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Clinical outcomes of surgically corrected atrial septal defects

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Abstract

Objective: To examine the outcomes of surgical repair of atrial septal defects in paediatric and adult patients.

Methods: The retrospective study comprised data of 84 patients who had undergone surgical correction of atrial septal defect at the Aga Khan University Hospital, Karachi, between June 2006 and December 2011. All patients with isolated atrial septal defect (ostium secundum, ostium primum and sinus venosus with or without partial anomalous pulmonary venous connection) were included. Clinical and transthoracic echocardiographic data was reviewed. SPSS 17 was used for statistical analysis.

Results: There were no deaths in the study population. The mean time for follow-up was 6.5±9.9 months. Most of the patients (n=80; 95.2%) were in New York Heart Association class I at follow-up, while the remaining 4(4.8%) were in New York Heart Association class II. Post-operatively, 8 (9.5%) patients developed brief episodes of arrhythmias. There were 3 (3.57%) patients who were re-admitted within 30 days; 2 (66.7%) had superficial wound infection, while 1 (33.3%) had to be re-opened because of cardiac tamponade.

Conclusion: Surgical repair of atrial septal defects is a safe procedure which is associated with excellent results and low morbidity.

Keywords: Heart septal defects, Atrial, Heart defects, Congenital, Thoracic surgery. (JPMA 63: 662; 2013)

Introduction

Atrial septal defect (ASD) is the cause of approximately one-third of the cases of congenital heart disease (CHD) diagnosed in adults and almost 10% of all CHDs.[1] Due to the lack of specific symptoms, diagnosis can be missed during childhood.[2] Patients with ASD and left-to-right shunts are at an increased risk of developing pulmonary arterial hypertension.[3] While large atrial septal defects may present in childhood with signs of heart failure, a significant proportion of patients present in the third or fourth decade of life.[4] Progressively limiting, untreated ASD can lead to early death in middle-aged adults.[5]

ASD has been surgically repaired for almost 60 years.[6] The distinct feature of ASDs which sets them apart from other CHDs is the slow progress of the clinical course, which does not lead to debilitating symptoms until after the fourth or fifth decade of life.[7] When defects are closed in adults, the majority of those with pre-operative arrhythmias do not revert to sinus rhythm and new atrial flutter/fibrillation develops in 8% of those over 40 years (follow-up 3.8 years).[8] The objectives of surgical closure of an ASD are the reversal of haemodynamic abnormalities and the prevention of complications, including heart failure and irreversible pulmonary vascular obstructive changes, hence leading to improvement of symptoms.[9] Surgical closure of ASD provides good early post-operative and long-term results.[10]

The current study reviewed data of 84 patients who had undergone surgical repair of ASD, including the three major forms: ostium primum, ostium secundum and sinus venosus (with or without partial anomalous pulmonary venous connection or PAPVD).

Patients and Methods

The retrospective study reviewed data of all the patients with surgical closure of ASD who returned for follow-up from June 2006 to December 2011 at the Aga Khan University Hospital, Karachi. Pre-operative, operative and post-operative data of 84 patients was reviewed.

All patients with isolated ASD (ostium secundum, ostium primum and sinus venosus with or without PAPVD) were included. Excluded from the study were patients with ASD associated with one or more other congenital heart anomalies (ventricular septal defect, Ebstein's Anomaly, Tetralogy of Fallot, complete atrioventricular septal defect, pulmonary valve stenosis etc). Clinical and echocardiographic data from baseline evaluation, early

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follow-up and late follow-up were obtained by chart review. Baseline clinical data included gender, symptoms, diagnosis, age at surgery, and the size of the defect.

The diagnosis was established by transthoracic cross-sectional echocardiography. Variables assessed pre-operatively included the type and size of the ASD. Other clinical and haemodynamic echocardiographic factors were also assessed. Operative data consisted of the duration of surgery (time of incision to the time of dressing), cardiopulmonary bypass (CPB) time and aortic cross-clamp (ACx) time, minimum temperature, surgical technique employed to close the ASD and type of myocardial preservation (crystalloid or blood cardioplegia). The post-operative data included the presence of any residual ASD shunt via a post-op echo (significant >2mm), the use of inotropic support, length of cardiac intensive care unit (CICU) stay, length of hospital stay, use of ventilation support for >48 hours and any morbidity or mortality.

