HEMODYNAMIC CHANGES AND OUTCOME AFTER SURGICAL CLOSURE OF VENTRICULAR SEPTAL DEFECT IN INFANTS

Hijazi IS[1] & Al-Hakim FA[2]

ABSTRACT

Objective: This study aimed to review a ten years experience of surgical closure of ventricular septal defect in infants, assessing the postoperative clinical and hemodynamic results.

Patients and Methods: During the study period between Jan 1991 to Jan 2001,a total of 153 patients aged less than two years, underwent surgical closure of ventricular septal defect. Follow up evaluation include clinical symptoms, electrocardiogram, 2-dimensional echocardiography and cardiac catheterisation. The main indications for follow up cardiac catheterisation were: persistent respiratory symptoms, a clinical or echocardiography signs of residual leak across the VSD and improper weight gain.

Results: Follow up was completed in 150 patients. Cardiac catheterisation was performed in 42 patients. 132 patients (88.0%) had complete resolution of their preoperative symptoms. Complete closure of the ventricular septum was achieved in 115 patients (76.6%). 28 patients (18.7%) had small residual leak across the septum, 6(4%) patients had significant leak across the septum and needed a second operation to close the defect, and five patients (3.3%) had small leak and mild pulmonary hypertension, but the calculated left to right shunt was <1.5:1 and they needed medical treatment for one year. Persistent severe pulmonary hypertension was noticed in one patient who underwent atrial septostomy. The mean pulmonary arterial pressure dropped from 60 mmHg to 27 mmHg. Three patients (2.0%) had complete heart block and needed an insertion of permanent pacemaker. The hospital mortality of the entire group was 1.30 % and there was no late mortality.

Conclusion: Surgical closure of ventricular septal defects at an early age resulted in resolution of symptoms, satisfactory 2-dimensional echocardiography and Doppler findings and further reduction in pulmonary artery pressure as early as three months in most patients.

KEY WORDS: ventricular, infants, septum, pulmonary, hypertension.

Pak J Med Sci January-March 2005 Vol. 21 No. 1 17-21

1. Dr. Issa Saleh Hijazi MD
Paediatric Cardiologist
2. Dr. Fakhri Ahmad Al- Hakim MD, FACC
Chief & Consultant Paediatric Cardiologist
1-2: Paediatric Cardiology Division
Queen Alia Heart Institute,
King Hussein Medical Center,
Amman, Jordan

Correspondence:

Dr. Issa Saleh Hijazi
P. O. Box: 134,
Amman-11733, JORDAN
Email: ihijazi@hotmail.com

* Received for publication: September 22, 2004
Accepted: October 25, 2004

INTRODUCTION

Isolated ventricular septal defect (VSD) is the commonest lesion in the congenital heart disease, with an incidence of at least 12 per ten thousands live birth, and surgical closure of VSD is one of the most common open heart procedures performed in paediatric cardiac surgery[1].

The primary surgical repair of the ventricular septal defect in infancy has become increasingly common in recent years and is now the procedure of choice in many centres. Several

studies[2-4] have demonstrated a lower operative mortality for primary closure when compared with a two-staged approach with initial pulmonary artery banding. As outlined in earlier reports[5,6], direct surgical closure of a large VSD has been recommended for infants demonstrating intractable congestive heart failure, persistent or recurrent pneumonia, marked growth retardation and severe pulmonary hypertension (that is, pulmonary arterial pressure greater than 75% of the systemic pressure). This study aimed to review a 10 years experience with primary closure of ventricular septal defect (VSD) in infants, assessing pre and postoperative hemodynamic data and clinical follow up.

PATIENTS AND METHODS

The patient population consisted of 153 consecutive patients who had undergone surgical closure of isolated ventricular septal defect in Queen Alia Heart Institute, King Hussein Medical Centre, in Jordan during the period between 1992-2002 (Queen Alia Heart Institute is the national heart canter in Jordan with an average 130-150 surgery for congenital heart diseases each year). All patients have had regular follow up visit at two weeks, 6 weeks, and 12 weeks postoperatively and then accordingly every 3-9 months. During each visit the patient's evaluation include: clinical evaluation, 12-leads electrocardiogram, two-dimensional and Doppler echocardiography assessment of left ventricular end diastolic diameter (LVEDD), and leak across the VSD.A total of 42 patients underwent follow up cardiac catheterisation after the surgery. The time interval between surgery and re-catheterisation was ranging from 3 to 6 months. All the 42 patients underwent right and left heart catheterisation, oxygen saturations, pressure measurement, and calculation of pulmonary vascular resistance at rest and during oxygen administration for 20 minutes if needed; left ventricular angiogram to demonstrate the presence of residual leak were done.

