



Clinical Improvement in an Adolescent with Severe Multivalvular Rheumatic Heart Disease During Conservative Management: A Case Report

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ABSTRACT

Rheumatic heart disease (RHD) remains an important cause of cardiovascular morbidity in children and adolescents, particularly in developing countries. Severe multivalvular involvement, especially severe mitral regurgitation (MR) accompanied by severe aortic regurgitation (AR), may result in substantial hemodynamic burden and frequently requires evaluation for definitive valve intervention. However, the clinical course of selected adolescents with severe multivalvular RHD managed conservatively remains insufficiently described. Case Report: A 14-year-old boy presented with fever, progressive dyspnea, chest pain, palpitations, and migratory joint pain with swelling, preceded by recurrent

sore throat and episodes of migratory arthralgia. Laboratory investigations demonstrated elevated C-reactive protein, erythrocyte sedimentation rate, and antistreptolysin O titer. The clinical presentation was consistent with acute rheumatic fever, with carditis and polyarthritis fulfilling major Jones criteria and fever fulfilling a minor criterion, supported by evidence of preceding streptococcal infection. Echocardiography demonstrated severe MR, severe AR, and mild tricuspid regurgitation, with preserved left ventricular systolic function and low probability of pulmonary hypertension. The patient received medical treatment consisting of benzathine penicillin G, prednisone, aspirin, furosemide, and captopril, followed by regular secondary prophylaxis with benzathine penicillin G and clinical follow-up. During follow-up, the patient's fever, dyspnea, chest pain, palpitations, and migratory joint symptoms resolved, and he was able to perform light physical activities without symptoms. Serial echocardiography showed improvement of AR from severe to moderate, while severe MR persisted. At the latest follow-up, left ventricular ejection fraction remained preserved at 71%. Conclusion: This case demonstrates sustained clinical and functional improvement in an adolescent with severe multivalvular RHD during individualized conservative management and secondary antibiotic prophylaxis. However, clinical improvement did not indicate resolution of structural valvular disease, as severe MR persisted despite improvement of AR and preservation of left ventricular systolic function. Conservative management should therefore not be considered a substitute for definitive valve intervention but may provide clinical stability in selected patients while the indication and timing of intervention are reassessed through close clinical and echocardiographic follow-up.

1. INTRODUCTION

Rheumatic heart disease (RHD) remains a major global health burden, particularly in developing countries, where it continues to contribute substantially to cardiovascular morbidity and mortality. Globally, RHD is estimated to affect more than 40.5 million individuals and is responsible for approximately 306,000 deaths annually (Zhang et al., 2025). Indonesia is among the countries with a substantial burden of RHD, with an estimated 1.18 million cases reported in 2015 (Amalia and Platini, 2022). The prevalence of RHD increases with age during childhood and adolescence, rising from 4.7 per 1,000 children at 5 years of age to 21.0 per 1,000 children by 16 years of age (Rozani, 2023; Dass and Kanmanthareddy, 2023). This age-related burden is clinically important because valvular damage may progress during childhood and adolescence, potentially resulting in functional limitations and long-term cardiovascular consequences.

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Rheumatic heart disease is a chronic consequence of acute rheumatic fever (ARF), characterized by structural and functional abnormalities of the heart valves (Zahari et al., 2022). It results from an abnormal immune response following infection with Group A beta-hemolytic *Streptococcus* (GABHS). Through molecular mimicry, immune responses directed against streptococcal antigens may cross-react with cardiac tissues, resulting in persistent inflammation and progressive valvular injury. Structural valve damage may subsequently lead to regurgitation due to incomplete valve closure or stenosis due to restricted valve opening (Chowdhury et al., 2025). In children and adolescents, progressive valvular disease may have important consequences for physical activity, daily functioning, and educational activities. Marpaung et al. (2021) reported impaired school functioning in all adolescents with RHD in their study, while 85% experienced impairment in physical function.

Valvular involvement in RHD may affect a single valve or multiple valves, with the mitral valve being the most frequently affected. Lilyasari et al. (2020) reported that among patients aged 3–30 years treated at a tertiary referral hospital in Indonesia, mitral valve involvement occurred in 92.83% of cases, followed by tricuspid valve involvement in 69.17%, aortic valve involvement in 40.14%, and pulmonary valve involvement in 16.48%. Multivalvular disease is particularly important when severe mitral regurgitation (MR) occurs together with severe aortic regurgitation (AR), because simultaneous dysfunction of the left-sided valves can impose substantial hemodynamic stress and increase the risk of clinical deterioration. In a study involving patients with RHD from low- and middle-income countries, approximately 25.9% had involvement of two or more valves with moderate-to-severe disease (Karthikeyan et al., 2024). Severe multivalvular disease may therefore require careful assessment of symptoms, ventricular function, chamber remodeling, and pulmonary pressure to determine the appropriate timing of definitive intervention.

