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Predictors and outcomes of early left ventricular dysfunction in paediatric patients postoperative for ventricular septal defect: a retrospective cohort study based on myocardial strain and work

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Abstract

Background Early postoperative left ventricular (LV) dysfunction commonly occurs after ventricular septal defect (VSD) repair in paediatric patients. This study aimed to investigate its risk factors and impact on follow-up cardiac functional status.

Methods We retrospectively enrolled patients with preserved LV ejection fraction (LVEF) ≥ 55% undergoing isolated surgical VSD repair. Early postoperative LV dysfunction was defined as LVEF < 55% during hospitalisation. LV function parameters including myocardial strain and work were obtained. Cardiac functional recovery was assessed at 1, 3, 6, and 12 months post-discharge based on Ross Classifications.

Results The cohort comprised 409 patients (3.00 [1.00–5.75] years; 50.4% male), with 82 (20.05%) developing postoperative LV dysfunction. Multivariate analysis showed that preoperative LV global longitudinal strain (LV GLS), global work index (LV GWI), global work efficiency (LV GWE), LV end-diastolic volume index (LVEDVi), moderate-to-severe sPAP, and large VSD size, as well as prolonged cardiopulmonary bypass (CPB) time, were significantly associated with postoperative LV dysfunction (all $p < 0.05$). ROC analyses found that integrating LV GWI and GWE with clinical parameters (LVEDVi, VSD size, or sPAP) enhanced prediction (AUC 0.848–0.883, $P < 0.001$). During follow-up, patients with postoperative LV dysfunction exhibited less favourable Ross classification recovery at 12 months after surgery.

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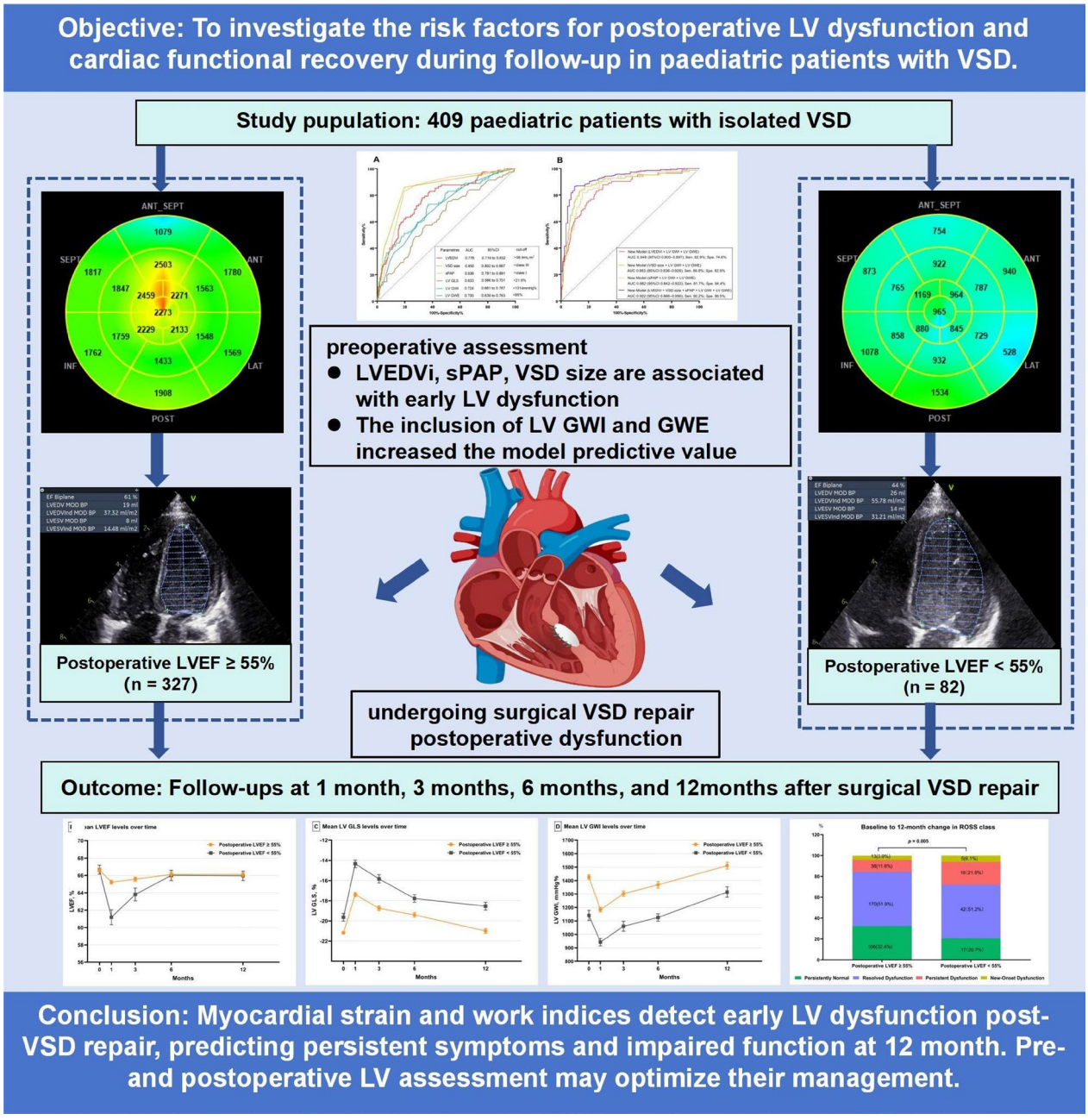
Conclusions Myocardial strain and work indices were related to early postoperative LV dysfunction in paediatric patients after isolated VSD repair, which was correlated with persistent symptoms and cardiac dysfunction at 12-month follow-up. Pre- and postoperative LV function assessment may offer useful information for managing this patient population.

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Keywords Myocardial strain, Myocardial work, Postoperative left ventricular dysfunction, Ventricular septal defect

Graphical abstract

LV, left ventricular; LVEF, left ventricular ejection fraction; LVEDVi, left ventricular end-diastolic volume index; sPAP, systolic pulmonary arterial pressure; VSD, ventricular septal defect; GLS, global longitudinal strain; GWI, global work index; GWE, global work efficiency



Introduction

Ventricular septal defect (VSD) is the most common congenital heart disease (CHD), comprising approximately 40% of all cardiac malformations [1]. Hemodynamically significant VSD can result in a series of complications, including left ventricular volume overload, pulmonary hypertension, arrhythmias, and heart failure, especially in pediatric patients [2, 3]. Surgical repair is currently the guideline-recommended treatment for patients with VSD, associated with favorable long-term survival [4]. However, a substantial proportion of patients (approximately 20–30%) may suffer from the early postoperative left ventricular (LV) dysfunction after VSD closure, which has significantly prolonged the duration of hospitalisation, mechanical ventilation and intensive care unit (ICU) stay [5–7].

