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A CASE REPORT OF A LIFE-THREATENING CONDITION: COMPLETE HEART BLOCK INDUCED BY SYSTEMIC LUPUS ERYTHEMATOSUS

Yogi Puji Rachmawan[1]*, Arsha Pramudya[2], Witri Pratiwi[3], Irnizarifka[4], Fawzia Haznah Nurul Imani[5]
Universitas Swadaya Gunung Jati, Cirebon, Indonesia[1,3]
Hasna Medika Cardiovascular Hospital, Cirebon, Indonesia[2]
Universitas Sebelas Maret, Surakarta, Indonesia[4]
Universitas Diponegoro, Semarang, Indonesia[5]
Email: yogikage@gmail.com[1]*, we3.borneo@gmail.com[2], irnizarifka@gmail.com[3], arshapram@gmail.com[4], faw.fhni@gmail.com[5]

Abstract
Systemic lupus erythematosus (SLE) is often accompanied by complications in the cardiovascular system such as pericarditis, valvular disease, and coronary artery disease, but complications in the conduction pathway are a rare condition. Conduction system disorders such as complete heart block (CHB) can cause life-threatening conditions and require immediate treatment. This case report describes a 43-year-old woman who presented with recurrent syncope over one week, accompanied by palpitations and lightheadedness. Initial evaluation revealed marked bradycardia (heart rate 40 bpm), a malar rash, and ECG findings of complete heart block with atrioventricular dissociation. Echocardiography showed preserved left ventricular function (LVEF 68%), and laboratory evaluation revealed positive antinuclear antibody (ANA) with speckled pattern. The patient received high-dose corticosteroid therapy (methylprednisolone 62.5 mg daily) but the complete heart block did not improve, so a permanent pacemaker was implanted. Post-implantation, the patient showed rapid clinical improvement with no further syncopal episodes and normalization of hemodynamic parameters (blood pressure 135/70 mmHg, heart rate 60 bpm). Outpatient follow-up ECG revealed reduced pacing dependency, suggesting partial recovery of intrinsic conduction. This case demonstrates that SLE complications in the cardiovascular system, including the cardiac conduction system, must be watched out for because they are life-threatening conditions. The case also highlights that autoimmune screening should be considered in younger patients presenting with unexplained complete heart block, particularly in the absence of structural heart disease, and that immunosuppressive therapy may partially reverse conduction system injury when initiated promptly.
Keywords: Complete heart block, Conduction Block, Myocarditis, Pacemaker, Systemic lupus erythematous, SLE

INTRODUCTION

Complete heart block (CHB) represents a rare but potentially life-threatening cardiac manifestation in adults with systemic lupus erythematosus (SLE), a chronic autoimmune disease characterized by immune dysregulation, autoantibody production, and widespread inflammatory tissue injury. SLE predominantly affects women of reproductive age and is associated with substantial morbidity and premature mortality due to cumulative organ damage. Among the various organ systems involved, cardiovascular complications are particularly important contributors to long-term prognosis and survival. Over the past decades, advances in immunosuppressive therapy have improved overall disease control; however, cardiac involvement continues to pose significant diagnostic and therapeutic challenges (Buch et al., 2024; Fan et al., 2025; Opałka et al., 2023; Tana et al., 2025).

The heart may be affected in SLE through multiple mechanisms, including immune-mediated inflammation, vascular injury, and accelerated atherosclerosis. The most frequently reported cardiac manifestations include pericarditis, which is considered the most common, followed by valvular abnormalities such as Libman–Sacks endocarditis, myocarditis, and premature coronary artery disease. These conditions may occur independently or in combination, reflecting the systemic nature of lupus-related inflammation. Despite increasing awareness of myocardial and valvular involvement, abnormalities of the cardiac conduction

system remain relatively uncommon and are therefore often overlooked in routine clinical assessment (Natsheh et al., 2019).

