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# Predictors of mortality and morbidity in acyanotic congenital heart disease with pulmonary hypertension: A cross-sectional study of patients at Dr. Soetomo General Academic Hospital Surabaya



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## ABSTRACT

**Introduction:** Congenital heart defects (CHDs) are the most common major congenital malformations in neonates. Pulmonary hypertension frequently develops as a complication, particularly in chronic or uncorrected defects. The interaction between anatomical abnormalities, hemodynamic alterations, nutritional status, and surgical outcomes highlights the need for a multidisciplinary approach to management. This study aimed to evaluate the clinical characteristics, pulmonary hypertension severity, nutritional status, and outcomes of patients with acyanotic congenital heart disease complicated by pulmonary hypertension.

**Methods:** A cross-sectional study was conducted from August 2024 to August 2025, utilizing secondary data obtained from electronic medical records. Acyanotic congenital heart disease diagnosis, pulmonary hypertension severity, nutritional status, cardio-pulmonary bypass duration, aorta clamping duration, duration of stay, and mortality were noted.

**Results:** Out of 80 recorded cases of acyanotic congenital heart disease, 37 patients with complete records were identified to have accompanying pulmonary hypertension. The mean age was 13.2 ± 14.3 years, with the majority aged 1–5 years (40.5%), and females comprising 71.3% of the study. Malnutrition was highly prevalent, with a mean Body Mass Index (BMI) of 14.9 ± 4.8 kg/m$^2$; 83.8% were underweight. Ventricular septal defect (64.9%) was the most common lesion, followed by atrial septal defect (45.9%) and atrioventricular septal defect (18.9%). Severe pulmonary hypertension was observed in 56.8% of patients, most frequently observed with ventricular septal defects (VSD). Valvular abnormalities were also common, notably tricuspid regurgitation (40.5%) and mitral regurgitation (27%). The overall mortality rate was 21.6%, occurring exclusively in underweight patients, suggesting a strong link between poor nutritional status and adverse outcomes.

**Conclusion:** Severe pulmonary hypertension and malnutrition were observed with adverse outcomes, underscoring the need for early intervention and nutritional optimization in acyanotic congenital heart disease.

**Keywords:** acyanotic congenital heart disease, malnutrition, mortality, nutritional status, pulmonary hypertension, surgical outcome.

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## INTRODUCTION

Congenital heart defects (CHDs) represent the most prevalent type of major congenital anomaly, comprising roughly one-third of all birth malformations. They are a significant contributor to morbidity, mortality, and the substantial healthcare costs linked to congenital disorders.[1] Acyanotic congenital heart disease (CHD) represents approximately 75-80% of all congenital heart defects, with a global prevalence of 8-9 per 1,000 live births.[2] Among these, ventricular septal defects (VSD), atrial septal defects (ASD), and patent ductus arteriosus (PDA) represent the most common lesions, collectively accounting for the majority of left-to-right shunt defects.[3] Despite being classified as "acyanotic", these conditions carry significant morbidity and mortality risks, particularly when complicated by the development of pulmonary hypertension.[4]

In recent classification systems, pulmonary arterial hypertension (PAH) is a subset of pulmonary arteriopathy, causing elevated pulmonary vascular resistance. This includes heritable PAH, which is related to congenital heart disease.[5,6] Group 1 PH was traditionally defined by the hemodynamic definition: mean pulmonary arterial pressure (mPAP) ⩾ 25 mmHg, pulmonary arterial wedge pressure (PAWP) ⩽ 15 mmHg, and pulmonary vascular resistance (PVR) > 3 Wood units (WU).[7] However, recent understandings are based on contemporary outcome data and a focus on early disease detection. Now, pulmonary hypertension includes patients with a

mean pulmonary artery pressure (mPAP) > 20 mmHg as measured with right heart catheterization.[8] The pathophysiology of pulmonary hypertension in CHD involves the progressive remodeling of the pulmonary vasculature secondary to chronic volume and pressure overload from left-to-right shunting. This process begins with endothelial dysfunction and progresses through medial hypertrophy, intimal proliferation, and ultimately irreversible plexiform lesion, leading to elevated pulmonary vasculature resistance.[9,10]

Malnutrition represents a prevalent and clinically significant comorbidity in patients with acyanotic congenital heart defects, with studies reporting undernutrition rates approaching 49-54% for underweight, 47.8% for stunting, and 31.4% for wasting.[11] The pathogenesis of nutritional deficits in these patients is multifactorial, involving both decreased nutrient intake and altered metabolic processes characteristic of cardiac cachexia.