Management of severe multivalvular RHD may involve surgical valve repair or replacement, particularly when severe valvular dysfunction is accompanied by significant clinical symptoms or adverse cardiac consequences. However, the decision to proceed with invasive intervention in adolescents may be complex because it must consider not only the severity of valvular lesions but also the patient's clinical status, ventricular function, and the potential long-term implications of valve surgery. Surgical treatment can provide substantial clinical benefit, but valve replacement in children and adolescents may also be associated with long-term considerations, including prosthetic valve durability, the possibility of reoperation, and the need for lifelong anticoagulation in selected patients. Therefore, in clinically selected patients who remain hemodynamically stable and can be closely monitored, optimal medical therapy may represent an important component of management while the need and timing of definitive intervention are continuously reassessed.

In this context, conservative management refers to non-surgical treatment directed toward controlling cardiac symptoms and preventing recurrent rheumatic fever. Pharmacological management may include secondary antibiotic prophylaxis and treatment of cardiac involvement with diuretics and angiotensin-converting enzyme inhibitors (ACE-I), according to the patient's clinical condition (Hasanah and Suryati, 2020). Secondary prophylaxis is particularly important because adherence to penicillin therapy reduces the risk of recurrent ARF and subsequent progression of RHD (Ambari et al., 2024). Thus, conservative management should not be regarded as definitive correction of structural valvular disease, but rather as a strategy that may provide clinical stabilization and improvement in selected patients under close clinical and echocardiographic surveillance.

Although severe RHD in children has been associated with a substantial likelihood of requiring surgical intervention, the clinical course may vary among individual patients. Cannon et al. (2017) reported that approximately 41.6% of pediatric patients with severe RHD required surgical intervention within one year after diagnosis, increasing to 59.7% within five years and 62.7% within ten years. These findings emphasize the progressive nature of severe RHD and the importance of appropriate follow-up. However, evidence describing meaningful clinical improvement in adolescents with severe multivalvular RHD managed without early definitive valve intervention remains limited. In particular, reports describing clinical improvement

accompanied by favorable functional status and echocardiographic changes during prolonged conservative management in an adolescent with severe MR and severe AR are scarce.

Therefore, this case is clinically relevant because it describes an adolescent with severe multivalvular RHD involving severe MR, severe AR, and mild TR who demonstrated substantial clinical improvement during conservative management and close follow-up. In the present case, the initial echocardiographic examination showed severe MR and severe AR with preserved left ventricular systolic function, whereas subsequent follow-up demonstrated persistent severe MR but improvement of AR to moderate severity, together with resolution of the symptoms that had previously interfered with daily activities. This case highlights the potential role of individualized conservative management and intensive follow-up in selected adolescents with severe multivalvular RHD while the indication and timing of definitive valve intervention are carefully evaluated. Accordingly, this case report aims to describe the clinical course, conservative management strategy, and significant clinical improvement in an adolescent with severe multivalvular RHD, and to highlight the clinical considerations involved in determining the timing of definitive intervention.

2. METHOD

Case Report

A 14-year-old male, initials NHAF, presented to the Emergency Department of Primary Healthcare Center on September 18, 2024, accompanied by his father, with a major complaint of fever for three days prior to admission, which was persistent and interfered with daily activities. The fever did not improve despite the use of antipyretic medications. The patient also complained of shortness of breath for three days prior to admission. Initially, the symptom occurred during fatigue after daily activities. However, over time, the symptom progressively worsened and occurred even during mild activities, such as walking from the bedroom to the bathroom, and improved with rest. The patient also complained of chest pain accompanied by palpitations for three days prior to admission. The chest pain was described as dull, heavy, predominantly on the left side, and non-radiating. The chest pain was mainly experienced during physical activity. These symptoms occurred simultaneously with fever. In addition, the patient complained of migratory joint pain involving different parts of the body for five days prior to admission.

