KESANS: International Journal of Health and Science
2808-7178 / 2808-7380
http://kesans.rifainstitute.com/index.php/kesans/index

Symptomatic Bradycardia Due to Progressive Atrioventricular Block in a Young Woman Without Structural Heart Disease: A Case Report

[1]I Gede Putu Ryan Adipathyama Perdana*, [2]Febriyanti Angghita Putri Duarsa, [3]I Wayan Prasetya Wisuda
[1]Internship Doctor, Negara General Hospital, Bali, Indonesia*; email: ryanadipathy@gmail.com
[2]Internship Doctor, Negara General Hospital, Bali, Indonesia; email: febriduarsa@gmail.com
[3]Cardiologist, Negara General Hospital, Bali, Indonesia; email: prasetyawisuda2006@gmail.com
*Correspondence

Article Information
Submitted: **20 May 2026**
Accepted: **09 June 2026**
Publish: **15 June 2026**

Keyword: Symptomatic Bradycardia; Atrioventricular Block; Complete Heart Block; Syncope; Pacing;

Abstract
Introduction: *Symptomatic bradycardia in young adults without structural heart disease is uncommon and may delay diagnosis because of its nonspecific presentation.* ***Case Report:*** *A 19-year-old woman presented with generalized weakness, dyspnea, chest discomfort, and recurrent syncope since adolescence. Initial electrocardiography showed second-degree atrioventricular block Mobitz type I with a ventricular rate of 36 beats per minute. Laboratory examination revealed mild hypokalemia and elevated anti-streptolysin O titer, while troponin T was normal. Transthoracic echocardiography showed normal cardiac structure and preserved left ventricular systolic function. Intravenous atropine produced minimal response, and dopamine infusion resulted only in modest heart rate improvement. Serial electrocardiography during hospitalization showed progression to complete atrioventricular block. The patient was referred to a tertiary cardiac center for further electrophysiological evaluation and pacing consideration.* ***Discussion:*** *This case shows that Mobitz type I atrioventricular block in young patients may not always represent a benign condition, particularly when accompanied by recurrent syncope, poor response to atropine, and progressive conduction abnormalities.* ***Conclusion:*** *Progressive atrioventricular block should be considered in young patients with symptomatic bradycardia despite the absence of structural heart disease. Early recognition, close monitoring, and timely pacing consideration are essential to prevent serious complications.*

How to Cite **I Gede Putu Ryan Adipathyama Perdana, Febriyanti Angghita Putri Duarsa, I Wayan Prasetya Wisuda/Symptomatic Bradycardia Due to Progressive Atrioventricular Block in a Young Woman Without Structural Heart Disease: A Case Report/Vol. 5, No. 9, 2026**
DOI https://doi.org/10.54543/kesans.v5i9.647
e-ISSN/p-ISSN **2808-7178 / 2808-7380**
Published by **CV. Rifainstitut/KESANS: International Journal of Health and Science**

Introduction

Symptomatic bradycardia is a clinically significant arrhythmia characterized by a reduced heart rate accompanied by symptoms such as syncope, presyncope, generalized weakness, dyspnea, chest discomfort, and, in severe cases, hemodynamic instability. The clinical manifestations arise when cardiac output becomes insufficient to maintain adequate tissue perfusion. Although sinus bradycardia may be physiological in certain individuals, symptomatic bradycardia requires prompt evaluation to identify its underlying cause and determine the need for urgent intervention. This condition is most commonly encountered in older adults as a result of age-related degeneration of the cardiac conduction system, structural heart disease, ischemia, and medication effects. In contrast, symptomatic bradycardia in young adults without structural cardiac abnormalities is relatively uncommon. Because of its rarity and often nonspecific presentation, diagnosis may be delayed, potentially leading to serious clinical consequences (Kawji, 2022).