Pulmonary hypertension itself emerges as an independent risk factor for undernutrition, reflecting the systemic impact of chronic cardiovascular stress on growth and development. The metabolic consequences extend beyond simple caloric deficiency, also involving the complex interaction of protein-energy malnutrition, micronutrient deficiencies, and disrupted anabolic-catabolic balance mediated by inflammatory cytokines, neurohormonal activation, and tissue hypoxia.[12] Intestinal congestion secondary to elevated central venous pressure contributes to malabsorption, early satiety, and enteric protein loss, creating a self-perpetuating cycle of nutritional deficiencies. The relationship between cardiac function and nutritional status is also bidirectional, as malnutrition can precipitate cardiomyopathy, arrhythmias, and impaired immune function, while cardiac dysfunction exacerbates nutritional compromise through reduced tissue perfusion and metabolic inefficiency.[13]

The complex interplay between anatomical factors, hemodynamic consequences, nutritional status, and surgical outcomes necessitates a multidisciplinary approach to patient care, with particular attention to identifying high-risk patients who may benefit from early intervention and intensive perioperative management strategies. Understanding these relationships is crucial for developing evidence-based protocols that can improve both short-term surgical outcomes and long-term quality of life for this vulnerable patient population. Therefore, this study aimed to evaluate the clinical characteristics, pulmonary hypertension severity, nutritional status, and outcomes of patients with acyanotic congenital heart disease complicated by pulmonary hypertension,

## RESEARCH DESIGN AND METHODS

### Types of Research

This study is an observational analytic research, employing a cross sectional method on secondary data from medical records of acyanotic congenital heart disease patients with pulmonary hypertension treated at the cardiology inpatient unit of a tertiary referral hospital in Indonesia, from August 2024 to August 2025. The primary outcome of this study was in-hospital mortality among patients with acyanotic congenital heart disease complicated by pulmonary hypertension. The secondary outcomes included morbidity-related clinical and perioperative parameters, namely pulmonary hypertension severity, nutritional status based on BMI category, cardiopulmonary bypass duration, aortic cross-clamp duration, length of hospital stay, and the presence and severity of associated valvular abnormalities, including mitral, tricuspid, aortic, and pulmonary regurgitation. We used total sampling in this study.

### Research Population

The population of this study comprises patients clinically diagnosed with acyanotic congenital heart disease accompanied by pulmonary hypertension in the cardiology inpatient unit of Dr. Soetomo General Academic Hospital during the research period. The study sample consists of medical records of patients diagnosed with acyanotic congenital heart disease that meet the inclusion and exclusion criteria. For the study, patients are included in the study with a complete medical history that comprises age, gender, acyanotic congenital heart disease diagnosis, pulmonary hypertension diagnosis, complete record of surgery procedures, variables, and history, anthropometry and nutritional status, and the outcome of surgery. Patients who present with uncertainty or no diagnosis of pulmonary hypertension, cyanotic congenital heart disease, or patients presenting with other significant comorbidities were all excluded.

### Data Collection

Complete medical data comprises a clear description of the type of acyanotic congenital heart disease, as well as a complete assessment of pulmonary hypertension severity. Additionally, information such as sex, age, mass, nutritional status, cardio-pulmonary bypass duration (CPB Time), aorta clamping duration (AoX time), duration of stay, and mortality were recorded.