Prior to this episode, the patient reported having experienced migratory joint pain several times at the ages of 7 and 10 years. At that time, the patient did not seek medical treatment because the symptoms were not significantly bothersome. During this illness period, the patient was admitted to the primary healthcare center for three days and was informed of having a bacterial infection, although the patient did not recall the treatment given. The patient was discharged after his clinical condition improved.

According to the patient's mother, the patient had a history of recurrent sore throat since nine months before hospitalization in the Primary Healthcare Center. The sore throat worsened during eating and drinking. The patient sought medical attention and received antibiotics, although the name of the antibiotic was not recalled. However, the patient frequently did not complete the antibiotic course because he felt symptomatically improved.

On September 26, 2024, the patient visited a general practitioner clinic and was advised to present to the Emergency Department of Secondary Referral Hospital. The patient presented with shortness of breath that had been present for nine days prior to admission and worsened five days prior to admission. The symptom worsened particularly after mild physical activity and did not improve with rest. Shortness of breath was also worse in the left lateral position and improved when lying on the right side or with additional pillows. The patient also complained of persistent fever for nine days prior to admission, which improved with antipyretics. In addition, the patient complained of dull, heavy, non-radiating left-sided chest pain accompanied by palpitations for nine days prior to admission, mainly during activity. The patient also complained of migratory joint pain for nine days prior to admission, accompanied by swelling of the left lower extremity. The patient denied subcutaneous nodules, skin rash, or involuntary movements. Based

on these findings, the pediatrician decided to admit the patient. The patient was hospitalized with a diagnosis of rheumatic heart disease and was discharged after his clinical condition improved.

Echocardiography performed at Secondary Referral Hospital on September 27, 2024, showed dilatation of the left atrium (LA) and left ventricle (LV), normal LV systolic function with an ejection fraction (EF) of 73%, severe aortic regurgitation (AR) with calcification of the RCC and NCC, severe mitral regurgitation (MR) with mild prolapse of the anterior mitral leaflet (AML), and mild tricuspid regurgitation (TR) with TVG of 21 mmHg (Figure 1). The echocardiographic conclusion was severe MR, severe AR, and mild TR with low probability of pulmonary hypertension (PH), suggestive of rheumatic heart disease.

On October 5, 2024, the patient presented for post-hospitalization follow-up at the pediatric clinic of Secondary Referral Hospital with persistent shortness of breath and chest pain. These symptoms interfered with daily activities and sleep. The symptoms worsened during mild activity and when lying in the left lateral position and improved with rest, especially in the supine or right lateral position. The pediatrician referred the patient to a Tertiary Referral Hospital for pediatric cardiology consultant. However, the patient's mother declined immediate referral because she was in Jakarta and requested referral in December 2024.

According to medical records provided by the family, on December 5, 2024, the patient came to the pediatric cardiology clinic at Tertiary Referral Hospital as a referral from Secondary Referral Hospital with a diagnosis of rheumatic heart disease. The patient complained of shortness of breath for 1.5 months prior to admission, which currently occurred only during fatigue after physical activity and improved with rest. The patient also complained of left-sided chest pain described as heaviness accompanied by palpitations for 1.5 months prior to admission, currently occurring only during fatigue and improving with rest. The patient also reported easy fatigability during moderate physical activity, such as cleaning his room. Migratory joint pain, swelling, and fever were no longer present. The patient denied involuntary movements, subcutaneous nodules, or erythematous ring-shaped rash. Based on these findings, the pediatric cardiologist decided to admit the patient. The patient was Tertiary Referral Hospitalized for 14 days with a diagnosis of rheumatic heart disease and showed significant clinical improvement, including resolution of fever, shortness of breath, and chest pain.

Vital signs showed a blood pressure of 124/78 mmHg, heart rate of 95 beats per minute, body temperature of 36.9°C, respiratory rate of 20 breaths per minute, and oxygen saturation of 99%. Anthropometric measurements revealed a weight of 49 kg and height of 159.3 cm, resulting in a body mass index of 19.38 kg/m² and a body surface area of 1.48 m². Abdominal examination was soft, non-tender, with normal bowel sounds and no hepatosplenomegaly. Extremities showed no edema and CRT was less than 2 seconds. At the Tertiary Referral Hospital, patient was hospitalized. Laboratory examination showed elevated erythrocyte sedimentation rate (ESR) of 91 mm/hour, elevated CRP of 37.1 mg/L, and positive antistreptolysin O (ASTO) titer. Echocardiography revealed moderate-to-severe MR with thickened leaflets, moderate AR, mild TR, trivial pulmonary regurgitation, and mild-to-moderate pericardial effusion, and left ventricular ejection fraction (LVEF) of 60%. The patient was diagnosed with rheumatic heart disease and treated with intramuscular benzathine penicillin G 1.2 million IU (body weight >27 kg), prednisone 2 mg/kg/day (~20 mg every 8 hours), and captopril 0.3 mg/kg every 12 hours (~12.5 mg every 12 hours). The patient Tertiary Referral Hospitalized for 14 days and discharged on December 18, 2024, with clinical improvement. The patient subsequently attended follow-up visits one week and one month after discharge, and in February 2025 was referred back to Secondary Referral Hospital and subsequently to Secondary Referral Teaching Hospital for benzathine penicillin G prophylaxis injections.

