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When the Heart Stops Conducting: AV Block in Acute Rheumatic Fever

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ABSTRACT

Acute rheumatic fever (ARF) is an autoimmune inflammatory complication that occurs following pharyngitis caused by group A β-hemolytic Streptococcus (GAS). Although ARF predominantly affects children aged 5–15 years, initial presentations in young adults pose significant diagnostic challenges because of atypical clinical manifestations and low clinical suspicion. Cardiac conduction abnormalities commonly manifest as first-degree atrioventricular (AV) block, whereas complete AV block is exceptionally rare. This case report describes ARF in a young adult presenting with first-degree AV block and subclinical carditis, accompanied by a structured review of rheumatic conduction abnormalities. A 22-year-old Indonesian woman presented to the emergency department with a one-week history of severe migratory polyarthralgia involving the lower extremities, resulting in difficulty walking. She reported an untreated episode of sore throat and fever four weeks before presentation. Initial 12-lead electrocardiography (ECG) demonstrated first-degree AV block, with a prolonged PR interval of 240 ms (0.24 seconds) and a heart rate of 84 beats/min. Laboratory investigations revealed elevated inflammatory markers, including an erythrocyte sedimentation rate (ESR) of 88 mm/h and a C-reactive protein (CRP) level of 14.34 mg/L, along with leukocytosis (11,200 cells/μL) and an elevated antistreptolysin O (ASO) antibody titer of 1:400. Transthoracic echocardiography revealed mild mitral regurgitation with a preserved left ventricular ejection fraction of 67%, consistent with subclinical carditis. The diagnosis of ARF was established using the 2015 revised Jones criteria, based on one major and three minor manifestations. Treatment with oral methylprednisolone (24 mg/day), acetaminophen, and a single intramuscular dose of benzathine penicillin G (1.2 million units) was followed by normalization of the PR interval to 160 ms (0.16 seconds) and resolution of symptoms within 72 hours. This case highlights the diagnostic significance of PR interval prolongation in young adults presenting with migratory joint symptoms and suspected ARF. Although first-degree AV block may accompany rheumatic carditis, it is not independently diagnostic of valvular involvement. Early recognition, appropriate cardiac evaluation, and timely treatment are essential for identifying cardiac manifestations and guiding the management of ARF.

INTRODUCTION

Acute rheumatic fever (ARF) and its chronic sequela, rheumatic heart disease (RHD), remain major global public health concerns and significant causes of acquired cardiovascular morbidity and mortality among children, adolescents, and young adults in low- and middle-income countries (Nascimento et al., 2023; Watkins et al., 2017). Global Burden of Disease estimates indicate that more than 470,000 new episodes of ARF occur annually, over 33.4 million individuals live with chronic RHD, and approximately 319,400 RHD-related deaths

occur each year (Kotit et al., 2021; Watkins et al., 2017). Most cases and deaths occur among socioeconomically disadvantaged populations, where access to primary healthcare services, timely antibiotic treatment of streptococcal infections, and secondary antibiotic prophylaxis remains inadequate (Guilherme et al., 2017; Karthikeyan & Kothari, 2023; Watkins et al., 2017).

In Southeast Asia, particularly Indonesia, the clinical burden of ARF and RHD remains substantial (Arafuri et al., 2022; Ardini et al., 2025; Çevlik et al., 2025). Historical epidemiological surveys conducted across several Indonesian provinces reported an ARF prevalence ranging from 0.3 to 0.8 cases per 100 school-aged children (Çevlik et al., 2025). Subsequent national epidemiological assessments estimated that more than one million individuals in Indonesia were living with chronic RHD in 2015, with the highest prevalence observed among adults aged 20–50 years (Çevlik et al., 2025). Survival analyses of Indonesian cohorts have also reported an RHD-related mortality rate of 4.8 deaths per 100,000 individuals at risk (Arafuri et al., 2022). The continued transmission of group A Streptococcus (GAS), particularly in overcrowded and socioeconomically disadvantaged communities, underscores the importance of heightened clinical vigilance, timely diagnosis, appropriate treatment, and structured secondary antibiotic prophylaxis (Çevlik et al., 2025; Guilherme et al., 2017).

