RESEARCH PAPER

Evaluation of Left Ventricular Function Following Surgical Versus Interventional Ventricular Septal Defects Closure in Children: A Double Center Study in Egypt

Manar A. El Desouky[1], Eman Sharaf [1], Rania Abu Shokka [2], Doha Hesham Sayed Abbas[3]

[1] *Pediatric Department, October 6 University, Egypt.*
[2] *Consultant of Cardiology, National Heart Institute, Egypt.*
[3] *Pediatrics Resident, National Heart Institute, Egypt.*

ABSTRACT

Ventricular septal defect (VSD) is the most common congenital heart disease in pediatrics, and its closure can be achieved either surgically or via transcatheter intervention. Both approaches effectively eliminate left-to-right shunting but may have different impacts on early and late left ventricular (LV) function. Objective: to assess the early and follow-up changes in left ventricular systolic function and ventricular dimensions after surgical versus transcatheter closure of isolated ventricular septal defects (VSDs), as well as the time required for recovery of left ventricular systolic function and structural reverse remodeling following defect closure. Methods: This record-based study included 50 children aged 6 months to 5 years who underwent isolated VSD closure (25 surgical, 25 transcatheter) at two centers in Egypt between 2019 and 2025. Anthropometric, Clinical, and comprehensive echocardiographic data were systematically collected and reviewed. Statistical analysis was performed using appropriate parametric and non-parametric tests, with significance set at $p \leq 0.05$. Results: Both groups demonstrated significant improvement in LV dimensions and PAP over time ($p<0.001$). However, the surgical group exhibited a more pronounced early decline in EF (61.88 ± 3.07%) compared to the transcatheter group (66.20 ± 2.36%, $p<0.001$). Recovery of LV systolic function occurred significantly faster in the transcatheter group (3.28 ± 1.06 months) than in the surgical group (9.36 ± 0.95 months, $p<0.001$). Intensive care unit ICU stay was significantly longer in the surgical group ($p<0.001$). At follow-up, both groups showed recovery of LV function, although EF remained slightly higher in the transcatheter group ($p=0.037$). Larger VSD size was significantly associated with greater reductions in LV dimensions and PAP ($p<0.001$). Conclusion: Both surgical and transcatheter VSD closure are effective in achieving favorable long-term cardiac reverse remodeling and functional recovery. However, transcatheter closure is associated with milder early LV decline in function, faster recovery of LV systolic function, and shorter hospital stay. These findings support the preference for transcatheter intervention in appropriately selected patients.

Keywords: Ventricular septal defect; VSD closure; Transcatheter intervention; Surgical repair; Left ventricular function; Ejection fraction; Pediatric cardiology; Cardiac remodeling; Echocardiography; Congenital heart disease

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INTRODUCTION

Congenital heart disease (CHD) denotes anatomical and functional anomalies of the heart that are evident at birth. The worldwide incidence of congenital heart disease (CHD) is estimated at roughly 8 instances per 1,000 live births.1 Ventricular septal defects (VSDs) are the most prevalent congenital heart anomalies, constituting 20-30% of all congenital heart disease cases. They are categorized using various systems according to anatomical position, size, and hemodynamic significance.2 There are many nomenclature systems that have been developed for VSD types based on embryologic and anatomic principles. One of the famous classifications of VSD types is van Praagh classification Which classified the VSD into four major types3: (1)Cono-ventricular type (para-membranous or peri-membranous):it's the most common type of all VSD, (2)Atrioventricular canal types of VSD (Inlet type), (3)Conal or outlet defects, (4)Muscular VSD or (trabecular defects). The symptomatology and the clinical course of VSDs Patients primarily depending on the size of the defect, magnitude of left to right shunt, and the pulmonary vascular resistance. Infants and children with VSD mainly suffer tachypnea, Feeding Intolerance, Failure to thrive especially if the defect is significant. When auscultating the heart, a pansystolic murmur along the lower left sternal border is often heard louder in smaller defects. In larger defects, however, the murmurs may be quieter but there may be clear signs of ventricular overload and failure.4 If the VSD defect neglected can cause many complications. These include left ventricular volume overload, resulting in

**Author for Correspondence:*

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ventricular dilation and dysfunction; Congestion in pulmonary circulation leading to pulmonary hypertension, that is turned over time to irreversible vascular alterations (Eisenmenger's syndrome). Other complications include arrhythmias as a result of ventricular enlargement or distortion of the conduction system, infective endocarditis (especially in defects with high flow), and valve regurgitation (aortic or tricuspid) if the anatomical defect involves valvular structures.5 The treatment for VSD varies according to the case, depending on the size and anatomy of the defect, the significance of the shunt, the symptoms, the status of the ventricles and lungs, and any other associated cardiac defects. A small, hemodynamically insignificant ventricular septal defect (VSD) may be managed conservatively with regular follow up due to the potential for spontaneous closure.6 For children with moderate to large defects who are showing symptoms (such as congestive heart failure, failure to thrive, and rising pulmonary pressures), medical treatment: anti-failure (such as diuretics, nutritional support, and afterload reduction) may be started while management plans are being made for permanent closure. Open-heart surgery (patch closure) or, if the case is candidate for transcatheter (interventional) closure can both lead to definitive closure.7 For several decades, open-heart surgery remained the standard technique for repair of ventricular septal defects (VSDs). This major procedure involves thoracotomy and the use of cardiopulmonary bypass, with possible blood transfusion requirements. Surgical VSD repair has demonstrated high closure success and very low perioperative mortality rates in appropriately selected patients. 8 Over the past two decades, transcatheter device closure has become an increasingly effective minimally invasive alternative for both peri-membranous and muscular (trabecular) VSDs. The muscular subtype tends to respond particularly well to this approach, showing excellent closure rates and low procedural mortality across multiple studies.9 This evolution in management underscores the importance of comparing surgical and transcatheter techniques, not only in terms of efficacy and safety but also regarding long-term ventricular performance. Although catheter-based closure avoids many of the morbidities of open surgery, it introduces its own spectrum of complications, including residual shunt, arrhythmias, device migration, and potential ventricular dysfunction. Consequently, further research is warranted to clarify the impact of transcatheter closure on ventricular function, identify predictive factors for postprocedural dysfunction, and refine patient selection criteria to ensure optimal outcome.10