At the age of 16 years, the patient presented to the Pediatric Cardiology Clinic at Secondary Referral Teaching Hospital on January 10, 2026, accompanied by his uncle for routine follow-up and prophylactic antibiotic injection every four weeks. The patient reported no current complaints. The patient denied fever, sore throat, migratory joint pain, shortness of breath, chest pain, palpitations, subcutaneous nodules, rash, or involuntary movements.

Based on previous medical history, the patient was diagnosed with rheumatic heart disease on September 26, 2024, at Secondary Referral Hospital and underwent hospitalization. At

that time, symptoms included fever, shortness of breath, left-sided chest pain, palpitations, and migratory joint pain. Echocardiography performed on September 26, 2024, showed severe MR, severe AR, mild TR, and LVEF of 73%. The patient currently continues regular follow-up for benzathine penicillin G prophylaxis injections.

Physical examination showed good general condition. Cardiac percussion revealed the right cardiac border at the fifth intercostal space (ICS) at the right parasternal line, upper cardiac border at the second ICS at the right parasternal line, and left cardiac border at the fifth ICS at the left axillary line. Electrocardiography at Secondary Referral Teaching Hospital showed sinus rhythm (Figure 2). Echocardiography performed on February 7, 2026, showed severe MR with thickening and prolapse of the AML, moderate AR, and LVEF of 71%.

3. RESULT AND DISCUSSION

Result

Table 1. Patient laboratory findings at Tertiary Referral Hospital (December 5, 2024)

Parameter	Result	Unit	Normal Range
Erythrocyte Sedimentation Rate (ESR)	91	mm/h	0-15
C-reactive Protein (CRP)	37.1	mg/L	< 5
ASTO	Positive		Negative

