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Clinical Profile and Outcome of Patients with Chronic Postinfarction Left Ventricular False Aneurysm Treated Surgically

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ABSTRACT

Background: Rupture of the left ventricular (LV) free wall is usually fatal. In rare instances, hemorrhage is confined, and a false aneurysm (pseudoaneurysm) forms. In this study we reviewed our experience with patients treated surgically for chronic LV pseudoaneurysm.

Methods: Between 1997 and 2001, pseudoaneurysm was diagnosed in 8 patients (6 men and 2 women) 55.9 ± 7.6 years of age, and the patients underwent surgery. Before operation electrocardiogram (ECG), chest x-ray, echocardiography, and cardiac catheterization were performed. Repair was accomplished by resection of the pseudoaneurysm with use of cardiopulmonary bypass and in moderate systemic hypothermia (26°C-28°C). In 3 cases coronary artery bypass grafts were implanted, and in another a postinfarction ventricular septal rupture was closed.

Results: Before operation, 4 (50.0%) of the patients had congestive heart failure, 2 patients had unstable angina, and 2 were relatively asymptomatic. Six patients had ECG abnormalities (signs of myocardial infarction, persistent ST elevation). On chest x-ray 5 patients had cardiomegaly, and 2 of them had pulmonary edema. The definite diagnosis was established before operation in 7 patients and during the procedure in 1 patient. All patients survived operation, and none needed reoperation. An intraaortic balloon pump was used in 1 case. The postoperative courses of the other patients were uncomplicated. At the end of follow-up (mean, 31.0 ± 12.4 months) all patients were alive in New York Heart Association functional class I (6 patients) or II (2 patients).

Conclusion: The clinical presentation of chronic pseudoaneurysm often is nonspecific. The results of surgical treatment of chronic LV pseudoaneurysm are satisfactory.

INTRODUCTION

Rupture of the left ventricular (LV) free wall is one of the most dangerous complications of myocardial infarction and in most cases is fatal because of massive hemorrhage into the pericardial cavity [Keller 1987, Pollak 1993]. In rare instances, bleeding is confined to a limited space, and fibrous tissue organizing around hematoma forms a pseudoaneurysm [López-Sendon 1995]. Pseudoaneurysm, also called expanding intrapericardial hematoma, enlarges gradually because during systole blood has been propelled through its orifice [Yeo 1998]. Because it lacks any component of the normal cardiac wall, the wall of a pseudoaneurysm, unlike a true LV aneurysm, has a high propensity for further rupture [Frances 1998]. Thus this condition is considered an indication for urgent operation [Rittenhouse 1979, López-Sendon 1995]. Even in asymptomatic patients prophylactic repair is recommended soon after diagnosis [Ivert 1994]. Early and appropriate diagnosis of this uncommon and potentially fatal disease is of particular importance because patients can undergo successful surgical treatment [Yeo 1998].

We reviewed the clinical profiles and outcome among patients treated surgically for LV pseudoaneurysm.

MATERIALS AND METHODS

Patients

From March 1997 to May 2001, 8 patients (6 men and 2 women) with subacute LV free wall rupture followed by pseudoaneurysm formation underwent surgery in our department (less than 0.004% of open heart procedures). Mean patient age was 55.9 ± 7.6 years (range, 43-68 years).

Preoperative Status

Before operation, ECG, chest x-ray (posteroanterior and side projections), transthoracic (TTE) and transesophageal echocardiography (TEE) (cross-sectional and Doppler), and left-sided cardiac catheterization (ventriculography and coronary angiography) were carried out.

Surgical Procedure

All operations were performed through median sternotomy and with cardiopulmonary bypass. Moderate systemic hypothermia (26°C-28°C) was used. The chest was opened by median sternotomy. After total cardiopulmonary bypass had been instituted, the aorta was cross clamped, and cold crystalloid cardioplegic solution (St. Thomas Hospital formula) was administered into the root of the aorta to induce diastolic heart arrest. After pericardial adhesions were dissected free, pseudoaneurysms of the LV were found. Pseudoaneurysms were resected along the border between the necrotic wall of the aneurysm and viable myocardium of the LV. Because all pseudoaneurysms were chronic, this border was seen in all cases, so it was possible to close pseudoaneurysm orifices with 2 layers of nonabsorbable running suture (2-0 polypropylene [Prolene]) reinforced with felt patches. In patients with a LV free wall defect close to the basal aspect, special attention was paid to avoidance of

