RESEARCH PAPER

Effects of Six-Week Exercise on Right Ventricular Hemodynamic Parameters in Congenital Heart Disease–Associated Pulmonary Hypertension

Jauhar Mario[1]*, Zaenab Djafar[1,2], Aussie Fitriani Ghaznawie[1,2], Yulius Pattimang[1,2], Melda Warliani[3] and Andi Alfian Zainuddin[4]

[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
[2]Cardiac Center, Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia
[3]Department of Physical Medicine and Rehabilitation, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
[4]Department of Public Health and Community Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
Corresponding Author: E-mail: mariojauhar@gmail.com.*

Received: 24th April, 2026; Revised: 20th May 2026; Accepted: 30th June, 2026; Available Online: 10th July, 2026

ABSTRACT

Background and Objective: Pulmonary hypertension associated with congenital heart disease involves pulmonary vascular remodeling and increased right ventricular afterload, impairing functional capacity and worsening outcomes. Although exercise rehabilitation improves function in pulmonary hypertension, its effects on echocardiographic right ventricular hemodynamics are unclear. This study evaluated a six-week exercise program in adults.

Methods: pre–post study enrolled 14 adults with pulmonary hypertension and congenital heart disease. Participants underwent exercise training three times weekly for six weeks. Echocardiography before/after intervention assessed pulmonary artery systolic pressure, tricuspid regurgitation velocity, mean pulmonary artery pressure, and pulmonary vascular resistance. Paired t-tests or Wilcoxon signed-rank tests were applied by distribution.

Results: Following the intervention, pulmonary artery systolic pressure decreased significantly from 83.93 ± 31.80 to 68.55 ± 24.45 mmHg ($p = 0.044$), while mean pulmonary artery pressure declined from 53.20 ± 19.39 to 43.81 ± 14.91 mmHg ($p = 0.044$). Pulmonary vascular resistance also decreased significantly from 0.45 (0.05–0.60) to 0.33 (0.07–0.59) Wood units ($p = 0.039$). Tricuspid regurgitation maximum velocity showed a reduction from 4.22 ± 0.95 to 3.78 ± 0.83 m/s, although the difference did not reach statistical significance ($p = 0.055$).

Conclusion: A six-week structured exercise program was associated with improved right ventricular hemodynamics in adults with pulmonary hypertension associated with congenital heart disease, supporting exercise rehabilitation as a safe adjunct to standard medical therapy.

Keywords: *Adult Congenital Heart Disease; Echocardiography; Exercise Therapy; Pulmonary Hypertension; Ventricular Function.*
How to cite this article: Mario J, Djafar Z, Ghaznawie AF, Pattimang Y, Warliani M and Zainuddin AA., Effects of Six-Week Exercise on Right Ventricular Hemodynamic Parameters in Congenital Heart Disease–Associated Pulmonary Hypertension. Int J Drug Deliv Technol. 2026;16(7): 824-830; DOI: 10.25258/ijddt.16.7.87
Source of support: Nil.
Conflict of interest: None

INTRODUCTION

Congenital heart disease (CHD) is the most common congenital anomaly worldwide, affecting approximately 8–10 per 1,000 live births. Advances in pediatric cardiology, cardiac surgery, and perioperative care have enabled more than 90% of children with CHD to survive into adulthood, resulting in a rapidly expanding population of adults with congenital heart disease (ACHD) [1,2]. Pulmonary hypertension (PH) remains one of the most serious long-term complications in adults with CHD because persistent systemic-to-pulmonary shunts, residual cardiac lesions, and pulmonary vascular remodeling progressively increase pulmonary arterial pressure and right ventricular afterload [3,4]. CHD-associated PH is associated with reduced exercise capacity, impaired quality of life, right ventricular dysfunction, frequent hospitalization, and increased mortality, making early identification and appropriate management essential [5]. Since right ventricular adaptation is the strongest determinant of prognosis in PH, serial assessment of right ventricular hemodynamics has become an important component of patient follow-up [6,7].