PATIENTS AND METHODS:

A record-based study included 50 children of both sexes, aged 6 months to 5 years, who underwent isolated ventricular septal defect (VSD) closure either surgically or by transcatheter intervention at the National Heart Institute and the Pediatric Cardiology Unit of October 6 University Hospital, Egypt, between 2019 and 2025. Clinical and echocardiographic data were obtained before VSD closure, immediately after the procedure during ICU stay, and during subsequent follow-up visits. The follow-up duration varied, as all patients were monitored until recovery of left ventricular (LV) function. Inclusion was restricted to patients with complete echocardiographic data at all assessed time points. Patients with incomplete records, associated congenital cardiac anomalies, myocardial involvement, or known pre-existing ventricular dysfunction were excluded to reduce potential confounding effects on cardiac function outcomes. All relevant medical records were systematically collected and reviewed. Demographic and anthropometric data included age at closure (in months), sex, weight (kg), height (cm), body surface area (BSA), and body mass index (BMI), all recorded on the day of admission prior to the closure either by surgery or catheter. Body surface area was calculated using the Mosteller formula.11 Anthropometric measurements were evaluated in accordance with World Health Organization (WHO) pediatric growth standards, with z-scores and percentiles determined for weight-for-age, height-for-age, and BMI-for-age. Clinical and hemodynamic data were also reviewed before and after the closure for clinical assessment of symptoms and signs of congestive heart failure, such as tachypnea, hepatomegaly, feeding difficulties, and failure to thrive, in addition to oxygen saturation and blood pressure measurements. Echocardiographic evaluation was performed at baseline before VSD closure, immediately after the procedure during the intensive care unit stay, and at late follow-up, when recovery of left ventricular ejection fraction and reverse remodeling of cardiac chamber dimensions were documented. Ventricular septal defect morphology, including defect site and size, was recorded. Assessment included two-dimensional, M-mode, color flow, and Doppler echocardiography using pediatric probes (3.5–7.5 MHz). The measured parameters were left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), left ventricular ejection fraction (EF%) calculated by M-mode, and pulmonary artery systolic pressure (PASP). Any associated valvular or structural cardiac lesions identified on echocardiography were also documented.

Ethical consideration

The study received approval from the Ethical Committee of the Faculty of Medicine, 6th of October University. The study objectives were explained to the parents of all participating children, and written informed consent was obtained prior to inclusion. Data confidentiality was strictly maintained throughout the study, and no additional risks were imposed on the participants. There is no conflict of interest related to this study. (Ethical Code: SCCREIRB-MEDICIN6OCT-PU-001-121224-018).

Statistical Analysis:

Statistical analysis was conducted utilizing IBM SPSS software version 20.0 (Armonk, NY: IBM Corp). Qualitative factors were described using frequencies and percentages, whilst quantitative variables were reported as mean ± standard deviation, median, and range (minimum–maximum) based on data distribution. The Kolmogorov–Smirnov test was employed to evaluate the normality of quantitative variables. Comparisons of categorical variables were conducted with the Chi-square test. The independent two-sample t-test was employed for regularly distributed

quantitative variables, whilst the Mann–Whitney U test was utilized for non-normally distributed variables. Spearman's correlation coefficient was employed for correlation analysis when applicable. Regression analyses were used to assess predictors of clinical outcomes. Binary outcomes were analyzed using univariate and multivariate logistic regression models, with results presented as odds ratios (OR) accompanied by 95% confidence intervals (CI). Linear regression models were employed for continuous outcomes, with results presented as unstandardized beta coefficients (β). The threshold for statistical significance was established at $p < 0.05$.

RESULTS:

Our study included 50 pediatric patients who underwent ventricular septal defect (VSD) closure at the National Heart Institute and the Pediatric Cardiology Unit at October 6 University Hospital in Egypt. Our sample was equally divided into two groups: 25 patients in the surgical group (group 1) and 25 in the transcatheter group (group 2). The study population included 35 males (70%) and 15 females (30%).

The mean age at the time of closure was 14.40 ± 14.59 months in group (1) and 44.00 ± 14.49 months in group (2). Regarding anthropometric measurements, the mean body weight was 7.56 ± 3.30 kg in group (1) and 14.00 ± 2.16 kg in group (2) , the mean body length was 70.76 ± 12.71 in group (1) and 95.44 ± 9.12 in group (2) , with a mean body surface area (BSA) of 0.38 ± 0.12 m^2 and 0.61 ± 0.08 m^2 in group (1) and (2) respectively , and with a mean body mass index (BMI) of 14.44 ± 1.96 in group (1) and 15.37 ± 0.93 in group (2) as shown in table 1 and figure 2.

Table 1: Descriptive analysis of the studied cases according to demographic and anthropometric measurement

	Group (1)	*Group (2)*
	N=25	***N=25***
Age of closure (months)	14.40 ± 14.59	44.00 ± 14.49
Weight (kg)	7.56 ± 3.30	14.00 ± 2.16
Weight Percentile	9.50 ± 9.94	21.62 ± 8.50
Length (cm)	70.76 ± 12.71	95.44 ± 9.12
Length Percentile	9.62 ± 10.84	17.30 ± 8.97
Weight Z-score	−1.40 ± 0.96	−0.63 ± 0.46
Length Z-score	−1.79 ± 0.99	−1.23 ± 0.33
BSA (m^2)	0.38 ± 0.12	0.61 ± 0.08
Body Mass Index	14.44 ± 1.96	15.37 ± 0.93

Data are presented as mean ± standard deviation (SD). BSA: body surface area; BMI: body mass index; kg: kilogram; cm: centimeter; m^2: square meter.