Given these adverse impacts, there is an increasing need for dedicated markers to better predict the onset of short-term LV dysfunction. Moreover, it remains largely unknown if this rapid decline in LV function after surgery would affect the longer-term function recovery after discharge. Speckle-tracking-derived myocardial strain and myocardial work are emerging technologies for evaluating cardiac function, providing a more precise assessment of myocardial mechanics [8, 9]. Strain quantifies myocardial deformation using the contraction and relaxation phases [8], while work integrates strain with loading conditions for a comprehensive mechanical evaluation [9]. These techniques enable more sensitive assessment of volume overload and impaired cardiac function in paediatric populations [10, 11], and thus may hold value in this specific clinical scenario. We hypothesize that impaired preoperative myocardial strain and work indices are associated with early postoperative LV dysfunction, which correlates with persistent symptoms and impaired long-term recovery.

Consequently, this study aimed to (1) evaluate the association of LV myocardial strain and work with early postoperative LV dysfunction and assess their additive predictive values; (2) investigate the impact of the postoperative LV dysfunction on patient cardiac functional recovery after discharge based on Ross function class.

Methods

Study population

We retrospectively enrolled patients (aged 0–14 years) with preserved LVEF (≥ 55%) who underwent isolated surgical repair of VSD at Yunnan Fuwai Cardiovascular Hospital between September 2020 and December 2023.

Patients were excluded for: (1) additional CHD other than patent foramen ovale; (2) comorbidities affecting outcomes (severe arrhythmias/cardiomyopathy/left ventricular outflow tract obstruction); (3) transcatheter closure or non-surgery (severe irreversible pulmonary vascular resistance); (4) aortic regurgitation as primary indication; (5) prior cardiac surgery; (6) perioperative complications (residual shunt > 3 mm, low cardiac output syndrome, pulmonary hypertension crisis, cardiac tamponade); (7) missing/poor preoperative images; (8) post-discharge loss to follow-up. Exclusion criteria is detailed in Fig. 1.

Definition of early postoperative LV dysfunction

According to the Children's Heart Failure Study Group criteria established in the 2013 Canadian Cardiovascular Society guidelines, early postoperative left ventricular (LV) dysfunction was defined as an LV ejection fraction (LVEF) < 55% during the postoperative hospitalization period [12]. Moderate LV dysfunction was defined as LVEF < 50%, severe LV dysfunction was defined as LVEF < 40%, and preserved LV function was defined as LVEF ≥ 55% within the same timeframe.

Clinical data and surgical techniques

Demographic and clinical data were obtained from the institutional electronic medical records, including age, sex, height, weight, body surface area (BSA), systolic and diastolic blood pressure, heart rate (HR) and peripheral oxygen saturation (SpO_2). Standardized questionnaires were administered to assess heart failure symptoms, which were subsequently classified in severity using the Ross Classification for Children [13], with detailed criteria for each class provided in Table S1. All patients underwent isolated surgical VSD repair using cardiopulmonary bypass (CPB) under a standardized institutional protocol, including uniform cardioplegia and temperature management strategies. The recorded data included the date of surgery, operative approach, CPB time, aortic cross-clamp (ACC) time and number of ACC applications, duration of endotracheal intubation, perioperative complications, medications, and the lengths of hospital and ICU stay.

Surgical intervention details were documented in comprehensively, including VSD type and size, intracardiac incision approach, repair method, and patch material. VSD subtypes were classified according to standard recommendations [14], and defect size was categorised as small (< 1/3 of the aortic annulus diameter), moderate (1/3–1/2), or large (≥ 1/2) [15].

Echocardiographic data

All of the patients underwent preoperative transthoracic echocardiography (TTE) using a Vivid E95 ultrasound system (GE Vingmed Ultrasound AS, Horten, Norway) in accordance with the guidelines of the American Society of Echocardiography [16]. LVEF and LV volume were measured using the modified biplane Simpson method [16]. The severity of the associated secondary mitral

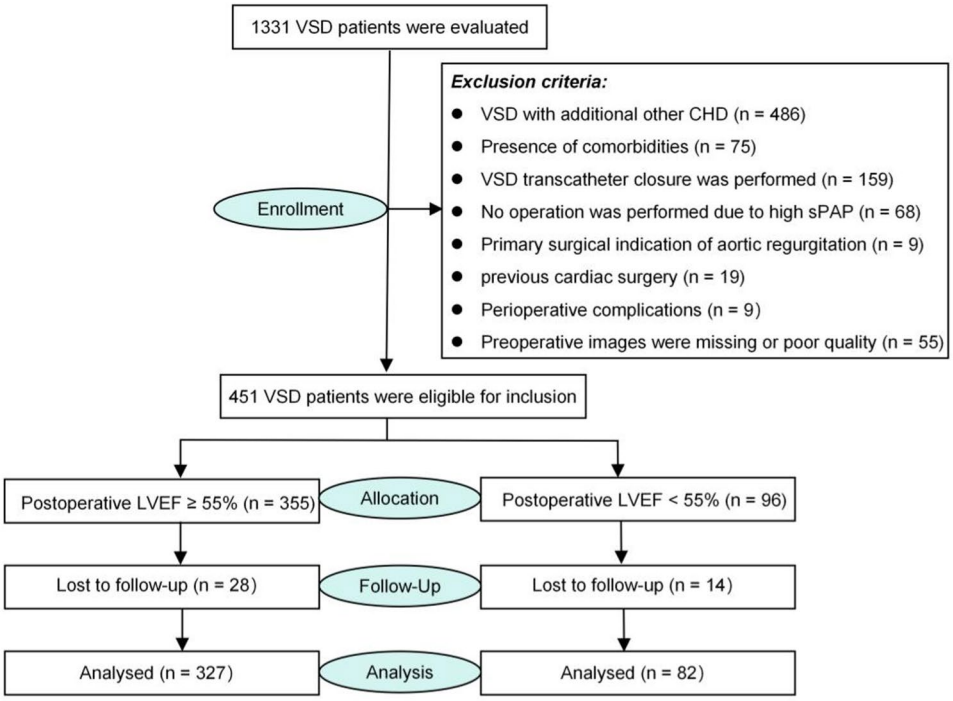


Fig. 1 Study flow chart. VSD, ventricular septal defect; CHD, congenital heart disease; sPAP, systolic pulmonary arterial pressure; LVEF, left ventricular ejection fraction

regurgitation (MR), tricuspid regurgitation (TR) and systolic pulmonary arterial pressure (sPAP) were evaluated and graded using a standardised approach and following current recommendations [17, 18].