**Author for Correspondence: mariojauhar@gmail.com*

Exercise intolerance is a cardinal manifestation of PH associated with CHD and results from impaired right ventricular contractile reserve, elevated pulmonary vascular resistance, endothelial dysfunction, and abnormal skeletal muscle metabolism [8]. Although patients with PH were previously advised to avoid physical exertion because of concerns regarding adverse cardiovascular events, growing evidence indicates that supervised exercise training is safe and improves functional capacity, exercise tolerance, and health-related quality of life in clinically stable patients [9]. Consequently, contemporary international guidelines recommend individualized exercise rehabilitation as an adjunct to optimal medical therapy in selected patients with pulmonary hypertension [10,11]. Recent systematic reviews and meta-analyses have further demonstrated that structured exercise programs improve cardiopulmonary performance without increasing the risk of clinical deterioration [12].

Transthoracic echocardiography remains the most widely used non-invasive imaging modality for evaluating right ventricular hemodynamics through pulmonary artery systolic pressure (PASP), tricuspid regurgitation maximum velocity (TR Vmax), mean pulmonary artery pressure (mPAP), and pulmonary vascular resistance (PVR) [13]. Although exercise training has consistently been shown to improve functional outcomes, evidence regarding its effect on echocardiographic right ventricular hemodynamic parameters in adults with CHD-associated PH remains limited because previous studies have included heterogeneous patient populations, small sample sizes, and varying exercise protocols [14,15]. Therefore, this study aimed to evaluate the effects of a six-week structured exercise program on echocardiographic right ventricular hemodynamic parameters, including PASP, TR Vmax, mPAP, and PVR, in adults with pulmonary hypertension associated with congenital heart disease.

MATERIALS AND METHODS

Study Design and Participants

This single-arm interventional study employed a pre–post design to evaluate the effects of a six-week structured exercise program on right ventricular hemodynamic echocardiographic parameters in adults with pulmonary hypertension associated with congenital heart disease. The study was conducted at the Integrated Cardiac Center, Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, between May and July 2025. Eligible participants were consecutive adult outpatients diagnosed with pulmonary hypertension associated with congenital heart disease based on right heart catheterization and transthoracic echocardiography. Patients were enrolled consecutively until the required sample size was achieved. All participants provided written informed consent before enrollment.

Eligibility Criteria

Participants were eligible if they were aged ≥18 years, had pulmonary hypertension confirmed by right heart catheterization, and had congenital heart disease confirmed by echocardiography and/or right heart catheterization. Patients were excluded if they had significant left-sided heart disease, severe pulmonary disease, acute coronary syndrome within the preceding 40 days, percutaneous coronary intervention within two weeks, coronary artery bypass grafting within the previous three months, intracardiac thrombus or recent embolic events, resting heart rate >90 beats/minute, exercise-induced arrhythmias or significant conduction disturbances, acute myocarditis or pericarditis, hypertrophic cardiomyopathy, anemia (hemoglobin <11 g/dL), severe musculoskeletal or neurological disability limiting exercise participation, thrombophlebitis, uncontrolled systemic disease including thyrotoxicosis, renal failure, or diabetes mellitus, active malignancy, aortic aneurysm, severe psychiatric disorders, or inability or unwillingness to participate in the rehabilitation program. Participants with adherence below 80% or those who failed to complete the follow-up evaluation were considered dropouts.

Sample Size

The sample size was calculated using the formula for paired continuous outcomes based on the expected clinically meaningful change, pilot-study standard deviation, and a two-sided confidence level of 95% ($Z = 1.96$). The minimum required sample size was 16 participants. Assuming a dropout rate of 20%, the target enrollment was increased to 20 participants to preserve statistical power and improve the reliability of the study findings.

Exercise Intervention

Before initiating the intervention, all participants underwent baseline clinical assessment, including demographic data collection, medical history, medication review, anthropometric measurements, resting heart rate, and blood pressure evaluation. Standardized cardiovascular rehabilitation education was provided to all participants, including medication adherence, smoking cessation, cardiovascular risk factor modification, dietary counseling, stress management, and exercise education.

