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Mitral and Tricuspid Valve Regurgitation as a Subclinical Manifestation of Rheumatic Heart Disease: A Pediatric Case Report

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ABSTRACT

Background: *Rheumatic heart disease (RHD) accounts for approximately 15.6 million cases globally, with 282,000 new cases and 471,000 episodes of acute rheumatic fever (ARF) annually, predominantly affecting children aged 5–15 years in the Pacific region. In 2015, the incidence in Indonesia reached 1.18 million cases, mostly among children and young adults. RHD results from valvular damage due to an autoimmune inflammatory response to Group A Streptococcus (GAS) infection. It primarily affects the mitral valve (75%), aortic valve (25%), and rarely the tricuspid valve. ARF typically precedes RHD, marked by valvular abnormalities. Initial symptoms include sore throat, progressing in 2–4 weeks to fever and clinical signs such as polyarthralgia, polyarthritis, chorea, and erythema marginatum. Complications may include infective endocarditis, heart failure, stroke, and atrial fibrillation. The World Heart Federation recommends prophylactic administration of Benzathine benzylpenicillin G (BPG) every 3–4 weeks to prevent recurrent streptococcal infections.*

Case Report*: An 8-year-old boy presented with left-sided chest pain described as pressure-like and non-radiating, along with fatigue, nausea, and joint pain for four days. He had a history of recurrent pharyngitis beginning a year earlier. A positive Anti-Streptolysin O (ASTO) test was previously recorded. Due to persistent joint pain and chest discomfort, the patient was referred to a tertiary hospital and hospitalized for four days. Physical examination showed a body weight of 20.5 kg, height 119 cm, with no murmur or tachycardia, but arthritis and erythema marginatum were observed. Laboratory findings indicated leukocytosis (14,110/mm³), and echocardiography revealed mild mitral and tricuspid regurgitation consistent with RHD. He was treated with intramuscular BPG (600,000 IU) and oral aspirin. The patient continues monthly outpatient follow-ups for BPG injections. Conclusion: RHD remains a significant contributor to childhood morbidity and mortality. This case highlights an atypical subclinical presentation without murmur, identified through echocardiography showing dual mild valvular insufficiency. Treatment included BPG and aspirin..*

1. INTRODUCTION

Cardiovascular diseases, including rheumatic heart disease (RHD), coronary artery disease, and cerebrovascular disorders, contribute to approximately 17.9 million deaths annually worldwide. RHD alone accounts for around 288,348 deaths per year and is one of the leading causes of mortality globally. In 2015, Indonesia reported 1.18 million cases of RHD, ranking fourth globally in terms of estimated case numbers (Amalia & Platini, 2022; Dougherty et al., 2023). Between 2012 and 2018, there were 279 reported cases of RHD, consisting of 108 pediatric patients aged 3–18 years and 171 young adult patients aged 18–30 years. RHD is strongly associated with low-income and developing countries, influenced by limited access to healthcare, low health awareness, and high population density (Watkins et al., 2017).

RHD is characterized by irreversible structural and functional damage to the heart valves and is a chronic sequela of acute rheumatic fever (ARF). It is most commonly found in children from developing countries (Pangestu et al., 2024). Repeated episodes of ARF can lead to valve

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deformities, including stenosis and regurgitation, due to an abnormal immune response following a Group A Streptococcal (GAS) infection. ARF typically occurs two to three weeks after a streptococcal pharyngitis and can affect joints, skin, the central nervous system, and the heart. Fibrotic changes following recurrent inflammation may result in progressive valvular dysfunction, heart failure, or even death (Dass & Kanmanthareddy, 2023). The immune system generates antibodies to fight GAS; however, these antibodies may cross-react with the host's own tissue due to molecular mimicry, especially affecting cardiac valves (Hasanah & Suryati, 2020). This results in chronic valve damage, leading to stenosis (impaired valve opening) or regurgitation (impaired valve closure), ultimately disturbing cardiac output (Rumanti, 2018). Early ARF symptoms include joint pain and involuntary movements (chorea), and when cardiac involvement ensues, symptoms may include shortness of breath, fatigue, and peripheral edema (Dougherty et al., 2023).