Pressure-restrictive VSD was defined by continuous-wave Doppler as a peak interventricular gradient > 60 mmHg (equivalent to VSD peak velocity > 4.0 m/s) [19]. Image analysis was performed using an EchoPAC workstation (v. 204, GE Healthcare, Chicago, IL, USA) by investigators blinded to clinical outcomes and follow-up data. LV global longitudinal strain (GLS) was assessed with speckle-tracking echocardiography, and manual endocardial border tracing was conducted in standard apical two-, three-, and four-chamber views [8]. GLS will be described as an absolute value to standardize data analysis. LV global work index were derived by integrating LV GLS with a noninvasively estimated LV pressure curve to construct LV pressure-strain loops, the area of which represents total myocardial work per cardiac cycle. Peak systolic arterial pressure was obtained via real-time measurement (measured twice and averaged) and assumed equivalent to peak systolic LV pressure [9, 20, 21]. The following myocardial work indices were calculated: (1) LV global work index (GWI, mmHg%), the total work from mitral valve closure to opening; (2) LV global constructive work (GCW, mmHg%), work from systolic shortening and diastolic lengthening contributing to ejection; (3) LV global wasted work (GWW, mmHg%), energy expended from systolic lengthening and diastolic shortening opposing ejection; (4) LV global work efficiency (GWE, %), calculated as GCW/(GCW + GWW), reflecting constructive work proportion [9, 20, 21].

Follow-up

All patients underwent scheduled clinical follow-up at post-discharge, which included included: (1)High-quality TTE images underwent offline analysis per preoperative protocols at 1, 3, 6, and 12 months; (2)Standardized questionnaires were administered to assess symptoms and determine the Ross functional class pre- and postoperatively. To characterize the dynamic changes in Ross functional class, patients were categorized into four categories: Persistently Normal (Class I both pre- and postoperatively), Resolved Dysfunction (preoperative Class II/III improving to Class I postoperatively), Persistent Dysfunction(preoperative Class II/III remaining in Class II/III postoperatively), and New-Onset Dysfunction (preoperative Class I deteriorating to Class II/III postoperatively); (3)Discharge medications, cardiovascular complications and adverse events were systematically recorded, e.g., new-onset arrhythmia, rehospitalisation, heart failure and mortality at 12 month.

Observer variability

Intra- and inter-observer variabilities of LV GLS and myocardial work indices were assessed in a random sample of 20 patients by two echocardiographers in a blinded

fashion, and quantified using intraclass correlation coefficients (ICC).

Statistical methods

Continuous data are presented as mean ± standard deviation (SD) or median (interquartile range [IQR]), and categorical variables as n (%). Group comparisons (with vs. without postoperative LV dysfunction) utilized Student's t-test or Mann-Whitney U test for continuous variables (based on normality) and Chi-square or Fisher's exact test for categorical variables. Comparisons across LVEF threshold groups employed one-way ANOVA or Kruskal-Wallis H test (continuous) and Chi-square test (categorical). Univariable logistic regression was first performed to screen potential predictors of LV dysfunction. Variables with $p < 0.05$ were then considered for multivariable analyses. To minimize overfitting and address collinearity between correlated LV GLS and myocardial work indices, separate multivariable logistic regression models were constructed: each model included one of these parameters together with the other candidate variables to identify independent predictors. Multicollinearity was evaluated using variance inflation factors (VIF). Model fit and complexity were compared using Akaike information criterion (AIC) and Bayesian information criterion (BIC), and adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were visualized in forest plots (R). Receiver operating characteristic (ROC) curve analysis was used to assess the diagnostic accuracy of individual parameters and combined models, as well as the incremental value of echocardiographic parameters beyond clinical variables. The area under the curve (AUC), optimal threshold values, sensitivity, and specificity were calculated to quantify predictive performance.

Generalised estimating equations (GEE) was used for longitudinal repeated-measures analyses to evaluate changes in LV volumes and functional parameters over time, accounting for within-patient correlation and comparing time trends between groups. Changes in Ross classifications were analysed using the Wilcoxon signed-rank test. Intra- and inter-observer variability were quantified via intraclass correlation coefficients (ICCs), with 95% confidence intervals derived through the Bland–Altman method. *P*-values < 0.05 were considered statistically significant. All statistical analyses were conducted using SPSS for Windows, v. 27.0 (IBM Corp., Armonk, NY, USA) and R. v. 4.4.2 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Baseline clinical characteristics of study population

A total of 1,331 patients with VSD, 409 met the study criteria and were included in the analysis. The baseline demographic, clinical characteristics of the study population are presented in Table 1. The median age was 3.00 (1.00, 5.75) years, with 206 (50.49%) male patients. Postoperative LV dysfunction occurred in 82 (20.05%) patients. These patients were younger, had smaller BSA, higher HR, and were more frequently symptomatic (Ross class II/III) (all $p < 0.05$) compared to those without postoperative LV dysfunction. Perioperatively, they also exhibited larger VSD sizes, significantly longer CPB times, endotracheal intubation durations, greater requirement for inotropic medications, and longer hospital and ICU lengths of stay, with a majority undergoing patch repair (all $p < 0.05$).

Baseline echocardiographic characteristics of study population

The baseline echocardiographic characteristics are presented in Table 2. No significant differences in preoperative LVEF were observed between those who did and did not suffer postoperative LV dysfunction ($p > 0.05$). However, patients with postoperative LV dysfunction exhibited significantly larger LV end-diastolic volume index (LVEDVi) and cardiac index (all $p < 0.001$). Notably, lower LV GLS, GWI, GCW and GWE, and higher LV GWW were observed in patients with postoperative LV dysfunction (all $p < 0.05$). Moreover, they exhibited higher sPAP, a lower proportion of pressure-restrictive VSD, and more severe TR than those without postoperative LV dysfunction (all $p < 0.05$).

Additionally, patients were stratified by postoperative LVEF into four groups: LVEF ≥ 55% ($n = 327$, 79.95%), 50% ≤ LVEF < 55% ($n = 30$, 7.33%), 40% ≤ LVEF < 50% ($n = 32$, 7.82%), and LVEF < 40% ($n = 20$, 4.89%). Echocardiographic characteristics are shown in Table S2.

