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Original Research Article

Effects on pulmonary arterial systolic pressure after surgical closure of ventricular septal defect

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ABSTRACT

Background: Pulmonary arterial hypertension (PAH) is a common complication of ventricular septal defect (VSD) caused by chronic left-to-right shunting and increased pulmonary blood flow. This study evaluated the effect of surgical VSD closure on pulmonary arterial systolic pressure (PASP) in patients with varying degrees of pulmonary hypertension.
Methods: This prospective observational study was conducted in the Department of Cardiac Surgery, Bangladesh Medical University (BMU), Dhaka, Bangladesh, from July 2022 to June 2024. Sixty patients with isolated VSD and pulmonary hypertension who underwent elective surgical closure were enrolled and divided into three groups according to preoperative PASP: Group A (36–45 mmHg), Group B (46–60 mmHg) and Group C (>60 mmHg). PASP was assessed by transthoracic Doppler echocardiography before surgery, on the first postoperative day, at one month and at three months after surgery. Data were analyzed using SPSS version 26 and a p value <0.05 was considered statistically significant.
Results: The mean age ranged from 5.98±4.31 to 6.33±3.88 years, with no significant differences in age or sex distribution among the groups. PASP decreased progressively following surgical closure in all groups. Significant reductions were observed on the first postoperative day and at one month (p<0.001). By three months, mean PASP decreased to 27.80±7.96 mmHg, 28.40±4.95 mmHg and 30.05±9.83 mmHg in Groups A, B and C, respectively, with no significant difference among the groups (p=0.285). One postoperative death occurred in the severe pulmonary hypertension group.
Conclusions: Surgical closure of VSD significantly reduces pulmonary arterial systolic pressure across all grades of pulmonary hypertension. Early surgical intervention may facilitate reversal of pulmonary hypertension and improve postoperative hemodynamic outcomes.

Keywords: Congenital heart disease, Echocardiography, Pulmonary hypertension, Pulmonary arterial systolic pressure, Surgical closure, Ventricular septal defect

INTRODUCTION

VSD is the most common congenital cardiac anomaly, accounting for approximately 20–40% of congenital heart defects diagnosed during infancy and childhood.[1] The defect permits left-to-right shunting of blood, producing pulmonary over circulation and increased pulmonary vascular pressure. The magnitude of the shunt depends on the size of the defect, ventricular compliance and pulmonary vascular resistance.[2] Small defects often remain hemodynamically insignificant, whereas moderate and large defects may cause progressive pulmonary

vascular remodeling, PAH, congestive heart failure, growth retardation and, ultimately, Eisenmenger syndrome if left untreated.[3] Pulmonary hypertension associated with congenital heart disease develops through chronic exposure of the pulmonary vasculature to increased blood flow and pressure. Sustained endothelial injury promotes smooth muscle proliferation, vascular remodeling, increased pulmonary vascular resistance and right ventricular pressure overload.[4] Once irreversible pulmonary vascular disease develops, surgical correction may no longer be beneficial and may even be contraindicated. Consequently, early identification and timely closure of significant VSDs remain the cornerstone of preventing irreversible pulmonary vascular changes.[5,6]

Advances in pediatric cardiac surgery, perioperative care and echocardiographic assessment have substantially improved outcomes after VSD closure. Surgical repair effectively eliminates left-to-right shunting, reduces pulmonary blood flow and facilitates gradual normalization of pulmonary arterial pressure.[7] Previous studies have demonstrated favorable postoperative reductions in pulmonary artery pressure; however, the rate and extent of recovery vary depending on patient age, preoperative pulmonary vascular resistance, defect characteristics and severity of pulmonary hypertension.[8,9] PASP, estimated noninvasively by Doppler echocardiography, is widely used for evaluating pulmonary hypertension before and after congenital heart surgery. Serial assessment of PASP provides valuable information regarding the reversibility of pulmonary vascular disease and postoperative recovery.[10] Despite increasing experience with congenital cardiac surgery in developing countries, evidence regarding postoperative PASP changes among Bangladeshi patients remains limited. Most published studies have focused on either severe pulmonary hypertension or long-term postoperative outcomes, whereas comparative data across different grades of pulmonary hypertension are scarce.[11,12] Furthermore, local evidence from Bangladesh is limited despite the substantial burden of congenital heart disease. Understanding the early postoperative hemodynamic response following VSD closure may facilitate better patient selection, optimize surgical timing and improve postoperative surveillance.