Acute rheumatic fever typically develops two to four weeks after untreated or inadequately treated pharyngitis caused by group A β-hemolytic Streptococcus pyogenes (Gewitz et al., 2015; Guilherme et al., 2017). Its underlying immunopathogenesis involves molecular mimicry, in which bacterial antigens, particularly epitopes of the streptococcal M protein and N-acetyl-β-D-glucosamine (GlcNAc), exhibit structural similarities to host proteins, including cardiac myosin, tropomyosin, laminin, vimentin, and certain neuronal antigens (Guilherme et al., 2017; Zhuang et al., 2025). This molecular resemblance contributes to an autoimmune response that targets cardiac and other host tissues (Pan et al., 2022; Song et al., 2025).

In genetically susceptible individuals, infection with rheumatogenic GAS strains can activate cross-reactive CD4+ T lymphocytes and induce the production of autoreactive immunoglobulin G (IgG) antibodies (Guilherme et al., 2017; Zhuang et al., 2025). These antibodies interact with cardiac endothelial structures, promote the expression of vascular cell adhesion molecule 1 (VCAM-1), and facilitate the recruitment and migration of inflammatory T lymphocytes into cardiac tissues (Guilherme et al., 2017; Zhuang et al., 2025). Persistent inflammatory infiltration may contribute to the formation of Aschoff bodies, characteristic granulomatous lesions containing activated macrophages, including Anitschkow cells, multinucleated giant cells, and areas of fibrinoid necrosis (Çevlik et al., 2025; Guilherme et al., 2017; Zhuang et al., 2025). Additionally, increased production of proinflammatory cytokines, including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α), contributes to inflammatory tissue injury and may be associated with disturbances in cardiac conduction (Guilherme et al., 2017; Zhuang et al., 2025).

Cardiac involvement in ARF primarily manifests as rheumatic valvulitis and may also involve the myocardium and pericardium, occasionally resulting in pancarditis (Gewitz et al., 2015; Guilherme et al., 2017). In addition to structural and functional valvular abnormalities, disturbances of the cardiac conduction system are recognized manifestations of acute

rheumatic inflammation (Epçaçan & Dönmez, 2019; Gewitz et al., 2015). Electrocardiographic abnormalities reported during acute rheumatic episodes include sinus tachycardia, sinus node dysfunction, junctional escape rhythms, premature atrial or ventricular contractions, bundle branch blocks, and varying degrees of atrioventricular (AV) block (Epçaçan & Dönmez, 2019; Suwansumrit & Jittham, 2022).

Atrioventricular conduction delay is among the most frequently reported electrocardiographic abnormalities in patients with ARF (Epçaçan & Dönmez, 2019; Gewitz et al., 2015). First-degree AV block, characterized in adults by a PR interval exceeding 200 ms (0.20 seconds), has been reported in up to 72.5% of patients in certain cohorts with acute rheumatic carditis (Epçaçan & Dönmez, 2019; Praneeth et al., 2020). However, the prevalence varies across populations, and PR interval prolongation does not necessarily indicate the presence or severity of valvular carditis. An age-adjusted prolonged PR interval is included as a minor manifestation in the 2015 revised Jones criteria, provided that carditis is not counted as a major manifestation (Gewitz et al., 2015). In contrast, advanced conduction abnormalities, including second-degree AV block (Mobitz type I or type II) and complete (third-degree) AV block, are uncommon manifestations of ARF, with reported frequencies ranging from approximately 0.016% to 4.6% across selected clinical series (Epçaçan & Dönmez, 2019; Kibar et al., 2015; Sunbanu, 2024).

Although ARF predominantly affects children aged 5–15 years, initial episodes and recurrences can also occur in adolescents and young adults (Guilherme et al., 2017; Praneeth et al., 2020; Umapathy & Saxena, 2018). Initial presentations in adulthood may pose diagnostic challenges because of lower clinical suspicion and variable clinical manifestations (Praneeth et al., 2020; Umapathy & Saxena, 2018). Compared with pediatric patients, some adults with ARF may present without a clearly documented history of preceding streptococcal pharyngitis or may exhibit less typical joint manifestations and conduction abnormalities (Fakhreddine et al., 2025; Praneeth et al., 2020; Umapathy & Saxena, 2018). These presentations may overlap with other inflammatory, infectious, and infiltrative cardiac conditions, including viral myocarditis, Lyme carditis, infective endocarditis, and cardiac sarcoidosis (Fakhreddine et al., 2025; Umapathy & Saxena, 2018). Consequently, careful clinical assessment, documentation of preceding GAS infection, application of the revised Jones criteria, electrocardiography, and echocardiography are essential for establishing the diagnosis and identifying cardiac involvement.

