



Decompensated Heart Failure in a 10-Year-Old Male: A Case of Rheumatic Heart Disease with Severe Mitral Regurgitation and Pulmonary Hypertension

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ABSTRACT

Introduction: Rheumatic Heart Disease (RHD) remains a principal cause of acquired heart disease and subsequent heart failure among children and young adults in developing nations, despite being largely preventable. This report details a case of advanced RHD presenting as decompensated heart failure in a pediatric patient, highlighting the severe consequences of the disease.

Case Illustration: A 10-year-old male presented with a two-month history of progressive abdominal distension, dyspnea on exertion, and lower limb edema. Physical examination was significant for hypertension, tachycardia, a pansystolic murmur, ascites, and bilateral pitting edema. A comprehensive diagnostic workup confirmed the clinical suspicion. Electrocardiography revealed sinus tachycardia and left atrial enlargement (P mitrale). Chest radiography demonstrated marked cardiomegaly.

Transthoracic echocardiography was pivotal, identifying severe mitral regurgitation secondary to anterior mitral leaflet prolapse, severe left atrial dilation, and a high probability of pulmonary hypertension. Pertinent laboratory findings included mild anemia, leukocytosis, hypokalemia, borderline hypoalbuminemia, and proteinuria.

Discussion: The patient's clinical presentation is a classic manifestation of chronic volume and pressure overload resulting from severe mitral regurgitation, which has culminated in biventricular failure. The discussion explores the pathophysiological cascade from valvular incompetence to systemic congestion, the significance of the counterintuitive finding of hypertension in decompensated heart failure, and the multi-organ involvement indicated by the laboratory abnormalities. The management strategy, employing guideline-directed medical therapy with diuretics, renin-angiotensin-aldosterone system inhibitors, and beta-blockers, is detailed.

Conclusion: This case underscores the devastating consequences of untreated or recurrent acute rheumatic fever. It highlights the critical and urgent need for robust primary and secondary prevention strategies for RHD, particularly in endemic regions such as Indonesia, to avert preventable morbidity and mortality in the young.

Keywords: Pediatric Heart Failure, Rheumatic Heart Disease, Mitral Regurgitation, Pulmonary Hypertension, Case Report.

INTRODUCTION

Rheumatic heart disease (RHD) is a chronic, acquired cardiovascular condition that results from valvular damage following one or more episodes of acute rheumatic fever (ARF).¹ ARF itself is a delayed, non-suppurative autoimmune sequela of a pharyngeal infection with Group A Streptococcus (GAS).³ Despite being entirely preventable, RHD persists as a major public health problem and is the most common cause of cardiovascular morbidity and premature death in children and young adults globally.¹ The global burden is substantial, with an estimated 40.5 million people living with RHD, leading to over 300,000 deaths annually.⁴

The disease disproportionately affects low- and middle-income countries (LMICs), where socioeconomic factors such as poverty, household overcrowding, and limited access to healthcare create a fertile environment for GAS transmission and hinder timely diagnosis and treatment of streptococcal pharyngitis.⁴ Indonesia, as a populous developing nation, is considered an endemic area for RHD and faces significant challenges in its management, including a lack of nationwide epidemiological data and low adherence to preventative therapies.⁶

The pathophysiology of RHD begins with an abnormal immune response to GAS infection, driven by molecular mimicry. Antibodies generated against streptococcal M-protein cross-react with structurally similar human proteins in the heart, joints, brain, and skin.¹ While the extra-cardiac manifestations of ARF typically resolve, the cardiac inflammation (carditis) can lead to permanent valvular damage. Recurrent ARF episodes inflict cumulative damage, causing progressive fibrosis, thickening, and calcification of valve leaflets and chordae.¹ The mitral valve is most commonly affected, followed by the aortic valve.¹ In children and young adults, this damage often manifests as valvular regurgitation. Over years, chronic severe mitral regurgitation imposes a relentless volume overload on the left ventricle, leading to progressive chamber dilation, myocardial dysfunction, and ultimately, congestive heart failure—the primary driver of disability and mortality in RHD.⁵