Conduction system involvement in SLE may manifest as sinus node dysfunction, first-degree atrioventricular (AV) block, bundle branch block, or more severe disturbances such as complete heart block. Among these, CHB represents the most clinically significant form due to its association with profound bradycardia, reduced cardiac output, syncope, and risk of sudden cardiac death. Although CHB is well recognized in neonatal lupus associated with maternal autoantibodies, its occurrence in adults with established SLE is exceedingly rare. Consequently, the true incidence remains unknown, and most knowledge is derived from isolated case reports and small observational series.

Despite its rarity, conduction system disease in SLE carries considerable clinical implications. Patients may present with nonspecific symptoms such as fatigue, dizziness, palpitations, or exertional intolerance, which can easily be attributed to anemia, medication effects, or systemic disease activity. In more severe cases, abrupt onset of syncope, hemodynamic instability, or ventricular arrhythmias may occur, requiring immediate medical intervention. Failure to promptly recognize CHB can result in catastrophic outcomes, underscoring the importance of maintaining a high index of suspicion in lupus patients presenting with unexplained bradyarrhythmia's or transient loss of consciousness (Du Toit et al., 2023).

The novelty of this research lies in four key aspects. First, this case report documents SLE-related complete heart block in a 43-year-old woman from a region (Bululawang, Malang Regency, Indonesia) with limited prior documentation of this complication, contributing to the global literature on the geographical distribution of SLE cardiac manifestations. Second, the case demonstrates that a presumptive diagnosis of SLE-related CHB can be made using basic diagnostic tools (ECG, echocardiography, ANA testing) when advanced modalities are unavailable, providing a practical diagnostic framework for resource-limited settings.

Third, the case shows that immunosuppressive therapy (high-dose corticosteroids) initiated promptly after pacemaker implantation resulted in reduced pacing dependency on follow-up, suggesting partial reversibility of conduction system injury and supporting the role of immunosuppression even in chronic-appearing CHB. Fourth, the case highlights the importance of multidisciplinary collaboration between cardiology and rheumatology in managing SLE-related cardiac complications, with implications for clinical practice guidelines in similar healthcare settings. This novelty addresses the research gap identified from the works of Natsheh et al. (2019), Mishra & Chatterjee (2023), and Du Toit et al. (2023).

Diagnosis of conduction abnormalities in SLE is further complicated by the relatively young age of most affected patients, in whom degenerative conduction disease and ischemic heart disease are unlikely. As a result, autoimmune-mediated mechanisms may not be initially considered, particularly in individuals without a prior diagnosis of SLE or without prominent systemic manifestations. This diagnostic delay may postpone appropriate immunosuppressive therapy and timely pacing intervention, potentially worsening clinical outcomes.

The underlying pathophysiological mechanisms responsible for conduction disturbances in SLE are complex and multifactorial. Immune-mediated myocardial inflammation, small-vessel vasculitis, immune complex deposition, and progressive fibrosis of conduction tissue are thought to play central roles. Inflammatory infiltrates within the myocardium may extend to specialized conduction pathways, including the sinoatrial node, atrioventricular node, and His-Purkinje system, disrupting impulse generation and propagation. These changes may occur acutely during periods of active disease or evolve gradually as a result of chronic immune injury.

Autoantibodies represent another important contributor to cardiac involvement in SLE. Various lupus-related antibodies have been implicated in myocardial damage and electrical

instability. In the context of myocarditis, inflammatory cytokines and immune cell infiltration may lead to myocyte necrosis, edema, and subsequent fibrotic remodeling, all of which can interfere with electrical conduction. When myocardial inflammation involves perinodal tissue, atrioventricular conduction may become particularly vulnerable, resulting in varying degrees of heart block (Mohanty & Sunder, 2020).

The relationship between lupus myocarditis and conduction system disease is of particular clinical relevance. Lupus myocarditis is a rare but severe manifestation of SLE that may present with heart failure, arrhythmias, or cardiogenic shock. Inflammatory injury in this setting may be reversible with aggressive immunosuppressive therapy if promptly diagnosed. However, delayed treatment can result in permanent structural damage and persistent conduction defects. Given that myocarditis may not always be accompanied by overt systolic dysfunction on echocardiography, advanced imaging modalities such as cardiac magnetic resonance imaging are often required for definitive diagnosis, though access may be limited in many healthcare settings.