All procedures had been done using cardiopulmonary bypass. The chest was opened via median sternotomy. After total cardiopulmonary bypass had been achieved, either cold blood or crystalloid cardioplegic solution was given antegrade through the root of aorta. The method used to close the defect depended on its size and anatomical type. Isolated ASDs (ostium secundum only) underwent direct suture closure, while bovine pericardium and autologous patch with Prolene 5-0 suture were also used. Partial anomalous pulmonary drainage was repaired in existing cases and superior vena cava augmentation was carried out using an autologous patch. Anterior mitral valve leaflet was also repaired in cases where it was found to be regurgitant secondary to a cleft (ostium primum only).

After the procedure, residual intra-atrial shunting (significant >2 mm) was examined by colour Doppler. Inotropic support was provided where required and consisted of either one of epinephrine, dopamine, milrinone, dobutamine or a combination of epinephrine and milrinone.

Data was analysed using the SPSS version 17.0 and was expressed as median and ranges as appropriate.

Results

Of the 84 patients, 48 (57.1%) were males and 36 (42.9%) were females. The mean age at operation was 12.6±13.6 years and 76 (90.5%) had symptoms at presentation that included repeated chest infections, atypical chest pain, palpitations, dyspnoea, syncope and fatigue. Overall, 14 (16.7%) patients had moderate or severe pulmonary hypertension. Only 8 (9.5%) patients were asymptomatic. The size of the defect ranged from 5mm to 63mm, with a mean of 21.5±10.8mm. The relative frequencies of different ASDs were secundum 58 (69.0%), primum 8 (9.5%), and sinus venosus 18 (21.4%). The various pre-operative, operative and post-operative variables were assessed (Table-1).

The mean CPB time was 70.9±33.0 minutes and ACx time was 40.1±21.0 minutes. The mean minimum temperature reached was 36.7±0.4oC. Individual types of ASD, intra-operative variables and common morbidities were noted (Table-2). The foramen primum repair required a much longer CPB time. After each surgery, a post-operative on-table echo was done to check for the presence of any haemodynamic abnormality or for any residual patch defect (significant >2mm). There were no cases in which the residual patch shunt was >2mm.

Major post-operative complications and frequencies were also reviewed (Table-3). There was no significant difference in the morbidities between the different types of ASDs. Patients who developed pericardial and pleural diffusion were treated by tube thoracostomy and no further complications were reported. There was only 1 (1.2%) patient who required ventilation support for greater than 48 hours. Of the 3 (3.6%) patients who were readmitted within 30 days, 2 (66.7%) presented with a wound discharge from the incision site, while 1 (33.3%) was re-opened due to cardiac tamponade. There was 1 (1.2%) case of complete heart block which resolved spontaneously.

The mean time for follow-up was 6.5±9.9 months. Most of

Table-1: Primary pre-operative, operative and post-operative data (n=84).

Preoperative Variables	Mean ± SD
Height (cm)	121.2 ± 36.4
Weight (kg)	33.3 ± 29.7
Age (years)	12.6 ± 13.6
Size of ASD (mm)	21.5 ± 10.8
Operative Variables	
Duration of Surgery (min)	185.4 ± 49.0
CPB time (min)	70.9 ± 33.0
Aortic cross-clamp time (min)	40.1 ± 21.0
Mean Minimum Temperature (°C)	36.7 ± 0.4
Postoperative Variables	
Length of CICU stay (days)	1.4 ± 1.0
Length of Hospital Stay (days)	4.3 ± 2.7
Postoperative Results	
Complications (on patient-basis)	16 (19.0%)
Residual ASDs requiring reoperation (>2mm)	0

ASD: Atrial Septal Defect. CPB: Cardiopulmonary Bypass.

Table-2: individual type of ASD and the common morbidities.

Type of ASD	Frequency	Age (years)	Size of Defect (mm)	CPB Time (mins)	Arrhythmias	Pericardial Effusion	Pleural Effusion	Single Patch Repair	Two Patch Repair
Ostium Secundum	58	14.1 ± 15.2	24.5 ± 10.5 5- 63*	61.6 ± 26.4	A Fib. 1 SVT 1 JET 1	0	2	-	-
Ostium Primum	8	12.0 ± 8.5	11.7 ± 5.1 5-15*	124.5 ± 45.0	SVT 1 JET 1 CHB 1	1	0	-	-
Sinus Venosus	18	9.0 ± 11.7	12.3 ± 4.8 6-20*	77.3 ± 21.1	SVT 2	1	0	6	12

*Range of size of defect.
A. Fib= Atrial Fibrillation. SVT= Supraventricular Tachycardia.
JET= Junctional Ectopic Tachycardia. CHB= Complete Heart Block. CPB= Cardiopulmonary Bypass.