This report presents a detailed case of a 10-year-old boy with severe, decompensated heart failure secondary to previously undiagnosed RHD. The patient's young age and the advanced stage of his disease at presentation suggest an aggressive disease course, likely fueled by multiple, unrecognized episodes of ARF. This scenario points to a critical breakdown in both primary prevention (treatment of strep throat) and secondary prevention (prophylaxis after an initial ARF episode), making this case a sentinel event that reflects broader public health challenges. The objective is to illustrate the classic clinical progression, highlight key diagnostic findings, discuss the complex underlying pathophysiology, and review the principles of medical management in the context of a resource-limited setting.

CASE ILLUSTRATION

Patient Demographics and Chief Complaints

A 10-year-old Indonesian male, Muhammad Hafiz (Patient ID: 321752), presented to the Emergency Department of RSUD Kabupaten Bekasi on May 27, 2025. The primary complaints were a progressively enlarging abdomen and shortness of breath that had been worsening over the preceding two months. The symptoms were markedly exacerbated by physical activity, resulting in severe fatigue and an inability to participate in normal childhood activities.

History of Present Illness

The patient's abdominal distension and dyspnea were accompanied by palpitations and the recent development of bilateral leg swelling. A significant detail in the history was an episode of a productive cough lasting for one month prior to presentation, which had not been treated with antibiotics. The family denied any prior history of similar symptoms, known heart conditions, or chest pain.

Past Medical and Birth History

The patient had a notable history of recurrent episodes of cough and runny nose over the

past year, suggesting frequent upper respiratory infections. He was born via Caesarean section with a birth weight of 3.4 kg, and his childhood immunizations were reported to be complete.

Physical Examination

Upon admission on May 27, 2025, the patient was alert and conscious (compos mentis) but appeared ill and in moderate respiratory distress. His weight was 37.4 kg.

- **Vital Signs:**
 - Blood Pressure: 141/104 mmHg, with subsequent readings of 130/80 mmHg and 120/100 mmHg. The presence of hypertension was a consistent finding.
 - Heart Rate: 116 beats per minute (sinus tachycardia).
 - Respiratory Rate: 24 breaths per minute (tachypnea).
 - Temperature: 36.4 °C (afebrile).
 - Oxygen Saturation: 99% on room air; however, a nasal cannula was placed delivering oxygen at 2 L/min.
- **Cardiovascular Examination:** Auscultation revealed regular S1 and S2 heart sounds with an audible S3 gallop. A prominent grade III/VI pansystolic (holosystolic) murmur was heard best at the apex, radiating to the axilla, characteristic of mitral regurgitation.
- **Respiratory Examination:** Breath sounds were vesicular and clear bilaterally, with no rales or wheezing noted.
- **Abdominal Examination:** The abdomen was markedly distended, with a positive shifting dullness test confirming the presence of significant ascites. The waist circumference was 77 cm. The abdomen was soft and non-tender to palpation.
- **Extremities:** There was bilateral pitting edema of the lower limbs. Peripheral pulses were warm and well-perfused.

Diagnostic Investigations

Laboratory Findings

Blood and urine samples were analyzed on May 31, 2025. Key findings are summarized in Table 1. The results were significant for mild normocytic anemia, mild leukocytosis, hypokalemia, borderline hypoalbuminemia with low total protein, and the presence of proteinuria and microscopic hematuria.