In Indonesia, RHD is still frequently encountered and remains a pressing public health problem (Pangestu et al., 2024). The mitral valve is most commonly affected (75%), followed by the aortic valve (25%) (Hasanah & Suryati, 2020). Clinical manifestations of RHD may include fever associated with GAS pharyngitis, pain in large joints such as knees and elbows, exertional dyspnea, and rash on the extremities. Electrocardiography and chest X-ray assist in initial patient assessment. ECG findings may include left atrial or ventricular enlargement and, in more severe cases, atrial fibrillation due to mitral valve disease. Echocardiography plays a crucial role in confirming diagnosis, assessing severity of valve damage, and guiding treatment plans. Management of RHD includes both preventive and long-term strategies. According to the World Heart Federation, intramuscular injection of Benzathine benzylpenicillin G (BPG) every three to four weeks is effective in preventing recurrent streptococcal infections and halting disease progression.

2. METHOD

Case Report

An 8-year-old boy, referred to as An. K, was brought by his parents to the emergency department of Hospital E on Thursday, January 9, 2025, with complaints of joint swelling in both legs. Symptoms began after returning from school with right foot swelling, warmth, and redness, followed by similar symptoms in the left foot. He also complained of wrist pain and reddish patches that significantly interfered with his daily activities and sleep. He had difficulty holding objects and walking due to pain and swelling. According to his father, this was the first occurrence of such symptoms. The patient had a history of recurrent sore throats accompanied by fever since a year prior and was previously given antibiotics. However, his father discontinued the medication after two days, believing the symptoms had improved and fearing dependency. There was no history of congenital illness, and no family members had similar symptoms.

On January 11, 2025, an Anti-Streptolysin O (ASTO) test performed at Hospital E returned positive. That evening, the father reported the appearance of red, coin-shaped rashes with pale centers on the left foot, and the patient experienced six episodes of loose, bloody stools. Abdominal radiography showed fecal impaction, leading to a referral to RSMS Hospital. At 9:30 p.m. the same day, the patient presented to the emergency department of RSMS. Due to unavailable inpatient beds, the patient was referred to Hospital H for consultation with a pediatric surgeon. Based on prior abdominal X-ray results, the pediatric surgeon ruled out gastrointestinal bleeding and diagnosed fecal retention, prescribing L-Bio vitamins.

On the morning of January 13, 2025, the patient experienced a one-minute episode of non-radiating, pressure-like chest pain while sleeping, which caused him to wake up. He also reported fatigue, nausea, and persistent joint pain. He was admitted to RSMS for four days. According to the parents, the patient had decreased appetite and nausea, but no fever. Echocardiography on January 16 revealed mild mitral and tricuspid regurgitation. Physical examination showed the patient appeared weak and in pain, with vital signs as follows: heart rate 86 bpm, respiratory rate 26 breaths/min, body temperature 36.6°C, and SpO2 99% on room air. Weight was 20.5 kg and height 119 cm. Auscultation revealed normal heart sounds without murmurs. Examination noted

erythema marginatum on the left dorsum of the foot, both arms, and hands, and joint swelling in both lower limbs. Laboratory results from January 11 revealed leukocytosis (14,110/mm^3), albumin 3.65 g/dL, and calcium 8.6 mg/dL. Based on these findings, the patient was diagnosed with RHD and received intramuscular BPG (600,000 IU) and oral aspirin. He continues monthly follow-ups for prophylactic BPG injections at the pediatric outpatient clinic of RSMS.