Therefore, the present study was undertaken to evaluate the effects of surgical closure of ventricular septal defects on pulmonary arterial systolic pressure among patients with mild, moderate and severe pulmonary hypertension and to compare postoperative hemodynamic improvement across different severity groups.

METHODS

Study design and setting

This prospective observational study was conducted in the Department of Cardiac Surgery, Bangladesh Medical University (BMU), Dhaka, Bangladesh, from July 2022 to June 2024.

Study population

The study included patients diagnosed with isolated ventricular septal defect associated with pulmonary hypertension who underwent elective surgical closure during the study period.

Inclusion criteria

It includes patients with isolated ventricular septal defect. Presence of pulmonary hypertension confirmed by Doppler echocardiography. Patients scheduled for elective surgical closure. Written informed consent from patients or legal guardians.

Exclusion criteria

Patients with associated congenital cardiac anomalies including atrial septal defect, tetralogy of Fallot, double-outlet right ventricle, significant valvular heart disease, right-to-left shunting, residual postoperative VSD, irreversible pulmonary vascular disease, pulmonary hypertension from non-cardiac causes or those requiring emergency surgery were excluded.

Sample size and sampling

A total of 60 patients fulfilling the eligibility criteria were enrolled using convenience sampling.

Sample size estimation was based on the expected prevalence of pulmonary hypertension among patients with ventricular septal defect and the standard formula for estimating a population proportion with a 95% confidence interval.

Patient grouping

Participants were categorized according to preoperative pulmonary arterial systolic pressure measured by transthoracic doppler echocardiography.

Group A

PASP 36–45 mmHg (mild pulmonary hypertension; n=20).

Group B

PASP 46–60 mmHg (moderate pulmonary hypertension; n=20).

Group C

PASP>60 mmHg (severe pulmonary hypertension; n=20).

Surgical procedure

All patients underwent standard median sternotomy under general anesthesia with cardiopulmonary bypass. Myocardial protection was achieved using cardioplegia. Ventricular septal defects were repaired according to standard institutional protocols using direct closure or patch closure depending on defect size and morphology. Cross-clamp time and total cardiopulmonary bypass time were recorded.

Data collection

Demographic information, clinical findings, operative variables and postoperative outcomes were recorded using a structured case record form. Preoperative echocardiographic evaluation included assessment of PASP and left ventricular ejection fraction. PASP was estimated using continuous-wave Doppler measurement of tricuspid regurgitation velocity according to the modified Bernoulli equation:

$$PASP = 4\,(TR\ velocity)^2 + estimated\ right\ atrial\ pressure$$

Measurements were performed at four predefined time points before surgery, first postoperative day, one month after surgery, three months after surgery. Additional perioperative variables included mechanical ventilation duration, cardiopulmonary bypass time, aortic cross-clamp time, postoperative hospital stay and mortality.

Outcome measures

The primary outcome was the change in pulmonary arterial systolic pressure following surgical closure of ventricular septal defect.

Secondary outcomes included postoperative ventilation duration, length of hospital stay, perioperative mortality and comparison of PASP reduction among patients with different grades of pulmonary hypertension.

Statistical analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 26. Continuous variables were expressed as mean±standard deviation, whereas categorical variables were presented as frequencies and percentages. Comparisons among groups were performed using one-way analysis of variance (ANOVA) for continuous variables and Chi-square test for categorical variables. Post hoc Bonferroni analysis was performed where appropriate. A p value<0.05 was considered statistically significant.

Ethical considerations

Ethical approval was obtained from the Institutional Review Board of Bangladesh Medical University before commencement of the study. Written informed consent was obtained from all participants or their legal guardians. Confidentiality was maintained throughout the study and all procedures complied with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

A total of 60 patients with isolated ventricular septal defect and pulmonary hypertension underwent surgical closure and completed the study. Patients were equally distributed into three groups according to preoperative pulmonary arterial systolic pressure (PASP): Group A (36–45 mmHg), Group B (46–60 mmHg) and Group C (>60 mmHg), with 20 patients in each group. The mean age was 5.98±4.31 years in Group A, 6.28±3.82 years in Group B and 6.33±3.88 years in Group C. Most patients (46.7%) were between one and five years of age. There was no statistically significant difference in age distribution among the groups (p>0.05). Male patients predominated slightly in all three groups, accounting for 55%, 60% and 50% of Groups A, B and C, respectively. The sex distribution was comparable among the study groups (p=0.675) (Table 1).

