Case Reports

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Left Ventricular Aneurysm Repair

with Use of a Bovine Pericardial Patch

Left ventricular aneurysm, which can impair systolic function, has a reported incidence of 10% to 35% in patients after myocardial infarction. In a 58-year-old woman who had a history of myocardial infarction, we excised a large left ventricular aneurysm and restored left ventricular geometry with use of a bovine pericardial patch. The aneurysm's characteristics and the patient's preoperative left ventricular ejection fraction of 0.25 had indicated surgical intervention. The patient had an uneventful postoperative course, and her left ventricular ejection fraction was 0.50 to 0.55 on the 4th postoperative day. This case illustrates the value of surgical treatment for patients who have a debilitating left ventricular aneurysm. **(Tex Heart Inst J 2014;41(4):407-10)**

eft ventricular (LV) aneurysm is widely described in the medical literature; its reported incidence after myocardial infarction ranges between 10% and 35%.¹ In the current era of early revascularization, the overall prevalence of this complication might be decreasing. We describe the case of a patient in whom we repaired a large LV aneurysm with use of a bovine pericardial patch, and we discuss the history and nature of LV aneurysm.

Case Report

In June 2013, a 58-year-old woman was referred to our institution. She reported a 2-month history of dyspnea and heart palpitations. Her medical history included hypertension, hypothyroidism, gastroesophageal reflux disease, tobacco use, coronary artery disease, and a myocardial infarction (the date of which the patient could not recall). An electrocardiogram revealed evidence of a past myocardial infarction. A chest radiograph showed an enlarged cardiac silhouette. During the patient's echocardiographic evaluation, a wide-complex tachycardia (heart rate, approximately 190 beats/ min) prompted her transfer to the emergency department, where she underwent cardioversion to sinus rhythm. No studies were performed to confirm the cardiac rhythm during the palpitations; however, the wide-complex arrhythmia during echocardiography was thought to have a ventricular focus. Coronary angiograms revealed a 90% stenosis in the left anterior descending coronary artery (LAD), and a drug-eluting stent was placed. No disease was found in the left circumflex coronary artery. A large aneurysm was seen on the posterolateral wall of the LV (Fig. 1). Echocardiographic results confirmed the presence of a large LV aneurysm and an LV ejection fraction (LVEF) of 0.25. Computed tomographic angiograms showed the aneurysm (Fig. 2).

At surgery, cardiopulmonary bypass (CPB) was instituted, and the aorta was crossclamped. The heart was elevated, and a large, thin-walled, posterolateral LV aneurysm was seen (Fig. 3). The aneurysm was opened by means of a longitudinal incision, with care taken to avoid the LAD. No intraluminal thrombus was seen, and demarcation was clear between the smooth, thin-walled aneurysmal tissue and the healthy, trabeculated myocardium (Fig. 4). The papillary muscles were not involved. To exclude the thin aneurysmal ventricular wall and preserve the ventricular geometry, a 4×5 -cm elliptical bovine pericardial patch was sutured to the healthy myocardium with use of 2 layers of running 3-0 and 4-0 polypropylene suture (Fig. 5). No Foley catheter or other method to estimate LV volume was used during the repair; the appropriate patch size was determined by estimating the size of the defect in the LV wall and using the estimate to guide the creation of a patch that would fill this space and restore the native chamber geometry. The redundant aneurysmal wall was then excised sharply, and the remaining tissue was closed over the patch with use of 2 layers of running 2-0 polypropylene suture (Fig. 6). The aortic cross-clamp was removed. As the patient was weaned from CPB, de-airing was accomplished through the left atrial sump and a 19G needle in the ascending aorta. The patient's LVEF appeared to have improved, and additional support with an intra-aortic balloon pump was indeed unnecessary.

The patient recovered uneventfully. An echocardiogram on postoperative day 4 showed an intact patch repair and normal LV size (end-diastolic volume, 89.7 mL). The patient's LVEF was estimated to be 0.50 to



Fig. 1 Coronary angiogram shows the left ventricle (arrowhead) during systole and the left ventricular aneurysm (arrow).



Fig. 2 Preoperative computed tomogram shows the left ventricular aneurysm (arrow).

0.55. She was discharged from the hospital on day 6, in good condition. As of June 2014, she had a preserved LVEF and no dyspnea or palpitations.



Fig. 3 Intraoperative photograph shows the large left ventricular aneurysm.



Fig. 4 Intraoperative photograph shows a clear demarcation between healthy and nonviable myocardium.