Patients with no definite diagnosis of pulmonary hypertension were excluded from the data analysis of this study. Excluded patients were deleted from the database to limit the scope of this research to patients presenting with acyanotic congenital heart disease with the presence of pulmonary hypertension. Pulmonary hypertension severity was classified based on echocardiographic estimation of pulmonary artery pressure. Mild pulmonary hypertension was defined as estimated pulmonary artery systolic pressure of 36–45 mmHg, moderate pulmonary hypertension as 46–60 mmHg, and severe pulmonary hypertension as >60 mmHg. When available, additional echocardiographic findings such as tricuspid regurgitation velocity, right ventricular enlargement, septal flattening, or other signs of elevated pulmonary pressure were considered to support the classification.

### Data Analysis

Data collected from electronic medical records were analyzed by differentiating the diagnosis of accompanying acyanotic heart disease as well as their pulmonary hypertension severity. Descriptive

statistics are presented as mean ± standard deviation, where this method was used to describe factors such as the patient's demographic, acyanotic heart disease types, severity of pulmonary hypertension, CPB time, AoX time, as well as mortality.

## RESULTS

### Patient's Risk Factor and Case Presentation

Out of 80 recorded cases with various acyanotic heart disease conditions, 37 patients were found to have acyanotic heart disease conditions accompanied by pulmonary hypertension, with complete medical record data. **Table 1** shows the demographic and clinical characteristics of the study population.

**Table 1** summarizes the overall risk factors among patients with congenital heart disease (CHD) admitted to the Inpatient Unit of Dr. Soetomo General Academic Hospital between August 2024 and August 2025. A total of 37 patients with acyanotic congenital heart defects complicated by pulmonary hypertension were included in the study. The mean age was 13.2 ± 14.3 years, with the largest proportion belonging to the 1–5 year age group (40.5%), followed by 5–18 years (29.7%) and adults >18 years (24.3%), while only 5.4% were infants. Females constituted the majority of the study (71.3%), resulting in a female-to-male ratio of approximately 2.4:1.

The nutritional assessment revealed a high prevalence of malnutrition, with a mean BMI of 14.9 ± 4.8 kg/m², corresponding to underweight status. Data revealed that 83.8% of patients were underweight, whereas only 8.1% each had normal or overweight BMI. The mean body weight and height were 25.3 ± 21.7 kg and 117.2 ± 36.0 cm, respectively.

Ventricular Septal Defect (VSD) was the most prevalent lesion (64.9%), with Perimembranous VSD being the predominant subtype (29.7%). Atrial Septal Defect (ASD) was observed in 45.9% of patients, most frequently Secundum ASD (32.4%). Other notable congenital heart defects included Atrioventricular Septal Defect (AVSD; 18.9%), Patent Ductus Arteriosus (PDA; 13.5%), and Anomalous Pulmonary Venous Drainage (APVD; 5.4%). Less frequent anomalies

**Table 1. Patient Characteristics**

| Parameter | Acyanotic Heart Defects + Pulmonary Hypertension (n=37) |
|---|---|
| **Age, year, mean (SD)** | 13.24 (14.35) |
| 0 - 1 years old, n (%) | 2 (5.4%) |
| 1 - 5 years old, n (%) | 15 (40.5%) |
| 5 - 18 years old, n (%) | 11 (29.7%) |
| >18 years old, n (%) | 9 (24.3%) |
| **Sex** | |
| Male, n (%) | 11 (29.7%) |
| Female, n (%) | 26 (71.3%) |
| **Antropometry** | |
| Body Mass, kg, mean (SD) | 25.3 (21.7) |
| Height, cm, mean (SD) | 117.2 (36) |
| BMI, kg/m^2, mean (SD) | 14.9 (4.8) [underweight] |
| **BMI Categories** | |
| Underweight (BMI <18,5), n (%) | 31 (83.8%) |
| Normal weight (BMI 18,5 - 24,9), n (%) | 3 (8.1%) |
| Overweight (BMI 25,0 - 29,9), n (%) | 3 (8.1%) |
| **Surgery Profile** | |
| CPB Time, mean (SD) | 131.4 (76.8) |
| AoX Time, mean (SD) | 80.1 (56.2) |
| Length of Stay, mean (SD) | 8.4 (3.9) |
| Mortality, n (%) | 8 (21.6%) |
| **Types of Acyanotic Heart Defects** | |
| **ASD, total n (%)** | **17 (45.9%)** |
| ASD Secundum, n (%) | 12 (32.4%) |
| ASD Sinus Venosus, n (%) | 1 (2.7%) |
| ASD with PAPVD, n (%) | 2 (5.4%) |
| Bidirectional Shunt, n (%) | 2 (5.4%) |
| **VSD, total n (%)** | **24 (64.9%)** |
| VSD Perimembranous, n (%) | 11 (29.7%) |
| VSD Malaligned, n (%) | 1 (2.7%) |
| VSD SADC, n (%) | 2 (5.4%) |
| VSD Subaortic, n (%) | 1 (2.7%) |
| VSD Inlet, n (%) | 4 (10.8%) |
| Bidirectional Shunt, n (%) | 2 (5.4%) |
| Unspecified, n (%) | 3 (10.8%) |
| **AVSD, total n (%)** | **7 (18.9%)** |
| Partial, n (%) | 3 (8.1%) |
| Complete / CAVSD, n (%) | 2 (5.4%) |
| Intermediate, n (%) | 1 (2.7%) |
| Unspecified, n (%) | 1 (2.7%) |
| **PDA, total n (%)** | **5 (13.5%)** |
| Large, n (%) | 1 (2.7%) |
| Post ADO, n (%) | 3 (8.1%) |
| Unspecified, n (%) | 1 (2.7%) |
| **APVD, total n (%)** | **2 (5.4%)** |
| Supracardiac, n (%) | 1 (2.7%) |
| Partial, n (%) | 1 (2.7%) |