As expected, preoperative PASP differed significantly among the three groups according to the predefined classification (p<0.001). Mean preoperative PASP was 41.70±2.98 mmHg in Group A, 54.20±4.65 mmHg in Group B and 69.90±8.88 mmHg in Group C. However, left ventricular ejection fraction remained preserved in all patients, with no statistically significant intergroup difference (p=0.465). The mean aortic cross-clamp time was similar among the three groups, with no statistically significant difference (39.80±10.67 minutes in Group A, 38.90±10.99 minutes in Group B and 41.10±11.64 minutes in Group C; p=0.342). Patients with severe pulmonary hypertension (Group C) required significantly longer cardiopulmonary bypass time than those with mild or moderate pulmonary hypertension (88.70±18.70 minutes vs. 76.75±15.29 and 77.05±18.62 minutes, respectively; p<0.001).

The duration of postoperative mechanical ventilation did not differ significantly among the groups (6.48±2.16, 6.80±4.65 and 7.03±3.15 hours for Groups A, B and C, respectively; p=0.361). Similarly, postoperative hospital stay was comparable across the three groups, averaging approximately nine days (p=0.192). One postoperative death (5%) occurred in Group C, whereas no mortality was observed in Groups A or B. The difference was not statistically significant (p=0.541) (Table 2).

Serial echocardiographic assessment demonstrated a progressive decline in PASP following surgical closure of ventricular septal defect in all study groups. On the first postoperative day, mean PASP decreased to 39.10±7.77 mmHg in Group A, 40.15±6.94 mmHg in Group B and 55.10±11.36 mmHg in Group C (p<0.001). At one month after surgery, PASP declined further to 31.75±7.44 mmHg, 32.50±6.34 mmHg and 45.10±11.43 mmHg in

Groups A, B and C, respectively (p<0.001). By the third postoperative month, PASP had decreased to near-normal levels in all groups (27.80±7.96 mmHg, 28.40±4.95 mmHg and 30.05±9.83 mmHg, respectively). No statistically significant difference remained among the three groups (p=0.285), indicating substantial postoperative recovery irrespective of the initial severity of pulmonary hypertension (Table 3). PASP decreased progressively in all three groups following surgical closure of VSD. Significant intergroup differences were observed preoperatively, on the first postoperative day and at one month. However, by three months after surgery, PASP had decreased to near-normal values in all groups and the difference among the groups was no longer statistically significant (p=0.285), indicating substantial postoperative recovery irrespective of baseline disease severity. The mean PASP decreased significantly across all three groups at 3-month follow-up. Group A showed a reduction from 41.70 to 27.80 mmHg, representing a mean decrease of 13.90 mmHg (33.3%). Group B demonstrated a greater reduction, from 54.20 to 28.40 mmHg, with a mean decrease of 25.80 mmHg (47.6%). The greatest improvement was observed in Group C, where PASP decreased from 69.90 to 30.05 mmHg, corresponding to a mean reduction of 39.85 mmHg (57.0%), indicating that patients with higher baseline PASP experienced the largest absolute and relative reductions after surgery (Table 4).

Table 1: Baseline demographic characteristics of the study population.

Variables	Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Age (years), mean±SD	5.98±4.31	6.28±3.82	6.33±3.88	0.397
Age group, N (%) (in years)				
1–5	11 (55.0)	9 (45.0)	8 (40.0)	0.359
6–10	5 (25.0)	7 (35.0)	10 (50.0)	
11–17	4 (20.0)	4 (20.0)	2 (10.0)	
Sex, n (%)				
Male	11 (55.0)	12 (60.0)	10 (50.0)	0.675
Female	9 (45.0)	8 (40.0)	10 (50.0)	

Table 2: Comparison of postoperative outcomes.

Variables	Group A	Group B	Group C	P value
Mechanical ventilation (in hours)	6.48±2.16	6.80±4.65	7.03±3.15	0.361
Hospital stay (in days)	8.96±2.33	9.06±2.12	9.40±2.57	0.192
Mortality, N (%)	0 (0)	0 (0)	1 (5.0)	0.541

Table 3: Serial changes in pulmonary arterial systolic pressure after surgical closure.