While congenital heart block related to maternal anti-Ro antibodies has been extensively studied, adult-onset CHB in autoimmune disease remains poorly characterized. Available literature suggests considerable heterogeneity in clinical presentation, serological profiles, and response to therapy. Some patients demonstrate persistent conduction block requiring permanent pacemaker implantation, whereas others exhibit partial or complete recovery following immunosuppressive treatment. These variable outcomes suggest that the underlying pathology may range from reversible inflammatory injury to irreversible fibrotic destruction of conduction tissue.

The scarcity of large-scale studies and standardized management guidelines further complicates clinical decision-making. Currently, treatment strategies rely on a combination of supportive cardiac care, including temporary or permanent pacing, and immunosuppressive therapy targeting the underlying autoimmune process. However, the optimal timing, intensity, and duration of immunosuppression remain uncertain, particularly in patients without overt myocarditis or systemic disease flare.

Given these diagnostic and therapeutic challenges, individual case reports play an important role in expanding current understanding of SLE-related conduction system disease. Detailed clinical descriptions provide valuable insights into potential mechanisms, clinical trajectories, and treatment responses, thereby contributing to improved recognition and management of this rare complication.

In this report, we describe a relatively young woman who presented with recurrent syncope due to complete heart block and was subsequently diagnosed with systemic lupus erythematosus. This case illustrates an uncommon but clinically significant manifestation of SLE and highlights the importance of considering autoimmune etiologies in patients with unexplained conduction disturbances. By presenting this case alongside a review of previously reported literature, we aim to enhance awareness of SLE-associated CHB, discuss potential pathophysiological mechanisms, and emphasize the need for early diagnosis and appropriate multidisciplinary management to improve patient outcomes.

RESEARCH METHODS

CASE REPORT

A 43-year-old woman with no known history of cardiovascular disease was brought to the emergency department (ED) by her family after experiencing several episodes of syncope over the preceding week. Each syncopal episode occurred during routine daily activities, lasted approximately five minutes, and was preceded by palpitations and lightheadedness. The patient reported that similar symptoms of intermittent palpitations and near-syncope had been occurring intermittently over the past month but had progressively worsened in frequency and

severity. There was no history of chest pain, dyspnea at rest, seizure-like activity, or focal neurological deficits. The patient had never been previously diagnosed with systemic lupus erythematosus or any other autoimmune disorder and was not taking any regular medications.

Upon arrival at the ED, the patient appeared anxious with a reduced level of consciousness but was responsive to verbal stimuli. Initial vital signs revealed a blood pressure of 113/60 mmHg and marked bradycardia with a heart rate of 40 beats per minute. Oxygen saturation was within normal limits on room air. Physical examination revealed a characteristic malar rash involving both cheeks and the bridge of the nose, consistent with a butterfly-shaped lupus facial rash. No oral ulcers, joint swelling, peripheral edema, or signs of heart failure were observed. Cardiac auscultation revealed a slow but regular rhythm without murmurs, gallops, or rubs. Lung examination was clear bilaterally, and abdominal and neurological examinations were unremarkable.

An urgent 12-lead electrocardiogram demonstrated atrioventricular block with a 2:1 conduction pattern alternating with episodes of complete heart block, characterized by atrioventricular dissociation and a slow ventricular escape rhythm (Figure 1). Given the patient's symptomatic bradycardia and recurrent syncope, immediate pharmacologic therapy was initiated in accordance with advanced cardiac life support protocols. Intravenous atropine sulfate was administered at an initial dose of 1 mg and repeated every five minutes to a cumulative dose of 3 mg. Despite maximal atropine therapy, the patient's heart rate remained persistently low at approximately 42 beats per minute, and repeat ECG continued to demonstrate complete heart block.