comprised truncus arteriosus type 1, subaortic stenosis, and superior vena cava (SVC) variants.

Severe pulmonary hypertension was the most prevalent finding, affecting 56.8% of patients, while moderate and mild pulmonary hypertension were observed in 18.9% each. The mean cardiopulmonary bypass (CPB) time was 131.4 ± 76.8 minutes, with a mean aortic cross-clamp (AoX) time of 80.1 ± 56.2 minutes. The mean length of stay was 8.4 ± 3.9 days, ranging from 2 to 18 days. Mortality was found in 8 patients (21,6%).

Valvular involvement was also frequent. Tricuspid regurgitation (TR) was present in 40.5% of patients, most often classified as severe (18.9%). Mitral regurgitation (MR) was identified in 27% of cases, predominantly moderate in severity (10.8%). Aortic regurgitation (AR) was less common (8.1%), observed exclusively at moderate severity, while pulmonary regurgitation (PR) was detected in 21.6% of patients, largely in the mild or trivial category (16.2%).

**Table 1. Patient Characteristics (Continued)**

| Parameter | Acyanotic Heart Defects + Pulmonary Hypertension (n=37) |
|---|---|
| **Other Defects, total n (%)** | **5 (13.5%)** |
| Truncus arteriosus type 1, n (%) | 1 (2.7%) |
| Subaortic stenosis, n (%) | 1 (2.7%) |
| SVC Variant, n (%) | 3 (8.1 %) |
| **Grade of Pulmonary Hypertension** | |
| Severe Pulmonary Hypertension, n (%) | 21 (56.8%) |
| Moderate Pulmonary Hypertension, n (%) | 7 (18.9%) |
| Mild Pulmonary Hypertension, n (%) | 7 (18.9%) |
| **Valvular Anomalies** | |
| **Mitral Regurgitation, total n (%)** | **10 (27%)** |
| MR Severe, n (%) | 3 (8.1%) |
| MR Moderate, n (%) | 4 (10.8%) |
| MR Mild/Trivial, n (%) | 3 (8.1%) |
| **Tricuspid Regurgitation, total n (%)** | **15 (40.5%)** |
| TR Severe, n (%) | 7 (18.9%) |
| TR Moderate, n (%) | 1 (2.7%) |
| TR Mild/Trivial, n (%) | 7 (18.9%) |
| **Aortic Regurgitation, total n (%)** | **3 (8.1%)** |
| AR Moderate, n (%) | 3 (8.1%) |
| **Pulmonary Regurgitation, total n (%)** | **8 (21.6%)** |
| PR Moderate, n (%) | 2 (5.4 %) |
| PR Mild/Trivial, n (%) | 6 (16.2%) |