Time points	Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Preoperative	41.70±2.98	54.20±4.65	69.90±8.88	<0.001
First postoperative day	39.10±7.77	40.15±6.94	55.10±11.36	<0.001
One month	31.75±7.44	32.50±6.34	45.10±11.43	<0.001
Three months	27.80±7.96	28.40±4.95	30.05±9.83	0.285

Table 4: Within-group reduction in pulmonary arterial systolic pressure following surgical closure.

Groups	Preoperative PASP	3-month PASP	Mean reduction (mmHg)	% Reduction
Group A	41.70	27.80	13.90	33.3
Group B	54.20	28.40	25.80	47.6
Group C	69.90	30.05	39.85	57.0

DISCUSSION

Pulmonary hypertension remains one of the most important determinants of morbidity and mortality among patients with ventricular septal defect. Persistent exposure of the pulmonary circulation to increased blood flow leads to endothelial dysfunction, vascular remodeling and progressive elevation of pulmonary vascular resistance.[13] Timely surgical closure of the defect interrupts this pathological process and promotes reversal of pulmonary hypertension before irreversible pulmonary vascular disease develops. In the present study, the majority of patients were younger than six years of age, reflecting the current trend toward earlier diagnosis and surgical intervention. Similar age distributions have been reported in previous studies

evaluating surgical outcomes of congenital heart disease, where early repair has been associated with improved postoperative hemodynamic recovery.[14,15] The baseline demographic characteristics were comparable among the three study groups, minimizing potential confounding effects. Left ventricular systolic function was preserved in all patients before surgery, suggesting that the observed postoperative improvements were primarily attributable to elimination of the left-to-right shunt rather than recovery of ventricular function. One of the principal findings of this study was the significant and progressive reduction in pulmonary arterial systolic pressure following surgical closure of ventricular septal defect. Improvement was observed as early as the first postoperative day and continued throughout the three-month follow-up period. These findings demonstrate that relief of pulmonary over circulation rapidly reduces pulmonary arterial pressure and allows gradual recovery of pulmonary vascular physiology.

Patients with severe preoperative pulmonary hypertension experienced the greatest absolute reduction in PASP. Although pulmonary pressure remained higher in this group during the early postoperative period, by three months there was no statistically significant difference compared with patients who had mild or moderate pulmonary hypertension before surgery. This observation suggests that even patients with markedly elevated pulmonary pressures may benefit substantially from surgical repair when irreversible pulmonary vascular disease has not yet developed.[16]

The findings are consistent with those reported by Talwar et al who demonstrated significant reductions in pulmonary artery systolic pressure immediately following VSD closure among patients with severe pulmonary hypertension.[17] Similarly, D'Alto et al reported marked postoperative improvement in pulmonary hemodynamics after closure of congenital systemic-to-pulmonary shunts.[18] More recently, Akilov et al also observed significant postoperative reductions in pulmonary artery pressure following surgical correction of congenital heart defects complicated by pulmonary hypertension.[19] Together, these studies support the concept that pulmonary vascular remodeling remains at least partially reversible following timely elimination of abnormal pulmonary blood flow.[20]

The significantly longer cardiopulmonary bypass time observed among patients with severe pulmonary hypertension probably reflects greater operative complexity and more meticulous intraoperative management required in these high-risk patients. Nevertheless, postoperative ventilation duration and hospital stay were comparable among the three groups, suggesting that increased operative complexity did not translate into prolonged postoperative recovery. Only one postoperative death occurred, involving a patient with severe pulmonary hypertension. Although mortality was numerically higher in this group, statistical significance was not reached because of the relatively small sample size. Larger multicenter studies are needed to determine predictors of postoperative mortality among patients with advanced pulmonary hypertension undergoing VSD closure.

The present study has several clinical implications. First, it reinforces the importance of early diagnosis and timely referral for surgical correction before irreversible pulmonary vascular disease develops. Second, serial echocardiographic assessment of PASP provides a practical and non-invasive method for monitoring postoperative recovery. Finally, even patients presenting with severe pulmonary hypertension may achieve excellent short-term hemodynamic improvement when appropriately selected for surgery.

