Surgical Management of Recurrent Postinfarction Ventricular Septal Defect

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SUMMARY

A 57 year old man presented with postinfarction ventricular septal infarct (VSD) a week after myocardial infarction and thrombolytic therapy. Coronary angiography confirmed double vessel disease. He underwent surgical repair of the VSD and coronary artery bypass grafting. Two days postoperatively, he deteriorated due to recurrence of VSD. Reoperation was carried out with satisfactory results. The surgical management is described with a review of the relevant literature.

KEY WORDS:
Recurrent postventricular septal defect, Surgical management

INTRODUCTION

Postinfarction VSD occurs in 1-2% of patients following acute myocardial infarction[1]. The management of such patients is difficult. Without surgery, most patients die[2]. Surgical mortality is also considerable. In the UK, it is estimated to be 38%[2]. Since an average cardiac surgeon sees fewer than one case per year, the surgical experience is limited in most centers[2].

Recurrence of postinfarction VSD occurs in 10-25% of cases following surgery[1]. When this occurs, the management can be even more challenging. While a small recurrence may be managed conservatively, if the patient deteriorates, consideration should be given for repeat repair.

CASE REPORT

A 57 year old man presented with a five-day history of chest pain and breathlessness. He had a history of diabetes, hypertension and hypercholestoremia. His admission electrocardiogram showed old inferior infarct and recent anterior infarct. He was given thrombolytic therapy using streptokinase at a peripheral hospital. He was then transferred to our institution for further management.

He was in congestive cardiac failure clinically and radiologically. Initial echocardiography showed impaired left ventricle with an ejection fraction of 35% and normal valves. Two days later, a harsh systolic murmur was heard. Repeat echocardiography confirmed anterior VSD. Coronary angiography confirmed 90% stenosis of the left anterior descending and total occlusion of the right coronary arteries. Following full preoperative preparations, he underwent surgical repair on cardiopulmonary bypass with moderate hypothermia, antegrade and retrograde blood cardioplegia. To reduce postoperative bleeding, Aprotinin was given with a loading dose of 1 million KIU and another million KIU in the pump prime. Infusion at 50 ml/hour was continued through the surgery. Left ventriculotomy was performed via the infarct. There was a 2cm VSD in the anterior septum. This was closed by infarct exclusion using a Dacron patch. The repair was reinforced with sandwich closure of the left ventricular free wall, patch, ventricular septum and right ventricular free wall. Coronary bypasses using vein grafts were performed to the left anterior descending and right coronary arteries. The patient came off bypass with low dose inotropic support. Intraoperative transoesophageal echocardiography showed no evidence of residual VSD with good ventricular contractility.

The patient's initial postoperative course was uneventful and he was extubated on the first postoperative day. Late in the second postoperative day however, he became increasingly breathless. He also required increasing inotropic support. There was re-emergence of the harsh systolic murmur not detected earlier in the postoperative period. Echocardiography confirmed a recurrence of the VSD. Although it was smaller than previously, the patient was deteriorating. It was decided to re-operate.

At re-operation, the left ventriculotomy was reopened. The previously sutured patch was intact. There was a 1cm VSD anterio-superiorly away from the suture line. Another Dacron patch was inserted. The repair was reinforced with apposition of the left ventricular free wall, septum and right ventricular free wall in such a way as to exclude the repair from the ventricular cavity. All sutures were reinforced with Teflon strips. The patient was weaned off cardiopulmonary bypass with intra-aortic balloon pump (IABP). Postoperatively the patient recovered well. The IABP was removed on the second postoperative day. His subsequent recovery was uneventful and he was discharged well sixteen days after his initial admission. His pre-discharge echocardiogram showed no residual VSD with improved left ventricular contractility and an ejection fraction of 78%.

DISCUSSION

The management of patients who develop mechanical complications of myocardial infarction is difficult and carries high risk of morbidity and mortality. Without surgical intervention, most of these patients succumb. The occasional

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patient may survive and the VSD may in fact close spontaneously[3]. Patient selection obviously plays a part in the surgical results[4]. Unfortunately, waiting until patients develop multi-organ dysfunction will certainly worsen the outcome. Early surgical repair should therefore be the method of choice[2].

Since many of these patients have multi-vessel coronary artery disease, performing coronary angiography and coronary artery bypass should be seriously considered. This appears to have a survival advantage for patients[5]. If necessary, IABP may be inserted prior to coronary angiography.

Most surgical series describe some form of patch repair with infarct exclusion[1,5]. It is important to place the sutures as far away from the infarcted muscle as feasible in order to achieve a secure repair. Recurrence of VSD following surgical repair gives rise to a more severe surgical challenge and a dilemma in management. This appears to occur in 10-25% of patients[1]. A small shunt in a hemodynamically stable patient may not need further intervention. Transcatheter closure of recurrent VSD following surgical repair may be feasible in some cases. In patients who are unstable, this carries a significant risk, as in the case of the primary catheter closure.

Early recurrence appears to be easier to operate on since the ventriculotomy can be easily taken down, as in this case. Reinforcement of the repair with ventricular free walls not only makes the repair more secure but also results in useful ventricular remodeling. There is always some remodeling whichever method of repair is adopted. Since most of these patients have ventricular dysfunction associated with dilatation, tailoring the repair to achieve ventricular remodeling is a logical step.

While early re-operation carries with it a significant mortality risk, in suitable cases there should be no hesitation to undertake this procedure, as demonstrated in this case. Even after the second surgery, the ejection fraction was much improved compared to previously.

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