Against this background, this paper aimed to present a case report of a 22-year-old woman with ARF characterized by first-degree AV block, subclinical carditis, and migratory polyarthralgia. It also aimed to synthesize the existing literature on the pathophysiology, clinical manifestations, diagnostic evaluation, and evidence-based management of AV conduction abnormalities associated with acute rheumatic carditis. By highlighting an uncommon presentation of ARF in a young adult, this report sought to improve clinical recognition of rheumatic conduction disturbances and emphasize their potential reversibility following resolution of the underlying inflammatory process. Although rheumatic AV block is frequently transient, persistent or high-grade conduction abnormalities require individualized assessment, appropriate cardiac monitoring, and consideration of temporary or permanent pacing according to established clinical indications (Çevlik et al., 2025; Praneeth et al., 2020).

METHOD

This study employed a case report design to describe the clinical presentation, diagnosis, management, and outcomes of a 22-year-old woman with acute rheumatic fever complicated by first-degree atrioventricular (AV) block at Chasbullah Abdulmadjid Hospital in Bekasi, Indonesia. Clinical data were retrospectively obtained from the patient's medical records, including medical history, physical examination findings, laboratory results, electrocardiographic findings, echocardiographic assessments, treatment, and clinical follow-up. The diagnostic evaluation and therapeutic management were reviewed in accordance with relevant clinical guidelines and published literature.

RESULTS AND DISCUSSION

Patient History and Clinical Presentation

A 22-year-old Indonesian female presented to the Emergency Department of Chasbullah Abdulmadjid Hospital, Bekasi City, with a chief complaint of severe, progressive pain affecting multiple joints in her lower extremities over the preceding 7 days. The patient reported that her articular symptoms initially commenced 2 weeks prior as moderate pain and stiffness in her left upper extremity, specifically localized to the shoulder and elbow joints. Over the subsequent days, the upper extremity joint pain spontaneously subsided but migrated sequentially to involve her bilateral knees and ankles. The lower extremity joint pain progressively intensified, rendering weight-bearing extremely painful and resulting in severe gait impairment that prompted emergency medical evaluation.

Upon detailed anamnesis, the patient reported experiencing an episode of moderate-grade fever, sore throat, and non-productive cough approximately 4 weeks prior to emergency presentation, for which she did not seek formal medical evaluation or receive antibiotic therapy. She denied any history of chest pain, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, palpitations, presyncope, or syncope. She further denied any history of cutaneous rashes, subcutaneous nodules, abnormal involuntary choreiform movements, or prior episodes of acute rheumatic fever during childhood. Her past medical, surgical, and family histories were unremarkable, and she was taking no chronic medications prior to admission.

Physical Examination Findings

At presentation, physical examination demonstrated an alert, oriented young female in moderate painful distress secondary to joint discomfort. Her baseline vital signs were as follows: blood pressure of 107/73 mmHg, heart rate of 97 beats per minute (regular rhythm), body temperature of 36.8°C (afebrile at presentation), respiratory rate of 18 breaths per minute, and oxygen saturation of 99% on ambient room air. Her body weight was 48 kg, and body mass index was 19.2 kg/m (Kotit et al., 2021).

Head, neck, and throat examination demonstrated mild pharyngeal erythema without active tonsillar exudates, petechiae, or cervical lymphadenopathy. Cardiovascular examination revealed normal first (S1) and second (S2) heart sounds without gallops, pericardial friction rubs, or overt click. A subtle Grade 1/6 apical systolic murmur was noted upon meticulous auscultation in the left lateral decubitus position. Jugular venous pressure was normal (5-2 cm H2O), and peripheral pulses were symmetric and equal bilaterally. Pulmonary auscultation demonstrated clear breath sounds throughout all lung fields without rales or wheezes.

Abdominal examination was soft, non-tender, and without hepatosplenomegaly. Musculoskeletal examination revealed tenderness upon palpation and passive range of motion of the bilateral knee and ankle joints, without marked local erythema, overt joint effusion, or warmth. Neurological examination was entirely normal, with no evidence of Sydenham chorea. Dermatological evaluation showed no evidence of erythema marginatum or subcutaneous nodules.