Patients of group (1) had significantly larger VSD size compared to group (2) (6.8 ± 1.6 vs 5.1 ± 0.8 mm, $p<0.001$), and also presented at an earlier age and had significantly shorter duration between onset of symptoms and closure of defect ($p<0.001$ for both). Comparison of clinical symptoms between the two groups showed that feeding intolerance was present in 68% of patients in the surgical group and 40% in the transcatheter group ($p = 0.088$). Recurrent infections occurred in 60% and 44% of patients in the surgical and transcatheter groups, respectively ($p = 0.396$). Rapid breathing was observed in 36% of the surgical group and 48% of the transcatheter group ($p = 0.567$). Failure to thrive was reported in 44% of surgical patients and 28% of transcatheter patients ($p = 0.377$). No statistically significant differences were found between the two groups regarding these symptoms.

Table 2: Clinical Data in all patients

	Group (1)	*Group (2)*	*P value*
	N=25	***N=25***	
VSD Size (mm)	6.80 ± 1.59	5.11 ± 0.79	<0.001*
Age of Onset (months)	3.48 ± 3.70	12.64 ± 5.47	<0.001*
Time interval between onset and closure (months)	10.80 ± 11.35	31.68 ± 12.00	<0.001*
Clinical symptoms			
Feeding intolerance	17 (68%)	10 (40%)	0.088
Recurrent infections	15 (60%)	11 (44%)	0.396
Rapid breathing	9 (36%)	12 (48%)	0.567
Failure to thrive	11 (44%)	7 (28%)	0.377

VSD: ventricular septal defect; N: number of patients; mm: millimeter.

* p-value ≤0.05 was considered statistically significant (95% confidence interval).

Within Group (1), statistically significant changes were observed in all echocardiographic parameters over time. LVEDD decreased progressively from 2.88 ± 0.59 preoperatively to 2.70 ± 0.58 postoperatively and further to 2.22 ± 0.46 at follow-up ($p<0.001$). Similarly, LVESD declined from 1.94 ± 0.46 to 1.90 ± 0.51 and then to 1.39 ±

0.36 (p<0.001). PAP showed a marked reduction from 37.88 ± 5.30 preoperatively to 30.20 ± 4.00 postoperatively and 23.28 ± 1.88 at follow-up (p<0.001). EF showed an early decline from 67.43 ± 3.59% to 61.88 ± 3.07%, followed by recovery to 67.04 ± 2.57% at follow-up (p<0.001). One patient (4%) developed transient early postoperative left ventricular systolic dysfunction (EF <55%).

Table 3: Serial echocardiographic changes within each study group at pre-closure, immediate post-closure, and follow-up

	Pre-closure	Post-closure	Follow-up	P value
Group 1				
LVEDD	2.88 ± 0.59	2.70 ± 0.58	2.22 ± 0.46	<0.001*
LVESD	1.94 ± 0.46	1.90 ± 0.51	1.39 ± 0.36	<0.001*
PAP	37.88 ± 5.30	30.20 ± 4.00	23.28 ± 1.88	<0.001*
EF (%)	67.43 ± 3.59	61.88 ± 3.07	67.04 ± 2.57	<0.001*
Group 2				
LVEDD	3.72 ± 0.36	3.61 ± 0.34	3.23 ± 0.29	<0.001*
LVESD	2.57 ± 0.25	2.53 ± 0.29	2.11 ± 0.20	<0.001*
PAP	34.20 ± 4.41	27.72 ± 2.17	23.12 ± 1.45	<0.001*
EF (%)	68.58 ± 2.62	66.20 ± 2.36	68.32 ± 1.46	<0.001*

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Table 4: Serial Echocardiographic Parameters comparison between the Two Groups at Pre-closure, Immediate Post-closure, and Follow-up

	Group (1)	*Group (2)*	*P value*
	N=25	***N=25***	
Pre-closure			
LVEDD	2.88 ± 0.59	3.72 ± 0.36	<0.001*
LVESD	1.94 ± 0.46	2.57 ± 0.25	<0.001*
PAP	37.88 ± 5.30	34.20 ± 4.41	0.01*
EF (%)	67.43 ± 3.59	68.58 ± 2.62	0.201
Immediately post-closure			
LVEDD	2.70 ± 0.58	3.61 ± 0.34	<0.001*
LVESD	1.90 ± 0.51	2.53 ± 0.29	<0.001*
PAP	30.20 ± 4.00	27.72 ± 2.17	0.01*
EF (%)	61.88 ± 3.07	66.20 ± 2.36	<0.001*
Follow-up			
LVEDD	2.22 ± 0.46	3.23 ± 0.29	<0.001*
LVESD	1.39 ± 0.36	2.11 ± 0.20	<0.001*
PAP	23.28 ± 1.88	23.12 ± 1.45	0.738
EF (%)	67.04 ± 2.57	68.32 ± 1.46	0.037*

LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; PAP: pulmonary artery pressure; EF: ejection fraction. * p-value ≤0.05 was considered statistically significant (95% confidence interval).

Before the closure, patients in group (2) showed significantly larger LVEDD and LVESD compared to group (2) (p < 0.001), while pulmonary artery pressure was significantly higher in group (1) (p = 0.01). No statistically significant difference was observed in ejection fraction between the two groups (p = 0.201). Immediately after the closure during the hospital stay, statistically significant differences were observed between the two groups in all

measured parameters. Group (2) maintained significantly larger LVEDD and LVESD values ($p < 0.001$ for both), whereas Group (1) had significantly higher PAP ($p = 0.01$) and significantly lower EF ($p < 0.001$). In group (1), one patient (4%) developed transient early postoperative left ventricular systolic dysfunction (EF <55%), whereas no cases of postoperative LV dysfunction were observed group (2). AT follow-up, both groups demonstrated improvement in ventricular dimensions and pulmonary artery pressure. However, LVEDD and LVESD remained significantly greater in Group (2) ($p < 0.001$), whereas PAP no longer showed a statistically significant difference between the two groups ($p = 0.738$). EF at follow-up was significantly higher in Group (2) than in Group (1) ($p = 0.037$), as showed in in table 4.