RESULTS

Three patients were lost to follow up and they were excluded from the study. The follow up was completed in 150 patients (98%). There were 81 males and 69 females. The mean age at diagnosis was 1.4 months (Range, one day to 22 months). The mean age at surgery was 8.0 months (Range, 4.0 months to 24 months). Pulmonary artery banding was performed before corrective surgery in 4 children, all of which were performed in young infants with very low weights and in the same session underwent closure of associated patent ductus arteriosus in two patients and correction of coarctation of aorta in two patients. In the early postoperative period the hospital mortality rate of the all-entire group was 1.30%; we lost two patients: the first one aged 4 months died secondary to intractable heart failure and persistent low cardiac output, while the second patient aged 4.0 months died of Gram-negative septicaemia. Fortunately no late deaths were reported.

Clinically 132 patients (88.0%) had complete resolution of their preoperative symptoms while 11 patients continued to show respiratory symptoms, albeit less than their preoperative state, and 7 patients had failure to gain appropriate weight. Electrocardiogram showed complete heart block in three patients, all of which were managed by implantation of permanent pacemaker device. The finding on 2-dimensional echocardiography and Doppler colour study were: Complete closure of the defect in 116 patients (76.6%), small nonsignificant leak across the septum in 28 patients (18.6%), and large residual leak in 6 patients (4.0%). At follow up visit, the mean left ventricle end diastolic dimension (LVEDD) and left ventricle end diastolic dimension indexed for body surface area LVEDDi (LVEDD/BSA) were 2.4±0.3 cm and 7.5±0.7 cm respectively Considering both LVEDD and LVEDDi, the left ventricle size was normal in 121 patients (80.7%), borderline in 18 patients (12.0 %) and above normal in 11(7.30%). We performed

cardiac catheterisation follow up in 42 patients, the indications of the study were: failure to gain appropriate weight and / or persistence of respiratory symptoms in 11 patients and residual leak across the defect in 31 patients. The recatheterization study showed: small leak across the defect and trivial left to right heart shunt (pulmonary flow: systemic flow <1.3:1.0) in 30 patients while 6 patients had significant leak across the VSD, left to right shunt, (pulmonary flow: systemic flow) >2:1 and high pulmonary arterial pressure; needed a second attempt of closure. Five patients (3.3%) had small leak and mild pulmonary hypertension, but the calculated left to right shunt was significant >1.5:1 and they needed a treatment of digoxin and furosemide treatment for one year. One patient aged 1.2 year had a persistent severe pulmonary hypertension; needed surgical atrial septostomy to decompress the right heart and was kept on digoxin and furosemide treatment. Table-I shows the postoperative hemodynamic data following the VSD closure.

DISCUSSION

No controversy exists regarding early closure of an unrestrictive VSD in order to avoid pulmonary vascular obstructive disease, but surgical closure of a restrictive VSD in an asymptomatic patient with a modest or even small shunt is more controversial. In our group of patients all had a sizable VSD, left to right shunt, pulmonary hypertension and cardio respiratory symptoms, which made the surgical closure of the defects mandatory .In addition to the clinical improvement of cardio- respiratory status and growth, the two dimensional echocardiography and Doppler examination postoperative follow up of these patients had showed normal findings of the left ventricle mechanics in most patients. In his study, Pacileo G[7] et al. data suggest that, in the presence of a large ventricular septal defect, early successful surgical repair <2 years of age results in complete recovery of left ventricular mechanics in the postoperative follow-up. In contrast, surgical closure at >2 years of age, even for a moderately sized ventricular septal defect, deleteriously affects postoperative left ventricular geometry and shape. Pulmonary hypertension and high pulmonary vascular resistance were present preoperatively almost universally in this group of patients (table-II). While at follow up catheterisation; significant pulmonary hypertension, significant residual

Table-I: The postoperative hemodynamic data of the 42 patients, PAP (pulmonary arterial pressure), SP (systemic pressure), QP / QS (pulmonary / systemic flow ratio), PVR (pulmonary vascular resistance)