Table 1: Summary of Laboratory Investigations

Parameter	Patient's Result	Reference Range	Unit
Complete Blood Count			
Hemoglobin	11.7	13.0 - 18.0	g/dL
Hematocrit	37	40.0 - 54.0	%
Leukocytes	10.7	4.0 - 10.5	10 ³ /μL
Thrombocytes	399	150 - 450	10 ³ /μL
Biochemistry			
Ureum	20	15 - 36	mg/dL
Creatinine	0.4	< 0.87	mg/dL

Electrolytes			
Sodium (Na)	143	136 - 146	mmol/L
Potassium (K)	3.3	3.5 - 5.0	mmol/L
Proteins			
Albumin	3.5	3.2 - 4.5	g/dL
Total Protein	6.1	6.4 - 8.3	g/dL
Urinalysis			
Protein	Positive 1	Negative	-
Erythrocytes (Sediment)	3 - 4	< 5	/LPB

Electrocardiography (ECG)

The 12-lead ECG performed on May 28, 2025, showed sinus tachycardia at a rate of approximately 115 bpm [Image 3]. The P waves in limb lead II were broad (>0.10 seconds) and notched, a classic finding known as **P mitrale**, which is indicative of left atrial enlargement. The terminal negative component of the P wave in precordial lead V1 was also prominent, further supporting this finding. These changes are the electrocardiographic signature of the chronic volume

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overload imposed on the left atrium by severe mitral regurgitation.

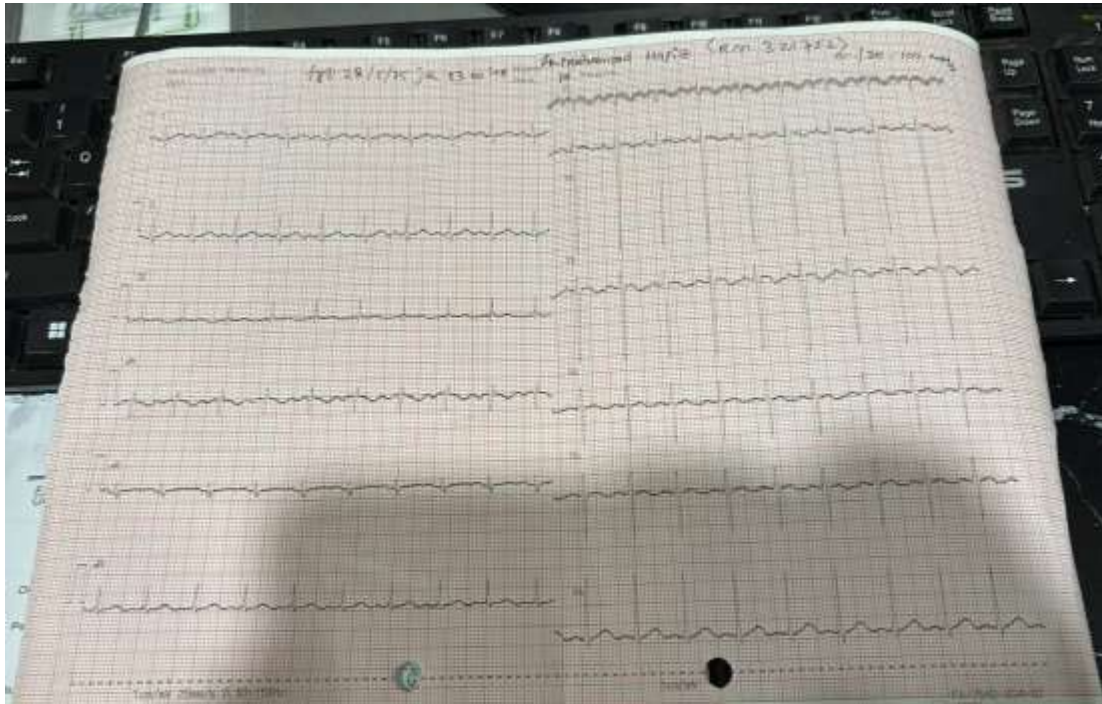


Figure 1. Electrocardiography (ECG)

Chest Radiography

An anteroposterior chest radiograph taken on May 27, 2025, was officially interpreted by a radiologist. The report concluded: "**Cardiomegaly**, suspicious for pericardial effusion. Lungs show no abnormality." The image revealed a markedly enlarged cardiac silhouette, consistent with multi-chamber enlargement from chronic volume overload. While pericardial effusion was suspected, the profound enlargement is more likely attributable to severe left atrial and ventricular dilation secondary to the underlying valvulopathy.