Each participant received an exercise package consisting of a 1-kg dumbbell, an incentive spirometer, a digital sphygmomanometer, a pulse oximeter, an exercise logbook, and educational materials. The intervention consisted of supervised low-intensity exercise training performed three sessions per week for six consecutive weeks, with each session lasting approximately 30 minutes under the supervision of the cardiac rehabilitation team at the Integrated Cardiac Center, Dr. Wahidin Sudirohusodo Hospital. Exercise adherence was monitored using individual logbooks that were reviewed regularly throughout the intervention period.

Echocardiographic Assessment

Comprehensive transthoracic echocardiography was performed before initiation of the exercise program and repeated after completion of the six-week intervention using a GE Vivid E95 ultrasound system. The evaluated

right ventricular hemodynamic parameters included PASP, TR Vmax, mPAP, and PVR. PASP was estimated from the peak TR Vmax using the modified Bernoulli equation with estimated right atrial pressure. Mean pulmonary artery pressure was subsequently derived from PASP using a validated echocardiographic method, whereas PVR was estimated non-invasively using the ratio of TR Vmax to the right ventricular outflow tract velocity–time integral according to established echocardiographic recommendations. All examinations were performed following the current British Society of Echocardiography and ESC/ERS guideline recommendations by experienced cardiologists using standardized imaging protocols.

Study Variables

The independent variable was participation in the six-week structured exercise program. The primary outcome variables were changes in PASP, TR Vmax, mPAP, and PVR measured by transthoracic echocardiography. Demographic and clinical characteristics, including age, sex, body mass index, comorbidities, and socioeconomic status, were recorded as potential confounding variables.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 21. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages. Baseline and post-intervention echocardiographic measurements were compared using appropriate paired statistical tests according to data distribution. All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant. The study findings were presented as text accompanied by tables and figures where appropriate.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia (Approval No. 1001/UN4.6.4.5.31/PP36/2025; Protocol No. UH25100843). Written informed consent was obtained from all participants before enrollment in the study.

RESULTS

Baseline Characteristics

A total of 14 patients completed the study and were included in the final analysis. The mean age was 32.0 ± 8.3 years, with a predominance of female participants (78.6%). Overall, participants had a relatively low body mass index, with a mean value of 19.6 ± 2.9 kg/m². Most patients had atrial septal defect (78.6%), whereas ventricular septal defect and patent ductus arteriosus were less common. Bidirectional shunting was observed in the majority of participants (78.6%), and the mean defect diameter was 3.0 ± 1.8 cm. Baseline demographic and clinical characteristics are summarized in Table 1.

Table 1. Baseline demographic and clinical characteristics of the study participants (n = 14)

Characteristic	Value (n = 14)
Age, years	32.0 ± 8.3
Female sex, n (%)	11 (78.6)
Body mass index, kg/m²	19.62 ± 2.91
Congenital heart disease diagnosis, n (%)	
Atrial septal defect	11 (78.6)
Ventricular septal defect	2 (14.3)
Patent ductus arteriosus	1 (7.1)
Shunt direction, n (%)	
Bidirectional shunt	11 (78.6)
Left-to-right shunt	3 (21.4)
Defect size, cm	2.96 ± 1.79

Echocardiographic Findings

Comprehensive echocardiographic findings before and after the six-week exercise intervention are presented in Supplementary Table 1. Overall, the intervention was associated with favorable trends in right heart remodeling and pulmonary hemodynamics. Structural assessment demonstrated reductions in right atrial and right ventricular dimensions, including decreases in basal, mid-cavity, and longitudinal right ventricular diameters, accompanied by a reduction in the main pulmonary artery diameter. In contrast, left ventricular systolic function remained unchanged throughout the study period, with a preserved ejection fraction before and after the intervention. Right ventricular functional indices showed a trend toward improvement, as reflected by increases in tricuspid annular plane systolic excursion, lateral S′ velocity, and fractional area change.