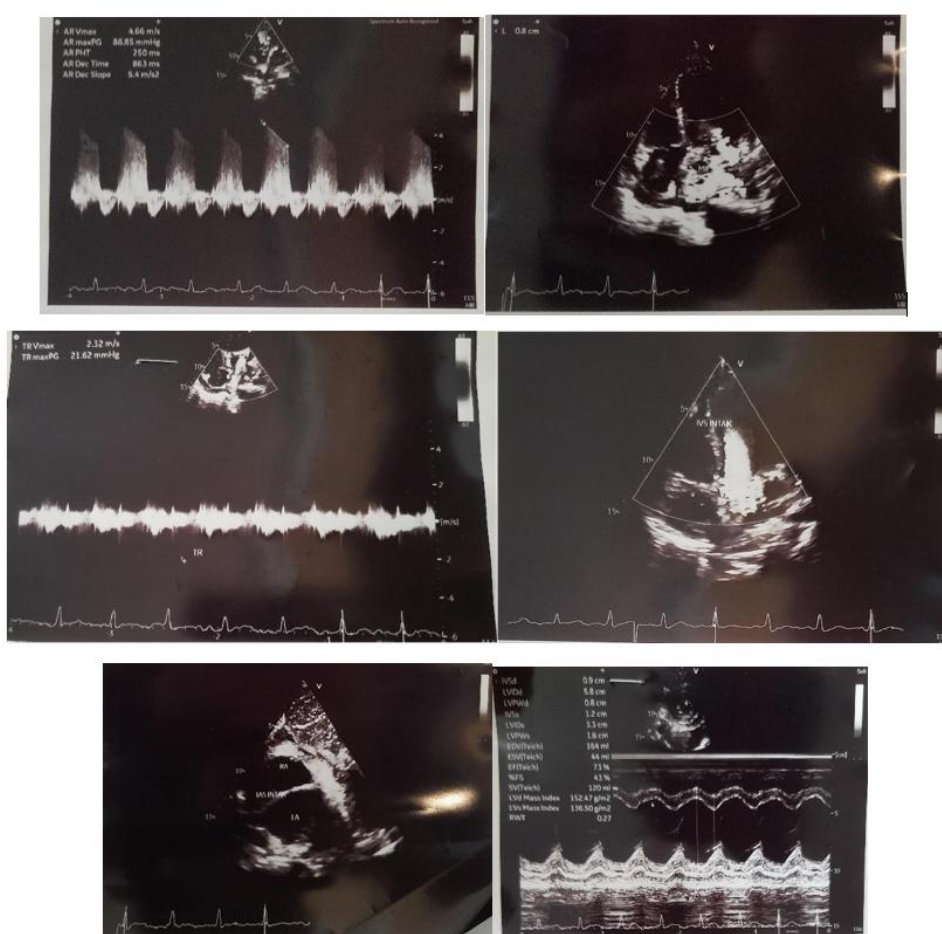


Figure 1. Echocardiography on September 24, 2024

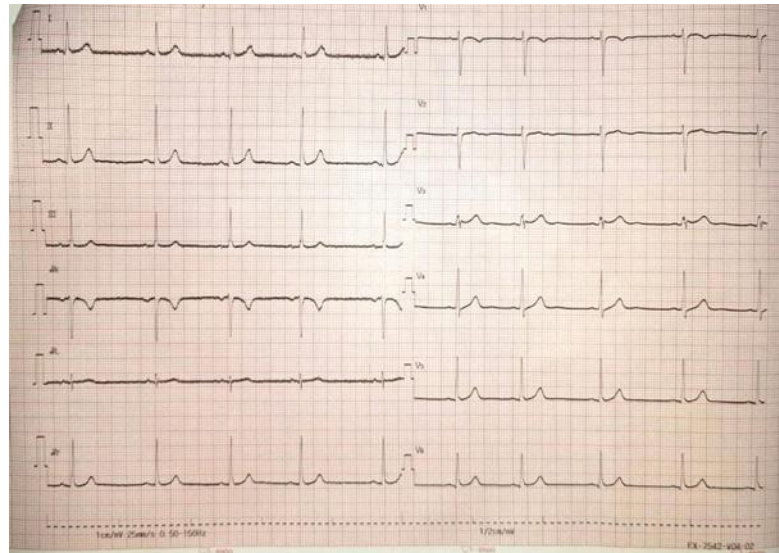


Figure 2. Electrocardiography on February 7, 2026



Figure 3. Echocardiography on February 7, 2026

Discussion

Rheumatic heart disease (RHD) is a chronic sequela of acute rheumatic fever (ARF) resulting from an abnormal immune-mediated response following Group A Streptococcus infection. The clinical course of ARF typically begins several weeks after streptococcal infection and may manifest as fever, migratory arthritis or polyarthritides, and carditis (Nuswantoro et al., 2024). In the present case, the clinical history was consistent with this sequence. The patient had a history of recurrent sore throat accompanied by fever, followed by fever, migratory joint pain, dyspnea, chest pain, and palpitations. The occurrence of migratory polyarthritides together with symptoms suggestive of cardiac involvement provided an important clinical basis for considering ARF and subsequent rheumatic valvular disease. The patient had previously experienced episodes of migratory joint pain at the ages of 7 and 10 years and reported recurrent sore throat during the months preceding the acute presentation, although documentation of microbiologically confirmed Group A Streptococcus infection was not available.

The diagnosis of ARF in this patient can be interpreted using the 2015 American Heart Association (AHA) revised Jones Criteria. The patient fulfilled two major manifestations, namely carditis and polyarthritides, and at least one minor criterion, namely fever $\geq 38.5^{\circ}\text{C}$. The patient also had laboratory evidence suggestive of recent streptococcal exposure, demonstrated by a positive antistreptolysin O (ASTO) titer. Elevated inflammatory markers, with an ESR of 91 mm/hour and CRP of 37.1 mg/L, provided additional evidence of an active inflammatory process. The positive ASTO should be interpreted as evidence supporting preceding streptococcal infection rather than as a Jones criterion itself. Similarly, the history of recurrent sore throat supports the clinical

context but cannot independently establish preceding Group A *Streptococcus* infection in the absence of microbiological confirmation. The diagnosis should therefore be interpreted in the context of the total clinical presentation rather than any single laboratory finding. The use of retrospective medical records also represents an important limitation because some historical clinical and microbiological information was incomplete.