Figure 2. Chest Radiography

Transthoracic Echocardiography

Echocardiography performed on May 31, 2025, provided the definitive diagnosis. The key findings were:

- **Severe Left Atrial Dilation:** The left atrium was severely enlarged, with a left atrium-to-aortic root ratio (LA/Ao) of 1.90 (normal <1.5).
- **Mitral Valve Pathology:** The study identified **prolapse of the anterior mitral leaflet** as the underlying anatomical abnormality. This is a common manifestation of chronic rheumatic valvulitis.
- **Severe Mitral Regurgitation:** The prolapsing leaflet resulted in a failure of coaptation, leading to severe mitral regurgitation, which is the primary hemodynamic lesion.
- **Pulmonary Hypertension:** Doppler interrogation of the tricuspid regurgitation jet revealed a peak velocity of 3.53 m/s. This corresponds to a peak systolic pressure gradient between the right ventricle and right atrium of 39.9 mmHg ($4 \times (3.53)^2$). This value indicates a **high**

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probability of pulmonary hypertension, a direct consequence of chronically elevated left atrial pressure.

- **Right Ventricular Function:** The Tricuspid Annular Plane Systolic Excursion (TAPSE) was 1.97 cm, suggesting grossly preserved right ventricular systolic function at the time of the study, despite the high afterload.



Figure 3. Transthoracic Echocardiography

Hospital Course and Management

The patient was admitted with an initial diagnosis of suspected congenital heart disease with decompensated heart failure (anasarca, ascites, and dyspnea).¹⁴ Following a pediatric cardiology consultation, the diagnosis was refined to heart failure secondary to severe mitral regurgitation from **Mitral Valve Prolapse of the Anterior Mitral Leaflet**, consistent with RHD.

A comprehensive medical regimen for heart failure was initiated :

- **Diuretics:** Intravenous Furosemide was started, followed by an oral regimen of **Furosemide 20 mg twice daily** to reduce systemic and pulmonary congestion. **Spironolactone 25 mg once daily** was added for its neurohormonal benefits and potassium-sparing effects.
- **Renin-Angiotensin-Aldosterone System (RAAS) Inhibition:** **Ramipril 1.25 mg once daily**, an ACE inhibitor, was initiated to reduce both preload and afterload, thereby decreasing the mitral regurgitant fraction and mitigating maladaptive cardiac remodeling.
- **Beta-Blockade:** **Bisoprolol 1.25 mg once daily** was started with a plan to titrate upwards to 2.5 mg. This was to counteract the deleterious effects of chronic sympathetic nervous system activation.

The patient's clinical condition improved with medical therapy over three days of hospitalization, and he was discharged to continue his medications on an outpatient basis with close follow-up scheduled.

DISCUSSION

This case provides a compelling and classic illustration of the natural history of RHD in a pediatric patient. The diagnosis is established not by a single finding but by the convergence of evidence from the patient's history, physical examination, and a series of targeted investigations. The history of recurrent, untreated upper respiratory infections is the likely starting point for the

cascade of autoimmune-mediated valvular injury.³ The progression to severe mitral regurgitation and, ultimately, decompensated heart failure is a well-described outcome of chronic, untreated RHD.¹²

Pathophysiological Cascade: From Infection to Biventricular Failure

The central hemodynamic abnormality in this patient is severe, chronic mitral regurgitation (MR). During ventricular systole, a significant volume of blood is ejected backward from the high-pressure left ventricle (LV) into the low-pressure left atrium (LA). This imposes a state of chronic volume overload on both the LA and the LV, as the LV must handle both the forward stroke volume and the regurgitant volume with each beat.¹³

Initially, the heart compensates. The LA dilates markedly to accommodate the large regurgitant volume without a significant rise in pressure, a change reflected in this patient's LA/Ao ratio of 1.90 and the "P mitrale" on his ECG.¹⁶ The LV undergoes eccentric hypertrophy and dilation, allowing it to generate a larger total stroke volume to maintain adequate forward cardiac output. For years, these compensatory mechanisms can keep a patient asymptomatic.