Diagnostic Investigations

A baseline 12-lead electrocardiogram (ECG) recorded at presentation demonstrated a regular sinus rhythm at a heart rate of 84 beats per minute, normal cardiac axis (+45°), normal QRS duration (88 ms), and normal corrected QT interval (QTc 410 ms). Notably, the ECG revealed a distinct first-degree atrioventricular (AV) block, characterized by a prolonged PR interval duration of 0.24 seconds (240 ms), significantly exceeding the normal adult upper limit of 0.20 seconds (200 ms).

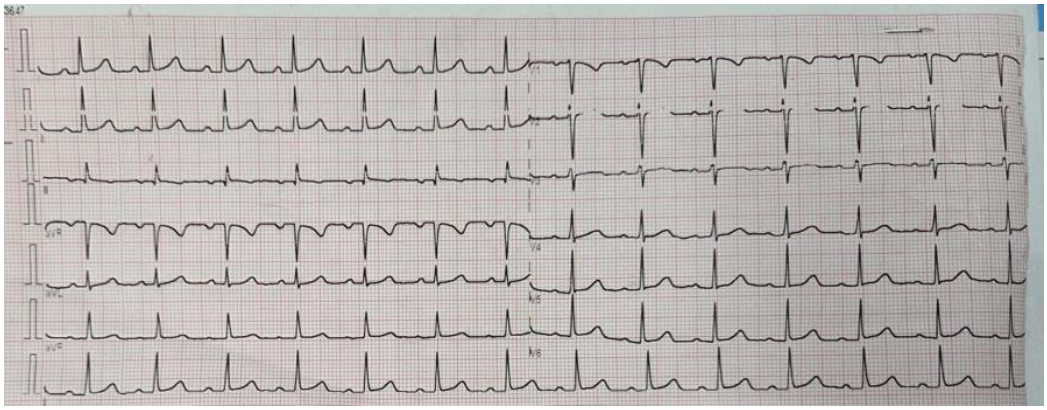


Figure 1. Initial ECG showing first degree AV block

Comprehensive emergency laboratory testing was performed upon admission. Hematological analysis demonstrated leukocytosis with a total white blood cell (WBC) count of 11,200 cells/µL (reference range: 4,000–10,000/µL) with 78% neutrophilia, hemoglobin of 12.1 g/dL, hematocrit of 36.5%, and platelet count of 312,000/µL. Systemic inflammatory markers were markedly elevated, with an erythrocyte sedimentation rate (ESR) of 88 mm/hr (reference: <20 mm/hr) and a high-sensitivity C-reactive protein (CRP) level of 14.34 mg/L (reference: <5.0 mg/L). Qualitative anti-streptolysin O (ASTO) serological testing was positive with a high titer of 1/400 (reference: negative or <200 IU/mL), confirming recent Group A Streptococcal exposure. Serum chemistry revealed mild hypoalbuminemia at 3.30 g/dL (reference: 3.5–5.0 g/dL), normal renal function (blood urea nitrogen 11.2 mg/dL, serum creatinine 0.68 mg/dL), normal serum electrolytes (sodium 139 mEq/L, potassium 4.2 mEq/L, chloride 101 mEq/L), and normal liver transaminases.

Chest radiography (PA view) demonstrated a normal cardiac silhouette with a cardiothoracic ratio (CTR) of 46%, normal pulmonary vascularity, and clear lung parenchymal fields. Transthoracic echocardiography (TTE) performed during admission revealed normal cardiac chamber dimensions, intact interatrial and interventricular septa, and preserved global left ventricular systolic function with an ejection fraction (LVEF) of 67%. Color Doppler flow interrogation identified mild mitral valve regurgitation with a central jet, absence of structural

valve prolapse or chordal rupture, and no pericardial effusion. Based on international diagnostic criteria, the echocardiographic finding of mild mitral regurgitation in the setting of active systemic inflammation fulfilled the definition of subclinical rheumatic carditis.