ADO, Amplatzer duct occluder; AoX, aortic cross-clamp; APVD, anomalous pulmonary venous drainage; AR, aortic regurgitation; ASD, atrial septal defect; AVSD, atrioventricular septal defect; CPB, cardiopulmonary bypass; MR, mitral regurgitation; PAPVD, partial anomalous pulmonary venous drainage; PDA, patent ductus arteriosus; PR, pulmonary regurgitation; SADC-VSD, subarterial doubly committed ventricular septal defect; SVC, superior vena cava; TR, tricuspid regurgitation; VSD, ventricular septal defect.

## Severity of Pulmonary Hypertension Across Congenital Heart Defects

Table 2 presents multiple-response data showing the distribution of pulmonary hypertension severity across overlapping congenital heart defect categories. Because several patients had more than one anatomical defect, the total number of lesions across CHD categories exceeds the total analytic study of 37 patients. Severe pulmonary hypertension was most frequently observed among patients with VSD, followed by ASD, other defects, and AVSD. Mild pulmonary hypertension was also common across several lesion categories, particularly VSD, ASD, PDA, and other defects.

The congenital heart defect categories in Table 1 and Table 2 were analyzed as multiple-response lesion-based data rather than mutually exclusive patient categories. Therefore, one patient could contribute to more than one diagnostic category when overlapping defects were present, such as ASD with VSD, AVSD with additional shunt lesions, PDA, APVD, or associated vascular variants. Consequently, the summed frequencies across defect types may exceed the total number of patients in the analytic study.

**Table 2. Pulmonary Hypertension and Congenital Heart Defects overlap**

| PH Severity | VSD | ASD | AVSD | PDA | Others |
|---|---|---|---|---|---|
| Severe | 10 | 4 | 2 | 0 | 3 |
| Moderate | 2 | 4 | 1 | 0 | 0 |
| Mild | 12 | 9 | 4 | 5 | 5 |

## Severity of Pulmonary Hypertension Across Nutritional Status

Table3 shows the distribution of pulmonary hypertension severity according to nutritional status. Among underweight patients, severe pulmonary hypertension was the most frequent category, observed in 18 patients, followed by mild pulmonary hypertension in 7 patients and moderate pulmonary hypertension in 6 patients. In the normal-weight group, severe pulmonary hypertension was observed in 2 patients and moderate pulmonary hypertension in 1 patient, while no mild cases were recorded. Among overweight patients, mild pulmonary hypertension was observed in 2 patients and severe pulmonary hypertension in 1 patient, with no moderate cases. Overall, underweight patients represented the largest proportion of the study and accounted for most cases of severe pulmonary hypertension.

### Mortality Across Grades of Nutritional Status

Table 4 shows that all mortality cases occurred exclusively in patients classified as having underweight nutritional status, indicated by BMI values below the age-appropriate cutoffs. This pattern was observed consistently across the eight patients who died, highlighting a clear correlation between low BMI and increased mortality risk in this congenital heart disease study with pulmonary hypertension. The absence of deaths among patients with normal or higher BMI further underscores the critical impact of undernutrition on adverse outcomes within this patient population.

**Table 3. Pulmonary Hypertension and BMI Categories**

| PH Severity | Underweight | Normal Weight | Overweight |
|---|---|---|---|
| Mild | 7 | 0 | 2 |
| Moderate | 6 | 1 | 0 |
| Severe | 18 | 2 | 1 |

**Table 4. Mortality and BMI Categories**

| Nutritional Status | Alive | Death |
|---|---|---|
| Underweight | 23 | 8 |
| Normal Weight | 3 | 0 |
| Overweight | 3 | 0 |

## DISCUSSION

The observed distribution of congenital heart defects in this study aligns with global and regional data, strengthening the validity of these results. Ventricular septal defect (VSD) is well established as the most common congenital lesion worldwide, accounting for 20-35% of all cases across studies in Asia, Europe, and North America.[14] In this dataset, 64.9% of patients admitted to the Inpatient Unit of Dr. Soetomo General Academic Hospital were diagnosed with VSD, and perimembranous VSD was the predominant subtype at 45,8%, occurring slightly less than the established data of being the most observed type among VSDs, occurring as high as 75-80% in VSD cases.[15]