Eventually, these adaptations fail. The LA becomes less compliant, and its pressure rises. This pressure is transmitted retrograde into the pulmonary venous circulation, leading to pulmonary congestion and the symptom of dyspnea.¹² Chronically elevated pulmonary venous pressure triggers reactive pulmonary arteriolar vasoconstriction and vascular remodeling, culminating in pulmonary arterial hypertension (PAH).⁵ The high TR gradient of 39.9 mmHg in this patient is clear evidence of this advanced stage.

Sustained PAH imposes a severe afterload on the right ventricle (RV). The RV, which is not designed to pump against high pressures, eventually fails. RV failure leads to elevated systemic venous pressures, causing the clinical signs of right-sided heart failure: peripheral pitting edema, congestive hepatomegaly (not explicitly documented but likely), and ascites.⁵ This patient's

presentation with anasarca represents the end stage of this cascade: decompensated biventricular heart failure.

Interpreting the Diagnostic Evidence: A Multimodal Approach

The diagnosis was solidified through a multimodal approach. The ECG finding of **P mitrale**—a broad (>0.10 s in children), notched P wave in lead II—is a direct electrical consequence of the delayed depolarization of a severely enlarged left atrium.¹⁷ The chest radiograph provided visual confirmation of gross cardiomegaly, reflecting the multi-chamber dilation that had occurred over time. However, transthoracic echocardiography was the cornerstone of diagnosis. It not only quantified the severe left atrial dilation and confirmed the high probability of pulmonary hypertension but also identified the precise etiology: prolapse of the anterior mitral leaflet, a classic morphological feature of chronic rheumatic inflammation, leading to severe regurgitation.⁵ This constellation of findings unequivocally points to advanced RHD.

The Systemic Impact of Decompensated Heart Failure: A Multi-Organ Perspective

The patient's laboratory results paint a picture of the widespread systemic consequences of severe heart failure. The triad of hypoalbuminemia, proteinuria, and hypokalemia is particularly revealing.

- **Cardiorenal and Cardiohepatic Syndromes:** Severe right-sided heart failure causes profound systemic venous congestion. This elevates pressure in the hepatic veins, leading to congestive hepatopathy, which can impair the liver's synthetic function and contribute to the borderline low albumin (3.5 g/dL).²¹ Simultaneously, elevated renal venous pressure, combined with reduced renal perfusion from low forward cardiac output, leads to congestive nephropathy. This renal dysfunction impairs the glomerulus's ability to retain protein, resulting in the observed proteinuria.²²
- **Protein-Losing Enteropathy and Malnutrition:** Venous congestion of the intestinal wall can lead to a protein-losing enteropathy, further exacerbating hypoalbuminemia. This is often

compounded by anorexia and the increased metabolic demands of chronic illness, leading to a state of cardiac cachexia.²¹

- **Hypokalemia:** The low serum potassium (3.3 mmol/L) is a direct consequence of secondary hyperaldosteronism. The intense activation of the RAAS leads to high circulating levels of aldosterone, which promotes renal potassium excretion.²²

The Paradox of Hypertension

The finding of significant hypertension (e.g., 141/104 mmHg) in a patient with severe decompensated heart failure can seem counterintuitive. However, it is a manifestation of profound neurohormonal dysregulation. The decreased effective forward cardiac output is sensed by baroreceptors and the kidneys, triggering intense activation of the sympathetic nervous system (SNS) and the renin-angiotensin-aldosterone system (RAAS). This results in potent peripheral vasoconstriction, which increases systemic vascular resistance, and avid sodium and water retention by the kidneys.²⁵ In a young patient with compliant arteries, this can manifest as hypertension. This response is highly maladaptive, as it increases the afterload against which the failing LV must pump, thereby worsening the degree of mitral regurgitation and perpetuating a vicious cycle of cardiac decline.¹²