Atrioventricular (AV) block is one of the most important causes of symptomatic bradycardia and reflects impaired conduction of electrical impulses from the atria to the ventricles. AV block is classified into first-degree AV block, second-degree AV block (Mobitz type I and Mobitz type II), and third-degree or complete AV block. The clinical significance of AV block varies depending on the site and severity of conduction impairment. In younger patients, the etiologies of AV block are more heterogeneous than in older populations. Potential causes include congenital conduction abnormalities, inflammatory and infectious diseases, autoimmune disorders, infiltrative cardiomyopathies, and idiopathic progressive cardiac conduction disease. Therefore, identifying the underlying etiology is important for risk stratification and therapeutic decision-making (Mkoko *et al*., 2023); (Dyssekilde *et al*., 2022).

Second-degree AV block Mobitz type I, also known as the Wenckebach phenomenon, is traditionally considered a benign rhythm disturbance, particularly in healthy young individuals with increased vagal tone. In many cases, this pattern is transient and asymptomatic, requiring no specific treatment. However, the presence of symptoms such as recurrent syncope, exercise intolerance, or persistent fatigue should raise concern for clinically significant conduction disease. Several studies have showed that Mobitz type I AV block is not always benign and may progress to high-grade AV block or complete heart block in selected patients. Poor response to atropine and progressive abnormalities on serial electrocardiography are additional warning signs of intrinsic conduction system disease. Failure to recognize these high-risk features may result in severe hemodynamic compromise and, in rare cases, sudden cardiac death (Bun *et al*., 2022).

The diagnostic approach to AV block in young adults should be comprehensive and aimed at excluding reversible causes and structural heart disease. Initial evaluation includes serial electrocardiography, continuous cardiac monitoring, serum electrolyte assessment, cardiac biomarkers, and review of medication or toxin exposure. Transthoracic echocardiography is essential to assess cardiac structure and function and to identify congenital or acquired abnormalities. Additional investigations, such as inflammatory markers, autoimmune testing, infectious serologies, Holter monitoring, cardiac magnetic resonance imaging, and genetic studies, may be considered when the etiology remains unclear. Cardiac magnetic resonance imaging is particularly useful when infiltrative cardiomyopathies, myocarditis, sarcoidosis, or other inflammatory

myocardial diseases are suspected, especially in patients with progressive conduction abnormalities despite normal echocardiographic findings. This systematic evaluation helps determine prognosis and guides decisions regarding temporary or permanent pacing. Current international guidelines emphasize that symptomatic complete AV block is a strong indication for pacemaker therapy regardless of patient age (Glikson *et al*., 2022); (Moukthika *et al*., 2022).

This report describes a 19-year-old woman with symptomatic bradycardia caused by progressive atrioventricular conduction disturbance in the absence of structural heart disease. The case is notable for a long-standing history of recurrent syncope since adolescence, minimal response to atropine, and progression from second-degree AV block Mobitz type I to complete AV block during hospitalization. Transthoracic echocardiography showed normal cardiac structure and preserved ventricular function, while anti-streptolysin O titer was markedly elevated without clinical evidence of rheumatic carditis. These findings highlight the diagnostic challenges posed by conduction abnormalities in young patients with otherwise normal hearts. This case underscores the importance of early recognition, close monitoring, and timely consideration of pacing therapy in young adults with symptomatic AV block and high-risk clinical features. Such reports contribute valuable clinical insights into uncommon presentations of potentially life-threatening conduction disorders (Glikson *et al*., 2022); (Moukthika *et al*., 2022).

Case Report

A 19-year-old woman presented to the Emergency Department of Negara General Hospital, Bali, Indonesia, on October 21, 2025, with a chief complaint of generalized weakness that began approximately one hour prior to admission. The weakness was accompanied by shortness of breath and a sensation of chest discomfort. She described the symptoms as persistent and progressively worsening with physical activity, with no improvement at rest. Since the previous day, she had noted increasing fatigue even during light daily activities. There was no history of fever, palpitations, orthopnea, or lower extremity edema. She was referred from Pakutatan I Primary Health Center after marked bradycardia was identified during initial evaluation.

The patient reported a history of recurrent syncopal episodes since high school, particularly during exertion, but had never undergone formal cardiac assessment. She denied any history of seizures, head trauma, or prolonged loss of consciousness. There was no history of hypertension, diabetes mellitus, congenital heart disease, autoimmune disorders, or other chronic illnesses. She was not taking any regular medications and denied the use of herbal supplements or over-the-counter drugs. Her past surgical history was unremarkable. Family history was negative for arrhythmias, structural heart disease, and sudden cardiac death.

