Comparison of VSD closure with single patch and flap valve patch in VSD patients with systemic pulmonary hypertensin at Rajaie Heart center.

G.R.OMRANI M.D, R BaghaiM.D,
B.Baharestani ; MD.MA Sheikhi ; MD.N.Givtage; MD

Abstract:

Back ground:

Back ground: closure of large ventricular septal defect (VSD) in children with elevalled pulmonary vascular resistance (PVR) is associated with significant morbidity and mortality. Surgical cure is likely to result in any infant in whom the VSD is repair before age 6 to 9 months, irrespective of degree of pulmonary vascular resistance .

Methods:

sixty-five patients (mean age of 21.20±52.08 month) with large size VSD and pulmonary hypertension Underwent VSD closure. That Double flap valve patch technique were used in 14 patients (21.7%).

Results:

After operation there was one Residual VSD needs Reoperation. Early mortality was 4.5% and RBBB and CHB were seen in 1.5% of patients.

Echocardiography 6months ofter operation showed that all double patches were closed.

Conclusion:

Double flap valve patch technique is a safe operation and can be used for closure of large VSD with the systemic pulmonary hypertension and morbidity of this technique is the same as single patch. It has a safety guard in early post operative period and we have to compare this two techniques in a double blind Experiment in the future

KEY word

VSD: ventricular septal defect is a hole or multiple holes in the inter ventricular septum. (1)

The closure of a large ventricular septal defect is usually performed, in most industrialized countries, at an early age before the onset of elevated pulmonary vascular resistance (PVR)1. Although the occurance of a pulmonary hypertensive crisis can contribute to morbidity and mortality. most children undergoing closure of a large VSD in infancy can expect excellent long-term results. In most medically sophisticated countries of the world / these pulmonary hypertensive events can be managed with either sophisticated pharmacologic agents such as nitric oxide or circulatory assist devices. Such as ECMO(2). Although children with a large VSD and elevated PVR are rarely seen in surgically advanced countries, they continue to represent a signtficant proportion of the congenital heart disease population in remainder of the word. The prognosis for children undergoing surgical closure of a large VSD with elevated PVR (pulmonary vascular resistant) is dependent upon the age and PVR at presentation (3) These children are at an increased risk for significant morbidity and mortality even when closure is performed in infancy (4)

Confronted with this problem. We designed a simple fenestrated flap valve VSD closure patch to reduce the morbidity and mortality associated with surging on children with a large VSD and increased PVR this report provides the intermediate results of this surgical method.

Patient and methods:

Sixty-five patients with a large VSD and pulmonary hypertension underwent VSD closure between 2001 and 2005 in Shaheed Rajaee hospital. Twenty-nine patients (44.6%) were female. The age of patients ranged from 6 months to 240 months with a median age of 21.20±5.08 months.

Department of Cardiac Surgery, Shahid Rajaee Heart Center, Tehran, Iran

Preoperative evaluation:

All children had a preoperative two-dimensional echo cardiographic Doppler evaluation, and cardiac catheterization.

We used double patch technique in 14 patients (21.7%) and single patch in 57 patients (77.3%)

Systolic pulmonary artery pressure and diastolic pulmonary pressure, that was measured by angiography was as same as intra operative data with non significant difference. Direction of shunt were left to right in 32 patients (50%) and right to left shunt in none.

Mean left ventricular ejection fraction that was estimated by preoperative echocardiography were 70.7±9% with minimum 80% and maximum 89%.

Other concomitant cardiac anomalies were seen in 26 patients (47.5%) and the most common concomitant cardiac anomaly was small PDA±LSVC.

Other non cardiac anomalies were seen in 4 patients (one cleft lip, one hirshprung disease , one club foot and one down syndrome

Most common type of the VSD's were perimembranous type in 33 patients (50%) and followed by sub aortic type in 19 patients (28.8%), inlet type in 6 patients (9.7%) muscular type in 4 patients (6.1%) and sub pulmonary in 3 patients (4.5%)

All of VSD's were large size**.**

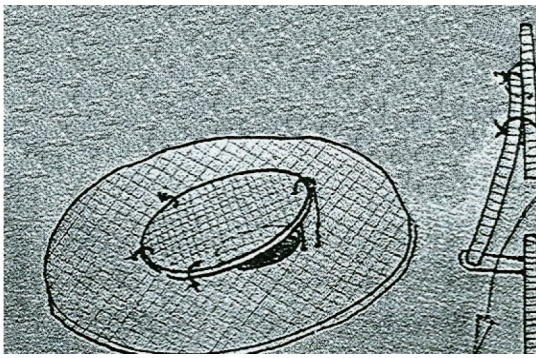
Operative managemeat

All surgical proccedures were performed by one surgeon. Routine CPB with hypothermia was employed for all cases.