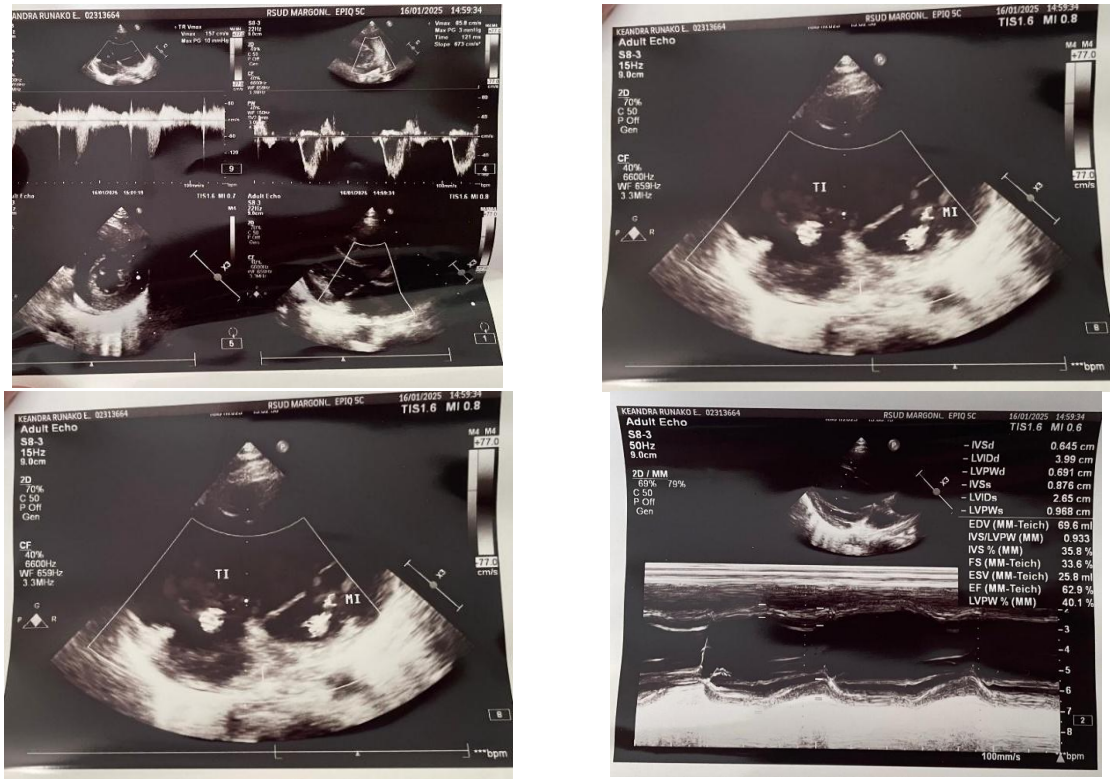

Figure 1. Echocardiographic Findings from RSMS (January 16, 2025)
(Mild Mitral Regurgitation, Mild Tricuspid Regurgitation)

Table 1. Laboratory Examination Results from RSMS

Examination	11/01	13/01	Reference range
Hemoglobin	11.0	10.8	10.8-15.6 g/dL
Leukosit	11690	**14110**	4500-13500 /mm^3
Hematokrit	33.2	31.9	33-45%
Eritrosit	4.14		3.8-5.8 10^6/µL
Trombosit	404000		181000-521000/ mm^3
MCV	80.2		69-93 fL
MCH	26.6		22-34 pg
MCHC	33.2		32-36 g/dL
RDW	13.8	14.7	11.5-14.5%
MPV	7.8	7.9	9.4-12.4 fL
Basofil	0.0	0.1	0-1%
Eosinofil	0.3	0.7	1-5%
Batang	0.4	0.4	3-6%
Segmen	77.8	64.3	25-60%
Limfosit	13.2		25-50%
Monosit	8.3	8.7	1-6%
Neutrofil	78.2	64.7	25-60%
Granulosit	12200	9090	1500-8500 /µl

Albumin	3.65		3.97-4.94 g/dL
Ureum	16.10		15-36 mg/dL
Kreatinin	0.29		0-0.6 mg/dL
Glukosa sewaktu	113		80-139 mg/dL
Kalsium	8.6		0-1 mg/dL
Natrium	136	136	80-139 mg/dL
Kalium	4.28		3.5-5.1 mmol/L
Klorida	102		97-107 mmol/L