The patient was a college student with light to moderate physical activity. She denied smoking, alcohol consumption, and illicit drug use. On arrival, she appeared weak but was fully alert with a Glasgow Coma Scale score of E4V5M6. Her blood pressure was 120/80 mmHg, heart rate was 43 beats per minute with a regular rhythm, respiratory rate was 20 breaths per minute, body temperature was 36°C, and oxygen saturation was 99% while receiving oxygen via nasal cannula at 2 L/min. Examination of the head, neck, abdomen, and extremities was unremarkable. Pulmonary examination revealed clear

breath sounds bilaterally, and cardiac auscultation showed regular S1 and S2 heart sounds without murmurs.

Initial electrocardiography showed a regular atrial rhythm with a ventricular rate of 36 beats per minute. Progressive prolongation of the PR interval followed by non-conducted P waves was observed, consistent with second-degree atrioventricular block Mobitz type I (Wenckebach phenomenon). The QRS complexes were narrow, with no evidence of bundle branch block or ventricular hypertrophy. Mild left axis deviation was noted. The ST segments were isoelectric, and the corrected QT interval was within normal limits.

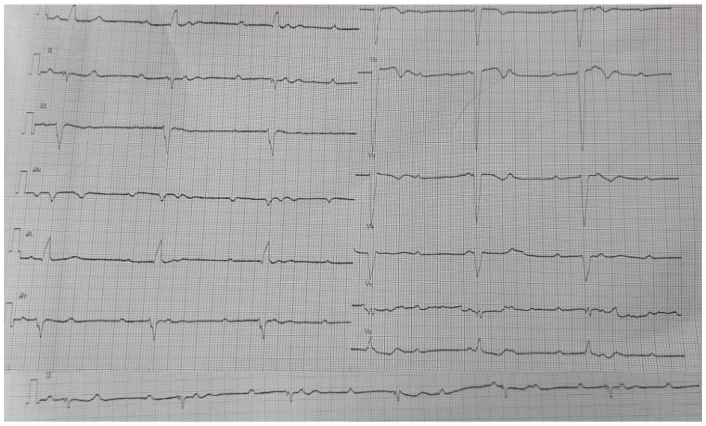

Figure 1. Initial Electrocardiogram Second-Degree Atrioventricular Block Mobitz Type I With Progressive PR Interval Prolongation and a Dropped Beat

Laboratory evaluation showed mild leukocytosis with a white blood cell count of 11.46 × 10^3/μL. Hemoglobin, platelet count, random blood glucose, renal function, and cardiac troponin T were all within normal limits. Serum electrolyte analysis revealed mild hypokalemia with a potassium level of 3.42 mmol/L. Anti-streptolysin O (ASO) titer was markedly elevated at 1600 IU/mL. No additional laboratory findings suggested acute infection, myocarditis, or systemic inflammatory disease.

Transthoracic echocardiography showed normal chamber dimensions and preserved biventricular function. Left ventricular ejection fraction was estimated at 70%. No valvular abnormalities, wall motion abnormalities, intracardiac shunts, or pericardial effusion were identified. There was also no echocardiographic evidence of rheumatic carditis or other structural heart disease.

