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HEMODYNAMIC CHANGES AFTER SURGICAL CLOSURE OF VENTRICULAR SEPTAL DEFECT

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Summary

This is a prospective study that was carried out during the period between January 1990 to January 2000, to assess the hemodynamic changes and outcome after surgical closure of ventricular septal defect (VSD). Out of 150 patients who underwent surgical closure of VSD, 52 patients had follow up cardiac catheterisation. The main indications for recatheterisation were: persistent respiratory symptoms, clinical or echocardiographic signs of residual leak across the VSD, and improper weight gain. The time interval between recath and surgery was ranging from 3 to 6 months. Complete closure was achieved in 35 patients out of 52 (76%) who had been recatheterized, persistent severe pulmonary hypertension was noticed in one patient who underwent atrial septostomy, 4(8%) patients had significant leak across the defect and high pulmonary hypertension needed a second operation, 12(23%) patients had small residual leak. The mean pulmonary arterial pressure has dropped from 60mmHg to 27 mmHg. Two patients had complete heart block needed an insertion of permanent pacemaker. The hospital mortality of the entire group was 2% and there was no late mortality. In conclusion, hemodynamic changes following the surgical closure of the ventricular septal defect had shown 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. Surgical atrial septostomy might be needed in patients with persistent severe pulmonary hypertension to decompress the right side of the heart.

Introduction

Isolated ventricular septal defect (VSD) is one of the commonest lesions in the congenital heart disease, with an incidence of at least 12 per 10.000 live birth[1]. Although many defects close spontaneously, surgical repair is indicated for large defects and for significant associated lesions.

The primary surgical repair of the ventricular septal defect in infancy has increasingly common in recent years and

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is now the procedure of the 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 an earlier reports [5], direct surgery 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 VSDs in early childhood, assessing pre and postoperative hemodynamic data and clinical follow up.

Patients and Methods

Consecutive children undergoing VSD repair between 1990 to 2000, 150 were reviewed, 52 patients underwent cardiac recatheterization after the surgery.

The time interval between recatheterization were: high preoperative pulmonary arterial pressure and persistent respiratory symptoms in 30 patients (58%), failure to gain proper weight 13 patients (25%) and signs of residual leak either on clinical ground or echocardiography 16 patients (31%). Two patients (3.8%) died aged 4,6 months respectively early in the postoperative period due to intractable heart failure and low cardiac output, and sepsis in the other patient.

All the 52 patients underwent right and left heart catheterization, oxygen saturation, pressure measurement, calculation of pulmonary vascular resistance at rest and during oxygen administration test for 20 minutes if needed, and left ventricular angiogram to demonstrate the presence of residual leak preoperative (Table I).

Table I. The preoperative hemodynamic data of the 52 patients, PAP (pulmonary arterial pressure), SP (systemic pressure), QP/QS (pulmonary /systemic flow ratio), PVR (pulmonary vascular resistance).

Age (Yrs) : Mean age	4(0.4-7.8)
Sex : Male / Female	30 / 20
Weight (kg) : Mean range	12 (4-20)
PAP(mmHg):Mean– range	60 (50-110)
SP (mmHg) : Mean-range	90 (65-110)
Qp/QS: Mean – range	2.5 (1.7-4.5)
PVR (unit): Mean-range	6.5 (3.4-15)

Results

The recatheterization study showed complete closure of the defect in 35 patients (67%), 4 patients (8%) had significant leak across the VSD and high pulmonary arterial pressure; needed a second attempt of closure, 12 patients (23%) had small insignificant leak, 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 antifailure treatment (Table II).

Table II. The postoperative homodynamic data of the 52 patients, (pulmonary arterial pressure), SP (systemic pressure), QP/QS (pulmonary /systemic flow ratio), PVR (pulmonary vascular resistance).

Age (Yrs) : Mean age	4.2 (0.6-8)
Sex : Male / Female	30/22
Weight (kg) : Mean range	14.5 (5-22)
PAP(mmHg):Mean– range	27 (19-70)
SP (mmHg) : Mean-range	90 (75-110)
Qp/QS: Mean – range	1.16 (1.1-2.2)
PVR (unit): Mean-range	2.5 (2.8-12)

The hospital mortality rate of the all-entire group was 3.8%, the main causes of death were low cardiac output and sepsis. No late deaths were reported. Four (8%) patients developed a complete heart block; needed permanent pace-maker insertion.

Discussion

Pulmonary hypertension and high pulmonary vascular resistance were present preoperatively almost universally in this group of patients, at follow up catheterization; significant pulmonary hypertension, significant residual leak and high persistent pulmonary hypertension without any leak; this result is consistent with other reports [6,7] 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 PVR and large shunt is very low in infant with VSD as their primary cardiac malformation. While King et al.[1] suggested that surgical closure before 9 months of age is indicated for large VSDs and by 2 years of age for moderate shunts to prevent pulmonary vascular obstructive disease and the consequences of long standing volume overload.

Scott et al. [8] reported a 6% significant residual leak and approximately 25% small insignificant residual leak; these are comparable with our result that showed significant leak in 8% which required a second attempt of closure and insignificant leak in 23%.

The reported incidence of complete heart block after closure of VSD was ranging from 0-11% [6,9-12]. Our result 8% was comparable with these figures. The occurrence of early postoperative arrthythmias after repair of congenital heart disease was significantly associated with procedure-related risk factors[13], the presumed mechanism is trauma to the atrioventricular node or the bundle of his before its bifurcation [14,15]. Recent review of primary closure of VSD in infant reported a mortality rate of 2.4% to 24%[6,9-12,16,16,17]. The mortality rate in our study was 3.8% which comparable to the published figures.

A consistently higher mortality rate has been encountered among infants undergoing operation during the first few months of life although the mortality rate was higher among younger patients in our experience as well, the circumstances surrounding the death must be emphasized, intractable heart failure and severe respiratory difficulties were much more common among the younger infants.

Conclusion

Hemodynamic changes following the surgical closure of the ventricular septal defect had shown 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.

Surgical atrial septostomy might be needed in patients with persistent severe pulmonary hypertension to decompress the right side of the heart.

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