Factors associated with postoperative LV dysfunction

Univariable regression analysis and multicollinearity assessment of candidate predictors are presented in Table S3 and Figure S1. Across different multivariable models, larger VSD size, sPAP class II/III, and increased LVEDVi, along with prolonged CPB time (an intraoperative variable), were independently associated with postoperative LV dysfunction. Additionally, LV GLS (OR 1.15; 95% CI 1.02–1.29; $P = 0.017$), LV GWI (OR 0.86; 95% CI 0.76–0.97; $P = 0.017$), and LV GWE (OR 0.87; 95% CI 0.80–0.95; $P = 0.002$) were independently associated with postoperative LV dysfunction (Fig. 2). Model comparison using information criteria showed that the LV GWE model provided the best fit (AIC = 231.71; BIC = 267.81), with higher AIC/BIC values for the LV GWI and LV GLS models (Table S4). Fig. 3 shows representative cases with and without postoperative LV dysfunction.

Significant preoperative variables (LV GLS, GWI, GWE) discriminated postoperative LV dysfunction in ROC analysis, with AUCs of 0.633, 0.724, and 0.700,

Table 1 Baseline clinical characteristics of study population

Variables	Population (*n* = 409)	Postoperative LVEF ≥ 55%(*n* = 327)	Postoperative LVEF < 55%(*n* = 82)	*P*-value
Demographics				
Age, years	3.00(1.00, 5.75)	3.92(1.58, 6.00)	1.00(0.42, 2.00)	< 0.001
Male, n(%)	206(50.4)	172(52.6)	34(41.5)	0.071
Clinical datas				
BSA, m^2	0.59(0.39, 0.79)	0.64(0.47, 0.82)	0.34(0.31, 0.48)	< 0.001
HR, beats/min	105 ± 18	102 ± 18	114 ± 16	< 0.001
Systolic BP, mmHg	97 ± 11	97 ± 11	95 ± 10	0.103
Diastolic BP, mmHg	62 ± 10	62 ± 10	60 ± 10	0.134
Ross class I/II/II, n(%)	141(34.5)/252(61.6)/16(3.9)	119(36.4)/201(61.5)/7(2.1)	22(26.8)/51(62.2)/9(11.0)	0.014
SpO_2, %	96 (95, 98)	96 (95, 98)	96 (95, 98)	0.942
Surgical intervention details				
VSD type 1/2/3/4, n(%)	325(79.5)/2(0.5)/10(2.4)/72(17.6)	262(80.1)/0(0.0)/7(2.1)/58(17.7)	63(76.8)/2(2.4)/3(3.7)/14(17.1)	0.069
VSD size I/II/III, n(%)	133(32.5)/164(40.1)/112(27.4)	129(39.4)/152(46.5)/46(14.1)	4(4.9)/12(14.6)/66(80.5)	< 0.001
Chest wall incision approach 1/2, n(%)	379(92.7)/30(7.3)	299(91.4)/28(8.6)	80(97.6)/2(2.4)	0.057
Intracardiac incisions approach 1/2/3, n(%)	346(84.6)/15(3.7)/48(11.7)	276(84.4)/9(2.8)/42(12.8)	70(85.4)/6(7.3)/6(7.3)	0.066
Repair method 1/2, n (%)	95(23.2)/314(76.8)	92(28.1)/235(71.9)	3(3.7)/79(96.3)	< 0.001
Patch material 1/2/3, n(%)	66(21.0)/54(17.2)/194(61.8)	51(21.7)/43(18.3)/141(60.0)	15(19.0)/11(13.9)/53(67.1)	0.510
CPB time, min	74(59, 96)	70(57, 89)	94(76, 116)	< 0.001
ACC time, min	40(30, 63)	37(28, 54)	63(43, 78)	< 0.001
Number of ACC>2, n%	10(2.4)	6(1.8)	4(4.9)	0.232
Perioperative status				
Duration of endotracheal intubation, hours	8(6, 12)	8(6, 10)	14(8, 31)	< 0.001
milrinone therapy, n%	227(55.5)	165(50.5)	62(75.6)	< 0.001
bosentan/ambrisentan or sildenafil therapy, n%	15(3.7)	9(2.8)	6(7.3)	0.101
ICU length of stay, days	1(1, 3)	1(1, 2)	3(1, 7)	< 0.001
Hospitallength of stay, days	15(11, 19)	14(11, 17)	19(13, 25)	< 0.001

Statistics are mean ± standard deviation, median (IQR), or number (%)

BSA Body surface area, *HR* Heart rate, *BP* Blood pressure, *SpO_2* Peripheral oxygen saturation, *VSD* Ventricular septal defect, *CPB* Cardiopulmonary bypass, *ACC* Aortic cross-clamp, *ICU* Intensive care unit

VSD type1, Central perimembranous; VSD type 2, Inlet; VSD type 3, Trabecular Musucular; VSD type 4, Outlet; VSD size I, Small; VSD size II, Moderate; VSD size III, Large; Chest wall incision approach 1, Median sternotomy; Chest wall incision approach 2, Right axillary incision; Intracardiac incisions approach 1, Right atrial incision; Intracardiac incisions approach 2, Right ventricular outflow tract incision; Intracardiac incisions approach 3, Main pulmonary artery incision; Repair method 1, direct suture; Repair method 2, patch repair; Patch material 1, autologous pericardium; Patch material 2, synthetic patch; Patch material 3, bovine pericardium

respectively (all $P < 0.001$) (Fig. 4A). Multi-model ROC curve analyses found that integrating LV GWI and GWE with clinical parameters (LVEDVi, VSD size, or sPAP) provided superior incremental predictive value for postoperative LV dysfunction. All combinations achieved excellent AUCs (range 0.848–0.883; $P < 0.001$ for each) with sensitivity (81.7–86.8%) and specificity (74.6–84.4%) (Fig. 4B). ROC analysis repeated using the lower LV dysfunction threshold (LVEF < 50% vs. <40%) confirmed that the predictive value of the identified preoperative variables (Table S5).

Follow-up assessment of progression in LV measurement

A total of 409 patients contributed 1,181 follow-up examinations, with completion rates of 91.7%, 70.9%, 63.3%, and 62.8% at 1, 3, 6, and 12 months, respectively. Echocardiographic evaluation revealed significant changes in LV volume and function 1, 3, 6 and 12 months postoperatively (Table S6, Fig. 5). Of the 82 patients who developed postoperative LV dysfunction, LVEF normalized in 68/82 (82.9%) by 1 month, 80/82 (97.6%) by 3 months, and 82/82 (100%) by 6 months. During the 1–3 months, the dysfunction group exhibited significantly elevated LVEDVi and reduced LVEF compared to the non-dysfunction group (all $p < 0.05$); by 6–12 months, both parameters normalized with no significant intergroup differences (all $p > 0.05$). However, the postoperative LV dysfunction group displayed persistently poorer LV GLS, GWI and GCW than the non-dysfunctional group throughout the follow-up period (all $p < 0.05$). Figure S2 illustrates representative cases with and without postoperative LV dysfunction, showing the pre- and post-operative changes in the myocardial work indices over time.