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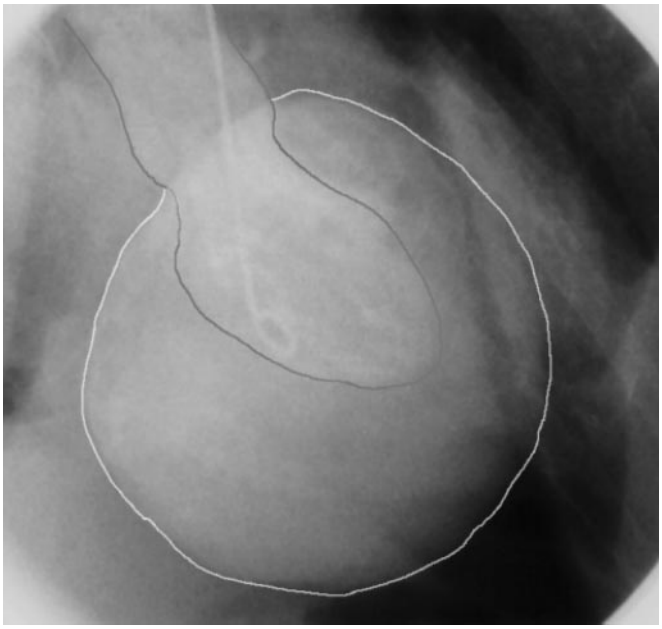

Figure 1. Giant left ventricular (LV) pseudoaneurysm involving posterior wall. Capacity of a false aneurysm was higher than LV volume. Patient was referred to the cardiac surgery department in cardiogenic shock.

distortion of the heart structures and excessive traction on the edges of the defect. Thus less myocardium was captured in the stitch. The area was fixed with tissue glue, and a second layer of xenopericardium was sutured on the epicardium. In 3 cases additional coronary artery bypass grafts were implanted. In 1 case bypass was from the left internal thoracic artery (LITA) to the left anterior descending artery (LAD). In another case bypass was from the LITA to the LAD with saphenous vein bypass grafts to the first diagonal branch from the LAD and the circumflex artery. In the third case, venous graft was applied to the right coronary artery (RCA). In 1 patient postinfarction ventricular septal defect (1.5 cm in diameter) near the apex was closed with Gore-Tex patch.

Data Management

Continuous variables are expressed as mean ± SD.

RESULTS

Preoperative Clinical Presentation

Four patients had symptoms of congestive heart failure, and 1 of them was in cardiogenic shock. Two patients had unstable angina; 2 others with ischemic heart disease were relatively asymptomatic at the time of diagnosis. Two patients were in New York Heart Association (NYHA) functional class IV, 1 in class III, 3 in class II, and 2 in class I. All had myocardial infarction in the history. The mean interval between myocardial infarction and operation was 11.6 ± 9.4 months (range, 1.5-23 months). One patient had undergone successful angioplasty of the circumflex (Cx) artery 17 months prior to pseudoaneurysm resection.

Before operation 6 patients had ECG abnormalities. Signs of myocardial infarction were found in 6 patients; infarction involved the inferior wall in 4 patients and the posterior wall in 2. Persistent ST-segment elevation in the leads over the infarcted areas was noted in 2 patients. In 2 patients ECG showed nonspecific ST changes without evidence of myocardial infarcts. In 5 patients, chest x-ray showed cardiomegaly. Two of these patients had radiological signs of pulmonary edema; 1 had intensified pulmonary vascularity.

At preoperative TTE and TEE (performed in 6 cases) the diagnosis of pseudoaneurysm was made in 3 cases. Before operation mitral regurgitation was detected in 4 patients and was graded as mild in 3 patients and moderate in 1 patients. In all cases mitral leaflets were morphologically normal, a finding confirmed during direct intraoperative inspection.

At coronary angiography 5 patients were found to have single-vessel coronary artery disease involving either the Cx artery or the RCA. One patient was found to have double-vessel disease, and 2 were found to have triple-vessel disease. Pseudoaneurysm was inferior in 5 patients and posterior in 3. Examples of ventriculograms are shown in Figures 1 and 2.