Pulmonary hemodynamic parameters also demonstrated improvement following exercise training. The mean TR Vmax decreased from 4.22 ± 0.95 to 3.78 ± 0.83 m/s, accompanied by reductions in TR pressure gradient (74.10 ± 32.02 vs. 60.09 ± 24.49 mmHg), PASP (83.93 ± 31.80 vs. 68.55 ± 24.45 mmHg), mPAP (53.20 ± 19.39 vs. 43.81 ± 14.91 mmHg), and PVR (0.37 ± 0.18 vs. 0.32 ± 0.17 Wood units). In addition, pulmonary valve acceleration time increased from 102.54 ± 28.32 to 121.07 ± 31.95 ms, suggesting improved pulmonary vascular hemodynamics. Complete echocardiographic measurements are provided

in Supplementary Table 1. Overall, these findings indicate favorable cardiac remodeling and improvements in pulmonary hemodynamics and right ventricular performance following the six-week exercise program.

Subgroup Analysis by Congenital Heart Disease Diagnosis

Subgroup analyses according to the underlying congenital heart disease diagnosis are presented in Table 2. Across all diagnostic subgroups, the six-week exercise training program demonstrated a consistent trend toward improved pulmonary hemodynamic parameters, including reductions in TR Vmax, PASP, mPAP, and PVR. The largest subgroup, comprising patients with atrial septal defect (ASD; n = 11), showed decreases in TR Vmax (4.18 ± 0.90 to 3.88 ± 0.67 m/s), PASP (81.38 ± 29.61 to 70.06 ± 21.72 mmHg), mPAP (51.64 ± 18.06 to 44.74 ± 13.25 mmHg), and PVR (0.38 ± 0.18 to 0.34 ± 0.18 Wood units) following the intervention. Similar improvements were observed among patients with ventricular septal defect (VSD; n = 2), who demonstrated reductions in TR Vmax, PASP, mPAP, and PVR after exercise training. The single participant with patent ductus arteriosus (PDA) likewise exhibited decreases across all measured hemodynamic parameters, including TR Vmax, PASP, mPAP, and PVR. Owing to the very small sample sizes in the VSD and PDA subgroups, these findings should be interpreted as descriptive and hypothesis-generating rather than inferential, and no formal between-group statistical comparisons were performed.

Table 2. Changes in primary echocardiographic hemodynamic parameters according to congenital heart disease diagnosis before and after the six-week exercise program

Variable	ASD (n = 11)		VSD (n = 2)		PDA (n = 1)	
	Pre	Post	Pre	Post	Pre	Post
TR Vmax (m/s)	4.18 ± 0.90	3.88 ± 0.67	4.00 ± 1.57	3.15 ± 1.90	5.26	4.00
PASP (mmHg)	81.38 ± 29.61	70.06 ± 21.72	80.63 ± 55.47	58.48 ± 53.06	118.67	72.00
mPAP (mmHg)	51.64 ± 18.06	44.74 ± 13.25	51.18 ± 33.83	37.67 ± 32.36	74.38	45.92
PVR (Wood units)	0.38 ± 0.18	0.34 ± 0.18	0.29 ± 0.26	0.19 ± 0.17	0.46	0.35

Data are presented as mean ± standard deviation. Owing to the very small number of participants in the VSD (n = 2) and PDA (n = 1) subgroups, these results are descriptive only and were not subjected to formal between-group statistical comparisons.

Effect of the Six-Week Exercise Program on Right Ventricular Hemodynamics

Changes in the primary echocardiographic hemodynamic parameters following the six-week exercise program are summarized in Table 3. Overall, the intervention was associated with significant improvements in pulmonary hemodynamics. PASP decreased significantly from 83.93 ± 31.80 mmHg at baseline to 68.55 ± 24.45 mmHg after the intervention, corresponding to a mean reduction of 15.38 ± 25.76 mmHg (p = 0.044). Similarly, mPAP declined significantly from 53.20 ± 19.39 mmHg to 43.81 ± 14.91 mmHg, with a mean decrease of 9.38 ± 15.71 mmHg (p = 0.044).