Table 2. Urinalysis Results from RSMS

Examination	**14/01**	**16/01**	**Reference range**
Total Urine			
Fisis			
Warna	Light yellow	Light yellow	-
Kejernihan	Jernih	Jernih	-
Bau	Khas	Khas	-
Kimia			
Urobilinogen	Normal	Normal	0.2-1.0 mg/dL
Glukosa urin	Negatif	Negatif	Negatif
Bilirubin	Negatif	Negatif	Negatif
Keton	Negatif	Negatif	Negatif
Berat jenis	1.010	1.010	1.002-1.03
Eritrosit	Negatif	Negatif	Negatif
pH	7.0	7.0	4.8-7.4
Protein urin	Negatif	Negatif	Negatif
Nitrit	Negatif	Negatif	Negatif
Leukosit	Negatif	Negatif	Negatif
Sedimen urin			
Eritrosit	0	0	Negatif
Leukosit	0	0	Negatif
Kristal	Negatif	Negatif	Negatif
Bakteri	1-10	1-10	Negatif
Jamur	Negatif	Negatif	Negatif
Trikomonas	Negatif	Negatif	Negatif
Epitel	0	0	Negatif
Silinder Leukosit	Negatif	Negatif	Negatif
Silinder Eritrosit	Negatif	Negatif	Negatif
Silinder Hialin	Negatif	Negatif	Negatif
Silinder Lilin	Negatif	Negatif	Negatif
Granuler Halus	Negatif	Negatif	Negatif
Granuler Kasar	Negatif	Negatif	Negatif

Based on the collected data, the patient was diagnosed with rheumatic heart disease and was recommended to undergo prophylactic antibiotic therapy with Benzathine benzylpenicillin G 600,000 IU to prevent further spread of the infection. The patient is the second child, born via spontaneous vaginal delivery by a midwife near the family's residence on January 3, 2017. He was delivered at 36 weeks of gestation from a 29-year-old mother (G2P2A0), with a birth weight of 3,600 grams and cried immediately after birth. The mother had regularly attended prenatal check-ups with an obstetrician. During the pregnancy, she had no known medical conditions.

3. DISCUSSION

Rheumatic heart disease (RHD) is a sequela of acute rheumatic fever characterized by abnormalities of the heart valves (Dass & Kanmanthareddy, 2023). These abnormalities commonly occur in children aged 5–15 years, with an incidence of 0.3–0.8% (Kusuma & Sudarmanto, 2022). Acute rheumatic fever is an inflammatory response that can affect the heart, joints, central nervous system, and subcutaneous tissue (Hasanah & Suryati, 2020). Group A beta-hemolytic Streptococcus (GAS) has 130 M protein serotypes capable of infecting humans, but only Group A is associated with the etiopathogenesis of rheumatic fever and RHD (Afif, 2017). Rheumatic fever results from an autoimmune response triggered by pharyngitis caused by GAS. This autoimmune response begins with the recognition of Streptococcus pyogenes antigens by T and B cells. Tissue damage is mediated by immune mechanisms initiated through molecular mimicry. Structural similarities between bacterial antigens and human proteins lead to cross-reactivity, whereby antibodies and T cells attack host proteins, particularly in organs such as the heart (Uswatun, 2020).

The primary mechanism underlying the pathogenesis of RHD is molecular mimicry, in which the immune system fails to distinguish between bacterial and self-antigens due to structural similarities. During GAS infection, the immune system produces antibodies and T cells that target bacterial antigens, especially the M protein and group A carbohydrate antigens such as N-acetyl-β-D-glucosamine (GlcNAc). However, the M protein shares a structure with host proteins such as cardiac myosin, laminin, tropomyosin, and type IV collagen, which also feature an alpha-helix coiled-coil structure (Cunningham, 2019; Zhuang et al., 2025). As a result, these immune components attack cardiac tissues, particularly the valves, leading to inflammation and progressive fibrosis (Zhuang et al., 2025).

Maladaptive immune responses also play a key role, including imbalances between Th17 and Treg cells and activation of inflammatory cytokine pathways such as IL-1β and GM-CSF, which exacerbate tissue damage via CD4+ T-cell recruitment and macrophage activation (Kim et al., 2018; Wang et al., 2020). Additionally, activation of adhesion molecules (VCAM-1) on valvular endothelium facilitates lymphocyte and antigen-presenting cell infiltration, reinforcing the autoimmune response (Watkins et al., 2018). Risk factors for RHD include genetic predisposition (e.g., HLA class II alleles, TNF-α, and TGF-β1), repeated exposure to virulent GAS strains (especially emm types 1, 3, 5, and 6), and environmental conditions such as overcrowding, poor hygiene, and limited access to healthcare (Zhuang et al., 2025; Watkins et al., 2018).