Table 1. Summary of Laboratory and Diagnostic Investigation Results

Diagnostic Parameter	Patient Result	Reference / Normal Range
White Blood Cell (WBC) Count	11,200 / µL	4,000 – 10,000 / µL
Hemoglobin	12.1 g/dL	12.0 – 15.5 g/dL
Erythrocyte Sedimentation Rate (ESR)	88 mm/hr	0 – 20 mm/hr
C-Reactive Protein (CRP)	14.34 mg/L	< 5.0 mg/L
Anti-Streptolysin O (ASTO) Titer	Positive (1/400)	Negative (< 200 IU/mL)
Serum Albumin	3.30 g/dL	3.5 – 5.0 g/dL
PR Interval (12-Lead ECG)	0.24 seconds (240 ms)	0.12 – 0.20 seconds
Left Ventricular Ejection Fraction (TTE)	67%	≥ 55%
Mitral Valve Interrogation (TTE)	Mild Mitral Regurgitation	No Regurgitation / Physiologic

Diagnostic Application of Revised Jones Criteria

Application of the 2015 Revised Jones Criteria for the Diagnosis of Acute Rheumatic Fever established a definitive diagnosis in this patient. Indonesia is categorized as a moderate-to-high risk population for ARF/RHD based on regional epidemiological prevalence. In moderate-to-high risk populations, the diagnostic threshold requires the presence of 2 major criteria, OR 1 major plus 2 minor criteria, in conjunction with confirmatory evidence of preceding GAS infection.

In our patient, the diagnosis was definitively fulfilled through the presence of 1 Major Criterion (Carditis, demonstrated echocardiographically as mild subclinical mitral regurgitation) and 3 Minor Criteria (Prolonged PR interval on ECG [0.24 s], Migratory Polyarthralgia, and Elevated Inflammatory Markers [ESR 88 mm/hr, CRP 14.34 mg/L]). Confirmatory evidence of recent Group A Streptococcal infection was established by the elevated qualitative ASTO serological titer (1/400). Alternative differential diagnoses, including viral myocarditis, systemic lupus erythematosus, infective endocarditis, and rheumatoid arthritis, were systematically evaluated and excluded.

Management and Clinical Course

Following diagnostic confirmation, a comprehensive therapeutic regimen was instituted. To control systemic inflammation and acute carditis, the patient was initiated on oral corticosteroid therapy with methylprednisolone at a dosage of 8 mg three times daily (24 mg/day). Symptomatic analgesia for articular discomfort was managed with oral paracetamol 500 mg three times daily. For primary streptococcal eradication and initial secondary prophylaxis, a single deep intramuscular injection of Benzathine Penicillin G (BPG) was administered at a standard dose of 1.2 million units.

The patient exhibited a rapid and favorable clinical response to medical therapy. Joint pain and gait impairment substantially improved within 24 hours of corticosteroid initiation. Serial 12-lead electrocardiographic monitoring performed on hospitalization Day 3 demonstrated complete resolution of the atrioventricular block, with the PR interval normalizing to 0.16 seconds (160 ms) in normal sinus rhythm at a heart rate of 76 beats per

minute. Systemic inflammatory markers checked prior to discharge demonstrated significant downward trends. The patient was discharged in stable clinical condition after 4 days of inpatient observation, with instructions to taper oral corticosteroids over 3 weeks and to receive secondary prophylaxis with intramuscular Benzathine Penicillin G (1.2 million units) every 4 weeks. At her 1-month and 3-month outpatient cardiology follow-up visits, she remained completely asymptomatic, with normal serial ECGs and preserved ventricular function.

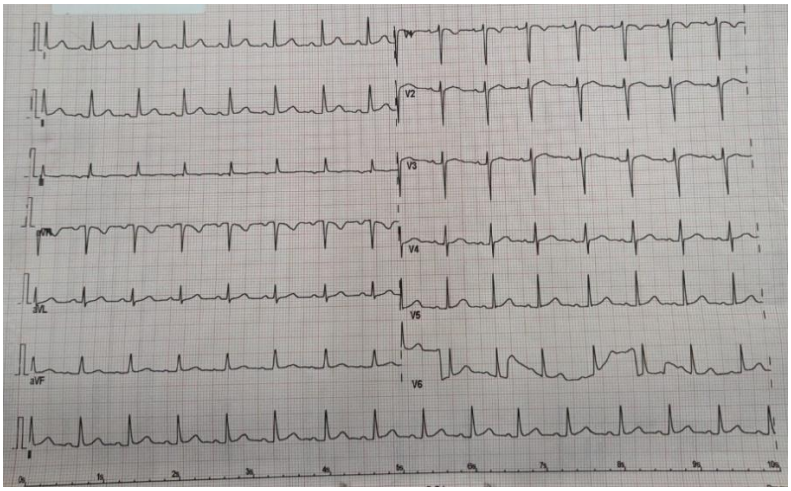


Figure 2. ECG on the third day showing sinus rhythm

The electrophysiological and pathophysiological mechanisms underlying atrioventricular (AV) conduction delays during acute rheumatic fever (ARF) involve a complex interplay between autonomic neurovegetative alterations and localized immune-mediated tissue inflammation (Guilherme et al.(Guilherme et al., 2017; Zhuang et al., 2025); Zhuang et al., 2025). Contemporary evidence supports a dual-mechanism hypothesis to explain the spectrum of rheumatic conduction disturbances (Guilherme et al.(Guilherme et al., 2017; Karacan et al., 2010)