The length of hospital stay was shorter in group (2) than in group (1) ($p<0.001$). No complications were evident in group (2) in contrast to group (1) which showed Cardiogenic shock, arrhythmias, and hypotension occurred, as seen in in figure 1.

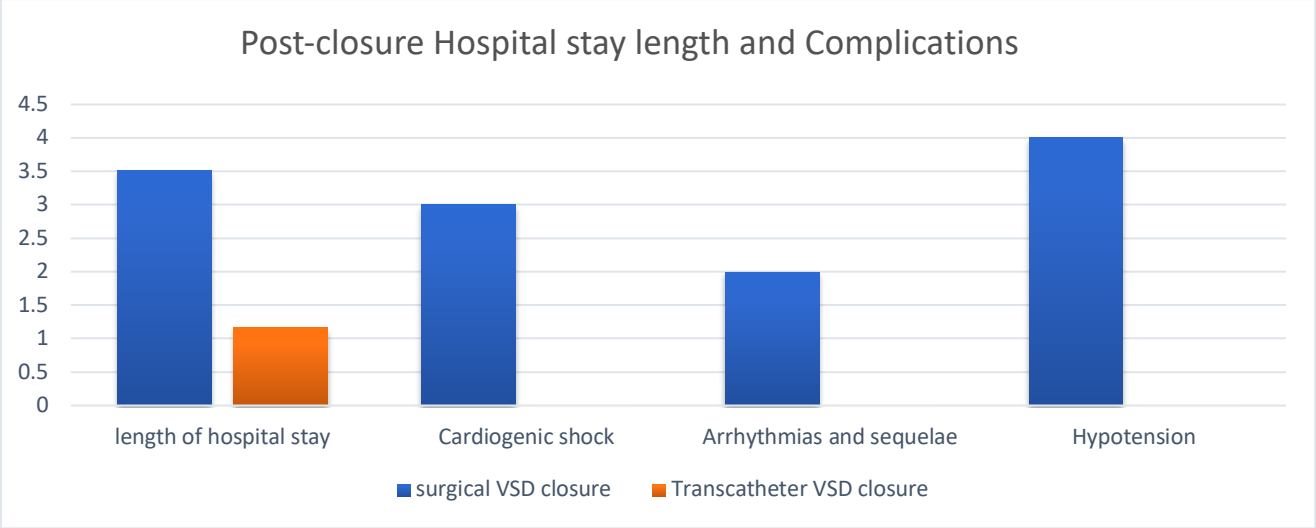

Figure 1: Post-closure Hospital stay length and Complications

Figure 2 showing the time to recovery of left ventricular systolic function was significantly shorter ($p < 0.001$) in the group (2) than in group (1). Patients in group (2) closure achieved recovery within 3.28 ± 1.06 months, whereas patients in group (1) required 9.36 ± 0.95 months.

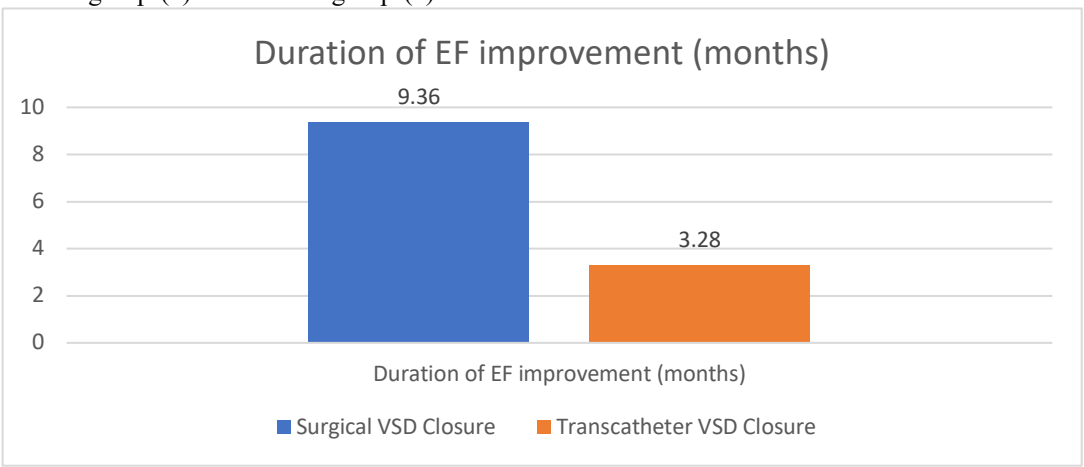

Figure 2: Time to Recovery of Left Ventricular Systolic Function in the two Groups

The correlation analysis demonstrated statistically significant positive correlations between VSD size and changes in left ventricular dimensions after closure, ΔLVEDD ($r = 0.591$, $p < 0.001$) and ΔLVESD ($r = 0.503$, $p < 0.001$) as seen in figure 3.