The primary cause is GAS infection. Initial symptoms typically include sore throat, which can progress within 2–4 weeks to polyarthralgia, polyarthritis, chorea, or erythema marginatum (Hasanah & Suryati, 2020). Erythema marginatum presents as non-pruritic pink lesions on the trunk and limbs, sparing the face. These lesions are centrifugal, with sharply demarcated outer edges and a diffuse inner area (Dewi & Pamela, 2019). In this patient, coin-shaped erythematous lesions with pale centers were found on the elbows and wrists bilaterally, and similar lesions subsequently appeared on the dorsum of the left foot. The patient also complained of joint pain and swelling in the hands and feet. This is consistent with findings by Firdaus & Yanuarso (2022), who reported patients with RHD presented with body rashes, swollen knees, and painful swallowing. Kusuma & Sudarmanto (2022) also noted that polyarthritis is the most common symptom in rheumatic fever, characterized by severe pain, swelling, and erythema in large joints such as the knees.

Clinical manifestations of rheumatic fever are categorized into major and minor criteria according to the 2015 American Heart Association (AHA) revision (Table 3). A diagnosis is established when either two major criteria or one major and two minor criteria are present, accompanied by evidence of a preceding GAS infection (Hasanah & Suryati, 2020).

Table 3. Diagnostic Criteria for Acute Rheumatic Fever (AHA Revision, 2015)

Major Criteria	Low-Risk Population	Moderate–High-Risk Population
Carditis	Clinical or subclinical carditis	Clinical or subclinical carditis
Arthritis	Polyarthritis only	Polyarthritis or monoarthritis, polyarthralgia

Major Criteria	Low-Risk Population	Moderate–High-Risk Population
Chorea	Chorea	Chorea
Erythema marginatum	Erythema marginatum	Erythema marginatum
Subcutaneous nodules	Subcutaneous nodules	Subcutaneous nodules

Diagnosis of rheumatic heart disease can be made through physical examination and supporting investigations. Clinically, a patient may not always have a history of acute rheumatic fever, but may present with symptoms such as shortness of breath or the presence of a heart murmur upon auscultation. Elevated anti-streptolysin O (ASO) titers are typically observed in the first week, peaking between the third and sixth week following exposure to infection (Kosaraju et al., 2023). The gold standard for diagnosing rheumatic heart disease is echocardiography. This modality can identify valvular damage and assess its impact on cardiac function (Kumar et al., 2020). Echocardiography can also evaluate the severity of valve stenosis or regurgitation, pericardial effusion, and ventricular dysfunction. Common echocardiographic findings include mitral annular dilation and chordae elongation (Kusuma & Sudarmanto, 2022).

Radiological examinations may reveal cardiomegaly and pulmonary congestion as signs of longstanding heart failure due to carditis. On physical examination, heart murmurs can be heard upon auscultation, and echocardiography may reveal mitral and tricuspid regurgitation (Kusuma & Sudarmanto, 2022). The erythema marginatum manifestation in RHD cases occurs due to autoimmune processes triggered by molecular mimicry. The immune system fails to distinguish between Streptococcus antigens and self-tissues, leading antibodies and immune cells—initially formed to combat Streptococcus—to attack synovial tissue and skin (Animasahun et al., 2021; Franczyk et al., 2022). Erythema marginatum is a rare but characteristic skin manifestation of RHD, with a prevalence of less than 10%. It presents as pink or bright red macular or plaque-like rashes with raised edges, serpiginous borders, and pale centers. It is often associated with rheumatic carditis (Animasahun et al., 2021).

Polyarthralgia in RHD is a clinical manifestation resulting from a systemic autoimmune response triggered by Group A Streptococcus infection. This infection initially activates the innate immune response via epithelial cells and macrophages, releasing cytokines such as IL-1β, IL-6, TNF-α, and IL-8. GAS components like M protein, peptidoglycan, and nucleic acids stimulate immune responses that inadvertently lead to tissue damage. The M protein shares antigenic structures with human proteins such as cardiac myosin, tropomyosin, and valvular and synovial proteins. This molecular mimicry causes antibodies and T cells produced against GAS to also attack joint tissues, triggering inflammation and joint pain (Franczyk et al., 2022).