Table-3: Major postoperative complications and frequencies.

Morbidity	Frequency
Wound Infections	3 (3.6%)
Ventilation Support >48 hours	1 (1.2%)
Pneumonia	2 (2.4%)
Pericardial Effusion (requiring tube thoracostomy)	2 (2.4%)
Pneumothorax (requiring tube thoracostomy)	2 (2.4%)
Pleural Effusion (requiring tube thoracostomy)	2 (2.4%)
Reopening	1 (1.2%)
Arrhythmias	8 (9.5%)
Atrial Fibrillation	1 (1.2%)
Supraventricular Tachycardia	4 (4.8%)
Junctional Ectopic Tachycardia	2 (2.4%)
Complete Heart Block	1 (1.2%)
Heart Failure	1 (1.2%)
Readmission within 30 days	3 (3.6%)

the patients (N=80; 95.2%) were in New York Heart Association (NYHA) class I at follow-up, while the remaining 4(4.8%) were in NYHA class II.

Discussion

ASDs account for 10% of all cardiac malformations in childhood.[1] If left uncorrected, they may lead to premature death from congestive heart failure.[11] Long-term follow-up after surgical closure suggests that survival is comparable with that of age matched controls if surgery is performed in the first two decades of life or before the onset of pulmonary hypertension.[6] The decision to repair any kind of ASD is based on clinical and compiled echocardiographic information, including size and location of the ASD; haemodynamic impact of the left-to-right shunt and associated right-sided cardiac volume overload; and the presence and degree of pulmonary hypertension.

Surgical repair remains the standard for closure of secundum ASD, although more recently satisfactory results have been reported for transcatheter occlusion of these defects with a number of devices.[12,13] There are significant advantages to transcatheter closure in terms of a fewer complications, avoidance of cardioplegia and cardiopulmonary bypass, shorter hospitalisation time, reduced need for blood products and less patient discomfort. However, device embolisation is a major complication of this procedure which requires immediate surgical intervention for retrieval and correction of the heart defect.[14] In adults, there is very little evidence to support the benefits of transcatheter closure over surgical correction.[15]

Most of the surgical candidates in the study had been unsuitable for device closure primarily because of deficient inferior rims, or because the size of the ASD was too large. It is stressed that five rims must be assessed to decide the suitability for device closure: the aortic rim, superior rim, superior vena cava rim, inferior rim, and the inferior vena cava rim. In addition, a 5-mm rim of tissue around the defect is a prerequisite to prevent obstruction of the coronary sinus, right pulmonary veins, vena cavae, or atrioventricular valves.[14] It is a common practice of cardiologists to refer ASDs >20mm directly for surgery.

The present study compared the outcomes of 84 patients. There were no deaths in the study. In most patients, there was considerable improvement of symptoms post-operatively. Improvement were also seen in patients who were in NYHA class I before operation, which was in conjunction with other studies that stated functional improvement of previously asymptomatic patients after secundum ASD closure.[16] In our data, there seemed to be no link between age and onset of pulmonary hypertension since it was found in both paediatric and adult age groups. It is, however, suggested that surgery should be performed in the younger age group and

probably before structural changes in the myocardium or pulmonary vasculature occur.[11]

It is interesting to note that 13 patients (15.5%) who were admitted had a family history of ASDs. Though a small figure, the results are suggestive that there may be some correlation between familial genetic factor and the probability of developing a congenital heart anomaly. However, more data is required before a sound link can be established.

Surprisingly, most of the cases of arrhythmias were found in the paediatric age group. The exception was a 54-year-old female who suffered from atrial fibrillation.

The study shows that surgical closure of atrial septal defect is associated with low morbidity and a majority of patients were of NYHA class I on follow-up. Most importantly, this study demonstrates that ASD correction is associated with extremely low morbidity and mortality in older patients and almost none in younger patients. Hence, although ideally it is desirable that ASDs should be diagnosed and treated as early as possible, adult correction should not be ruled out as it has also proven to be beneficial.[6]

In terms of limitations, the retrospective nature of the study and missing the pre-op echocardiogram (ECG) for certain patients made it impossible to compare ECG changes before and after the surgery. Secondly, a majority of the patients were referrals from various regions of Pakistan, and, therefore, not all patients were available for late follow-up (>6 months).

Conclusion

Surgical repair of ASDs is a safe procedure which is associated with excellent results and low morbidity in younger as well as adult age groups.

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