First, heightened autonomic vagal tone (hypertonia) plays a key role in early or transient PR interval prolongation (Karacan et al.(Karacan et al., 2010) Inflammatory activation of cardiac vagal mechanoreceptors and autonomic reflex arcs during acute rheumatic attacks enhances parasympathetic input to the AV node (Epçaçan & Dönmez, 2019; Karacan et al.(Epçaçan & Dönmez, 2019; Karacan et al., 2010) This parasympathetic overdrive depresses nodal conduction velocity and prolongs the refractory period (Karacan et al.(Karacan et al., 2010) The autonomic influence is supported by clinical observations demonstrating that mild PR prolongation in ARF can frequently be transiently reversed or shortened following atropine administration or during exercise-induced sympathetic stimulation (Ardani & Aditya, 2024; Karacan et al.(Ardani & Aditya, 2024; Karacan et al., 2010)

Second, direct immune-mediated inflammatory involvement of the specialized conduction tissue represents the primary driver of persistent or advanced AV block (Guilherme et al.(Guilherme et al., 2017; Zhuang et al., 2025); Zhuang et al., 2025). Autoreactive CD4+ T lymphocytes and cross-reactive IgG antibodies—generated via molecular mimicry between streptococcal M protein/NABG epitopes and human cardiac proteins—infiltrate subendocardial tissues (Guilherme et al.(Guilherme et al., 2017; Zhuang et al., 2025); Zhuang

et al., 2025). Histopathological evaluations reveal interstitial edema, dense lymphocytic infiltration, microvascular vasculitis affecting the AV nodal artery, and localized Aschoff granuloma formation within the AV junction and bundle of His. (Guilherme et al., 2017; Zhuang et al., 2025) Furthermore, the AV node is rich in glycogen, serving as a target during acute rheumatic inflammation (Guilherme et al.(Guilherme et al., 2017) Localized interstitial swelling, microvascular ischemia, and cytokine-mediated channelopathy impair electrical impulse propagation, giving rise to first-, second-, or third-degree heart block (Guilherme et al.(Guilherme et al., 2017; Kibar et al., 2015; Zhuang et al., 2025); Kibar et al., 2015; Zhuang et al., 2025).

Atrioventricular conduction disturbances represent the most prevalent electrocardiographic finding in acute rheumatic carditis (Epçaçan & Dönmez, 2019; Gewitz et al.(Epçaçan & Dönmez, 2019; Gewitz et al., 2015) First-degree AV block, characterized by PR interval prolongation beyond 0.20 seconds in adults, occurs in 50% to 72.5% of pediatric and adult patients during acute attacks and serves as a minor diagnostic criterion in the 2015 Revised Jones Criteria (Epçaçan & Dönmez, 2019; Gewitz et al.(Epçaçan & Dönmez, 2019; Gewitz et al., 2015; Karacan et al., 2010)

In contrast, advanced degrees of conduction delay—specifically second-degree AV block (Mobitz Type I or II) and complete (third-degree) AV block—are exceptionally rare, documented in only 0.016% to 4.6% of cases across major clinical series (Epçaçan & Dönmez, 2019; Karacan et al.(Epçaçan & Dönmez, 2019; Karacan et al., 2010) Holter monitoring studies by Karacan et al. (2010) identified first-degree AV block in the majority of pediatric ARF cases, Mobitz Type I second-degree block in 1.5%, and accelerated junctional rhythm in 14% (Karacan et al.(Karacan et al., 2010) Epçaçan et al. (2019) underscored that rheumatic conduction delays are overwhelmingly transient when managed with anti-inflammatory therapy (Epçaçan & Dönmez, 2019).(Epçaçan & Dönmez, 2019)