Fig. 5 Intraoperative photograph shows the use of the bovine pericardial patch in the repair.



Fig. 6 Intraoperative photograph shows the completed repair.

Discussion

Left ventricular aneurysm is a well-known sequela of myocardial infarction. Ventricular aneurysm was apparently recognized centuries ago^{2,3}; however, its association with coronary occlusion and myocardial infarction was not definitively noted until the early 1900s.⁴ The first verifiable attempt to correct an LV aneurysm surgically was in 1942 by Beck,⁵ who used parietal pericardium in the repair; his desperately ill patient died 5 weeks postoperatively. The first reported angiographic diagnosis of LV aneurysm was published in 1951.² In 1958, Cooley and colleagues⁶ described the first successful repair, in which they used a linear technique and CPB. Other techniques have since been described, and modern surgeons have several repair options.

True and false aneurysms can involve the LV. True aneurysms by definition are full-thickness lesions of the LV wall, whereas pseudoaneurysms of this chamber are essentially contained ruptures of the free wall. However, the nomenclature and official definitions remain imprecise, because these lesions' morphologic characteristics might be less important than the resultant physiologic changes—chief among these a substantial decrease in LVEF caused by akinetic or paradoxical motion of the ventricular wall.7 Although the exact mechanism of aneurysm formation is not known, a transmural infarction is required. Several investigators, including Hirai and associates,8 have suggested that poor coronary collateral flow after infarction leads to continued necrosis of the infarcted tissue and increases transmural scarring. The scar can become aneurysmal when it is subjected to increased systolic wall tension, in accordance with Laplace's law. In addition, it is unclear whether most patients have single-vessel or multivessel coronary disease, although a slight majority of studies suggest that patients tend to have multivessel stenosis.9,10

Our patient's symptoms, preoperative LVEF of 0.25, dysfunctional wall motion, and echocardiographic evidence of a large aneurysm all suggested the benefit of surgical repair. In addition, 15% to 30% of patients with this condition have ventricular arrhythmias,¹¹ as our patient did during preoperative echocardiography. However, she did not undergo preoperative electrophysiologic mapping. A 90% stenosis indicated stenting of the LAD; however, stenting would probably not prevent future ventricular arrhythmias that would most likely be secondary to an aneurysmal focus.

Surgical treatment of this condition is typically warranted in patients who present with angina, heart failure, or ventricular arrhythmia. Coronary angiography should be performed before surgery, and necessary revascularization can be performed at the time of aneurysmorrhaphy.

The linear repair described by Cooley and colleagues⁶ involves a longitudinal ventricular incision, excision of the aneurysmal tissue, and closure with heavy, nonabsorbable polypropylene suture reinforced with felt strips. This procedure has been used successfully since its introduction; however, it can decrease the patient's LVEF when the ventricle is closed after resection of the aneurysm.

Circular patch repair, which we used, was also described by Cooley¹² and by Dor and co-authors.¹³ This method arose from the idea that all apical scarring will be completely excluded from the ventricle, resulting in retained ventricular geometry and better postoperative LV function. Komeda and colleagues¹⁴ described the cases of 336 patients who underwent partial aneurysm resection and repair of the ventricular wall by conventional closure of the ventriculotomy (281 patients), inverted T closure (17 patients), or endocardial patching with a Dacron graft or with glutaraldehyde-preserved bovine pericardium (38 patients). In the subset of patients who had severely impaired LV function, the operative mortality rate was substantially higher with conventional repair (13 deaths among 103 patients; 12.6%) than with T closure or endocardial patching (3 deaths among 46 patients; 6.5%).

Multiple patch materials have been used, including Dacron, polytetrafluoroethylene, autologous pericardium, and bovine pericardium. The choice of patch material is at the discretion of the surgeon, because no studies have been conducted to determine the superiority of any one material. In addition, patch size is crucial: a small patch will allow for only small LV volumes, whereas oversized patches can lead to larger akinetic segments and increased wall stress.¹⁵ Before the suture lines are tied, blood is reintroduced retrograde into the heart through the cardioplegia cannula, to aid in de-airing. After it is confirmed that the patch is watertight, the aneurysmal tissue can be excised, and the remaining tissue can be closed over the patch with use of running polypropylene suture. Thrombus that then forms in the cavity between the patch and the outer wall will be excluded from the LV.

Functional status, comorbidities, and risk in the patient with a symptomatic LV aneurysm are the most important factors in determining surgical eligibility. In viable surgical candidates, repair of such an aneurysm can resolve debilitating symptoms and improve the patient's functional status.

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