Rationale for Pharmacological Management

The therapeutic regimen initiated for this patient is consistent with modern, evidence-based heart failure management guidelines, which advocate for a multi-pronged approach to interrupt the neurohormonal vicious cycle.²⁷

- **Furosemide:** As a potent loop diuretic, it was essential for managing the patient's severe volume overload, providing rapid symptomatic relief from dyspnea and edema by reducing preload.²⁹
- **Ramipril (ACE inhibitor):** By blocking the RAAS, it induces vasodilation, which reduces LV afterload. This is particularly beneficial in MR, as it decreases the impedance to forward flow,

thereby reducing the regurgitant fraction and improving effective cardiac output.³¹

- **Bisoprolol (Beta-blocker):** This agent blocks the cardiotoxic effects of chronic sympathetic overstimulation, reducing heart rate, decreasing myocardial oxygen consumption, and, over the long term, promoting favorable reverse remodeling of the ventricle.³¹
- **Spironolactone (MRA):** Its use is critical to block the aldosterone pathway, preventing sodium retention, potassium loss, and myocardial fibrosis. Its potassium-sparing property was particularly indicated given the patient's baseline hypokalemia.³¹

Prognosis and the Public Health Imperative in an Endemic Setting

While medical therapy stabilized the patient's acute decompensation, his long-term prognosis remains guarded. A recent cohort study of pediatric RHD in Jakarta reported a 5-year survival rate of 90%; however, poor prognostic factors included NYHA class III-IV and reduced ejection fraction at diagnosis, both of which are features of this case.¹⁹ Without surgical correction of the mitral valve, he faces a high risk of recurrent heart failure hospitalizations, arrhythmias such as atrial fibrillation, infective endocarditis, and premature death.¹⁸

The cornerstone of his long-term management is meticulous secondary prophylaxis with intramuscular benzathine penicillin G every 3-4 weeks to prevent recurrent ARF and further valvular damage.³⁷ Lifelong prophylaxis is likely necessary given the severity of his disease.³⁷ However, adherence to this regimen is a major challenge in Indonesia, with studies showing regular prophylaxis rates as low as 23-37%.³⁹ Common barriers include the pain of injection, logistical difficulties, and lack of family and social support.⁷ Overcoming these barriers through dedicated clinics, patient education, and robust follow-up systems will be paramount to altering this child's natural history and improving his long-term outcome. Ultimately, he will require referral to a tertiary cardiac center for evaluation for mitral valve surgery—preferably repair over replacement—which offers the only definitive treatment for his severe structural heart disease.¹⁹ This case is a microcosm of the broader public health crisis of RHD in Indonesia, underscoring the urgent need for improved primary and secondary prevention strategies to avert such devastating outcomes in the

nation's youth.²⁰

CONCLUSION

This report documents the case of a 10-year-old boy with severe, decompensated heart failure resulting from advanced, undiagnosed Rheumatic Heart Disease. His presentation with severe mitral regurgitation and pulmonary hypertension is a classic yet tragic endpoint of a preventable disease process. The case effectively demonstrates the complex, multi-system pathophysiology of chronic heart failure and the rationale for modern, guideline-directed medical therapy, which led to his acute stabilization.

More importantly, this case serves as a stark reminder of the persistent and devastating impact of RHD in endemic regions. It highlights a critical failure of public health infrastructure to implement primary prevention through the prompt treatment of streptococcal pharyngitis. It further underscores the urgent need for effective, patient-centered secondary prophylaxis programs to halt disease progression in affected individuals. Without a concerted effort to strengthen these preventative health strategies, children like this patient will continue to suffer the entirely avoidable and life-threatening consequences of rheumatic heart disease.

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