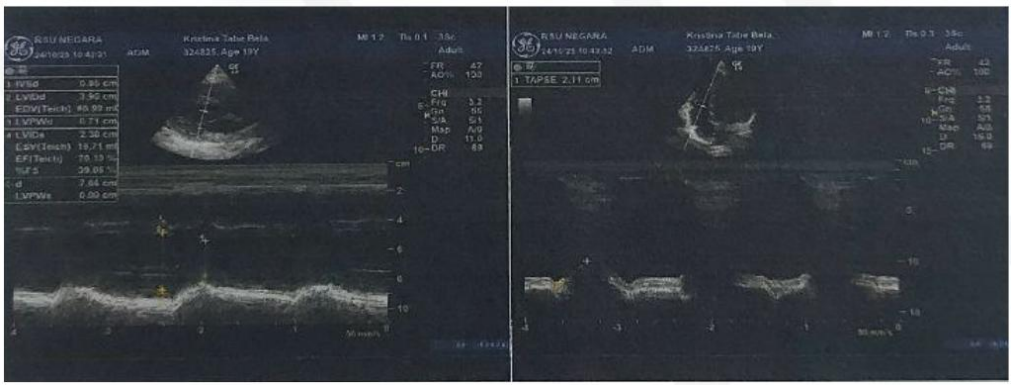

Figure 2. Transthoracic Echocardiography Normal Cardiac Structure and Preserved Systolic Function without Structural Abnormalities

Based on the initial findings, the patient was diagnosed with symptomatic bradycardia secondary to atrioventricular conduction disturbance, with a preliminary diagnosis of second-degree atrioventricular block Mobitz type I. Emergency management included continuous cardiac monitoring, intravenous administration of 0.9% normal saline, and incremental intravenous atropine up to a total dose of 3 mg. However, atropine produced only minimal chronotropic response, with heart rate remaining approximately 42 beats per minute. Because of the inadequate response, dopamine infusion was initiated at 5 µg/kg/min and gradually titrated to 12 µg/kg/min. This intervention resulted in only modest heart rate improvement to approximately 50-55 beats per minute.

During hospitalization, the patient continued to experience persistent weakness. Blood pressure ranged from 90/60 to 110/70 mmHg, while heart rate fluctuated between 34 and 55 beats per minute. Serial electrocardiographic monitoring revealed progression of the conduction abnormality to complete atrioventricular block with a ventricular escape rhythm and clear atrioventricular dissociation.

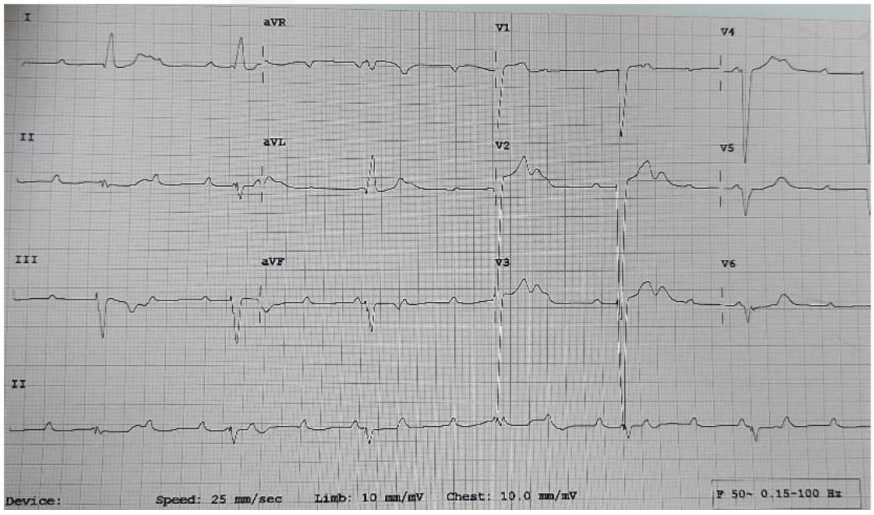

Figure 3. Serial Electrocardiogram Progression to Complete Atrioventricular Block with Atrioventricular Dissociation

Based on the clinical course and serial electrocardiographic findings, the final diagnosis was symptomatic bradycardia due to progressive atrioventricular block in the absence of structural heart disease. Differential diagnoses considered included congenital conduction disease, idiopathic progressive cardiac conduction disease, sick sinus syndrome, and post-streptococcal inflammatory involvement of the conduction system. Acute rheumatic fever was considered unlikely because the patient did not meet the major Jones criteria and had no structural evidence of carditis. Troponin T remained normal, making acute myocarditis less likely.

