Secundum atrial septal defect repair: long-term surgical outcome and the problem of late mitral regurgitation

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Summary: This study examines the clinical and surgical outcome of a group of 55 patients (mean age 33 years) with secundum atrial septal defect who underwent surgical repair of this defect between 1981 and 1990. A group of 25 of these patients underwent late echocardiographic follow-up.

Fifty-two patients underwent repair by direct suturing and three by patch closure. Surgical mortality was nil. There was one late death of a 58 year old who died from cardiac failure 4 years after surgery. Late postoperative morbidity consisted of two patients; one, age 63 at the time of surgery, required mitral and tricuspid valve replacement 6 years later and one, age 77 at surgery, developed cardiac failure 3 years later.

Atrial fibrillation persisted in the six patients who had the rhythm before surgery and developed postoperatively in two patients aged 54 and 58. Two patients aged 49 and 57 developed immediate postoperative sinus node dysfunction requiring permanent pacing. The mean age at surgery of those six patients who suffered cardiac morbidity was 60 years. The patients with preoperative angiographic evidence of mitral valve prolapse were significantly older (P<0.001) and had higher mean pulmonary artery pressures (P<0.001) than patients with normal valves. There was no significant relationship between shunt size and mitral valve prolapse.

Echocardiographic follow-up showed persistent mitral valve prolapse in all nine patients who developed the condition preoperatively. Five patients developed mitral valve prolapse with mitral regurgitation postoperatively, one of whom needed subsequent mitral and tricuspid valve replacement. These five patients were on average older (mean age 54) but the group was too small to prove significance.

The follow-up data illustrate the current low mortality and morbidity associated with surgical closure of atrial septal defects. Late postoperative echocardiography has revealed not only that mitral valve prolapse persists in those patients who developed the condition pre-operatively but that new cases of mitral valve prolapse with mitral regurgitation can occur after atrial septal defect closure.

Introduction

Atrial septal defect (ASD) is the cause of approximately one third of cases of congenital heart disease diagnosed in adults and just under 10% of all congenital heart disease.1 Secundum atrial septal defect is the most common type and accounts for more than 90% of atrial septal defects. It is usually an isolated lesion which lies near the fossa ovalis and does not border the atrioventricular valves.

If unrepaird, this defect leads to death at an average age of 39-49 years2,3 but, if repaired in younger patients, survival approaches that of population controls.4 We present the most recent study of the long-term outcome of surgical repair of this defect. This study is unique in carrying out a late postoperative echocardiographic assessment.

Patients and methods

During a 9 year period between 1981 and 1990, 59 patients underwent surgical closure of a secundum atrial septal defect in the cardiothoracic unit of a teaching hospital. They were diagnosed as having secundum rather than primum ASD on the basis of a normal electrocardiographic axis and no evidence of a 'goose neck' deformity of the left ventricle or cleft mitral valve on angiography. This report deals with the 55 patients for whom clinical and cardiac catheter data were available for review. They comprised 36 (65%) females and 19 (35%) males with a median age of 26 years (range 3-77) and a mean age of 33 years. Clinical details were obtained from medical records. Cardiac catheter data were reviewed by an observer blinded to the clinical data. All cardiac catheter procedures followed a routine pattern of pressure measurements from right atrium, right ventricle, pulmonary artery, left ventricle and aorta, with estimation of oxygen saturations by cuvette oximetry at these positions.

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and also from the superior and inferior vena cava. Angiograms were obtained of the left ventricle in the 45° right anterior oblique position (RAO) and of the right ventricle in the 5° RAO position with 10° cranial tilt, with follow-through into the laevo phase to visualize the left and right atria, and atrial septal defect. Angiographic mitral valve prolapse was defined as a ballooning deformity of the mitral valve on the left ventricular angiogram in the 45° RAO position2 (Figure 1). Angiographic mitral regurgitation was visually graded as mild, moderate or severe.

Follow-up data were obtained on the 55 patients from outpatient clinic attendances for a median period of 2 years (range 1–9 years). Prospective echocardiographic data were obtained on 25 patients. M-mode, two-dimensional and Doppler echocardiography was performed using an ATL Ultramark 6 ultrasound system with a phased-array scan head. Echocardiographic mitral valve prolapse was diagnosed on M-mode echocardiography when there was abrupt mid-systolic to late-systolic posterior displacement of part of the mitral systolic closure line to at least 2 mm below the line joining the point of valve closure in systole (C) to the point of valve opening in diastole (D), with superior leaflet displacement confirmed by cross-sectional echocardiography. It was also diagnosed if there was pansystolic prolapse with posterior displacement of at least 3 mm below a line joining C and D, with the nadir occurring in mid-systole confirming by bulging of the mitral annulus during systole on echocardiography.6

Statistical analysis

Statistical analysis was carried out using the unpaired Student’s t-test. A probability value of less than 0.05 was considered significant.

Results

Clinical data

Of the 55 patients in this study, 26 (47%) had symptoms at presentation (mean age 45, range 21–77). These symptoms included dyspnoea, palpitations, fatigue, syncope and atypical chest pain. Supraventricular arrhythmias in the form of atrial fibrillation (AF) were present in six cases pre-operatively (mean age 62, range 46–77).