Diagnosing acute rheumatic fever in young adults presents significant diagnostic hurdles (Fakhreddine et al.(Fakhreddine et al., 2025; Hartiono et al., 2025) Clinicians frequently maintain a low index of clinical suspicion for adult-onset ARF due to the classical perception of ARF as an exclusively pediatric illness (Hartiono et al.(Hartiono et al., 2025; Praneeth et al., 2020) Furthermore, adult patients often present with atypical articular complaints, subtle constitutional symptoms, or isolated conduction system delays without a distinct history of preceding pharyngitis (Ardani & Aditya, 2024; Fakhreddine et al.(Ardani & Aditya, 2024; Fakhreddine et al., 2025; Hartiono et al., 2025)

The differential diagnosis for new-onset atrioventricular block in a young adult is broad and encompasses viral myocarditis (e.g., Coxsackievirus, Parvovirus B19, SARS-CoV-2), Lyme carditis (Borrelia burgdorferi), infective endocarditis with perivalvular ring abscess formation, cardiac sarcoidosis, systemic lupus erythematosus, adult-onset Still's disease, and drug-induced conduction block (Ardani & Aditya, 2024; Fakhreddine et al.(Ardani & Aditya, 2024; Fakhreddine et al., 2025; Hartiono et al., 2025) Systematic diagnostic differentiation relies upon adhering strictly to the 2015 Revised Jones Criteria, obtaining serial streptococcal serologies (ASTO and anti-DNase B titers), and performing high-resolution Doppler transthoracic echocardiography (Gewitz et al.(Gewitz et al., 2015; Hartiono et al., 2025; Yakut et al., 2022)

The integration of Doppler transthoracic echocardiography into the 2015 AHA Revised Jones Criteria represents a monumental advancement in rheumatic fever diagnosis (Gewitz et al.(Gewitz et al., 2015) Historically, rheumatic carditis required clinical auscultation of a pathologic murmur or overt heart failure signs (Gewitz et al.(Gewitz et al., 2015) However, echocardiography provides substantially higher diagnostic sensitivity, detecting subclinical carditis (valvulitis without an auscultatory murmur) in up to 30% of ARF patients who would otherwise remain undiagnosed (Gewitz et al.(Gewitz et al., 2015; Kotit et al., 2021)

According to 2015 AHA Doppler criteria, pathological mitral regurgitation is defined by fulfilling all 4 of the following parameters: (1) Visualized in at least 2 immunofluorescent planes; (2) Jet length ≥2 cm; (3) Peak velocity >3 m/s; and (4) Pan-systolic duration (Gewitz et al.(Gewitz et al., 2015) Pathological aortic regurgitation requires: (1) Visualized in 2 planes; (2) Jet length ≥1 cm; (3) Peak velocity >3 m/s; and (4) Pan-diastolic duration (Gewitz et al.(Gewitz et al., 2015) In our patient, Doppler echocardiography identified mild subclinical mitral regurgitation, satisfying the major carditis criterion and confirming active endocardial involvement (Gewitz et al.(Gewitz et al., 2015; Yakut et al., 2022) Yakut et al., 2022).

Therapeutic management of ARF carditis with conduction disturbances centers upon rapid control of systemic inflammation, primary streptococcal eradication, temporary hemodynamic stabilization, and lifelong secondary prophylaxis (Gerber et al.(Gerber et al., 2009; Gewitz et al., 2015; Yakut et al., 2022)

Anti-inflammatory Pharmacotherapy: Systemic anti-inflammatory agents represent the mainstay of therapy (Gewitz et al.(Gewitz et al., 2015; Yakut et al., 2022) In patients with mild carditis or isolated PR prolongation, high-dose salicylates (aspirin 50–100 mg/kg/day in children; 3–4 g/day in adults) or NSAIDs provide rapid resolution of joint pain and conduction delay (Gerber et al.(Gerber et al., 2009; Gewitz et al., 2015; Yakut et al., 2022)09; Gewitz et al., 2015; Yakut et al., 2022). In cases of moderate-to-severe carditis, high-degree AV block, or hemodynamic compromise, systemic corticosteroids (oral prednisone 1–2 mg/kg/day or methylprednisolone 24–48 mg/day) are recommended (Gewitz et al.(Gewitz et al., 2015; Kibar et al., 2015; Suwansumrit & Jittham, 2022; Yakut et al., 2022) Corticosteroids suppress nodal interstitial edema and cytokine-mediated conduction impairment, facilitating rapid recovery of AV node conduction (Guilherme et al.(Guilherme et al., 2017; Hartiono et al., 2025; Kibar et al., 2015)