The patient was maintained on dopamine infusion and underwent continuous hemodynamic and rhythm monitoring. Given the persistence of symptomatic bradycardia and progression to complete atrioventricular block, she was referred to Sanglah General Hospital (Prof. Dr. I.G.N.G. Ngoerah Hospital), Denpasar, Bali, for advanced electrophysiological evaluation and consideration of temporary pacemaker implantation. Further assessment, including long-term rhythm monitoring and definitive pacing strategy, was planned at the tertiary cardiac center.

1. Discussion

Symptomatic bradycardia in young adults without structural heart disease is an uncommon clinical condition and may present a significant diagnostic challenge. Most cases of bradyarrhythmia in this age group are associated with increased vagal tone, physiological adaptation in athletes, or congenital conduction abnormalities rather than acquired degenerative disease (Kawji, 2022; Mkoko *et al*., 2023). In contrast to older patients, in whom fibrosis and degeneration of the conduction system are the predominant mechanisms, younger individuals often require broader etiological evaluation. The present case is notable because the patient was a previously healthy 19-year-old woman with no evidence of structural cardiac abnormalities on transthoracic echocardiography. Her symptoms of generalized weakness, dyspnea, chest discomfort, and recurrent syncope since adolescence suggested the presence of longstanding conduction system dysfunction. This clinical history shows that significant bradyarrhythmias may remain undiagnosed for years when symptoms are intermittent or attributed to non-cardiac causes.

The clinical manifestations of bradycardia depend largely on the degree of heart rate reduction, duration of the arrhythmia, and the patient's ability to maintain adequate cardiac output. Reduced cerebral perfusion caused by prolonged bradycardia is the most likely mechanism underlying recurrent syncope in this patient. Symptoms such as fatigue, exercise intolerance, dizziness, and presyncope are frequently nonspecific and may lead to delayed diagnosis, particularly in young adults who are otherwise healthy (Kusumoto *et al*., 2019). In this case, the recurrent syncopal episodes beginning during high school strongly suggest that clinically significant conduction abnormalities were present long before the current hospitalization. The persistence of symptoms despite preserved blood pressure shows the limited compensatory capacity of the cardiovascular system when severe bradycardia is sustained. Therefore, a detailed history of exertional syncope should prompt early electrocardiographic evaluation even in young patients without known heart disease.

The initial electrocardiogram showed second-degree atrioventricular block Mobitz type I, also known as the Wenckebach phenomenon. This rhythm disturbance is traditionally considered benign because it usually reflects reversible conduction delay within the atrioventricular node and is often seen in individuals with heightened vagal tone (Bun *et al*., 2022). However, the prognosis of Mobitz type I depends heavily on the clinical context. Several studies have shown that symptomatic patients with Mobitz type I may progress to high-grade atrioventricular block or complete heart block, especially when symptoms are recurrent or conduction abnormalities persist on serial recordings (Bun *et al*., 2022; Pachón-Mateos *et al*., 2022). In the present case, progression from Mobitz type I to complete atrioventricular block during hospitalization confirms that the initial electrocardiographic pattern did not represent a benign physiological finding. This observation emphasizes that Mobitz type I should not be automatically considered harmless when accompanied by concerning clinical features.

Several high-risk findings in this case suggested clinically significant conduction disease rather than isolated nodal Wenckebach physiology. First, the patient had recurrent syncope over several years, indicating intermittent periods of inadequate cardiac output. Second, the ventricular rate remained profoundly low despite administration of atropine up to the maximum recommended dose of 3 mg. Third, serial electrocardiograms documented progression to complete atrioventricular block with atrioventricular dissociation. Together, these findings strongly suggest intrinsic disease of the cardiac conduction system. The presence of these red flags is particularly important in younger patients, in whom clinicians may otherwise underestimate the severity of conduction abnormalities. Early recognition of these warning signs should prompt consideration of temporary pacing and referral to a tertiary cardiac center.