Table 2 Baseline echocardiographic characteristics of study population

Variables	Population (*n* = 409)	Postoperative LVEF ≥ 55%(*n* = 327)	Postoperative LVEF < 55%(*n* = 82)	*P*-value
VSD complications				
pressure-restrictive VSD	182(44.5)	179(54.7)	3(3.7)	< 0.001
sPAP class None/I/II/III, n(%)	287(70.2)/23(5.6)/40(9.8)/59(14.4)	271(82.9)/16(4.9)/20(6.1)/20(6.1)	16(19.5)/7(8.5)/20(24.4)/39(47.6)	< 0.001
MR 3 + ~ 4+, n(%)	8(2.0)	4(1.2)	4(4.9)	0.091
TR 3 + ~ 5+, n(%)	6(1.5)	2(0.6)	4(4.9)	0.018
Echocardiographic parameters				
IVSd, mm	5.0 ± 0.9	5.0 ± 0.9	4.9 ± 0.9	0.252
LVPWd, mm	5.0 ± 0.9	5.0 ± 0.9	4.8 ± 0.9	0.063
LVEDVi, mL/m^2	58.4 ± 23.3	53.6 ± 18.3	77.4 ± 30.7	< 0.001
Clz	3.8 ± 2.1	3.2 ± 1.9	5.9 ± 1.8	< 0.001
FS, %	36.1 ± 3.3	36.1 ± 3.2	36.0 ± 3.4	0.783
LVEF, %	66.7 ± 4.3	66.7 ± 4.2	67.0 ± 4.4	0.580
PSD, ms	29.9(25.0, 36.0)	29.2(24.5, 36.6)	30.0(25.0, 35.0)	0.666
LV GLS, %	20.9 ± 3.2	21.2 ± 3.00	19.6 ± 3.5	< 0.001
LV GWI, mmHg%	1374 ± 330	1429 ± 304	1153 ± 340	< 0.001
LV GCW, mmHg%	1848 ± 372	1899 ± 352	1645 ± 379	< 0.001
LV GWW, mmHg%	68(51, 99)	67(49, 94)	81(54, 119)	0.003
LV GWE, %	95(94, 97)	96(94, 97)	94(91, 96)	< 0.001

Statistics are mean ± standard deviation, median (IQR), or number (%)

VSD Ventricular septal defect, *sPAP* Systolic pulmonary arterial pressure, *MR* Mitral regurgitation, *TR* Tricuspid regurgitation, *sPAP class I* Mild, *sPAP class II* Moderate, *sPAP class III* Severe, *IVSd* Interventricular septal end-diastolic thickness, *LVPWd* Left ventricular posterior wall end-diastolic thickness, *LVEDVi* Left ventricular end-diastolic volume index, *FS* Shortening fraction, *Clz* Cardiac index Z-score, *LVEF* Left ventricular ejection fraction, *PSD* Peak strain dispersion, *LV* Left ventricular, *GLS* Global longitudinal strain, *GWI* Global work index, *GCW* Global contractive work, *GWW* Global waste work, *GWE* Global work efficiency

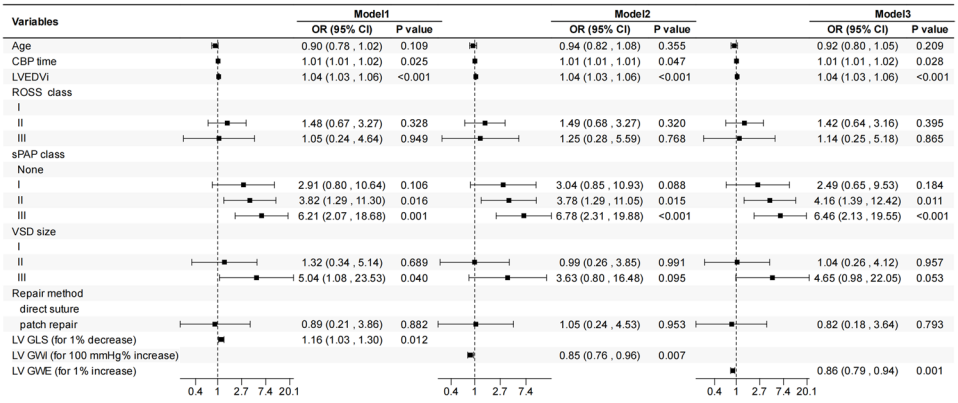


Fig. 2 Multivariable logistic regression models for independent predictors of postoperative LV dysfunction. Forest plot displays odds ratios (ORs) with 95% confidence intervals across three sequential model. CPB, Cardiopulmonary bypass time; LVEDVi, left ventricular end-diastolic volume index; sPAP, systolic pulmonary arterial pressure; VSD, ventricular septal defect; LV, left ventricular; GLS, global longitudinal strain; GWI, global work index; GWE, global work efficiency; VSD size I, Small; VSD size II, Moderate; VSD size III, Large; sPAP class I, Mild; sPAP class II, Moderate; sPAP class III, Severe

Cardiac function recovery based on Ross classification

During the 12-month postoperative follow-up period, 8 (1.96%) of these patients received digoxin to improve cardiac function following discharge, and 40 (9.78%) received bosentan/ambrisentan or sildenafil for pulmonary arterial hypertension reduction. No adverse events or deaths occurred within the cohort. Patients with postoperative LV dysfunction showed less favourable Ross classification recovery outcomes compared to those without postoperative LV dysfunction ($p = 0.005$) (Table S6, Fig. 5).

Intra- and inter-observer variability

The intra- and inter-observer reliability for LV GLS and the myocardial work indices was excellent, evidenced by high ICCs. Bland-Altman analysis of these parameters is shown in Figure S3.