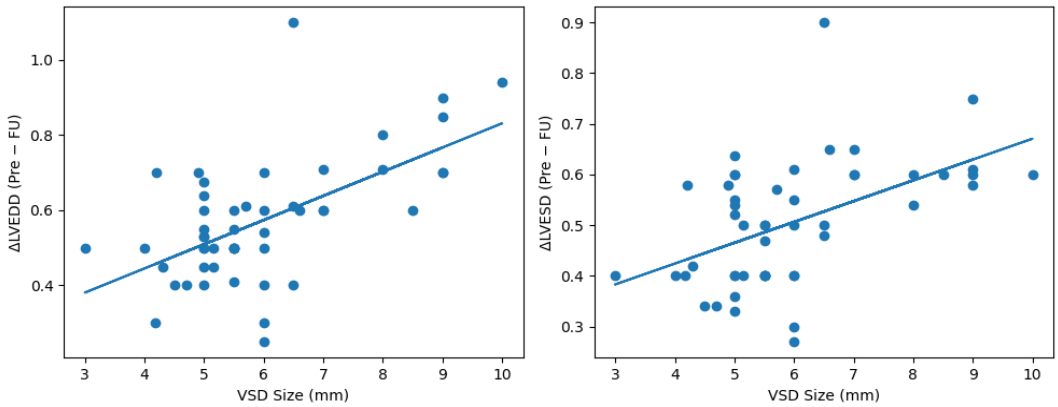

Figure 3: Combined scatter plots illustrating the relationship between ventricular septal defect (VSD) size and changes in left ventricular dimensions after closure. The plots demonstrate the associations between VSD size and the changes in left ventricular end-diastolic diameter (ΔLVEDD) and left ventricular end-systolic diameter (ΔLVESD) from pre-closure to follow-up

Figure 4 showing the Correlation analysis demonstrated a statistically significant positive correlation between VSD size and reduction in pulmonary artery pressure after closure ($r = 0.671$, $p < 0.001$).

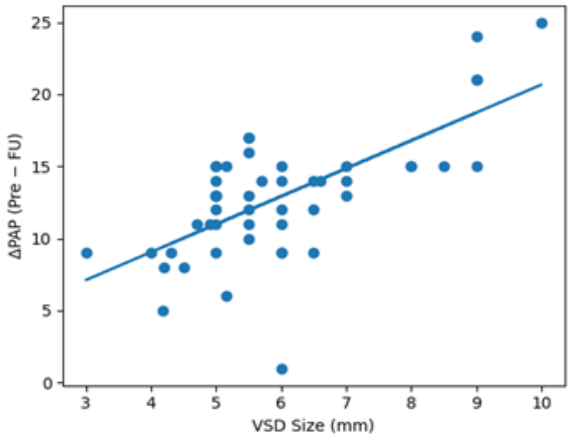

Figure 4: Combined scatter plots illustrating the relationship between ventricular septal defect (VSD) size and change in pulmonary artery pressure (ΔPAP) following closure. The plots demonstrate the correlation between VSD size and the difference in pulmonary artery pressure between pre-closure and follow-up measurements(ΔPAP)

Table 5 showing Multivariate logistic regression analysis was performed to identify independent predictors of postoperative complications. Age at closure was not a significant predictor (OR=0.716, 95% CI: 0.305–1.679, p=0.442). Although VSD size showed a relatively high odds ratio (OR=9.368), it did not reach statistical significance (p=0.168). Preoperative PAP was also not significantly associated with complications (OR=0.745, p=0.325). Cross clamp time demonstrated a borderline association with complication risk (OR=1.123, 95% CI: 0.987–1.277, p=0.079).

Table 5: Multivariate Logistic Regression for Risk of Any Postoperative Complication in Group 1

Predictor	*Odds Ratio (OR)*	*95% CI*	*p-value*
Age of closure (months)	0.716	0.305 – 1.679	0.442
VSD Size (mm)	9.368	0.390 – 224.905	0.168
Pre PAP	0.745	0.414 – 1.339	0.325
Cross clamp time	1.123	0.987 – 1.277	0.079

Author for Correspondence:

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OR: odds ratio; CI: confidence interval; VSD: ventricular septal defect; PAP: pulmonary artery pressure.
p-value ≤0.05 was considered statistically significant (95% confidence interval).

DISCUSSION:

Ventricular septal defect (VSD) is the most common congenital heart defect in children and is characterized by a defect in the interventricular septum leading to left-to-right shunting that imposes significant hemodynamic overload on the left ventricle (LV) and pulmonary vasculature12. In pediatric patients, hemodynamically significant VSDs may result in progressive left ventricular remodeling, congestive heart failure, failure to thrive, and pulmonary arterial hypertension necessitating prompt intervention to limit long term morbidity. Closure of hemodynamically significant VSDs can be achieved by either surgical or transcatheter intervention, with the choice of modality depending on patient characteristics, defect anatomy, and hemodynamic severity13, 14. Following closure, important hemodynamic and myocardial changes may occur due to elimination of the left-to-right shunt, which can transiently affect left ventricular systolic function before recovery and reverse remodeling take place15, 16.

Our study showed that the transcatheter group had more favorable anthropometric parameters than the surgical group, as reflected by higher weight percentiles and Z-scores for both weight and length, with the mean body weight was (7.56 ± 3.30 kg in group1 and 14.00 ± 2.16 kg in group2), the mean body length was 70.76 ± 12.71 in group (1) and 95.44 ± 9.12 in group (2). This is likely attributable to the older age at the time of closure in group (2) compared with the group (1) (44.00 ± 14.49 vs 14.40 ± 14.59 months), This finding is consistent with Kuswiyanto et al. (2022), who evaluated 132 children undergoing transcatheter VSD closure and reported a median age of 4.5 years and a median weight of 14.8 kg. These values are very close to those observed in our transcatheter group, supporting that our transcatheter cohort is comparable to other international device-closure series 17.