The poor response to atropine provides additional insight into the pathophysiology of this patient's conduction disturbance. Atropine exerts its effect by inhibiting parasympathetic tone, thereby increasing sinus node automaticity and improving conduction through the atrioventricular node (Tisdale *et al*., 2020). When bradycardia fails to respond adequately, more distal conduction system disease involving the His-Purkinje system should be suspected. In this patient, atropine resulted in only minimal heart rate improvement, supporting the possibility of intrinsic conduction tissue dysfunction. Dopamine infusion was subsequently administered as a chronotropic agent, but the increase in heart rate was modest and insufficient to normalize conduction. The limited response to both atropine and dopamine further reinforces the severity of the underlying conduction abnormality.

Current international guidelines from the European Society of Cardiology (ESC) and the American College of Cardiology/American Heart Association recommend pacing therapy for patients with symptomatic complete atrioventricular block regardless of age (Kusumoto *et al*., 2019; Glikson *et al*., 2021). Temporary pacing is indicated when bradycardia causes symptoms or hemodynamic compromise and does not respond adequately to pharmacological therapy. In this case, the patient remained symptomatic despite maximal atropine administration and dopamine infusion, and serial electrocardiography confirmed progression to complete heart block. These findings met clear criteria for advanced rhythm management. Referral to a tertiary cardiac center for temporary pacemaker consideration was therefore clinically appropriate. This management strategy was aimed at preventing recurrent syncope, worsening hemodynamic instability, and sudden cardiac death.

An important aspect of this case is the absence of structural heart disease on transthoracic echocardiography. Cardiac chamber dimensions were normal, left ventricular systolic function was preserved, and no valvular abnormalities or evidence of rheumatic carditis were identified. In such situations, the differential diagnosis shifts toward congenital atrioventricular block, idiopathic progressive cardiac conduction disease, inflammatory disorders, and inherited channelopathies. Congenital conduction disease may remain asymptomatic for years and become clinically apparent only when compensatory mechanisms fail (Yavuz *et al*., 2002). Similarly, genetic abnormalities affecting ion channels or conduction tissue proteins can present with progressive atrioventricular block in otherwise healthy young individuals. Because advanced diagnostic tools such as Holter monitoring, electrophysiological studies, and genetic testing were not available, the precise etiology in this case remains uncertain.

The markedly elevated anti-streptolysin O titer raised the possibility of a post-streptococcal inflammatory process affecting the conduction system. Elevated ASO indicates previous exposure to Group A beta-hemolytic streptococci but is not diagnostic of acute rheumatic fever in the absence of compatible clinical features. Although transient conduction abnormalities have been described in association with rheumatic carditis and post-infectious inflammation, this patient did not meet the Jones criteria for acute rheumatic fever (Varkutty *et al*., 2020). There was no evidence of carditis, migratory polyarthritis, chorea, erythema marginatum, or subcutaneous nodules. Furthermore, echocardiography showed no valvular involvement. Therefore, the elevated ASO titer was considered a nonspecific finding rather than a definitive explanation for the progressive atrioventricular block.

Other potential reversible causes were considered but were less likely to account for the severity of the conduction disturbance. Troponin T remained within normal limits, making acute myocarditis or myocardial ischemia less probable. Mild hypokalemia was present, but a potassium level of 3.42 mmol/L would not typically cause progressive conduction disease culminating in complete atrioventricular block. The patient denied exposure to medications known to impair atrioventricular conduction, such as beta-blockers, calcium channel blockers, or digoxin. There was also no history suggestive of toxin exposure or substance abuse. Consequently, intrinsic conduction system disease remains the most plausible diagnosis. This conclusion is supported by the chronic symptom history and progressive electrocardiographic changes.

This case underscores the importance of a comprehensive and cautious approach to symptomatic bradycardia in young adults. Although Mobitz type I atrioventricular block is often considered benign, clinicians should maintain a high index of suspicion when it is accompanied by recurrent syncope, inadequate response to atropine, and progression on serial electrocardiography (Bun *et al*., 2022; Pachón-Mateos *et al*., 2022). Normal echocardiographic findings do not exclude clinically significant conduction disease. Close monitoring and early pacing consideration are essential to prevent adverse outcomes, including severe hemodynamic compromise and sudden cardiac death (Kusumoto *et al*., 2019; Glikson *et al*., 2021). This case also shows the need for further etiological evaluation, including electrophysiological and genetic studies, when available. Recognition of these high-risk features is important to ensuring timely and appropriate management in young patients with symptomatic atrioventricular block.