Discussion

There were key findings in this study. (1) in patients with VSD, preoperative LV GLS, GWI, GWE, LVEDVi, moderate-to-severe sPAP, large VSD size, as well as prolonged CPB time, were found to be associated with early

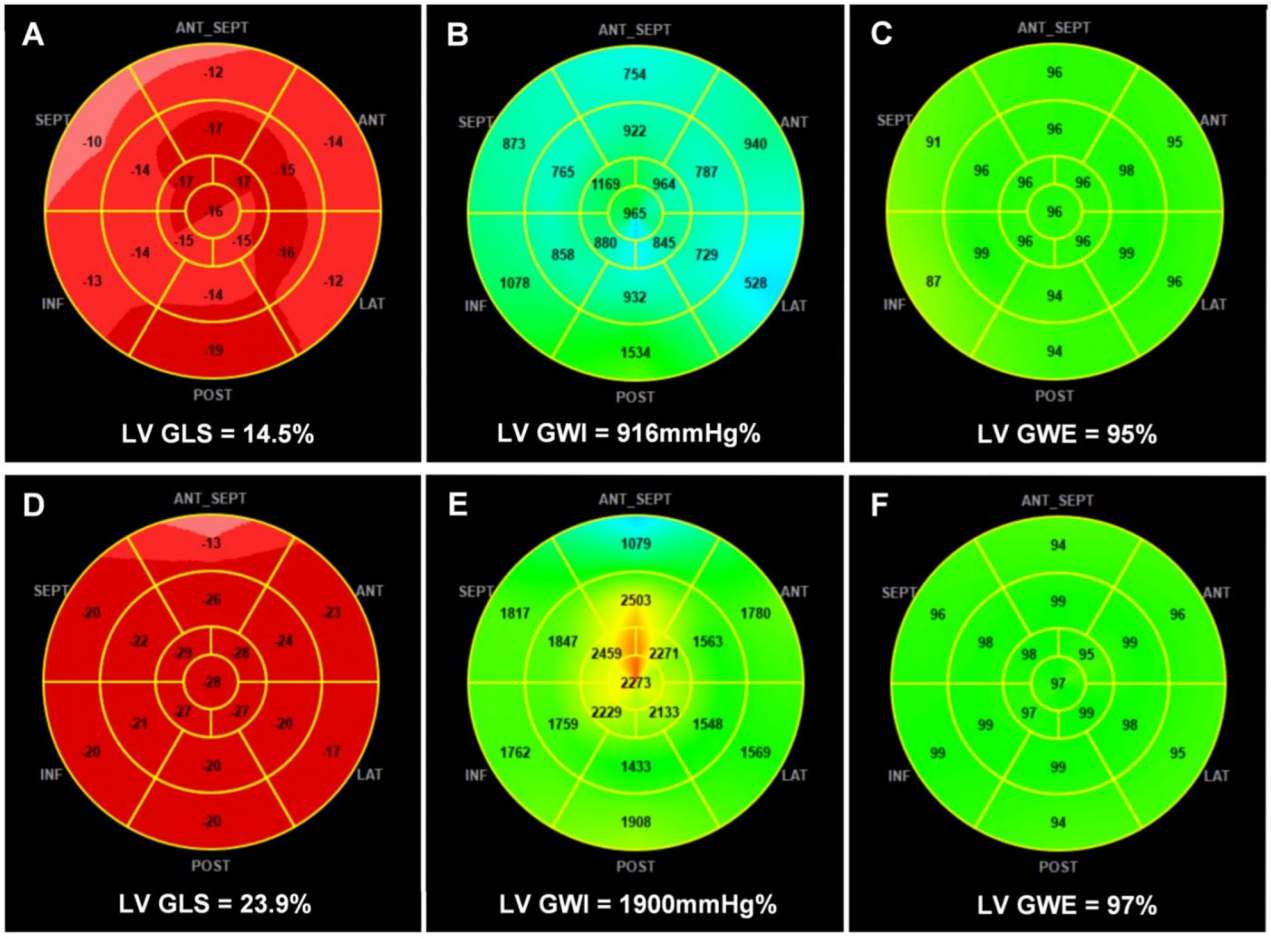


Fig. 3 Representative cases of myocardial strain, myocardial work indices, and bullseye maps in patients with(**A**-**C**) and without (**D**-**F**) post-operative LV dysfunction. LV, left ventricular; GLS, global longitudinal strain; GWI, global work index; GWE, global work efficiency

postoperative LV dysfunction after surgery; (2) The inclusion of myocardial strain and work had additive value in predicting the postoperative LV dysfunction; (3) The occurrence of postoperative LV dysfunction leads to a compromised patient functional recovery at 12 months after surgery.

Risk factors for postoperative LV dysfunction following surgical VSD repair

Consistent with previous studies, we found that over 20% of patients developed postoperative LV dysfunction after surgical VSD repair [5–7]. We acknowledge that a transient decline in LVEF is, to some extent, an expected haemodynamic consequence of acute preload reduction following VSD closure, and may not exclusively reflect intrinsic myocardial injury. Nevertheless, despite similar preoperative LVEF, impaired preoperative LV GLS, GWI, and GWE independently predicted postoperative LV dysfunction. Furthermore, sensitivity analyses using lower LV dysfunction thresholds (LVEF < 50% and LVEF < 40%) confirmed the robustness of our findings. LV GLS has been shown to predict cardiovascular events in CHD patients with transient LV systolic dysfunction [22], and is independently associated with LV dysfunction following surgical VSD repair [5, 7], consistent with our findings. However, strain measurements are influenced by afterload conditions. The novelty of this study is the inclusion of myocardial work, an emerging technique integrating ventricular deformation and afterload, providing a refined assessment of myocardial mechanics [9, 20, 21]. Mechanistically, chronic left-to-right shunting in VSD induces progressive volume overload, driving myocardial remodelling and ventricular dilation, if sustained, ultimately progresses to in LV myocardial impairment and clinical deterioration [23, 24]. Preoperative LV GLS, GWI, and GWE in the dysfunction group fell below recently established pediatric reference ranges [25–27], consistent with subclinical myocardial impairment prior to surgery.