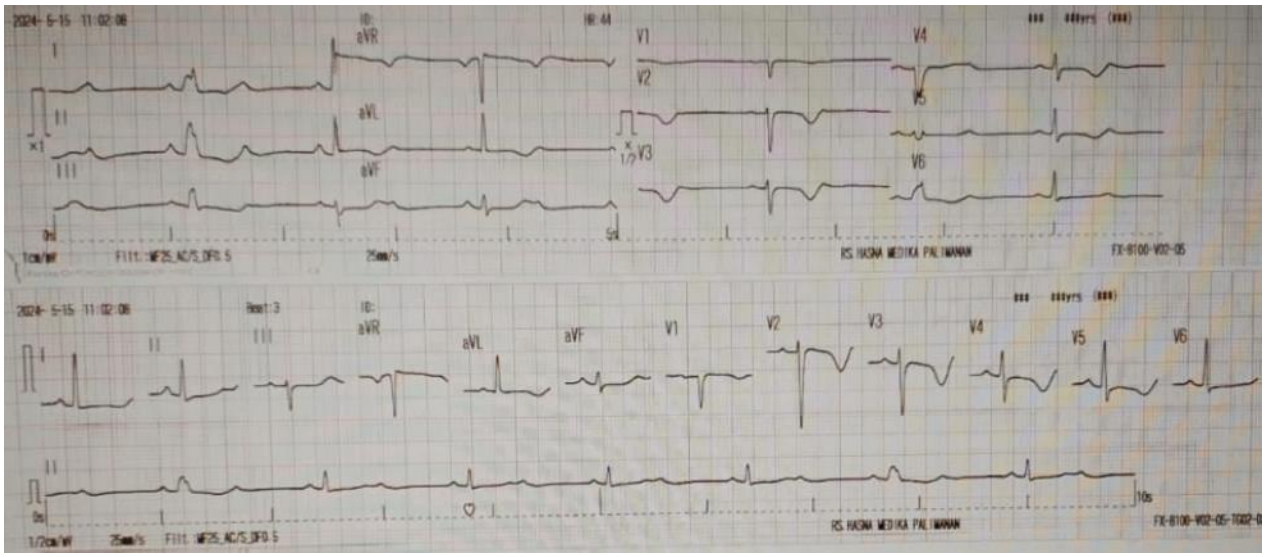

Figure 1. ECG of complete heart block at the emergency department

Due to the lack of response to atropine and ongoing hemodynamic risk, a dopamine infusion was initiated at 5 µg/kg/min to support heart rate and blood pressure. Simultaneously, preparations were made for emergent temporary pacemaker (TPM) insertion as definitive stabilization therapy. Transvenous temporary pacing was successfully performed without complications, resulting in restoration of an adequate paced ventricular rhythm and immediate symptomatic improvement.

Following hemodynamic stabilization, the patient was transferred to the cardiovascular intensive care unit for further evaluation of the underlying etiology of complete heart block. Given the absence of known structural heart disease, young age, and presence of suggestive cutaneous findings, an autoimmune cause was strongly considered.

Transthoracic echocardiography revealed preserved left ventricular systolic function with a left ventricular ejection fraction of 68% and normal right ventricular systolic performance, as indicated by a tricuspid annular plane systolic excursion of 27 mm. Cardiac chamber dimensions were within normal limits, and there were no regional wall motion abnormalities. Valvular structure and function were normal, with no evidence of regurgitation, stenosis, or vegetations. These findings suggested the absence of overt cardiomyopathy, significant valvular disease, or pericardial involvement (Table 1).

Table 1. Echocardiography results

DOPPLER		M-MODE	
MV E Vd	1.09 m/s	IVSd	0.56 cm
MV DecT	167.20 ms	LVIDd	4.79 cm
MV Dec Slope	6.51 m/s^2	LVPWd	0.96 cm
MV A Vel	1.02 m/s	IVSs	0.98 cm
MV E/A Ratio	1.07	LVIDs	2.96 cm
MV PHT	48.49 ms	LVPWs	1.44 cm
MVA by PHT	4.54 cm^2	EDV (Teich)	107.03 ml
e'Lat	0.07 m/s	ESV (Teich)	33.90 ml
e/e' Lat	15.60	EF (Teich)	68.32 %
e/e' Avg	0.54	%FS	38.19 %
		SV (Teich)	73.13 ml
		LVd Mass Index	81.31 g/m^2
		LVs Mass Index	73.98 g/m^2
		RWT	0.40
		TAPSE	2.70 cm
		RVIDd	1.95 cm