Atrial septal defect (ASD) was present in 45.9% of patients, with the Secundum type making up the majority at 71%. This matches findings from large epidemiological investigations demonstrating that Secundum ASD comprises 75-85% of all ASDs.[16] Studies in Indonesia and other Southeast Asian countries confirm high rates of both VSD and ASD, with similar proportions of Secundum ASD and perimembranous VSD identified via echocardiography.[17] The prevalence of ASD in local adult populations is further increased due to delayed detection and the asymptomatic nature of many cases in childhood.

Other significant defects in this study, such as AVSD (18.9%), PDA (13.5%), and APVD (5.4%), closely reflect published patterns in both local and global studies. PDA frequency in congenital studies typically ranges from 7-16%, while APVD remains relatively rare but carries high clinical significance due to its association with pulmonary hypertension and operative complexity.[18] The detection of rarer anomalies, including truncus arteriosus, subaortic stenosis, and SVC variants in the sample, complements the established spectrum observed in referral centers and supports the comprehensive nature of previous data.

Severe pulmonary hypertension (PH) was the most common presentation in this study of congenital heart disease patients, affecting 56.8%, while moderate and mild PH were observed less frequently at 18.9% each. This distribution shows a higher burden of advanced PH compared to regional studies, which report that mild PH is typically the most common, followed by moderate and severe forms. For example, a 2020 Indonesian cohort found mild PH in 52% of patients, moderate in 26%, and severe in 22%, notably lower than the current study's proportion of severe PH.[19]

The defect subtype and PH severity in this study mirrors previous findings that VSDs, especially large or uncorrected lesions, are most commonly linked to severe PH. At the same time, ASD cases tend to present with milder elevations in pulmonary arterial pressure or a more even distribution across severity grades. PDA, on the other hand, was found exclusively in mild PH here, consistent with other studies identifying small and isolated ductal lesions as less likely to cause severe PH. The relationship between defect subtype and PH severity reinforces the importance of early intervention and defect closure in high-risk subgroups.[19,20]

Nutritional status was significantly related to PH severity, with underweight patients forming the majority who experienced severe PH (48.6%). This is in line with recent investigations showing that malnutrition, low BMI, or low prognostic nutritional index are associated with more advanced pulmonary vascular disease and worse outcomes in PH.[21] Multiple studies suggest that while higher BMI is a risk factor for developing PH, patients with low BMI exhibit higher mortality and a more severe clinical trajectory—a pattern observed in this study as well.[22]

The mean cardiopulmonary bypass (CPB) and aortic cross-clamp (AoX) times—131.4 ± 76.8 minutes and 80.1 ± 56.2 minutes, respectively—were longer than some published regional averages, likely reflecting increased procedural complexity in patients with severe PH or multiple defects.[23] Lengths of hospital stay were also within the upper range of findings from other centers, emphasizing the burden of advanced disease and potentially complicated postoperative recovery in this group.[24]

Valvular involvement was a frequent comorbidity in this study, with tricuspid regurgitation (TR) present in 40.5% of patients and most commonly classified as severe in nearly one-fifth of cases (18.9%). The high prevalence of TR aligns with prior reports documenting that regurgitation, particularly when associated with congenital heart disease and pulmonary hypertension, is a common echocardiographic finding and major

determinant of prognosis in pediatric and adult populations. Studies have shown that severe or significant TR in congenital heart disease is often related to right ventricular overload and remodeling, particularly in conditions like VSD and AVSD, where pulmonary pressures are elevated.[25]

Mitral regurgitation (MR) was identified in 27% of patients, predominantly of moderate severity (10.8%). This observation is supported by literature noting that MR is prevalent in left-to-right shunt lesions and atrioventricular canal defects, where altered valve morphology or left-sided volume overload is present.[26] The presence of moderate or severe AV valve regurgitation is a known risk factor for unfavorable outcomes both pre- and postoperatively.