Intraoperative Findings

The pericardial cavity was found to be almost completely obliterated by adhesions in all patients. Pseudoaneurysms were filled with organizing thrombus. The average diameter of pseudoaneurysm assessed during operation was 6.9 ± 4.0 cm (range, 2.8-14.8 cm); orifice diameter, 3.3 ± 1.1 cm (range, 1.8-4.6 cm); and orifice/pseudoaneurysm diameter ratio, 0.54 ± 0.16 (range, 0.31-0.73). In 1 patient the diagnosis of pseudoaneurysm was made during the operation. The primary indication for surgery in this case was postinfarction ventricular septal defect.

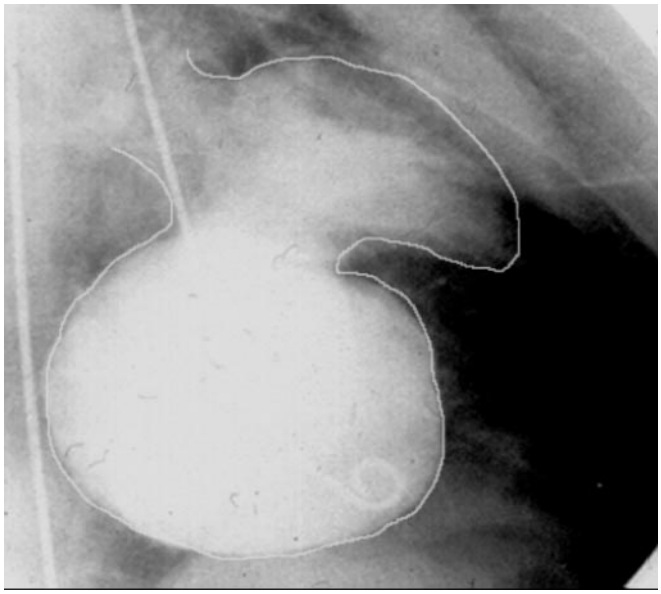

Figure 2. Inferior left ventricular pseudoaneurysm. Despite the volume of the lesion, the patient had only mild symptoms of congestive heart failure (New York Heart Association class II).

Postoperative Outcome

There were no hospital deaths. The postoperative course was uncomplicated in 7 cases. These patients were discharged from the hospital 7 to 11 days (mean, 8.5 ± 1.3 days) after the operation in satisfactory clinical status without evidence of congestive heart failure or angina pectoris. In 1 patient with triple-vessel disease, an intraaortic balloon pump (IABP) was inserted during surgery because of postcardiotomy low cardiac output syndrome. The patient recovered gradually. After 34 hours the IABP was removed, and after another 12 hours, the patient left the postoperative intensive care unit. Ten days later (ie, 12 days after surgical repair), the patient was referred to the rehabilitation center. No other postoperative organ or surgical complications were observed. Anticoagulant administration was started 2 days after surgery and was continued on a daily basis for at least 3 months postoperatively.

All patients were followed up for 11 to 58 months (mean, 31.0 ± 12.4 months). No late deaths occurred. Six patients were in NYHA functional class I and 2 were in class II. At last echocardiographic follow-up examination, only mild mitral regurgitation was found in 3 patients. Regional hypokinesis of the LV free wall corresponding to pseudoaneurysm localization was found in all cases.

DISCUSSION

The prognosis for acute heart rupture is fatal, and only a few cases of successful operations have been reported [López-Sendon 1995]. Although it is a rather rare complication, cardiac rupture accounts for approximately 10% of early deaths after acute myocardial infarction [Becker 1996]. Massive bleeding into the pericardial cavity leads to cardiac tamponade and to death within a short time. Less frequently, cardiac rupture is contained, and LV pseudoaneurysm formation occurs, but the patient has a greater chance of survival [Pollak 1993, Frances 1998]. A pseudoaneurysm forms when the necrotic area is relatively small, extracardiac leak is slow, and pericardial adhesions or intact epicardial wall limits bleeding [Keller 1987]. In most of our cases, preoperative coronary angiography revealed only isolated significant changes (occlusion or severe stenosis), thus the area of necrosis was limited. Moreover, in all cases pericardial adhesions were found after sternotomy. In addition to myocardial infarction, surgical cardiac procedures with the use of cardiopulmonary bypass, blunt or penetrating chest trauma, and infection (eg, endocarditis) can lead to formation of pseudoaneurysm [Gatewood 1980, Frances 1998].