TR Vmax also decreased following exercise training, from 4.22 ± 0.95 m/s to 3.78 ± 0.83 m/s; however, this reduction did not reach statistical significance (p = 0.055). In contrast, PVR demonstrated a significant reduction, with the median value decreasing from 0.45 (0.05–0.60) to 0.33 (0.07–0.59) Wood units (Z = −2.062, p = 0.039). Collectively, these findings indicate that the six-week exercise program was associated with significant improvements in pulmonary arterial pressure and pulmonary vascular resistance, whereas the reduction in TR Vmax showed a favorable but non-significant trend.

Table 3. Changes in primary echocardiographic hemodynamic parameters following the six-week exercise program (n = 14)

Variable	Baseline	Post-intervention	Change	95% CI	p value
PASP (mmHg)	83.93 ± 31.80	68.55 ± 24.45	−15.38 ± 25.76	0.50 to 30.26	0.044
TR Vmax (m/s)	4.22 ± 0.95	3.78 ± 0.83	−0.44 ± 0.78	−0.01 to 0.89	0.055
mPAP (mmHg)	53.20 ± 19.39	43.81 ± 14.91	−9.38 ± 15.71	0.31 to 18.46	0.044
PVR (Wood units)†	0.45 (0.05–0.60)	0.33 (0.07–0.59)	—	—	0.039

† Data are presented as median (range) and were analyzed using the Wilcoxon signed-rank test (Z = −2.062).

DISCUSSION

The present study demonstrated that a six-week structured exercise program was associated with significant improvements in pulmonary hemodynamics, as evidenced by reductions in PASP, mPAP, and PVR, whereas TR Vmax showed a favorable but statistically non-significant decrease. These findings suggest that short-term exercise training may reduce right ventricular afterload and improve pulmonary vascular function in adults with pulmonary hypertension associated with congenital heart disease. Although the study included a relatively small cohort, the consistent direction of change across all hemodynamic parameters supports the potential role of exercise rehabilitation as an adjunctive therapy in this high-risk population.

The baseline characteristics of the study population were consistent with previous registries of pulmonary hypertension associated with congenital heart disease. Participants were predominantly young adults and female, with atrial septal defect representing the most common underlying lesion. These findings are consistent with the COHARD-PH registry reported by DInarti et al., which identified atrial septal defect as the predominant congenital heart lesion among Indonesian adults with pulmonary hypertension, with a marked female predominance [16]. Similarly, Kaemmerer et al. reported that pulmonary hypertension develops most commonly in patients with unrepaired or late-repaired systemic-to-pulmonary shunts, particularly atrial septal defects, where chronic left-to-right shunting promotes progressive pulmonary vascular remodeling and increased pulmonary vascular resistance [4]. The high proportion of bidirectional shunting observed in the present study further reflects advanced pulmonary vascular disease, emphasizing the importance of adjunctive non-pharmacological interventions that may improve right ventricular adaptation despite established vascular remodeling [17].

The significant reduction in PASP represents one of the principal findings of this study. Elevated PASP reflects increased right ventricular systolic afterload and is closely associated with disease severity and prognosis in pulmonary hypertension. The observed reduction is biologically plausible because regular exercise has been shown to improve endothelial function through increased nitric oxide bioavailability, attenuation of oxidative stress, and suppression of inflammatory signaling, thereby promoting pulmonary vasodilation and reducing vascular stiffness. These findings are in line with Mereles et al. (2006), who demonstrated that supervised exercise training significantly improved pulmonary hemodynamics and exercise capacity in patients with severe pulmonary hypertension [18]. Similar improvements have also been reported by Ehlken et al., who found that exercise-based rehabilitation enhanced clinical status and functional performance without increasing adverse cardiovascular events [19]. Collectively, these studies support the concept that structured exercise may favorably modify pulmonary vascular physiology beyond improving functional capacity alone.