Aortic regurgitation (AR) was less frequent, occurring in 8.1% of the population and exclusively at moderate severity, echoing previous findings that significant AR is uncommon except in particular subtypes, such as those associated with subaortic stenosis or connective tissue involvement.[27] Pulmonary regurgitation (PR) was detected in 21.6% of cases, with most being mild or trivial (16.2%), a finding commonly observed in patients with post-intervention changes, RV dilation, or a history of surgical repair of right-sided outflow tract defects.

This observed pattern, where all mortality cases occurred exclusively among patients with underweight nutritional status, is strongly supported by existing literature. Multiple studies have shown that undernutrition is a critical determinant of poor outcomes in children with congenital heart disease, particularly those with pulmonary hypertension.[11] Undernourished children have diminished metabolic reserves, compromised immune function, and reduced ability to tolerate surgical interventions or acute illness, which significantly increases their risk of mortality and other adverse events.[28]

Research from diverse settings consistently demonstrates that low BMI and underweight status are associated with higher rates of hospital complications, prolonged recovery, and increased death rates compared to their well-nourished counterparts. The absence of mortality in patients with normal or higher BMI is in agreement with these findings, underscoring that adequate nutritional status confers a protective effect, supporting recovery and overall prognosis.[11] Thus, this study's outcomes strongly reinforce the global consensus that nutritional support and early correction of underweight status are vital components of care in congenital heart disease patients with pulmonary hypertension.

From a clinical standpoint, the findings of this study highlight the importance of early identification and management of patients with acyanotic congenital heart defects who are at risk of developing pulmonary hypertension and malnutrition. The high proportion of severe pulmonary hypertension and the predominance of underweight nutritional status as an outcome in this study emphasize the need for routine nutritional assessment, early cardiologic evaluation, and timely interventional correction of significant heart defects.

Several limitations of this study should be acknowledged. The relatively small sample size may limit the statistical power of the analysis and restrict the ability to draw broader cause-and-effect relationships beyond descriptive observations. In addition, because the research was conducted in a tertiary referral hospital, the study population likely represents a concentrated number of patient cases with more complex or advanced congenital heart disease who were referred for specialized management. This referral bias may explain the relatively high proportion of severe pulmonary hypertension observed in the study. It may limit the generalizability of the findings to the broader population of patients with acyanotic congenital heart disease. Future studies with larger multicenter studies and prospective designs are needed to characterize better the relationship between congenital heart defect subtypes, pulmonary hypertension severity, and nutritional status across different clinical settings.

## CONCLUSION

This study describes the clinical profile and outcomes of patients with acyanotic congenital heart disease complicated by pulmonary hypertension in a tertiary referral hospital. Ventricular septal defect and atrial septal defect were the most frequently observed lesions, while severe pulmonary hypertension and underweight nutritional status were common within the cohort. Valvular abnormalities, particularly tricuspid and mitral regurgitation, were also frequently identified. Mortality occurred only among underweight patients in this sample, suggesting that poor nutritional status may be clinically relevant in this population. However, given the descriptive design and limited sample size, these findings should be interpreted as observational patterns rather than causal or predictive associations. Larger prospective studies are needed to further evaluate the relationship between congenital defect type, pulmonary hypertension severity, nutritional status, and clinical outcomes.

## DISCLOSURES

### Ethical Clearance

This research has been approved by the ethical committee of Dr. Soetomo General Academic Hospital, Surabaya, with clearance number 4295/110/3/III/2026.

### Funding

The study is fully self-funded.

### Conflict of Interest

The authors declare that there is no potential conflict of interest regarding the publication of this manuscript. No funding was received to assist with the preparation of this manuscript.

### Author Contribution

HSB: Conceptualization, Writing, Methodology, Data Gathering, Supervision; AW: Conceptualization, Writing, Data Analysis; R: Supervision, Data Gathering, Data Curation. All authors prepare the manuscript and agree to this final version of the manuscript to be submitted to this journal.

### Use of Artificial Intelligence Generated Content

The use of Perplexity PRO AI was limited to minor language editing and minor readability improvements only. No figures, data, or scientific content and analysis were generated using AI tools.

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