The cardiac manifestations were further supported by echocardiographic findings. Echocardiography performed during the initial evaluation demonstrated severe mitral regurgitation (MR), severe aortic regurgitation (AR), and mild tricuspid regurgitation (TR), accompanied by left atrial and left ventricular dilatation but preserved left ventricular systolic function, with an LVEF of 73%. The low probability of pulmonary hypertension was also clinically relevant. These findings support the presence of established rheumatic valvular heart disease rather than isolated or transient carditis. Echocardiography is particularly important in RHD because it allows assessment not only of valvular morphology and severity of regurgitation but also of chamber remodeling, ventricular systolic function, pulmonary pressure, and other hemodynamic consequences.

The presence of severe MR and severe AR simultaneously at 14 years of age represents a substantial degree of multivalvular involvement. MR causes systolic regurgitation of blood from the left ventricle into the left atrium, whereas AR causes diastolic regurgitation from the aorta into the left ventricle. When these lesions coexist, the left ventricle is exposed to volume loading during both the systolic and diastolic phases of the cardiac cycle. Chronic volume overload may promote left ventricular and left atrial enlargement and eventually lead to ventricular dysfunction, pulmonary hypertension, atrial arrhythmias, and heart failure. In this patient, the initial left atrial and left ventricular dilatation indicated that significant hemodynamic consequences had already developed, despite the preserved LVEF. The coexistence of severe MR and severe AR therefore warranted careful assessment for possible valve intervention rather than being regarded as a benign echocardiographic finding.

The clinical course also illustrates the importance of distinguishing clinical improvement from structural echocardiographic improvement. At the initial presentation, the patient experienced dyspnea, chest pain, palpitations, fever, and migratory joint symptoms that interfered with daily activities. After treatment, these symptoms progressively resolved. By January 2026, the patient reported no fever, sore throat, migratory joint pain, dyspnea, chest pain, or palpitations. He was also able to perform light physical activities, including recreational swimming, without symptoms. Thus, the principal improvement observed in this case was clinical and functional.

Importantly, this clinical improvement should not be interpreted as resolution of the underlying valvular disease. The echocardiographic abnormalities persisted during follow-up. The initial examination on September 27, 2024, demonstrated severe MR, severe AR, and mild TR, with an LVEF of 73%. At tertiary referral assessment in December 2024, echocardiography showed moderate-to-severe MR, moderate AR, mild TR, mild-to-moderate pericardial effusion, and an LVEF of 60%. Subsequently, echocardiography on February 7, 2026, demonstrated persistent severe MR with thickening and prolapse of the anterior mitral leaflet, moderate AR, and an LVEF of 71%. Therefore, the reduction in AR severity from severe to moderate may represent an echocardiographic improvement in one component of the valvular disease, whereas MR remained severe. The overall structural disease consequently remained clinically important.

The trajectory demonstrates that clinical stabilization preceded complete resolution of the echocardiographic abnormalities. This distinction is important because improvement in symptoms does not necessarily indicate reversal of chronic rheumatic valve damage. Rather, the clinical response may reflect reduction of acute inflammation, reduction of hemodynamic loading, prevention of recurrent rheumatic fever, and adaptation to the residual valvular lesions.

The choice of conservative management in this patient requires careful interpretation. The presence of severe MR and severe AR would ordinarily prompt evaluation by a specialized valve team for possible intervention. Contemporary valvular heart disease guidance emphasizes that

intervention for severe regurgitant lesions is primarily intended to relieve symptoms and prevent irreversible ventricular remodeling. For severe MR, intervention is generally considered in symptomatic patients and in asymptomatic patients when objective evidence of LV dysfunction or other high-risk features is present. The 2025 ESC/EACTS guideline recommends mitral valve surgery for symptomatic severe primary MR and for asymptomatic severe MR when LV dysfunction is present, defined by LVEF $\leq 60\%$ or LV end-systolic dimensions above specified thresholds. Similarly, assessment of severe AR requires consideration of symptoms, LV systolic function, and ventricular dimensions when determining the timing of intervention.

In the present case, several clinical features may have contributed to the decision to continue conservative management. First, LV systolic function remained preserved at the initial assessment, with an LVEF of 73%, and the patient had a low probability of pulmonary hypertension. Second, during tertiary hospital assessment, the patient had no peripheral edema, pulmonary crackles, hepatosplenomegaly, or hypoxemia, and his symptoms subsequently improved during hospitalization. Third, the patient demonstrated sustained clinical improvement during follow-up, with an LVEF of 71% in February 2026. These findings suggest that the patient achieved clinical and hemodynamic stability with medical treatment.