Table 2. Laboratory results

Test	Reference Range	Value	Tests	Reference Range	Value
Hb	12 – 14 g/dL	12.9 g/dL	Na	135 – 148 mEq/L	139 mEq/L
Ht	37 – 43 %	40.6 %	K	3.5 – 5.3 mEq/L	4.1 mEq/L
Leucocyte	4 – 10 /µL	11000 /µL	Ca	1.13 – 1.32 mEq/L	1.3 mEq/L
Thrombocyte	150000 – 450000 /µL	377000 /µL	RBG	70 – 140 mg/dl	120 mg/dl
Ureum	16.6 – 48.5 mg/dl	37 mg/dl	Troponin T	<50 ng/L	<50 ng/L
Creatinine	0.6 – 1.3 mg/dl	0.8 mg/dl	Hs-CRP	< 1 mg/L	0.8 mg/L*
			ANA test	Negative	Positive*

*Abnormal results

Routine laboratory evaluation demonstrated normal renal function, electrolyte levels, and hematological parameters. Cardiac biomarkers showed troponin T levels below 50 ng/L, indicating no evidence of acute myocardial injury. High-sensitivity C-reactive protein was mildly elevated at 0.8 mg/L, suggesting low-grade inflammatory activity. Autoimmune screening revealed a positive antinuclear antibody (ANA) test with a speckled pattern, supporting the presence of an underlying autoimmune disorder consistent with systemic lupus erythematosus. Unfortunately, further serological testing, including anti-Ro/SSA antibodies and other lupus-specific autoantibodies, was not available at the treating facility (Table 2).

Advanced cardiac imaging, particularly cardiac magnetic resonance imaging (CMR), which could have provided detailed assessment of myocardial inflammation and fibrosis, was not available in the hospital. Endomyocardial biopsy was also not feasible. However, in the context of suggestive clinical features, positive ANA serology, and unexplained complete heart block, an inflammatory autoimmune-mediated cardiac involvement was strongly suspected.