This study has several limitations. It was conducted at a single tertiary-care center with a relatively small sample size and short follow-up period of three months. Pulmonary arterial pressure was estimated using Doppler echocardiography rather than routine right-heart catheterization during follow-up. Furthermore, convenience sampling may limit the generalizability of the findings. Future multicenter prospective studies with longer follow-up and invasive hemodynamic assessment are warranted to validate these results.

CONCLUSION

Surgical closure of ventricular septal defect significantly reduced pulmonary arterial systolic pressure in patients with mild, moderate and severe pulmonary hypertension. The findings support early surgical intervention to promote reversal of pulmonary hypertension and improve postoperative hemodynamic outcomes. Routine echocardiographic follow-up is recommended to monitor recovery after surgery.

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REFERENCES

1. Hoffman JIE, Kaplan S. The incidence of congenital heart disease. J Am Coll Cardiol. 2002;39(12):1890-900.
2. Penny DJ, Vick GW III. Ventricular septal defect. Lancet. 2011;377(9771):1103-12.
3. van der Linde D, Konings EEM, Slager MA, Witsenburg M, Helbing WA, Takkenberg JJM, et al. Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis. J Am Coll Cardiol. 2011;58(21):2241-7.
4. Baumgartner H, De Backer J, Babu-Narayan SV, Budts W, Chessa M, Diller GP, et al. 2020 ESC Guidelines for the management of adult congenital heart disease. Eur Heart J. 2021;42(6):563-645.

5. Galiè N, Humbert M, Vachiery JL, Gibbs S, Lang I, Torbicki A, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J. 2016;37(1):67-119.
6. Simonneau G, Montani D, Celermajer DS, Denton CP, Gatzoulis MA, Krowka M, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. Eur Respir J. 2019;53(1):1801913.
7. Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Colman JM, et al. 2018 AHA/ACC Guideline for the management of adults with congenital heart disease. Circulation. 2019;139(14).
8. Beghetti M, Galiè N. Eisenmenger syndrome: a clinical perspective in a new therapeutic era of pulmonary arterial hypertension. J Am Coll Cardiol. 2009;53(9):733-40.
9. Rosenzweig EB, Abman SH, Adatia I, Beghetti M, Bonnet D, Haworth S, et al. Paediatric pulmonary arterial hypertension. Eur Respir J. 2019;53(1):1801916.
10. Abman SH, Hansmann G, Archer SL, Ivy DD, Adatia I, Chung WK, et al. Pediatric pulmonary hypertension: guidelines from the American Heart Association and American Thoracic Society. Circulation. 2015;132(21):2037-99.
11. Kirklin JK, Barratt-Boyes BG. Cardiac Surgery. 4th ed. Philadelphia: Elsevier Saunders; 2013.
12. Mavroudis C, Backer CL. Pediatric Cardiac Surgery. 5th ed. Hoboken: Wiley-Blackwell. 2023.
13. Anderson RH, Baker EJ, Penny DJ, Redington AN, Rigby ML, Wernovsky G. Paediatric Cardiology. 4th ed. Philadelphia: Elsevier. 2020.
14. Haworth SG. Pulmonary hypertension in the young. Heart. 2002;88(6):658-64.
15. Dimopoulos K, Wort SJ, Gatzoulis MA. Pulmonary hypertension related to congenital heart disease. Eur Respir Rev. 2014;23(134):308-22.
16. Sluysmans T, Colan SD. Theoretical and empirical derivation of cardiovascular allometric relationships in children. J Appl Physiol. 2005;99(2):445-57.
17. Talwar S, Choudhary SK, Airan B. Surgical management of ventricular septal defect with pulmonary hypertension: early and mid-term outcomes. Asian Cardiovasc Thorac Ann. 2014;22(5):547-53.
18. D'Alto M, Mahadevan VS. Pulmonary arterial hypertension associated with congenital heart disease. Eur Respir Rev. 2012;21(126):328-37.
19. Akilov V, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2020;41(4):543-603.
20. Zoghbi WA, Adams D, Bonow RO, Enriquez-Sarano M, Foster E, Grayburn PA, et al. Recommendations for non-invasive evaluation of native valvular regurgitation. J Am Soc Echocardiogr. 2017;30(4):303-71.

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