However, the decision to defer valve intervention should not be interpreted as evidence that severe MR does not require intervention. In contemporary practice, the timing of intervention should be individualized based on symptoms, LV dimensions and function, pulmonary pressure, rhythm, progression of regurgitation, and the likelihood of durable valve repair. The absence of some important parameters in the available medical records, particularly serial LV end-systolic dimension, quantitative regurgitant volume, left atrial volume, and a formal multidisciplinary valve-team assessment, prevents a definitive conclusion regarding whether the patient fulfilled or did not fulfill all contemporary criteria for intervention. Therefore, the conservative approach in this case should be regarded as an individualized clinical decision made in the context of preserved systolic function and substantial clinical improvement, rather than as a general recommendation for patients with severe multivalvular RHD.

The pharmacological treatment may have contributed to the observed clinical improvement through several complementary mechanisms. Benzathine penicillin G was administered to eradicate or prevent recurrent Group A Streptococcus infection and subsequently continued as secondary prophylaxis. Prednisone and aspirin were used during the acute inflammatory phase, while furosemide and captopril were administered to reduce cardiac workload and manage symptoms associated with cardiac involvement. The patient's initial treatment at the tertiary hospital included benzathine penicillin G, prednisone, captopril, furosemide, and aspirin, followed by long-term benzathine penicillin prophylaxis.

The potential mechanisms of these treatments should nevertheless be interpreted cautiously. Benzathine penicillin G does not reverse established valvular fibrosis or structural deformation; its major long-term role is prevention of recurrent rheumatic fever and further rheumatic valve injury. Current WHO guidance strongly recommends antibiotic prophylaxis for children and adolescents with advanced RHD and identifies intramuscular benzathine benzylpenicillin as the preferred first-line approach for preventing recurrent rheumatic fever. In this patient, adherence to benzathine penicillin G every four weeks was an important component of long-term disease management. The absence of recurrent fever, sore throat, and migratory joint symptoms during follow-up suggests effective prevention of clinically apparent recurrent inflammatory episodes, although a causal relationship cannot be established from a single case.

Prednisone and aspirin may have contributed to control of the acute inflammatory manifestations of ARF and carditis, but they should not be considered therapies that directly repair chronic rheumatic valve damage. This distinction is particularly important because the patient continued to demonstrate severe MR despite clinical recovery. Similarly, captopril may reduce afterload and thereby reduce the hemodynamic burden associated with regurgitant lesions, while furosemide reduces congestion and preload when volume overload is present. These effects can improve symptoms and cardiac loading conditions but do not eliminate the underlying structural valve abnormalities.

Secondary prophylaxis therefore represents a particularly important component of this case. The patient subsequently received benzathine penicillin G prophylaxis every four weeks and remained under regular follow-up. Long-term prophylaxis is important because recurrent ARF can promote additional inflammatory injury and accelerate progression of existing valvular disease. WHO currently recommends long-term antibiotic prophylaxis for patients with advanced RHD to prevent recurrence of rheumatic fever and identifies IM benzathine benzylpenicillin as the preferred first-line prophylactic agent. Thus, the favorable clinical course observed in this patient should be interpreted in the context of both medical management and sustained secondary prevention rather than pharmacological treatment alone.

The clinical improvement observed in this case is also relevant when compared with the natural history of severe RHD. Cannon et al. (2017) reported that approximately 41.6% of pediatric patients with severe RHD were estimated to require surgical intervention within one year of diagnosis, increasing to 59.7% within five years and 62.7% within ten years. In addition, previous studies have demonstrated that moderate-to-severe multivalvular involvement is not uncommon among patients with RHD. In the present case, however, the patient was only 14 years old at diagnosis and had simultaneous severe MR and severe AR with preserved LV systolic function. After approximately 16 months of follow-up, he remained clinically asymptomatic despite persistent severe MR and moderate AR. This combination of young age, severe left-sided multivalvular disease, preserved systolic function, prolonged conservative management, and sustained clinical improvement represents the principal clinical interest of this case.