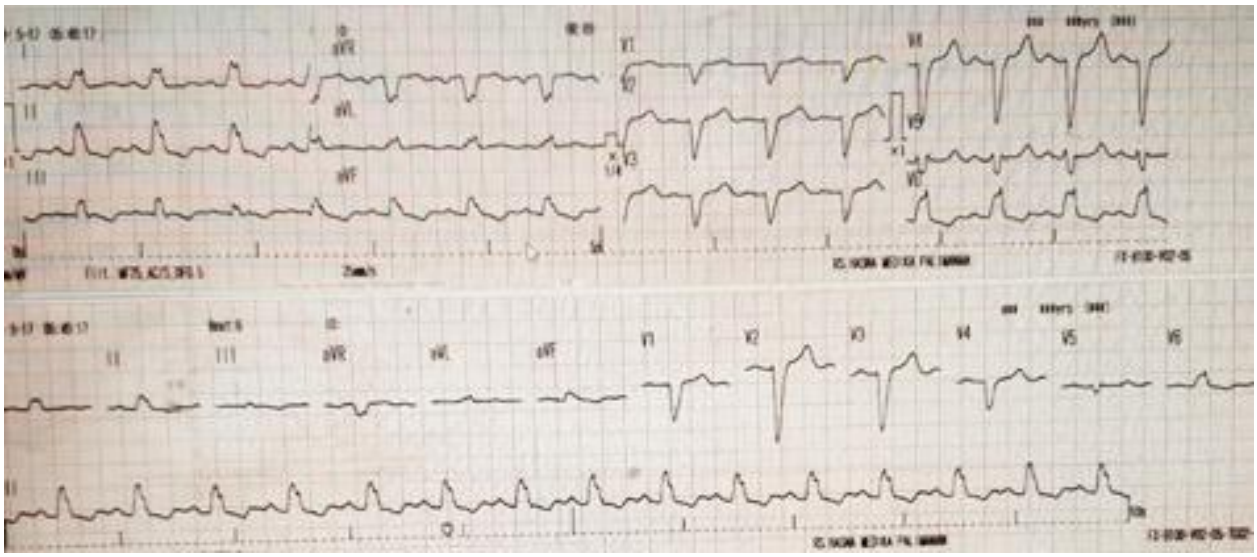

Figure 2. ECG after PPM insertion

Based on the presumptive diagnosis of SLE-related myocardial and conduction system involvement, high-dose intravenous methylprednisolone was initiated as immunosuppressive therapy. The patient received methylprednisolone at a dose of 62.5 mg daily. Despite corticosteroid treatment, intrinsic atrioventricular conduction did not recover during the initial observation period, and the patient remained dependent on pacing support.

Given the persistence of complete heart block and the high risk of recurrent syncope and sudden cardiac death, the decision was made to proceed with permanent pacemaker (PPM) implantation. The procedure was performed successfully without complications. Post-implantation ECG demonstrated appropriate pacing rhythm with stable heart rate control (Figure 2). Hemodynamic parameters normalized, with blood pressure of 135/70 mmHg and heart rate of approximately 60 beats per minute.

The patient showed rapid clinical improvement following pacing and immunosuppressive therapy. She experienced no further episodes of syncope, palpitations, or dizziness during hospitalization. After three days of observation, she was discharged in stable condition with instructions for outpatient cardiology and rheumatology follow-up. On subsequent outpatient ECG evaluation, there was evidence of reduced pacing dependency, suggesting partial recovery of intrinsic conduction, further supporting an inflammatory rather than purely degenerative etiology.

RESULTS AND DISCUSSION

Complete heart block (CHB) represents an uncommon but severe manifestation of systemic lupus erythematosus (SLE), reflecting the complex interplay between immune-mediated inflammation, myocardial injury, and disruption of cardiac conduction pathways. Although cardiovascular involvement is frequently encountered in SLE, conduction system abnormalities remain rare and are often overshadowed by more prevalent manifestations such as pericarditis, valvular disease, myocarditis, and accelerated atherosclerosis. Nevertheless, when present, conduction disturbances particularly CHB carry significant clinical consequences due to their association with syncope, hemodynamic instability, ventricular arrhythmias, and sudden cardiac death (Gómez-Barrado et al., 2002).

Epidemiology and Clinical Characteristics of SLE-Related Conduction Abnormalities

Available data regarding the prevalence of conduction system involvement in SLE are limited and largely derived from case series and retrospective analyses. Godeau et al. reported that approximately 14.5% of patients with SLE exhibited some form of conduction abnormality; however, complete atrioventricular block constituted only a small subset of these cases. This highlights the rarity of CHB within the broader spectrum of lupus-related cardiac involvement. Importantly, the majority of reported cases have occurred in women, consistent with the female predominance of SLE, and often affect relatively young individuals without traditional cardiovascular risk factors.

In the present case, a 43-year-old woman presented with recurrent syncope as the primary manifestation of CHB, ultimately leading to the diagnosis of SLE. This clinical scenario mirrors several previously reported cases in which CHB served as an early or initial manifestation of lupus, preceding or coinciding with the development of more typical systemic features. Such presentations pose a diagnostic challenge, as clinicians may not readily consider autoimmune etiologies in patients presenting with bradyarrhythmia's, particularly in the absence of established SLE or overt systemic disease activity.

Comparison With Previously Reported Cases

A review of published case reports summarized in Table 3 reveals several recurring themes that are relevant to the present case. First, most patients were young or middle-aged adults, predominantly female, and presented with syncope or presyncope as the initial symptom. Second, structural heart disease was generally absent, with normal echocardiographic findings in the majority of cases, suggesting that conduction abnormalities may occur independently of overt myocardial dysfunction. Third, autoimmune serology was frequently positive, even in patients lacking classic clinical manifestations of SLE at presentation.