Pretre and colleagues [2000b] divided pseudoaneurysms occurring after myocardial infarction into acute when discovered within 2 weeks and chronic when diagnosed more than 3 months after myocardial infarction. In our series all pseudoaneurysms were diagnosed more than 2 weeks after infarction and only 1 fewer than 3 months after infarction. The location of chronic pseudoaneurysm is partially related to its cause [Yeo 1998]. After myocardial infarction pseudoaneurysms form mainly in the inferior or posterolateral free LV wall [Frances 1998]. In accordance with this finding, in our group only posterior or inferior wall was involved. No patient had anterior wall pseudoaneurysm. Rupture of free LV anterior wall results in massive hemorrhage into the pericardial cavity, cardiac tamponade, and sudden death rather than in pseudoaneurysm formation [Rittenhouse 1979].

Definite diagnosis of pseudoaneurysm is difficult because of a lack of typical clinical symptoms suggesting the presence of this lesion [Pollak 1993]. Many patients remain asymptomatic for a long time, so the physician is not alerted to the possibility of the presence of a pseudoaneurysm [Badni 1991]. Yeo et al [1998] reported that in a study comprising not only cases with postinfarction pseudoaneurysm, 48% of pseudoaneurysms were found incidentally. Yeo et al also reported that patients with pseudoaneurysm occurring after myocardial infarction tended to present more often with recurrent anginal chest pain or congestive heart failure. In our group, 2 patients were relatively asymptomatic after myocardial infarction, and the diagnosis of pseudoaneurysm was established during elective coronary angiography. Congestive heart failure after myocardial infarction may suggest the presence of pseudoaneurysm [Komeda 1993, Pretre 2000b]. During systole a significant part of LV output is propelled to a pseudoaneurysm rather than to the ascending aorta. In our group, 37.5% of the patients were in NYHA functional class III and IV before operation, 1 in cardiogenic shock.

Problems related to surgical treatment of LV postinfarction pseudoaneurysm must be pointed out. First, there is a risk of thrombotic mobilization of material during dissection of the heart [Pretre 2000b]. To avoid this problem, dissection in all cases initially was limited to the ascending aorta and right atrium to allow institution of extracorporeal circulation. Dissection of LV and tissue adjacent to the pseudoaneurysm was performed after the ascending aorta had been clamped. Second, various techniques were proposed for closing the neck of a pseudoaneurysm [Pretre 1999, 2000b]. In cases of chronic pseudoaneurysm, direct closure often is possible because the edges of the lesion are fibrotic. To avoid excessive traction on the myocardium or distortion of the Cx artery or coronary sinus, some authors recommend use of a patch (pericardial or artificial) when the LV wall defect is located near the base of the heart [Pretre 2000b]. We managed to obliterate the neck of the pseudoaneurysm with the suture in all patients. Defects close to the cardiac base were small enough to enable direct closure. Moreover, we used special technique to reinforce the suture line.

In our study, neither in-hospital nor late mortality occurred. Thus the mortality rate was lower than reported by other authors [Ivert 1994, Pretre 2000a,b]. One reason for the low mortality in our patients was that surgery was not performed during the acute phase of rupture. Surgery during this phase bears a high perioperative mortality [Pretre 2000a]. Chronic LV pseudoaneurysm patients who undergo surgery in better preoperative clinical status do well, just as chronic postinfarction ventricular septal defect patients do better than those who undergo surgery while the lesion is in the acute phase. In addition, in our group no patient needed mitral

valve surgery, which is an important factor in increased mortality following surgery for pseudoaneurysm [Frances 1998]. Although cases of successful repair of ruptured postinfarction pseudoaneurysm have been described [Yaku 1995], higher propensity for fatal rupture than surgical risk should encourage surgical treatment of even asymptomatic patients [Ivert 1994, Pretre 2000b].

In general, the clinical status of patients who survive improves significantly after surgical repair of LV pseudoaneurysm. At the end of follow-up all our patients were in NYHA functional class I or II, an outcome consistent with earlier observation of survival with an excellent quality of life [Pretre 2000a,b].

CONCLUSIONS

On the base of our experience we conclude that the clinical presentation of chronic pseudoaneurysm is often nonspecific. The results of surgical treatment of chronic LV pseudoaneurysm are satisfactory.

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