Consistent with the reduction in PASP, both mPAP and PVR decreased significantly following the intervention. Because mPAP and PVR are fundamental indicators of pulmonary vascular load, simultaneous improvement in these parameters suggests a reduction in pulmonary vascular resistance rather than isolated changes in right ventricular performance. These findings are consistent with those reported by Firdaus et al., who observed significant reductions in mPAP following a structured exercise program in adults with congenital heart disease–associated pulmonary hypertension, although the magnitude of improvement in the present study was greater [20]. Likewise, Grünig et al. demonstrated that exercise rehabilitation reduced pulmonary vascular resistance while improving exercise tolerance and quality of life in patients with pulmonary hypertension [21]. Recent meta-analyses have further confirmed that structured exercise training improves pulmonary vascular function through enhanced endothelial responsiveness, increased skeletal muscle oxidative capacity, and improved peripheral oxygen utilization, collectively reducing cardiopulmonary workload during physical activity.

Unlike the other hemodynamic parameters, the reduction in TR Vmax did not achieve statistical significance despite showing a consistent downward trend. This finding should be interpreted cautiously. Because PASP is derived from the tricuspid regurgitant velocity together with estimated right atrial pressure, relatively small changes in velocity may still translate into clinically meaningful reductions in calculated pulmonary artery pressure. Furthermore, Doppler-derived measurements are influenced by insonation angle, signal quality, and regurgitant jet characteristics, which may contribute to greater measurement variability. The borderline statistical significance observed in the present study ($p = 0.055$) therefore likely reflects limited statistical power rather than absence of a physiological response, particularly given the concordant reductions observed in PASP, mPAP, and PVR.

Several mechanisms may explain the observed hemodynamic improvements following exercise training. Experimental and clinical studies have demonstrated that regular aerobic exercise enhances endothelial nitric oxide synthase activity, increases nitric oxide availability, suppresses endothelin-1 signaling, reduces oxidative stress, and promotes anti-inflammatory cytokine production. These adaptations improve pulmonary vascular compliance and reduce right ventricular afterload. Exercise also improves skeletal muscle efficiency and respiratory muscle performance, thereby lowering ventilatory demand and reducing cardiovascular stress during physical activity [13]. Together, these central and peripheral adaptations provide a plausible explanation for the favorable hemodynamic changes observed after only six weeks of intervention.

Overall, the present findings support the incorporation of structured exercise rehabilitation into the multidisciplinary management of adults with pulmonary hypertension associated with congenital heart disease. Beyond improving functional capacity, exercise appears to confer measurable benefits on pulmonary hemodynamics and right ventricular loading conditions, highlighting its potential role as a complementary strategy alongside guideline-directed medical therapy.

LIMITATIONS

This study has several limitations. First, the single-arm pre–post design without a control group limits causal inference and precludes definitive attribution of the observed hemodynamic improvements solely to the

exercise intervention. Second, the relatively small sample size may have reduced the statistical power to detect significant changes in certain echocardiographic parameters, particularly tricuspid regurgitation maximum velocity (TR Vmax), which demonstrated only borderline significance. Third, the relatively short follow-up period precluded assessment of the long-term sustainability of the observed hemodynamic improvements and their impact on clinical outcomes. Finally, hemodynamic parameters were evaluated using transthoracic echocardiography rather than repeat right heart catheterization, although echocardiography remains the preferred non-invasive modality for serial assessment in routine clinical practice. Nevertheless, the consistent improvement observed across multiple echocardiographic indices provides supportive evidence for the beneficial effects of exercise training. Future multicenter randomized controlled trials with larger sample sizes, longer follow-up, and invasive hemodynamic validation are warranted to confirm these findings and determine whether the observed short-term improvements translate into sustained reverse remodeling and improved long-term clinical outcomes.