Indications for Cardiac Pacing: Because rheumatic AV block is fundamentally a transient inflammatory process, permanent pacemaker implantation is rarely required (Hartiono et al.(Hartiono et al., 2025; Kibar et al., 2015; Praneeth et al., 2020; Umapathy & Saxena, 2018)). Temporary transvenous cardiac pacing should be reserved strictly for patients presenting with symptomatic advanced AV block, syncope (Stokes-Adams attacks), or severe bradycardia with hemodynamic collapse (Ardani & Aditya, 2024; Praneeth et al.(Ardani & Aditya, 2024; Praneeth et al., 2020; Suwansumrit & Jittham, 2022) In published series, temporary pacing support is typically required for 2 to 7 days, after which intrinsic nodal conduction recovers under anti-inflammatory coverage (Praneeth et al.(Praneeth et al., 2020; Suwansumrit & Jittham, 2022; Umapathy & Saxena, 2018)

Secondary Antibiotic Prophylaxis: Preventing recurrent Group A Streptococcal pharyngitis is mandatory to halt cumulative valvular degradation and chronic RHD progression (Arafuri et al.(Arafuri et al., 2022; Gerber et al., 2009; Gewitz et al., 2015; Kotit et al., 2021)22;

Gerber et al., 2009; Gewitz et al., 2015; Kotit et al., 2021). Intramuscular Benzathine Penicillin G (BPG) administered every 3 to 4 weeks (1.2 million units for individuals >27 kg) is the gold-standard regimen (Gerber et al.(Gerber et al., 2009; Gewitz et al., 2015)09; Gewitz et al., 2015). According to AHA guidelines, the duration of secondary prophylaxis is dictated by carditis severity:

1. ARF without carditis: 5 years or until age 21 (whichever is longer) (Gerber et al.(Gerber et al., 2009; Gewitz et al., 2015)
2. ARF with carditis but no residual valvular disease: 10 years or until age 21 (whichever is longer) (Gerber et al.(Gerber et al., 2009; Gewitz et al., 2015)
3. ARF with carditis and persistent valvular disease: 10 years or until age 40 (whichever is longer, or lifelong) (Gerber et al.(Gerber et al., 2009; Gewitz et al., 2015)

CONCLUSION

In conclusion, acute rheumatic fever (ARF) presenting with atrioventricular (AV) conduction abnormalities in young adults poses significant diagnostic challenges and requires heightened clinical awareness. A baseline 12-lead electrocardiogram demonstrating PR interval prolongation or higher-degree AV block may provide an important diagnostic clue to underlying rheumatic inflammation, although conduction abnormalities alone do not establish the presence of rheumatic carditis. Early diagnostic assessment using the 2015 revised Jones criteria, supported by Doppler echocardiography and serological evidence of preceding group A streptococcal infection, facilitates timely diagnosis and appropriate clinical management.

Atrioventricular conduction abnormalities associated with ARF are often transient and reversible as the underlying inflammatory process resolves. Anti-inflammatory treatment, including salicylates or corticosteroids when clinically indicated, may alleviate inflammatory manifestations, although its direct effect on AV conduction recovery remains uncertain. Most patients with transient conduction abnormalities do not require permanent pacemaker implantation; however, persistent or high-grade AV block warrants close cardiac monitoring and individualized pacing assessment. Appropriate antibiotic treatment to eradicate residual group A streptococcal infection, followed by regular intramuscular benzathine penicillin G for secondary prophylaxis according to established guidelines, is essential for preventing recurrent rheumatic fever and reducing the risk of progressive rheumatic valvular disease. These measures emphasize the importance of early recognition, comprehensive cardiac evaluation, and sustained secondary prevention in improving long-term cardiovascular outcomes.

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CONFLICT OF INTEREST

The author states that there is no conflict of interest in the preparation of this article.

AUTHOR CONTRIBUTIONS

All authors contributed to conceptualization, search and synthesis of literature, interpretation of results, manuscript preparation, and critical review of manuscripts. All authors have read and approved the final version of the manuscript.

GENERATIVE AI STATEMENT

The authors declare that Generative Artificial Intelligence (Generative AI) was used solely to assist with language refinement, grammatical correction, and improvement of manuscript readability. All scientific concepts, interpretations, literature synthesis, analyses, and conclusions were developed by the authors. All AI-assisted content was carefully reviewed, verified, and revised by the authors, who take full responsibility for the accuracy, integrity, and final content of the manuscript.

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