A 2020 study from Universitas Airlangga found that certain HLA-DRB1 alleles, including B112 and B115, are significantly associated with increased risk of RHD in children. These alleles enhance the immune system's tendency to mount an autoimmune response against heart tissues, increasing the risk of developing RHD (Tobing et al., 2020). Socioeconomic factors also play an important role in the occurrence of RHD in children, such as living conditions, habits, and level of knowledge. Overcrowded and unsanitary living environments facilitate the transmission of infections. Knowledge influences children's habits regarding nutritional intake and the importance of seeking healthcare services, which impact both the transmission and resolution of infections (Syam et al., 2020).

Rheumatic heart disease is a complication of acute rheumatic fever resulting from an abnormal immune response to Streptococcus infection that damages the heart valves. Streptococcus triggers neutrophil, macrophage, and dendritic cell activation, followed by antibody production by B lymphocytes (Passos, 2021). This process leads to fibrinoid degeneration and the formation of Aschoff nodules in the myocardium (Afif, 2017). Bacterial proteins like M protein resemble host proteins such as myosin and laminin, initiating cross-reactivity that damages the heart valves. Valve damage often starts with regurgitation and progresses to stenosis. Early stages may show small nodules with preserved valve function. Over time, fibrosis causes thickening, commissural fusion, and chordae tendineae shortening. The mitral valve, composed of valvular endothelial cells (VECs) and valvular interstitial cells (VICs), undergoes transformation of VICs

into myofibroblasts, leading to fibrosis and stiffness. These structural changes compromise valve function (Passos, 2021).

Both initial and recurrent episodes of rheumatic fever can damage heart valves, leading to RHD. Mitral valve lesions result in regurgitation, prompting compensatory left atrial and ventricular dilation to maintain cardiac output. However, as left ventricular dysfunction progresses, cardiac output decreases, resulting in fatigue and reduced exercise capacity (Hasanah & Suryati, 2020). In this case, echocardiography revealed mild mitral and tricuspid insufficiency. Kusuma & Sudarmanto (2022) reported that the mitral valve is most commonly affected (75%), followed by the aortic valve (20%), while the tricuspid valve is rarely involved. Molecular mimicry causes the immune system to target the mitral valve (bicuspid), which lies between the left atrium and left ventricle and is chronically exposed to high pressures and turbulent flow. Mechanical stress on the mitral valve is believed to expose hidden antigens, exacerbating the immune response (Kaplan, 2005).

Management of RHD in children requires a comprehensive approach, combining prevention, medical, and surgical interventions. Eradicating pharyngeal Streptococcus infection is essential to prevent recurrence (WHO, 2004). Prevention of recurrent GAS pharyngitis is an effective strategy for avoiding RHD. Benzathine benzylpenicillin G can be administered intramuscularly at 600,000 IU every 4 weeks in patients weighing <27 kg, and at 1,200,000 IU every 4 weeks in patients weighing >27 kg (Armstrong, 2010). Rheumatic fever symptoms, including carditis, typically respond rapidly to anti-inflammatory therapy. Aspirin is the first-line treatment. In cases of worsening carditis with heart failure and cardiomegaly, corticosteroids are recommended (Kusuma & Sudarmanto, 2022).

Table 4. Duration of Secondary Antibiotic Prophylaxis (Kumar et al., 2020)

Category	Duration
Rheumatic fever with carditis and residual heart disease	10 years or until age 40, whichever is longer
Rheumatic fever with carditis but no residual heart disease	10 years or until age 21, whichever is longer
Rheumatic fever without carditis	5 years or until age 21, whichever is longer

A 2022 trial published in The New England Journal of Medicine evaluated the administration of Benzathine Penicillin G (BPG) in children and adolescents aged 5–17 years diagnosed with RHD. The study concluded that routine BPG administration effectively prevents valve damage from recurrent Streptococcus infections and reduces RHD complications (Beaton et al., 2022).

In this case, the patient received a single intramuscular dose of 600,000 IU Benzathine benzylpenicillin G during hospitalization and continued receiving monthly injections during outpatient follow-up for secondary prophylaxis against GAS. The patient was also prescribed oral aspirin for pain relief and anti-inflammatory purposes (Kusuma & Sudarmanto, 2022). RHD complications include infective endocarditis, heart failure, stroke, and atrial fibrillation. Infective endocarditis (IE) is caused by microbial infection—mostly Gram-positive bacteria—of the endocardial surface of the heart. IE diagnosis is based on the modified Duke criteria (Tables 5 and 6). RHD is a major risk factor for IE (Willim, 2020).