CONCLUSIONS

A six-week structured exercise program was associated with overall improvements in right ventricular hemodynamics in adults with pulmonary hypertension associated with congenital heart disease, indicating reduced pulmonary vascular load and enhanced right ventricular adaptation. These findings support the incorporation of structured exercise rehabilitation as a safe and complementary strategy alongside standard medical therapy to optimize cardiovascular function in this patient population. Further large-scale randomized controlled studies are warranted to validate these findings and determine their long-term clinical implications.

DECLARATIONS

Consent for Publication

Not applicable.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Funding

The authors received no specific financial support for the research, authorship, and/or publication of this article.

Author Contributions (CRediT)

Jauhar Mario: Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, Visualization, Writing – Original Draft.

Zaenab Djafar: Conceptualization, Methodology, Supervision, Validation, Writing – Review & Editing.

Aussie Fitriani Ghaznawie: Methodology, Investigation, Validation, Writing – Review & Editing.

Yulius Pattimang: Investigation, Resources, Validation, Writing – Review & Editing.

Melda Warliani: Methodology, Investigation, Supervision, Writing – Review & Editing.

Andi Alfian Zainuddin: Formal Analysis, Methodology, Validation, Writing – Review & Editing.

All authors have read and approved the final version of the manuscript.

Acknowledgements

The authors would like to thank the staff of the Integrated Cardiac Center, Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, for their assistance with participant recruitment and data collection. The authors also sincerely thank all study participants for their valuable contribution to this research.

REFERENCES

1. Marelli AJ, Ionescu-Ittu R, Mackie AS, Guo L, Dendukuri N, Kaouache M. Lifetime prevalence of congenital heart disease in the general population from 2000 to 2010. Circulation. 2014;130(9):749-756. doi:10.1161/CIRCULATIONAHA.113.008396
2. Kong ASY, Khaw KY, Mood MC, Khan MA, Ayub Q, Maran S. The burden of congenital heart disease in Malaysia: a comprehensive review and meta-analytic synthesis, 1970–2024. BMC Public Health 2025 25:1. 2025;25(1):2595-. doi:10.1186/S12889-025-23800-2
3. Van Der Bom T, Zomer AC, Zwinderman AH, Meijboom FJ, Bouma BJ, Mulder BJM. The changing epidemiology of congenital heart disease. Nat Rev Cardiol. 2011;8(1):50-60. doi:10.1038/NRCARDIO.2010.166
4. Kaemmerer H, Diller GP, Dähnert I, et al. Pulmonary hypertension in adults with congenital heart defects (ACHDs)—in light of the 2022 ESC PAH guidelines—part I: definition, epidemiology, classification, diagnostics, genetics, risk stratification and follow-up, gender aspects. Cardiovasc Diagn Ther. 2024;14(5):935-948. doi:10.21037/CDT-24-148/COIF)
5. Liu Y, Chen S, Zühlke L, et al. Global birth prevalence of congenital heart defects 1970-2017: updated systematic review and meta-analysis of 260 studies. Int J Epidemiol. 2019;48(2):455-463. doi:10.1093/IJE/DYZ009
6. Bernardo RJ, Haddad F, Couture EJ, et al. Mechanics of right ventricular dysfunction in pulmonary arterial hypertension and heart failure with preserved ejection fraction. Cardiovasc Diagn Ther. 2020;10(5):1580. doi:10.21037/CDT-20-479