Cardiac catheter data

Angiographic evidence of mitral valve prolapse was found in 17 patients (31%, mean age 43), who were significantly older ($P < 0.001$) and tended to have higher mean pulmonary artery pressures ($P < 0.001$) (Figure 2). Of these 17 patients, six had mild mitral regurgitation. There were no patients with moderate or severe mitral regurgitation. There were 38 patients with no evidence of mitral valve prolapse (mean age 29). There was no significant relationship between shunt size and mitral valve prolapse.

Surgical data

Surgical closure was by direct suturing in 52 patients and by patch closure (Dacron) in three. Those patients who had patch closure did not have significantly larger ASDs in comparison to those who underwent repair by direct suture. There were no surgical deaths. Surgical morbidity consisted of two cases of tamponade due to pericardial bleeding, two cerebrovascular accidents (patients aged 39 and 63 who had closure by direct suture, both of
whom made a good recovery), one pneumothorax, four cases of transient tachyarrhythmia and two cases of bradyarrhythmia. The two patients (aged 49 and 57) with bradyarrhythmia had sinoatrial dysfunction with symptomatic bradycardia and sinus arrest requiring permanent pacemaker insertion. AF persisted postoperatively in those six patients with the arrhythmia preoperatively. Only two patients, aged 58 and 54, developed AF on long term follow-up. There was one late death due to cardiogenic shock in a 58 year old man 4 years after surgery. The remaining late postoperative morbidity consisted of cardiac failure in an 81 year old man (age 77 at ASD repair) and mitral and tricuspid valve replacement for severe regurgitation in a 70 year old woman (age 63 at ASD repair).

Prospective study: echocardiographic data

A randomly selected group of 25 of the 55 patients (mean age 18, median 30) underwent prospective follow-up echocardiographic examination (mean time from surgery 6 years, median 7 years). Echocardiographic mitral valve prolapse was persistent in all nine patients (36%, mean age 48) with preoperative angiographic evidence of mitral valve prolapse, four of whom had mild mitral regurgitation (Figure 2). The mean age of patients with no preoperative mitral valve prolapse was 29. In addition four patients (16%, mean age 38) had evidence of mitral valve prolapse with mitral regurgitation which had developed postoperatively (mean time 6 years, median 5 years). Those patients who developed mitral valve prolapse postoperatively were older (mean age 54), but the group was not large enough to prove significance.

Discussion

This study confirms the low mortality and morbidity associated with the surgical closure of atrial septal defects. Patients in the sample who suffered arrhythmias or other cardiac morbidity tended to be older (mean age 60) at the time of surgery and none was younger than 49 years old. For example, the mean age of patients who had atrial fibrillation (AF) preoperatively which then persisted after surgery was 62 years whereas the mean age of all patients was 33 years. Only two patients, aged 54 and 58, developed AF on long-term follow-up and in both cases it was unresponsive to electrical cardioversion. This study confirms the findings of other studies which show that patients who undergo surgical repair at a younger age do better.4,7

Surgical closure in 52 of the 55 patients in this study was by direct suture, a simpler procedure to carry out than patch closure, and one which avoids the increased risk of postoperative embolism associated with prosthetic material. The results confirm the low mortality and morbidity associated with direct suture.

The study is unique in including a late postoperative echocardiographic assessment of patients who have had an atrial septal defect repair. Mitral valve prolapse was shown to persist in all the patients (36%) who developed the condition preoperatively. This result is at odds with the echocardiographic study of Schreiber et al., looking at the early postoperative persistence of mitral valve prolapse at 7 days following atrial septal defect repair, which described a decrease in mitral valve prolapse in six out of seven patients.8 Schreiber et al. postulated that mitral valve prolapse occurred because of a distortion of the left ventricle, which is produced by a leftward shift of the interventricular septum as a result of right ventricular dilatation due to the interatrial shunt. If mitral valve prolapse occurred because of this, one would expect a significant relationship between mitral valve prolapse and shunt size, but none was found in the present study. One would also expect repair of the atrial septal defect to correct the left ventricular distortion and improve or rectify mitral valve prolapse. We have shown that not only did mitral valve prolapse persist in the patients who developed the condition preoperatively but it also developed postoperatively in four other patients (16%, mean age 38).

The association of mitral valve prolapse and atrial septal defect was first noted in 1966.9 Mitral valve dysfunction has been shown to be progressive, both in patients with primary mitral valve prolapse10,11 and where mitral valve prolapse is associated with atrial septal defect.8 We suggest that the valve deteriorates due to ageing and to the abnormal tensions placed upon it prior to ASD repair, though this is unproven.

The most important conclusion of this study is that the surgical closure of ASD has a low mortality and morbidity in older patients and almost none in younger patients. Murphy et al. have shown that younger patients benefit the most from surgical repair of ASD and that their life expectancy is identical to the normal population following surgery.4 Therefore, it is extremely important that ASD is diagnosed and repaired as early as possible. However, older patients also benefit from surgery.4

It is also interesting to note that new cases of mitral valve prolapse do occur after atrial septal defect closure. Accordingly, patients who have undergone atrial septal defect closure should be followed up with regular echocardiography to assess mitral valve function. They can then be advised of the need for antibiotic prophylaxis and offered surgery if they should develop severe mitral regurgitation.
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References

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