Age (month) Mean-range	*Sex Male/female*	*Weight (KG) Mean-range*	*PAP (mmHg) Mean-range*	*SP (mmHg) Mean-range*	*QP/QS Mean-range*	*PVR (unit) Mean-range*
11.6(8- 28)	23 / 19	7(5-14)	27(19-70)	90(75-110)	1.16(1.1-3.0)	2.5(2.8-8.6)

Table-II: The preoperative hemodynamic data of the 42 patients, PAP (pulmonary arterial pressure), SP (systemic pressure), QP / QS (pulmonary / systemic flow ratio), PVR (pulmonary vascular resistance)

Age (month) Mean-range	*Sex Male/female*	*Weight (KG) Mean-range*	*PAP (mmHg) Mean-range*	*SP (mmHg) Mean-range*	*QP/QS Mean-range*	*PVR (unit) Mean-range*
8(4-24)	81 / 69	6.0(3.5-11)	60(50-110)	90(65-110)	2.5(1.7-4.5)	6.5(3.4-9)

leak and high pulmonary vascular resistance were demonstrated in 4%, one patient developed severe persistent pulmonary hypertension without any leak; these results are consistent with other reports[7] which suggest that the risk of developing irreversible pulmonary vascular changes in the first year of life even in the presence of high PAP, high resting pulmonary vascular resistant (PVR) and large shunt is very low in infant with VSD as their primary cardiac malformation. Recently Zhu WH et al. came to a conclusion that an early operation may be the only way to decrease the incidence of pulmonary vascular disease in children with pulmonary hypertension due to ventricular septal defect.[8]

Re-operations because of patch leakage were performed in six children (4%), which is similar to the rate reported in other studies (0.6-4.7%).[9-15] Five patient without appropriate improvement after repair showed small leak, and normal pulmonary pressure but significant shunt across the defect; we could control their symptoms with medical therapy and in several months they grew out of the symptoms and the leak decreased significantly in two years. In G. Bol-Raap et al. patients trivial residual shunting disappeared spontaneously at a median follow-up time of 3.9 years and during follow-up no patient needed to be reoperated for residual VSD[16].

The occurrence of early postoperative arrhythmias after repair of congenital heart disease was significantly associated with procedure-related risk factors[17], the presumed mechanism is trauma to the atrioventricular node or the bundle of His before its bifurcation[18,19]. Twenty years ago the reported incidence of complete heart block after closure of VSD was ranging from 0-11%[6,20-23]. Our result 1.9% approaches those of recent studies[16].

In studies based on cases with surgery performed before 1980, primary closure of VSD in infant reported a mortality rate of 2.4% to 24%[6,19-21,25-27]; while in the last decade mortality rate is similar to the one seen in the present study; mortality rates ranging between 0% and 3.7% in later years.[6,9,16,28] The explanation for these improved surgical results is multifactor, related to factors such as improvements in the management of extracorporeal circulation in small children, improved surgical techniques, and better postoperative care.

CONCLUSION

Clinical,2-dimensional echocardiography and hemodynamic changes following the surgical closure of the ventricular septal defect at infancy showed significant improvement in symptoms and further reduction in the pulmonary pressure and pulmonary vascular resistance as early as 3 months in most patients, rarely some patients needed another attempt of closure of the residual VSD leak.

REFERENCES

1. King ME, de Moor M. Ventricular Septal Defect: Curr Treat Options Cardiovasc Med 1999; 1(4): 311-22.
2. Lamberti JJ, Lin CY, Cutilletta A, Anagnostopoulos CE, Arcilla RA, Replogle RL. Surface Cooling (20 C) and Circulatory Arrest in Infants Undergoing Cardiac Surgery. Arch Surg 1978;113:822-6.
3. Richardson JV, Schieken RM, Lauer RM, Stewart P, Doty DB. Repair of The Large Ventricular Septal Defects In Infants and Small Children. J Thorac Cardiovasc Surg 1981;195:318-22.
4. Hardin JT, Muskett AD, Canter CE, et al. Primary surgical closure of large ventricular septal defects in small infants. Ann Thorac Surg 1992;53:397-401.
5. Sigmann JM, Stern AM, Sloan HE: Early Surgical Correction of Large Ventricular Septal Defects. Pediatrics 1967; 39:4-13.
6. A Nygren, J Sunnegårdh, H Berggren. Preoperative evaluation and surgery in isolated ventricular septal defects: a 21 year perspective. Heart 2000;83:198-204.
7. Pacileo G, Pisacane C, Russo MG, Zingale F, et al. Left ventricular mechanics after closure of ventricular septal defect: influence of size of the defect and age at surgical repair.Cardiol Young 1998;8(3):320-8.
8. Zhu WH, Li JH, Zhu XK, Kang ML. Postoperative hemodynamic parameter follow-up in children with severe pulmonary resistance hypertension due to ventricular septal detect. Zhejiang Da Xue Xue Bao Yi Xue Ban. 2003;32(2):149-51, 166.
9. Knott-Craig CJ, Elkins RC, Ramakrishnan K, et al. Associated atrial septal defects increase perioperative morbidity after ventricular septal defect repair in infancy. Ann Thorac Surg 1995;59:573-8.