7. Kiyota T, Mendes P, Pereira MC. The role of the right ventricle in pulmonary arterial hypertension and imaging methods for non-invasive evaluation. Expert Rev Respir Med. 2026;20(5):543-567. doi:10.1080/17476348.2025.2601388
8. Mocumbi AO, Sliwa K. Women's cardiovascular health in Africa. Heart. 2012;98(6):450-455. doi:10.1136/HEARTJNL-2011-301025
9. Baumgartner H, Bonhoeffer P, De Groot NMS, et al. ESC Guidelines for the management of grown-up congenital heart disease (new version 2010). Eur Heart J. 2010;31(23):2915-2957. doi:10.1093/EURHEARTJ/EHQ249
10. Kaemmerer H, Gorenflo M, Huscher D, et al. Pulmonary Hypertension in Adults with Congenital Heart Disease: Real-World Data from the International COMPERA-CHD Registry. J Clin Med. 2020;9(5):1456. doi:10.3390/JCM9051456
11. Sengsim J, Vijarnsorn C, Chanthong P, et al. Survival comparison in adults with congenital systemic to pulmonary shunt and borderline elevated pulmonary vascular resistance versus Eisenmenger syndrome. Sci Rep. 2024;14(1):29891. doi:10.1038/S41598-024-81834-9
12. Galiè N, Humbert M, Vachiery JL, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension: The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS): Endo…. Eur Respir J. 2015;46(4):903-975. doi:10.1183/13993003.01032-2015
13. Soegistiono FH, Krevani CK, Hamdani R. The effect of a physical exercise program on functional capacity in patients with pulmonary arterial hypertension at Dr. M. Djamil Padang Hospital. Indonesian Journal of Cardiology. 2025;46(3):104-113. doi:10.30701/IJC.1594
14. Crisci G, De Luca M, D'Assante R, et al. Effects of Exercise on Heart Failure with Preserved Ejection Fraction: An Updated Review of Literature. Journal of Cardiovascular Development and Disease 2022, Vol 9, Page 241. 2022;9(8):241. doi:10.3390/JCDD9080241
15. Cuomo G, Lorenzo A Di, Tramontano A, et al. Exercise Training in Patients with Heart Failure: From Pathophysiology to Exercise Prescription. Rev Cardiovasc Med. 2022;23(4):144. doi:10.31083/J.RCM2304144
16. Dinarti LK, Hartopo AB, Kusuma AD, et al. The COngenital HeARt Disease in adult and Pulmonary Hypertension (COHARD-PH) registry: a descriptive study from single-center hospital registry of adult congenital heart disease and pulmonary hypertension in Indonesia. BMC Cardiovasc Disord. 2020;20(1):163. doi:10.1186/S12872-020-01434-Z
17. Kaemmerer H, Diller GP, Dähnert I, et al. Pulmonary hypertension in adults with congenital heart defects (ACHDs) in light of the 2022 ESC PAH guidelines—part II: supportive therapy, special situations (pregnancy, contraception, non-cardiac surgery), targeted pharmacotherapy, organ transplantatio…. Cardiovasc Diagn Ther. 2024;14(5):921-934. doi:10.21037/CDT-24-167/COIF
18. Mereles D, Ehlken N, Kreuscher S, et al. Exercise and respiratory training improve exercise capacity and quality of life in patients with severe chronic pulmonary hypertension. Circulation. 2006;114(14):1482-1489. doi:10.1161/CIRCULATIONAHA.106.618397
19. Ehlken N, Lichtblau M, Klose H, et al. Exercise training improves peak oxygen consumption and haemodynamics in patients with severe pulmonary arterial hypertension and inoperable chronic thrombo-embolic pulmonary hypertension: a prospective, randomized, controlled trial. Eur Heart J. 2016;37(1):35-44. doi:10.1093/EURHEARTJ/EHV337
20. Firdaus M, Martini H, Karolina W, Tjahjono CT, Yogibuana V. The effect of exercise training as adjuvant treatment on mean pulmonary arterial pressure by echocardiography and functional capacity in congenital heart disease with negative vaso reactivity test pulmonary hypertension patient at Saiful Anwar Hospital Malang. Heart Science Journal. 2025;6(2):83-88. doi:10.21776/ub.hsj.2025.006.02.13
21. Grünig E, Ehlken N, Ghofrani A, et al. Effect of exercise and respiratory training on clinical progression and survival in patients with severe chronic pulmonary hypertension. Respiration. 2011;81(5):394-401. doi:10.1159/000322475