In this study, we also identified elevated preoperative LVEDVi, moderate-to-severe sPAP, large VSD size, and prolonged CPB time as independent predictors of

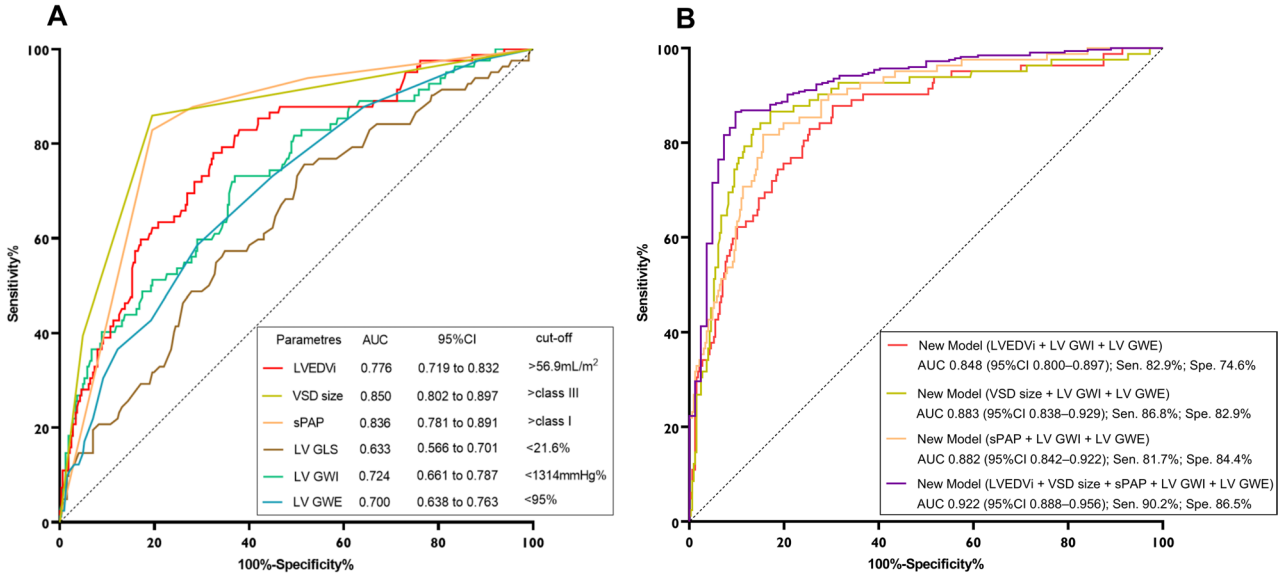


Fig. 4 **A** ROC curves for LVEDVi, VSD size, sPAP, LV GLS, LV GWI, and LV GWE to detect postoperative LV dysfunction. **B** ROC curves for detecting postoperative LV dysfunction improved in the new model with the inclusion of LV GWI and LV GWE. ROC, receiver operating characteristic; LVEDVi, left ventricular end-diastolic volume index; VSD, ventricular septal defect; sPAP, systolic pulmonary arterial pressure; LV, left ventricular; GLS, global longitudinal strain; GWI, global work index; GWE, global work efficiency; Sen., sensitivity; Spe.,specificity

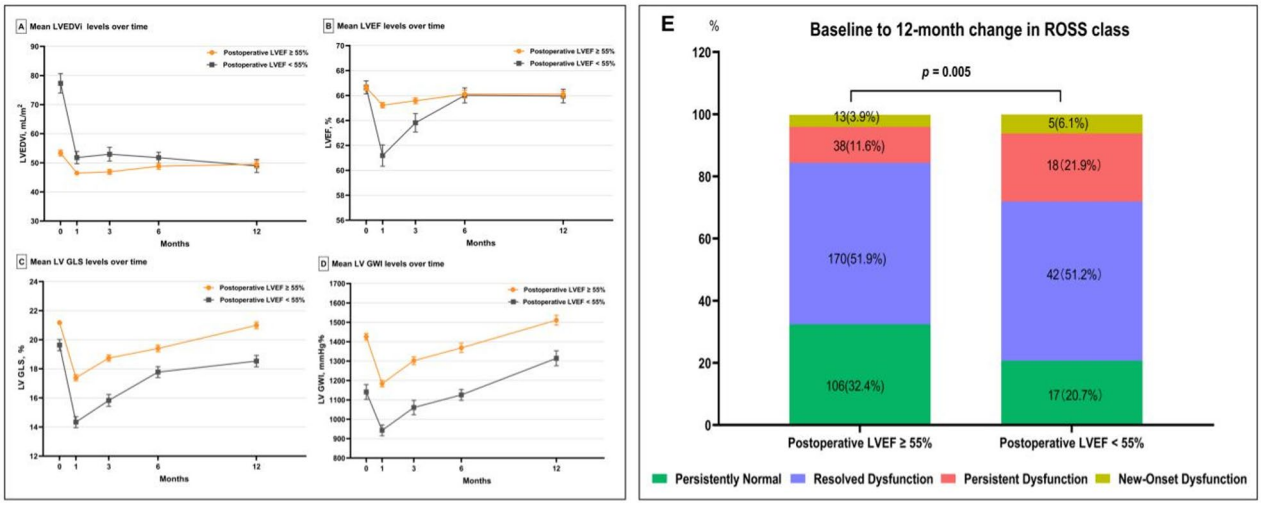


Fig. 5 Pre- and postoperative serial changes in (**A**) LVEDVi; (**B**) LVEF; (**C**) LV GLS; (**D**) LV GWI; (**E**) ROSS classifications between two groups. LVEDVi, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; LV, left ventricular; GLS, global longitudinal strain; GWI, global work index. Persistently Normal: Patients with pre- and postoperative ROSS class I. Resolved Dysfunction: Patients with preoperative ROSS class II/III who improved to class I postoperatively. Persistent Dysfunction: Patients with preoperative ROSS class II/III who remained in class II/III postoperatively. New-Onset Dysfunction: Patients with preoperative ROSS class I who deteriorated to class II/III postoperatively

postoperative LV dysfunction. The predominance of large VSDs in the dysfunction group suggests that higher LVEDVi and worse myocardial work indices (e.g., higher GWW) may largely reflect shunt-related volume overload and eccentric remodeling [5–7]. The current study is the first to establish the role of echocardiographic pulmonary artery pressure in this context. Pulmonary hypertension may persist after VSD closure and compromise postoperative LV function through interventricular dependence [28], highlighting the need for timely VSD closure before overt LV dilation or moderate-to-severe pulmonary hypertension develops. Prolonged CPB may exacerbate myocardial injury through inflammatory activation and ischemia-reperfusion [29, 30], and was likely attributable to larger defect sizes necessitating more extensive repair in the dysfunction group.

Importantly, combining LV GWI/GWE with LVEDVi, VSD size, and sPAP may add clinical value, because

LVEDVi and defect size reflect shunt-related volume overload, sPAP reflects pulmonary hemodynamic burden, and LV GWI/GWE provide complementary information on myocardial performance under afterload, whereas early postoperative LVEF decline may largely reflect unloading and geometric change. Therefore, patients with similar LV dilation, defect size, or sPAP but reduced LV GWI/GWE may represent a higher-risk subgroup despite preserved LVEF, warranting particular attention to haemodynamic status and early postoperative echocardiographic reassessment.