Patients undergoing surgical closure had significantly larger VSDs than those treated by the transcatheter approach (6.8 ± 1.6 vs 5.1 ± 0.8 mm, $p < 0.001$). This is consistent with Yi et al. (2018), whose network meta-analysis showed that VSDs reported in transcatheter series were generally smaller than those in open surgical cohorts18. In addition, the surgical group in our study had earlier symptom onset and a significantly shorter interval between symptom onset and defect closure ($p < 0.001$ for both), indicating that larger defects with earlier clinical presentation were more likely to be managed surgically. This interpretation is further supported by Turner et al. (2022), who identified defect size and morphology as major determinants of treatment selection, with larger or more complex defects being more suitable for surgical closure, whereas smaller defects with more favorable anatomy were more suitable for transcatheter closure. Together, these findings support the observation that larger and earlier symptomatic defects were more likely to undergo surgical closure13.

The current study demonstrated significant changes in left ventricular dimensions in both the surgical and transcatheter VSD closure groups, reflecting progressive reverse remodeling after defect closure. In the surgical group, LVEDD decreased from 2.88 ± 0.59 preoperatively to 2.70 ± 0.58 immediately postoperatively and further to 2.22 ± 0.46 at follow-up ($p < 0.001$), while LVESD declined from 1.94 ± 0.46 to 1.90 ± 0.51 and then to 1.39 ± 0.36 ($p < 0.001$). Similarly, in the transcatheter group, LVEDD decreased from 3.72 ± 0.36 pre-intervention to 3.61 ± 0.34 immediately post-intervention and further to 3.23 ± 0.29 at follow-up ($p < 0.001$), whereas LVESD declined from 2.57 ± 0.25 to 2.53 ± 0.29 and then to 2.11 ± 0.20 ($p < 0.001$). These changes are consistent with relief of chronic left ventricular volume overload, reduction in pulmonary over-circulation, and subsequent reverse remodeling following elimination of the left-to-right shunt. This interpretation is supported by Yuan et al. (2025), who reported rapid normalization of LV dimensions after transcatheter VSD closure in children, with the most pronounced reduction in LVEDD Z-scores occurring during the first postoperative month19. Notably, before closure, the transcatheter group had significantly larger baseline left ventricular dimensions, including both LVEDD and LVESD, than the surgical group ($p < 0.001$), which may be explained, at least in part, by the older age at intervention in the transcatheter group. A similar pattern noticed in Lasheen et al. 2023, which reported that in both surgical and transcatheter groups, LV diameters and volumes decreased over follow-up15.

The present cohort demonstrated significant changes in pulmonary artery pressure (PAP) within both groups. In the surgical group, PAP showed a marked reduction from 37.88 ± 5.30 preoperatively to 30.20 ± 4.00 immediately postoperatively and further to 23.28 ± 1.88 at follow-up ($p < 0.001$). Similarly, in the transcatheter group, PAP decreased significantly from 34.20 ± 4.41 pre-intervention to 27.72 ± 2.17 immediately post-intervention and further to 23.12 ± 1.45 at follow-up ($p < 0.001$). reflecting relief of chronic left ventricular volume overload, reduction in pulmonary over-circulation, and ongoing reverse remodeling after repair of the left-to-right shunt. Notably, before closure, PAP was significantly higher in the surgical group than in the transcatheter group ($p = 0.010$), suggesting a greater degree of pre-interventional pulmonary over-circulation and hemodynamic compromise in the surgical group. This is supported by Oleksandr et al. (2022), who studied children with VSD and severe pulmonary arterial hypertension undergoing surgical repair and showed that, despite acceptable mortality, pulmonary artery pressures often remained elevated long-term, with a substantial proportion of patients having systolic PAP ≥40

**Author for Correspondence:*

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mmHg at follow-up20. This persistence of higher PAP after surgery is concordant with the observation th at postoperative PAP in the present study remained significantly higher in the surgical group (p=0.010), supporting the view that these children constitute a higher-risk pulmonary vascular subset in whom PAP does not normalize immediately after defect closure. At follow-up echocardiography (showing recovery and left ventricular reverse remodeling there was absence of a significant difference in pulmonary artery pressure in both groups suggesting that both treatment modalities resulted in comparable hemodynamic recovery.

The findings of this study showed significant changes in left ventricular systolic function in both groups. In the surgical group, ejection fraction declined from 67.43 ± 3.59% preoperatively to 61.88 ± 3.07% immediately postoperatively, then recovered to 67.04 ± 2.57% at follow-up ($p < 0.001$), with one patient (4%) developing transient early postoperative left ventricular systolic dysfunction (EF < 55%). whereas the remaining patients exhibited only mild transient systolic decline in function that did not fulfill the criteria for true dysfunction. In the transcatheter group, ejection fraction also showed an early decline, from 68.58 ± 2.62% pre-intervention to 66.20 ± 2.36% immediately post-intervention, followed by recovery to 68.32 ± 1.46% at follow-up ($p < 0.001$), with no overt systolic dysfunction observed. The early decline in ejection fraction in both groups may be explained by acute alteration in left ventricular loading conditions following closure of the chronic left-to-right shunt. This is consistent with Adamson et al. 2020, that found postoperative LV dysfunction in 38% of infants after repair of large VSDs, with complete recovery of left ventricular ejection fraction during follow-up16. Before the VSD closure Ejection fraction was comparable in the two groups ($p = 0.201$); however, immediately after closure, ejection fraction was significantly lower in the surgical group than in the transcatheter group, indicating a greater early adverse effect on left ventricular systolic performance after surgery. This more pronounced early decline may be attributed to the effects of cardiopulmonary bypass, myocardial ischemia-reperfusion injury, and greater pre-closure shunt-related volume overload in the surgical group. At follow up echocardiography (showing recovery and left ventricular reverse remodeling), the ejection fraction recovered in both groups suggesting transient systolic adaptation with subsequent recovery. Although EF remained higher in the transcatheter group. (68.3% vs 67.0%, p=0.037). This pattern is consistent with the findings of Sinelnikov et al. (2019), who reported a significant immediate postoperative decline in ejection fraction and stroke volume in infants younger than 1 year after surgical VSD repair, with a subset developing marked systolic dysfunction. Compared with their series, the incidence and severity of dysfunction in our cohort were lower, which may be related to differences in age, defect characteristics, preoperative volume overload, or perioperative myocardial insult21.