10. Haneda K, Sato N, Togo T, et al. Late results after correction of ventricular septal defect with severe pulmonary hypertension. Tohoku J Exp Med 1994;174:41-8.
11. Moller JH, Patton C, Varco RL, et al. Late results (30 to 35 years) after operative closure of isolated ventricular septal defect from 1954 to 1960. Am J Cardiol 1991;68:1491-7.
12. Henze A, Koul BL, Wallgren G, et al. Repair of ventricular septal defect in the first year of life. Scand J Thorac Cardiovasc Surg 1984;18:151-4.
13. Kuribayashi R, Sekine S, Aida H, et al. Long-term results of primary closure for ventricular septal defects in the first year of life. Jpn J Surg 1994;24:389-92.
14. Blake RS, Chung EE, Wesley H, et al. Conduction defects, ventricular arrhythmias, and late death after surgical closure of ventricular septal defect. Br Heart J 1982;47:305-15.
15. Sairanen H, Leijala M, Louhimo I. Surgery for ventricular septal defect. Scand J Thorac Cardiovasc Surg 1991;25:1-5.
16. G Bol-Raap, J. Weerheim AP, Kappetein M, Witsenburg & Bogers AJJC. Follow-up after surgical closure of congenital ventricular septal defect. Eur J Cardio-Thoracic Surg 2003;24(4)511-4.
17. Pfammatter JP, Wagner B, Berdat P, Bachmann DC, Pavlovic M, Pfenninger J, Carrel. Procedural factors associated with early postoperative arrhythmias after repairOf congenital heart defects. J Thorac Cardiovasc Surg 2002;123(2):258-62.
18. Lev M. The Architecture of the Conduction system in the congenital heart disease Ventricular Septal Defect. Arch Pathol 1960;70:529-49.
19. Lev M, Fell EH, Arcilla R, Weinberg MH. Surgical Injury to the conduction system in ventricular septal defect .AM J Cardiol 1964;14:464-76.
20. Blackstone EH, Kirklin JW, Bradley EI, Dushane JW, Appelbaum A. Optimal Age and Results in Repair of Large Ventricular Septal Defects. J Thorac Cardio Vasc Surg 1976; 72:661-79.
21. Arciniegas E, Farooki ZQ, Hakimi M, Perry BL, Green EW. Surgical closure of ventricular septal defect during the first twelve months of life. J Thorac Cardio Vasc Surg 1980; 921-8.
22. Chia J, Shears JH, Wu DC, Tan NC. Surgical management of ventricular septal defect in infancy. Am Acad Med 1980;9:463-7.
23. Agosti J, Subramanian S. Corrective treatment of isolated ventricular septal defect in infancy. J Pediatr Surg 1975;10:785-93.
24. Chia J, Shears JH, Wu DC, Tan NC. Surgical management of ventricular septal defect in infancy. Am Acad Med 1980;9:463-7.
25. Agosti J, Subramanian S . Corrective treatment of isolated ventricular septal defect in infancy. J Pediatr Surg 1975;10:785-93.
26. Rein JG, Freed MD, Norwood WI, Castaneda AR. Early and late results of closure of Ventricular Septal Defect in Infancy. Ann Thorac Surg 1977; 24:19-27.
27. Vidne BA, Kovalivker MP, Levy MJ. Surgical experience of closure of ventricular septal defect with special reference to the trans atrial approach. Int Surg 1979;64:49-51.
28. Backer CL, Winters RC, Zales VR, et al. Restrictive ventricular septal defect: how small is too small to close? Ann Thorac Surg 1993;56:1014-9.