Conclusion

Symptomatic bradycardia due to progressive atrioventricular block in young adults without structural heart disease is an uncommon but clinically significant condition with the potential to progress to complete atrioventricular block. Recurrent syncope, poor response to atropine, and worsening conduction abnormalities on serial electrocardiography are important warning signs that show underlying conduction system disease and warrant close monitoring. In such cases, a comprehensive evaluation should be performed to exclude reversible and structural causes, even when echocardiographic findings are normal. Early referral to a tertiary cardiac center and timely consideration of pacing therapy are essential to prevent hemodynamic deterioration and sudden cardiac death. This case shows the importance of maintaining a high index of suspicion for serious conduction disorders in young patients presenting with symptomatic bradycardia despite the absence of overt structural cardiac abnormalities.

Reference

Bun, S. S., Asarisi, F., Heme, N., Squara, F., Scarlatti, D., Taghji, P., et al. (2022). Prevalence and clinical characteristics of patients with pause-dependent atrioventricular block. *Journal of Clinical Medicine, 11*(2), 449.

Dyssekilde, J. R., Frederiksen, T. C., Christiansen, M. K., Sørensen, R. H., Pedersen, L. N., Møller, P. L., et al. (2022). Diagnostic yield of genetic testing in young patients with atrioventricular block of unknown cause. *Journal of the American Heart Association, 11*(9), e025643.

Glikson, M., Nielsen, J. C., Kronborg, M. B., Michowitz, Y., Auricchio, A., Barbash, I. M., et al. (2022). 2021 ESC guidelines on cardiac pacing and cardiac resynchronization therapy. *Europace, 24*(1), 71-164.

Glikson, M., Nielsen, J. C., Kronborg, M. B., Michowitz, Y., Auricchio, A., Barbash, I. M., et al. (2021). ESC guidelines on cardiac pacing and cardiac resynchronization therapy. *European Heart Journal, 42*(35), 3427-3520.

Kawji, M. M. (2022). Symptomatic bradycardia. *JAMA Internal Medicine, 182*(10), 1107.

Kusumoto, F. M., Schoenfeld, M. H., Barrett, C., Edgerton, J. R., Ellenbogen, K. A., Gold, M. R., et al. (2019). 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. *Circulation, 140*, e382-e482.

Kusumoto, F. M., Schoenfeld, M. H., Barrett, C., Edgerton, J. R., Ellenbogen, K. A., Gold, M. R., et al. (2019). ACC/AHA/HRS guideline on bradycardia and cardiac conduction delay. *Circulation, 140*, e382-e482.

Mkoko, P., Rajoo, S. T., & Chin, A. (2023). Causes of heart block in young and middle-aged South Africans. *Current Problems in Cardiology, 48*(3), 101247.

Moukthika, K. V. N., Krishnappa, D., & Chandra, S. P. S. (2022). Inflammation begets isolated atrioventricular conduction disturbances in young: An observation from Indian registry data. *European Heart Journal, 43*(Suppl. 2), ehac544.499.

Pachón-Mateos, J. C., Ortencio, F. A., Pachon-M, J. C., Lobo, T. J., et al. (2022). Treatment of symptomatic functional atrioventricular block by cardioneuroablation as an alternative to pacemaker implantation. *JACC: Case Reports, 4*(15), 990-995.

Tisdale, J. E., Chung, M. K., Campbell, K. B., Hammadah, M., Joglar, J. A., Leclerc, J., et al. (2020). Drug-induced arrhythmias: A scientific statement from the American Heart Association. *Circulation, 142*(15), E214-E233.

Varkutty, B., Eybek, B., Erolu, E., & Karacan, M. (2020). A rare case of acute rheumatic fever with three different types of atrioventricular blocks in the same patient. *North Clinical Istanbul, 9*(3), 279-281.

Yavuz, T., Nazli, C., Kinay, O., & Kucukoglu, S. (2002). Congenital complete atrioventricular block in adulthood. *International Journal of Cardiology, 82*(3), 291-295.