Clinical and cardiac function outcomes following surgical VSD repair

Although surgical closure effectively alleviates symptoms in patients with VSD, the long-term survival benefits warrant further investigation [3]. Our study demonstrated progressive normalization of LVEDVi and LVEF within 6–12 months postoperatively in both cohorts, consistent with the expected reverse ventricular remodeling after elimination of chronic volume overload [5–7]. However, despite recovery trends in LV GLS, GWI, and GCW between 3 and 12 months postoperatively in both groups, persistent intergroup disparities in these parameters were observed during follow-up. Specifically, patients without postoperative dysfunction exhibited preserved preoperative LV GLS, GWI, and GCW, reflecting effective myocardial compensation for chronic volume overload. In contrast, patients who developed dysfunction had lower preoperative values of these parameters, indicative of subclinical impairment. We propose that this pre-existing subclinical impairment, compounded by more aggravated ventricular remodeling from chronic preoperative volume overload and sustained mechanical stress, fundamentally impaired intrinsic contractile properties and reserve capacity, leading to suboptimal LV functional recovery at mid-term follow-up [8, 9, 31].

Consequently, patients developing postoperative LV dysfunction showed significantly less symptomatic improvement at 12 months, highlighting the clinical relevance of this persistent pathophysiological state and the critical need for timely intervention before irreversible myocardial damage occurs.

Clinical implications

This study focused on patients with isolated VSD undergoing surgical repair, with stringent inclusion and exclusion criteria to ensure cohort homogeneity. We employed a multi-time point observational design and performed a comprehensive assessment of the prognostic value of novel technological indices. Our findings demonstrate that preoperative LV GLS, GWI, GCW, GWW, and GWE provide significant prognostic value for postoperative LV dysfunction, offering a scientific basis for optimizing therapeutic strategies to improve long-term outcomes. Our rigorous comparison of functional cardiac parameters at the follow-up time points between those who did and did not develop LV dysfunction demonstrates that even seemingly simple congenital defects, such as VSD, can lead to irreversible myocardial injury. Consistent with the Ross-class transition analysis, those with postoperative dysfunction exhibited less symptomatic improvement by 12 months. Our findings emphasise the need for prolonged monitoring in patients with postoperative LV dysfunction, as full functional recovery may take some time. Myocardial strain and myocardial work indices have shown distinct clinical utility as sensitive long-term indicators of cardiac function following surgical VSD repair.

Limitations and Future Directions

This study has several limitations. First, this was a single-centre cohort without external validation, and children underwent VSD repair at a relatively older age (median 3 years) than in some prior reports [32], likely reflecting regional disparities in CHD medical resources [4, 33]. Future multi-centre studies across settings with earlier repair and broader age distributions are warranted. Second, follow-up was incomplete at later time points, and although GEE accommodates incomplete repeated measures, findings may be biased if loss to follow-up was not random. Follow-up beyond 12 months is needed to define the longer-term trajectory of postoperative LV dysfunction. Furthermore, the primary endpoint, Ross functional class, is inherently subjective, although a standardized questionnaire (Supplementary Table S1) was used; objective endpoints (e.g., NT-proBNP, cardiopulmonary exercise testing) should be prioritized in future prospective studies. Third, pooling VSDs of different anatomic subtypes may have introduced clinical heterogeneity, and strain- and work-derived indices could be influenced by procedural factors such as repair strategy. Although residual confounding remains possible, these findings reflect real-world paediatric surgical practice. Fourth, the exclusion of patients with major perioperative complications, including low cardiac output syndrome, may have introduced selection bias, and future studies should evaluate this higher-risk subgroup. Finally, estimation of myocardial work relies on non-invasive cuff-based brachial blood pressure, which may not precisely reflect LV systolic pressure. Nevertheless, myocardial work has been widely used and has demonstrated utility for detecting subclinical myocardial dysfunction [9, 20, 21].

Conclusion

In paediatric patients who underwent surgical repair of isolated VSD with preserved LVEF, impaired myocardial strain and myocardial work indices were associated

with early postoperative LV dysfunction. At 12-month postoperative follow-up, patients with postoperative LV dysfunction tend to showed less symptomatic improvement and worse cardiac function status. Pre- and postoperative integrated assessment of LV function may provide insights into managing this patient population.

Abbreviations

ACC	Aortic cross-clamp
AIC	Akaike information criterion
AUC	Area under the curve
ANOVA	Analysis of variance
BIC	Bayesian information criterion
BP	Blood pressure
BSA	Body surface area
CI	Cardiac index
CIz	Cardiac index Z-score
CHD	Congenital heart disease
CPB	Cardiopulmonary bypass
FS	Shortening fraction
GEE	Generalised estimating equations
GLS	Global longitudinal strain
GWI	Global work index
GCW	Global constructive work
GWE	Global work efficiency
GWW	Global wasted work
HR	Heart rate
ICC	Intraclass correlation coefficient
ICU	Intensive care unit
IVSd	Interventricular septal end-diastolic thickness
LV	Left ventricular
LVEDV	Left ventricular end-diastolic volume
LVEDVi	Left ventricular end-diastolic volume index
LVEF	Left ventricular ejection fraction
LVPWd	Left ventricular posterior wall end-diastolic thickness
MR	Mitral regurgitation
PSD	Peak strain dispersion
ROC	Receiver operating characteristic
sPAP	Systolic pulmonary arterial pressure
SpO_2	Peripheral oxygen saturation
TR	Tricuspid regurgitation
TTE	Transthoracic echocardiography
VIF	Variance inflation factor
VSD	Ventricular septal defect

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12887-026-07470-6.

Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

Supplementary Material 4.

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Authors' contributions

All authors made substantial contributions to the research or article preparation. WZ and WL contributed to investigation, data curation, formal analysis, and drafting the original manuscript. TW, LG, JZ, YZ, and LZ contributed to data acquisition. QG contributed to formal analysis and investigation. SY contributed to image processing and software management. XD contributed to statistical analysis. JW contributed to conceptualization, methodology, and critical revision of the manuscript for important intellectual content. ZL contributed to resources, supervision, funding acquisition, critical revision of the manuscript.

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Data availability

Subject to patient privacy safeguards, de-identified datasets supporting this study are available from the corresponding author for legitimate research purposes.

Declarations

Ethics approval and consent to participate

This study adhered to the principles of the Declaration of Helsinki. The study protocol was approved by the Institutional Ethics Committee of Yunnan Fuwai Cardiovascular Hospital (approval no. 2024-079-01). Given that this was a retrospective cohort study using de-identified data obtained from the institutional electronic medical record system, the requirement for individual informed consent to participate was waived by the same ethics committee.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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