Our study also demonstrated a significant difference in the immediate post-procedural course between the two groups, with patients in the surgical group requiring a significantly longer ICU stay than those in the transcatheter group. This likely reflects the greater invasiveness of surgical repair and the need for closer postoperative hemodynamic monitoring and supportive care. This finding is consistent with Yang et al. (2014), who reported that transcatheter closure of perimembranous VSDs was associated with faster recovery and shorter hospital stay compared with surgical repair22.

It was observed that no cases of postoperative fits, chest infection, or sepsis were observed in either group, whereas cardiogenic shock, arrhythmias, and hypotension occurred only in the surgical group. This is likely related to the limited sample size, suggesting a greater early perioperative burden after surgical closure. This observation is broadly consistent with Singab et al. (2023), who reported different complications between the two modalities, including complete heart block requiring temporary pacing in the surgical group and failed device deployment in the transcatheter group. The variation in the exact type and frequency of complications may be explained by differences in patient age, defect anatomy, baseline hemodynamic burden, procedural complexity, and sample size. Importantly, in our cohort, the patients with more marked postoperative deterioration in the surgical group had additional high-risk factors, including redo surgery, pre-existing left ventricular systolic dysfunction, and a prolonged operative course, all of which likely contributed to the severity of their early postoperative course23.

An important finding was observed that a significant difference in the time required for recovery of left ventricular systolic function between the two treatment groups. Recovery occurred significantly earlier in the transcatheter group than in the surgical group, with a mean recovery time of 3.28 ± 1.06 months compared with 9.36 ± 0.95 months, respectively. This suggests that transcatheter closure was associated with faster myocardial functional recovery, likely owing to the milder degree of early systolic impairment and the absence of cardiopulmonary bypass-related myocardial injury. The longer recovery interval observed in that group is likely attributable to the more pronounced early postoperative decline in left ventricular systolic performance after surgical repair. This finding is in agreement with Adamson et al. (2019), who reported recovery of postoperative left ventricular systolic function within 9 months after surgical VSD repair, supporting that the duration of recovery observed in our surgical group is comparable to published data and that recovery after surgery may be more prolonged than after transcatheter closure16.

The present study found significant positive correlations between VSD size and the magnitude of change in left ventricular dimensions after closure. Larger defects were associated with greater reductions in both LVEDD and LVESD from pre-closure to follow-up with significant correlations observed for ΔLVEDD ($r = 0.591$, $p < 0.001$) and ΔLVESD ($r = 0.503$, $p < 0.001$), reflecting more pronounced reverse remodeling after elimination of the left-to-right shunt. The positive correlation between VSD size and the magnitude of reduction in LVEDD and LVESD

after closure is likely explained by the greater pre-closure shunt burden imposed by larger defects. Larger VSDs produce more marked left-to-right shunting, resulting in greater left ventricular volume overload and cavity dilatation before closure. Following elimination of the shunt, these patients experience a greater reduction in preload and wall stress, leading to more pronounced left ventricular reverse remodeling during follow-up. This is supported by Yuan et al. (2025), who demonstrated rapid left ventricular reverse remodeling after transcatheter VSD closure in children and identified an association between VSD size and improvement in LV dimensions19.

The current study performed multivariate logistic regression to identify independent predictors of postoperative complications. Procedure type was not significantly associated with complication risk, likely due to the small number of events and wide confidence intervals. Similarly, age at closure and preoperative PAP were not significant predictors (all $p>0.05$). Although VSD size showed a relatively high odds ratio, it did not reach statistical significance. Cross-clamp time demonstrated a borderline association with complications ($p=0.079$), suggesting a possible trend toward increased risk with prolonged ischemic time; however, this did not achieve statistical significance.

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DECLARATION OF CONFLICTS OF INTERESTS

Author declares that they have no conflict of interest.