Flap valve fenestrated (Double patch) patch was constructed of Gore-Tex used in 14 patient's (21.7%) and other patients repaired with single, Gore-tex patch (77.3%). Fenestration was placed in the lower third of VSD patch that tailored after inspection of the defect. The size of fenestration was determined according to the expected aortic Annulus diameter.

A separated flap patch at least 4mm larger than the fenestration was then constructed and sewn on to the superior margin of fenestration along one third of circumference. A separate tethering stitch was placed at the inferior apex of the flap valve and tied loosely oven a hegar dilator that was the same size as the fenestration. The VSD patch was then sewn into place using continuous suturing 4-0 prolen so that flap valve was placed on the left ventricular side and directing the flap so that it would open forward the LV apex.

Figure 12 : Illustration of double flap valve patch in profile with open valve



Post operative management:

Patients were allowed to awake from anoesthesia after operation, with millrinon infusion at medium dose. They extubated when they fulfilled criteriad for extubation. There were no significant differenc berween two groups for time of extobation and ICU stay.

Followup evaluation:

A minimum of 6 months followup with trous theracic echo cardiography poriodically were done for all patients.

Results:

Sixty-five patierts with a large VSD and pulmonary hypertension under went VSD obsure between 2001 and 2005 in Shaheed Rajaee hospital. Twenty-nine patients (44.6%) were female. The age of patients ranged from 6 months to 240 months with a mean age of 21.20±5.2 months, no significant differences were find in the sex and age of two groups.

After operation there was one residual inlet type VSD who returned to operating room for secondary closure: (It was missed by preoperative echocardiography). Trivial residual VSD were find in 17 patients that didn't needs reoperation and massive pericardial effusion needs drainage in 1 patients (1.5%) . RBBB in one patient CHB in 1 patient and early hospital death in 3 patient's (4.5%) that all of them were in single patch group. Fenestrations were closed at follow up echocardiography 6 months after operation.

Conclusion:

Pulmonary hypertentension is a sever complication of a large VSD. Historically, surgical closure of a large VSD with pullmonary hypertension is associated with high mortality rates. (4) and even in the recent era, post operative pulmonary hypertention remains a significant risk factor for morbidity and mortality however the evaluation of medical management of pulmonary hypertension after surgical correction of defects with preoperative pulmonary hypertension has resulted in a decrease in perioperative mortality, the cost of these treatment modalities is considerable. The creation of an intracardiac defect to prevent right ventricular failure has been used previously with variable success (5-6).

The modifiction we described allowed all patients to be extubated within 48 hours and there were no perioperative mortality in this group. (double patch group). The double patch flap valve modification provides a means of maintaining systemic cardiac output during pulmonary hypertentive episodes and preventing acute right ventricular volume overload.

We believe that this modification is analogous to the fenestration that were used previously. (7)

The results of closure of a large VSD in children with elevated PVR is well documented. (8) Carrent wisdom suggests that a PVR of at least 10 wood units is to be considered a contraindication to surgical intervention. The progression of pulmonary vascular disease after operative closure with the development of sever PHT and death is well known. (10) VSD closure in children with moderate to sever elevation in PVR preoperatively that decreases after closure of the defect has also been described.(11) previous authors (12-13) have reported that the immediate post repair and 1 year follow-up pulmonary artery pressure after closure of high-resistance, high pressure VSD's are related to the grade of Heath Edwards changes present at the time of repaire. The long-term survival of patients with severly elevated PVR and pulmonary artery pressure after closure of their defect is conteraversial. Cartaneda and colleagves (14) reviewed 55 patients with a high-pressure, high resistance VSD who underwent closure and found that of the 22 patients who underwent repeat cardiac catheterization at 1 year, the PVR and pulmonary artry pressures fell, but remained above normal valves.

Amajor ceneern when surgically treating patients with increased pulmonary artery pressure and eleveled PVR secondary to large left-to-right shunts is the possibility that patients with irreversible pulmonary vascular disease will under go operation.

Under such circumstances, the operation would unfavorably affect the natural history of Eisen minger's syndrome (15).