The present case should therefore be distinguished from the interpretation that conservative therapy resulted in anatomical resolution of RHD. The more appropriate interpretation is that clinical improvement occurred despite persistent structural valvular disease. This distinction represents the main educational value of the case. The patient's symptoms initially substantially restricted physical activity; however, during follow-up he was able to resume light recreational activities without symptoms. The preservation of LVEF at 71% in February 2026 further supports maintained systolic function, but it does not eliminate the risk associated with persistent severe MR.

The absence of valve surgery through the last follow-up requires particular consideration. The available records document sustained clinical improvement, preserved LVEF, absence of current symptoms, and regular follow-up; however, they do not provide a detailed formal explanation from a multidisciplinary valve team for why mitral valve repair or replacement was deferred. Furthermore, important quantitative parameters used in determining intervention timing, such as serial LV end-systolic diameter, LV end-systolic volume, quantitative MR severity, left atrial volume, and detailed pulmonary pressure measurements, are not consistently available. Therefore, it would be inappropriate to conclude that surgery was definitively contraindicated or unnecessary. Rather, the case demonstrates that conservative management was continued in a clinically stable adolescent while the residual valvular disease was monitored.

Long-term follow-up remains essential because symptomatic improvement does not eliminate the potential for progression of chronic valvular disease. Persistent severe MR and moderate AR may eventually result in progressive chamber remodeling, deterioration of LV systolic function, pulmonary hypertension, atrial or ventricular arrhythmias, heart failure, or the eventual need for valve repair or replacement. Contemporary guidance emphasizes serial clinical and echocardiographic assessment in patients with severe valvular regurgitation who are being managed without immediate intervention. In this patient, continued echocardiographic surveillance is therefore necessary even in the absence of symptoms.

This case has several limitations. First, it represents a single patient and therefore cannot establish the effectiveness of conservative management for severe multivalvular RHD. Second, because some historical information was obtained retrospectively, documentation of the preceding streptococcal infection and the exact chronology of earlier rheumatic episodes was incomplete. Third, echocardiographic examinations were not available at every interval, and some parameters relevant to valve intervention, including serial LV dimensions and quantitative

measures of regurgitation, were not consistently documented. Fourth, objective measurements of functional capacity, such as cardiopulmonary exercise testing or standardized exercise capacity assessment, were not performed. Finally, because several treatments were administered concurrently, including antibiotics, corticosteroids, diuretics, ACE inhibition, and secondary prophylaxis, the individual contribution of any particular treatment to the observed clinical improvement cannot be determined.

Overall, this case demonstrates that an adolescent with severe multivalvular RHD involving severe MR and severe AR may experience sustained clinical and functional improvement during carefully monitored conservative management when ventricular systolic function remains preserved and recurrent rheumatic episodes are prevented. However, the persistent severe MR and moderate AR on follow-up demonstrate that clinical improvement does not equate to structural resolution of valvular disease. The principal significance of this case is therefore not to suggest that conservative treatment replaces valve intervention, but to highlight the potential role of individualized medical management, secondary prophylaxis, and close echocardiographic surveillance in selected clinically stable adolescents while the timing of definitive valve intervention is continuously reassessed.

4. CONCLUSION

This case illustrates severe multivalvular rheumatic heart disease in an adolescent with severe mitral regurgitation and severe aortic regurgitation who demonstrated sustained clinical and functional improvement during conservative management and regular follow-up. The patient's clinical presentation, including fever, migratory polyarthritis, evidence of recent streptococcal exposure, elevated inflammatory markers, and echocardiographic evidence of significant valvular disease, supported the diagnosis of rheumatic heart disease. Long-term secondary prophylaxis with benzathine penicillin G, together with medical therapy for cardiac involvement, was associated with resolution of the patient's clinical symptoms and preservation of left ventricular systolic function.

Importantly, clinical improvement did not represent resolution of the underlying structural valvular disease. At the latest echocardiographic follow-up, severe mitral regurgitation persisted, while aortic regurgitation had improved from severe to moderate and left ventricular ejection fraction remained preserved at 71%. Therefore, the findings of this case should not be interpreted as evidence that conservative management replaces definitive valve intervention in severe multivalvular rheumatic heart disease. Rather, they highlight that individualized medical management, adherence to secondary antibiotic prophylaxis, and close clinical and echocardiographic surveillance may achieve sustained clinical stability in selected adolescents while the need and timing of definitive valve intervention are continuously reassessed. Long-term follow-up remains essential because persistent severe mitral regurgitation and moderate aortic regurgitation may still progress and lead to ventricular remodeling, heart failure, arrhythmia, or future need for valve intervention.

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