the US, constituting 48 % of cases in a report published in 1962 [3]. In 1990s tuberculosis has been found to be a very rare cause in the US [4]. Tuberculous constrictive pericarditis remains common in parts of the world, especially in developing countries. In a single-center review of 51 cases over 5 years (1990–2012) in China, constrictive pericarditis was confirmed in 65 % cases [5].

The clinical features of constrictive pericarditis upon physical examination are jugular venous pressure elevation and pericardial knock. Hepatomegaly might be pulsatile and sudden inspiratory splitting of the second heart sound might also be apparent [6-8]. However, at an early stage of constriction, these clinical

signs might be absent even when a patient has symptoms of fatigue or dyspnea. In our clinical experience, the absence of these signs is probably related to a predominant reduction in cardiac output with only mild elevation of filling pressures. Owing to the spectrum of clinical presentation and variation in symptom severity that can lead to the disease, constrictive pericarditis often remains unsuspected until other diagnostic avenues have been exhausted [9].

The clinical recognition of constrictive pericarditis is important but challenging. Constrictive pericarditis often requires a multimodality imaging tools. Now echocardiographic techniques have facilitated the noninvasive recognition of constrictive pericarditis and its differentiation from restrictive cardiomyopathy. If a patient with heart failure and a normal ejection fraction, echocardiographic finding of exaggerated interventricular interdependence, relatively preserved left ventricular longitudinal deformation, and attenuated circumferential deformation is diagnostic of constrictive pericarditis [10, 11]. MRI may be particularly useful for evaluating patients who are not well imaged with echocardiogram, or who have a negative echocardiogram but strong clinical suspicion for constrictive pericarditis. Typically, a patient with constrictive pericarditis has a thickened pericardium, measuring greater than 4 mm in MRI [12].

Pericardial calcification is most reliably demonstrated on CT [13]. Pericardial thickening and calcification are most findings associated with constrictive pericarditis.

Invasive haemodynamic studies are indicated in patients with suspected constrictive pericarditis who have nondiagnostic imaging studies. In patients with constrictive pericarditis, the typical findings on cardiac catheterization include: increased atrial pressure with prominent x and y-descents, equalization of end-diastolic pressures in the chambers and opposing changes in left and right ventricular filling and systolic pressures with ventilation. However, these changes might be not be apparent in patients with an early stage of constriction or in individuals who are hypovolaemic, such as those on diuretics [14]. But we didn't use this invasive diagnostic tool in our cases.

Although the immediate goal in the management of tuberculous pericarditis is relief of symptoms, the long-term goal is prevention of progression to constrictive pericarditis [15]. When constrictive pericarditis cannot be entirely excluded by comprehensive diagnostic testing, surgical exploration might be necessary to detect the typical pericardial changes of constriction described above. The normal pericardium is a thin, avascular sac enveloping the heart in the anterior mediastinum, its relative inelasticity provides constraint during diastolic filling that limits chamber dilation, particularly the thin-walled right atrium and ventricle. Classically, constrictive pericarditis is defined as an impedance to diastolic filling caused by a fibrotic pericardium. Pericardiectomy, involving partial or complete decortication, is the definitive therapy for constrictive pericarditis, resulting in complete relief of symptomsinmany patients. Care must be taken to resect every constrictive epicardial layer to avoid persistent pericardial constriction. The constricting envelope surrounds the entire heart and interferes with diastolic filling. The disease affects the filling of all cardiac chambers, and, as the constriction progresses, only early diastolic filling is possible. In patients with compromise of cardiac function, surgery to remove the constricting envelope is the only effective long-term treatment.

Median sternotomy provides good exposure of the right atrium and the venae cavae, and it enables extensive removal of the pericardium. Previously, a left anterolateral thoracotomy was used [16], but that approach renders the institution of cardiopulmonary bypass difficult in the event of unexpected bleeding. Through the median sternotomy, the patient can easily be connected to cardiopulmonary bypass, without which extensive bleeding cannot be controlled. Cardiopulmonary bypass aids surgical dissection by emptying the ventricular cavities, which clearly defines the appropriate plane of dissection and facilitates the management of inadvertent cardiac injury. We prefer the median sternotomy to perform pericardiectomy, which enables a safer and effective approach. But cardiopulmonary bypass was not used in our casess.

Pericardiectomy has been associated with a relatively high early mortality, morbidity and low long-term survival [17]. Tokuda and his colleagues revealed that the predictive factors for composite operative mortality or major morbidity were preoperative chronic lung disease, NYHA functional class IV, previous cardiac operation, preoperative renal failure, and cardiopulmonary bypass during the operation. Careful consideration should be given to these risk factors in the process of patient selection and perioperative management [18]. A small but not insignificant portion of patients with constrictive pericarditis will develop cardiac low-output syndrome after surgery, regardless of the operative methods or the extent of pericardial resection. Despite extensive pericardiectomy, there have always been some early mortality due to cardiac low-output syndrome. This is because outcome is related not only to the extent of surgery but to myocardial involvement. Autopsy findings showed that myocardial fibrosis and atrophy may result from chronic constrictive pericarditis. Cardiac low-output syndrome can also be caused by changes in cardiac architecture: long periods of myocardial compression contribute to

"remodeling" of the ventricles and to greater involvement of the myocardium in patients who have undergone long periods of symptomatic pericardial constriction [19]. So, continuous intravenous infusions of vasopressor or inotropic agents may be necessary to maintain hemodynamic stability. Our most cases were young, with mild conditions, so their outcomes were good. It is important to maintain perioperative cardiac function. After surgery, inotropes, diuretics and nutrition supply are also critical to improve the prognosis.

LIMITATIONS

This study is summarized our experience of treating constrictive pericarditis over the past 5 years. However, there were a few limitations. No patient underwent preoperative hemodynamic cardiac catheterization and hence there was an increased difficulty to differentiate between constriction and restrictive cardiomyopathy. We routinely made the differential diagnosis according to CT and MRI, which were considered as excellent methods for differentiating. We need more cases and long-term follow-up to determine the outcomes in this study.

CONCLUSIONS

Prompt treatment of constrictive pericarditis are crucial to reduce mortality and morbidity. Pericardiectomy should be performed early after diagnosis. After surgery, inotropes, diuretics and nutrition supply are also critical to improve the prognosis.

AUTHOR'S CONTRIBUTION

Haiyong Wang and Angui Li wrote the paper. Tianci Qian, Fugui Ruan, Jianbin Sun, Zhenzong Du, Jianfei Song and Xiaolin Sun supervised the composition of the paper. All authors read and approved the final paper.

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