And lead to an earlier demise (10) . However recent reports of patients with primary PHT who have improved hemodynamics and exercise capability after the use of intravenous and high-dose oral calcium-channel blockers (16-17) or continuous intravenous prostacyclin infusion (18) suggest that pulmonary remodeling may take place especially in children (27) .we have shows that a unidirectional flap valve VSD patch allows for a low-risk closure of a large VSD in the presence of PHT and elevated PVR. Long term survival of these patients will depend on the degree of regression in PVR. The possibility that calcium channel blocker and prostacyclin could further help frduce PVR and pulmonary artry pressure.

References :

1. *oppenheimer-pekker A, Githen verpor-de Groot AC, Battelin MM, wenink AC, Moene RJ, Vander Haten JJ, Abnormal architecture of the ventriclos in hearts with an overriding aortic valve and a perimembranous ventricular septal defect int J cardiol 1985; 9:34/*

2. *Gold man AP, Delius RE , Dean field JE, Macrae DJ. Nitric oxide is superior to prostacydin for pulmonary hypertersion after cardiac operation. Ann thorac surg 1995; 60:300-5 discussion 306.*

3. *Blackstone EH, Kirklin JW, Bredley EL, Du shane JW, Appen lbaun A. Optimal age and results in repair af garge VSD. J cardiovasc surg 1976;72:661-79.*

4. *Bando K, Turrentine MW, sun K, et al. Surgical management of complete atrioventricular septal defects. A Twenty year experience. J thorac cardiovasc surg 1995;110:1543-54.*

5. *lin SF chiu IS, Hsu RB. Creation of a one-way interatrial communication in the treatment of critical pulmonary stenosis with intact ventricular septum: a cate report. J cardiac surg 1996;11:368-70.*

6. *Zhou Q Lai Y/ wei H/ song R/ WUY/Z hang H. unidirectional valve patch for repair of cardiac swptal defects with pulmonary hypertension Ann. Thorac surg 1995;60:1245-9.*

7. *Kirklin JW. Tricuspid Atresia and the Fontan operation. In: Kirlin JW/ Barratl-Boyes BG/eds. Cardiac surgery: morphology, diagnostic criteria, natural history techniques, results, and indications. New york: Churchill livingstone. 1997:1055-104.*

8. *I kawa S/ shima Zak: y/ Nalano Sq etal pulmonary vascular resistance during exercise late after repair of large ventricular septal defects. J. thora cardiovase surg 1995;109:1218-24.*

9. *De leval M. VSD. In : stark J, de leval M, eds. Surgery for congenital heart defects. Philadelphia: WB saunders , 1994;355-72.*

10. *Momma K, Takao A, Ando M, Nakazawa M, Takmizawa K. Natural and post operative history of pulmonary vascular obstruction associated with ventricular septal defect . Jpn circ J 1981;45:230-7.*

11. *Lillehei CW, Anderson Rc, Eltot R S, wangy. Ferlic RM. Pre- and postoperative cardiac catheterization in 200 patients undergoing closure of ventricular septal defects. Surgery 1968;63:69-76.*

12. Freid R, falkousky G, new burger J, et al.pulmonary arterial changes in patients with VSD and sever pulmonary hypertension. Pediatric cardiology 1986;7:147-5

13. Rabinovitch M, keane JF, norwood WI, castuneda AR, Reid L. vascular structure in lung tissve obtained at biopsy correlated with pulmonary hemodynamic findings ofter repair of congenital heart defects. Circulation 1984;69:655-67.

14. Castaneda AR, Zamora R, Nicoloff DM,et al.High pressure, high-resistance VSD. Ann thorac surg 1971;12:29-38.

***15.** Kidd L, Driscoll DF, Gersony WM,et all. Second natural history study of congenital heart defects : results of treatment of patients with VSD circulation 1993;87 (suppl 1):I-38-51.*

***16.** Malcuic I, Richter D.Verapamil in primary pulmonary hypertension. Br heart J 1985;53:343-7.*

***17.** Rich S/ Kaufmann E/ levy PS.The effect of high doses of calcium channel blockers on survival in primary pulmonary hypertension . N England 5 Med 1992;327:76-81.*

***18.** Barst Rj, Rubin LJ, long WA, et al. A comparison of continuous epoprostenal (prostacycline) with conventional therapy for primary pulmonary hypertension . N Engl. J Medi 1996;334:296-301.*