Table 5. Definition of Infective Endocarditis (IE) Based on Modified Duke Criteria (Willim, 2020)

Category	**Criteria**
Definite IE	**Pathologic criteria**:
– Microorganisms confirmed by culture/histology from vegetations, emboli, or intracardiac abscess	
– Histologic evidence of endocarditis from vegetations or abscess	
Clinical criteria:	

Category	Criteria
– 2 major criteria, or – 1 major + 3 minor criteria, or – 5 minor criteria	
Possible IE	1 major + 1 minor criterion, or 3 minor criteria
Not IE	Alternative diagnosis more likely; or symptoms resolved after ≤4 days of antibiotics; or no pathologic evidence at surgery/autopsy; or does not meet possible IE criteria

Table 6. Major and Minor Criteria in Modified Duke Criteria (Willim, 2020)

Major Criteria
1. Positive blood culture for IE:
a. Typical microorganisms (e.g., *Streptococcus viridans*, *S. bovis*, HACEK group, *S. aureus*, *Enterococcus*) from 2 separate cultures
b. Persistent positive cultures (e.g., ≥2 positive cultures ≥12 hours apart or ≥3 of ≥4 cultures drawn ≥1 hour apart)
c. Single positive culture for *Coxiella burnetii* or IgG phase 1 antibody titer >1:800
2. Evidence of endocardial involvement: positive echocardiogram (vegetation, abscess, valve perforation, or prosthetic valve dehiscence)

Other RHD complications include heart failure, particularly in advanced stages. Mitral stenosis, mitral regurgitation, or aortic regurgitation cause excessive volume and pressure overload, leading to left ventricular dysfunction. According to Indonesia's 2013 Basic Health Research (Riskesdas), the prevalence of heart failure was approximately 0.13% or 229,696 individuals. Symptomatic treatment with diuretics, vasodilators, and rate-controlling agents is commonly used as supportive therapy or as a bridge to corrective intervention (Watkins et al., 2018).

Minor Criteria
1. Predisposing condition (heart disease or IV drug use)
2. Fever ≥38ºC
3. Vascular phenomena (e.g., emboli, pulmonary infarcts, mycotic aneurysm, Janeway lesions)
4. Immunologic phenomena (e.g., glomerulonephritis, Osler nodes, Roth spots, rheumatoid factor)
5. Positive blood culture not meeting major criteria

Stroke is a serious complication in RHD, especially in patients with mitral valve involvement and atrial fibrillation (AF). The stroke risk in AF patients is 2.4%, mainly due to cardioembolic events resulting from mitral stenosis. Mitral stenosis causes left atrial dilation and elevated pressure, promoting blood stasis and thrombus formation in the left atrial appendage (LAA). Chronic inflammation and atrial remodeling further worsen atrial structure and function. Oral anticoagulants such as vitamin K antagonists are recommended to prevent stroke in RHD patients with AF (Watkins et al., 2018). Atrial fibrillation is a common RHD complication and is strongly associated with poor prognosis, with or without valve intervention. Complications include heart failure, stroke, peripheral thromboembolism, and premature death (Kumar et al., 2020).

4. CONCLUTION

Rheumatic heart disease (RHD) is one of the conditions that significantly contributes to high mortality rates in children. It is a complication following acute rheumatic fever caused by an abnormal immune response to Streptococcus infection, which leads to valvular heart damage. Diagnosis is established based on the Jones criteria, which requires the presence of either two

major criteria or one major and two minor criteria, along with evidence of Group A β-hemolytic Streptococcus (GABHS) infection. Echocardiography is the key supporting investigation for detecting valvular abnormalities in RHD. Complications of RHD include infective endocarditis, heart failure, stroke, and atrial fibrillation. Secondary prevention can be achieved through intramuscular administration of Benzathine benzylpenicillin G (BPG) every 3 to 4 weeks to prevent recurrent infections and disease progression.

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