USE OF ARTIFICIAL INTELLIGENCE

Not applicable

REFERENCE

1. Eckerström, F.; Nyboe, C.; Maagaard, M.; Redington, A.; Hjortdal, V. E., Survival of patients with congenital ventricular septal defect. European Heart Journal 2023, 44 (1), 54-61.
2. Adan, A.; Eleyan, L.; Zaidi, M.; Ashry, A.; Dhannapuneni, R.; Harky, A., Ventricular septal defect: diagnosis and treatments in the neonates: a systematic review. Cardiology in the Young 2021, 31 (5), 756-761.
3. Brambilla, P. G.; Polli, M.; Pradelli, D.; Papa, M.; Rizzi, R.; Bagardi, M.; Bussadori, C., Epidemiological study of congenital heart diseases in dogs: Prevalence, popularity, and volatility throughout twenty years of clinical practice. PloS one 2020, 15 (7), e0230160.
4. Liu, H.; Wang, Z.; Xia, J.; Hu, R.; Wu, Z.; Hu, X.; Ren, W., Evaluation of different minimally invasive techniques in surgical treatment for ventricular septal defect. Heart, Lung and Circulation 2018, 27 (3), 365-370.
5. Anderson, R. H.; Spicer, D. E.; Mohun, T. J.; Hikspoors, J. P.; Lamers, W. H., Remodeling of the embryonic interventricular communication in regard to the description and classification of ventricular septal defects. The Anatomical Record 2019, 302 (1), 19-31.
6. Giblett, J. P.; Jenkins, D. P.; Calvert, P. A., Transcatheter treatment of postinfarct ventricular septal defects. Heart 2020, 106 (12), 878-884.
7. Lopez, L.; Houyel, L.; Colan, S. D.; Anderson, R. H.; Béland, M. J.; Aiello, V. D.; Bailliard, F.; Cohen, M. S.; Jacobs, J. P.; Kurosawa, H., Classification of ventricular septal defects for the eleventh iteration of the International Classification of Diseases—striving for consensus: a report from the International Society for Nomenclature of Paediatric and Congenital Heart Disease. The Annals of thoracic surgery 2018, 106 (5), 1578-1589.
8. Shan, W.; Yuanqing, X.; Jing, Z.; Xi, W.; Huifeng, G.; Yi, W., Risk factor analysis for adverse prognosis of the fetal ventricular septal defect (VSD). BMC Pregnancy and Childbirth 2023, 23 (1), 683.
9. Yamaguchi, A.; Shimamura, J.; Fukuhara, S.; Ueyama, H.; Takagi, H.; Kuno, T., Systematic Review and Meta-Analysis of Comparative Studies: Transcatheter Versus Surgical Closure for Postinfarct Ventricular Septal Defect: Transcatheter Versus Surgical Closure for PIVSD. Journal of Cardiac Surgery 2024, 2024 (1), 8159580.
10. Chen, C.-A.; Wang, J.-K.; Lin, M.-T.; Chiu, H.; Hsu, J.-Y.; Lin, S. M.; Chang, Y.-M.; Chiu, S.; Lu, C.-W.; Wu, M.-H.; Yang, M.-C., Exercise Capacity and Ventricular Remodeling After Transcatheter Ventricular Septal Defect Closure in Asymptomatic or Minimally Symptomatic Adolescents and Adults. 2020, 13.
11. Palamutoğlu, M. İ., BODY SURFACE AREA AND USAGE AREAS. Kocatepe Tıp Dergisi 2024.
12. Dakkak, W.; Alahmadi, M. H.; Oliver, T. I., Ventricular septal defect. In StatPearls [Internet], StatPearls Publishing: 2024.
13. Turner, M.; Bouhout, I.; Petit, C.; Kalfa, D., Transcatheter Closure of Atrial and Ventricular Septal Defects: JACC Focus Seminar. Journal of the American College of Cardiology 2022, 79 22, 2247-2258.
14. Jiang, D.; Han, B.; Zhao, L.-J.; Yi, Y.; Zhang, J.-J.; Fan, Y.; Lv, J.; ; WangJ.; Wang, Y., Transcatheter Device Closure of Perimembranous and Intracristal Ventricular Septal Defects in Children: Medium- and Long-Term Results. Journal of the American Heart Association: Cardiovascular and Cerebrovascular Disease 2021, 10.
15. Lasheen, S.; Ahmed, M.; Elgayar, M., Assessment of

Left Ventricular Systolic Function after Surgical and Transcatheter Device Closure of Ventricular Septal Defect Using Speckle Tracking Imaging. Menoufia Medical Journal 2023.

16. Adamson, G.; Arunamata, A.; Tacy, T.; Silverman, N.; ; Michael Maskatia, S.; Punn, R., Postoperative Recovery of Left Ventricular Function following Repair of Large Ventricular Septal Defects in Infants. Journal of the American Society of Echocardiography: official publication of the American Society of Echocardiography 2019.
17. Kuswiyanto, R.; Gunawijaya, E.; Djer, M.; Noormanto; Rahman, M.; Murni, I.; Sukardi, R.; Utamayasa, A.; Ardiansyah, R.; Nova, R.; Liliyanti, S.; Rahayuningsih, S.; Anggriawan, S.; Rahayuningsih, T. Y.; Koentartiwi, D.; Soewarniaty, R.; Yantie, V. K.; Nugroho, S.; Hidayat, T.; Ontoseno, T.; Tobing, T.; Ali, M.; Bashari, M.; Yosy, D.; Arafuri, N.; Hilmanto, D.; Yanuarso, P.; Advani, N.; Sastroasmoro, S.; Putra, S., Transcatheter Closure of Perimembranous Ventricular Septal Defect Using the Lifetech Konar-Multi Functional Occluder: Early to Midterm Results of the Indonesian Multicenter Study. Global Heart 2022, 17.
18. Yi, K.; You, T.; Ding, Z.-h.; Hou, X.-d.; Liu, X.-G.; Wang, X.-K.; Tian, J.-h., Comparison of transcatheter closure, mini-invasive closure, and open-heart surgical repair for treatment of perimembranous ventricular septal defects in children: a PRISMA-compliant network meta-analysis of randomized and observational studies. Medicine 2018, 97 (40), e12583.
19. Yuan, H.; Zhu, W.; Lv, J., Rapid left ventricular dimension normalization following transcatheter ventricular septal defect closure in children. Heliyon 2025, 11.
20. Oleksandr, G.; Lazorhyshynets, V.; Prokopovych, L.; Truba, Y.; Disessa, T.; Novick, W., Early and Long-term Results of Ventricular Septal Defect Repair in Children with Severe Pulmonary Hypertension and Elevated Pulmonary Vascular Resistance by Double or Traditional Patch Technique. European journal of cardio-thoracic surgery: official journal of the European Association for Cardio-thoracic Surgery 2022.
21. Sinelnikov, Y.; Orekhova, E.; Matanovskaya, T., Longitudinal mechanics as predictors of left ventricular systolic function in the early postoperative period after surgical perimembranous ventricular septal defect closure in children aged <1 year. Patologiya krovoobrashcheniya i kardiokhirurgiya 2019.
22. Yang J, Yang L, Yu S, Liu J, Zuo J, Chen W, et al. Transcatheter versus surgical closure of perimembranous ventricular septal defects in children: a randomized controlled trial. J Am Coll Cardiol. 2014;63(12):1159-1168.
23. Singab, H.; Elshahat, M. K.; Taha, A. S.;; Ali Y. A.; El-Emam, A. M.; Gamal, M. A., Transcatheter versus surgical closure of ventricular septal defect: a comparative study. The Cardiothoracic Surgeon 2023, 31 (1), 8.