Table 3. Several case reports regarding SLE related complete heart block

Reff	Year	Age, gender	Symptoms	Pre ECG	Myocard Disease	Serology	Medication	Evolution
Bharati (Bharati et al., 1975)	1975	12, Female	Syncope	Normal	Yes	LE, ANA	Prednisone	-
Maier (Maier et al., 1987)	1987	27, Female	Pre-syncope	NA	No	ANA, DNA, Sm, RNP, Ro	Indomethacin Prednisone	NA
Lo (Lo et al., 2018)	2018	27, Female (pregnant)	Dizziness, Syncope	NA	No	ANA, DNA, LOW C4	Prednisolone, hydroxychloroquine, PPM	Improve
Natsheh (Natshes, et al., 2019)	2019	19, Female	Syncope	NA	No	ANA	Naproxen, colchicine, hydroxychloroquine, Azathioprine, PPM	Improve

Shariff (Shariff et al., 2020)	2020	40, male	Giddiness, fall	NA	No	ANA, Ro, RNP	Hydrocortisone, PPM	Improve
Mishra (Mishra et al., 2023)	2023	30, Female	Shortness of breath	NA	No	ANA, Ro, Sm	PPM	Improve
Present case	2024	43, Female	Syncope	NA	No	ANA	Methylprednisolone, PPM	Improve

NA: not available; LE: lupus-erythematosus cell; ANA: antinuclear antibodies; DNA: anti dsDNA antibodies; Sm: Anti-Smith antibodies; La: Anti-La antibodies; Ro: Anti-Ro antibodies; RNP: anti-RNP (Ribonucleoprotein)

Natsheh et al. described a young woman who presented with recurrent syncope due to complete AV block as the first manifestation of SLE. Notably, systemic features such as pericarditis, arthritis, and photosensitivity developed only later in the disease course, underscoring the evolving nature of lupus and the potential for cardiac manifestations to precede systemic involvement (Natsheh et al., 2019). Similarly, Mishra et al. reported a 30-year-old woman with complete AV block as an early presentation of SLE, accompanied by positive ANA, anti-Ro, and anti-Smith antibodies, but lacking typical dermatologic or musculoskeletal features over time (Mishra & Chatterjee, 2023). These reports, together with the present case, emphasize the heterogeneity of SLE-related CHB and reinforce the importance of considering autoimmune screening in patients with unexplained conduction abnormalities, particularly when occurring at a young age or in the absence of structural heart disease.

Pathophysiological Mechanisms Underlying SLE-Related Complete Heart Block

The precise mechanisms responsible for conduction system involvement in SLE remain incompletely understood and are likely multifactorial. Proposed mechanisms include immune-mediated myocardial inflammation, immune complex deposition, small-vessel vasculitis, autoantibody-mediated electrophysiological disruption, and progressive fibrotic remodeling of conduction tissue.

Autoantibody-Mediated Injury

Autoantibodies, particularly anti-Ro/SSA antibodies, have been extensively implicated in the pathogenesis of congenital heart block associated with neonatal lupus. In fetal and neonatal hearts, these antibodies are thought to bind to surface antigens expressed on cardiomyocytes within the sinoatrial and atrioventricular nodes, triggering inflammatory cascades that lead to apoptosis, fibrosis, and irreversible conduction system damage (Wahren-Herlenius & Sonesson, 2006; Popescu et al., 2020).

In addition to inflammatory injury, anti-Ro antibodies have been shown to interfere directly with cardiac electrophysiology by targeting L-type calcium channels, particularly Cav1.2 and Cav1.3, which play a critical role in impulse conduction within the atrioventricular node. Inhibition of calcium influx disrupts action potential propagation, predisposing to conduction block (Karnabi & Boutjdir, 2010; Lazzerini et al., 2023).

Although the adult myocardium has traditionally been considered more resistant to the effects of anti-Ro antibodies compared to fetal tissue, growing evidence suggests that prolonged exposure to autoantibodies and chronic immune activation may induce similar pathological changes in adults. Lazzerini et al. proposed that anti-Ro/SSA antibodies may

contribute to idiopathic AV block in adults by exerting cumulative electrophysiological and inflammatory effects over time (Lazzerini et al., 2023). Jobling and Rajabally further supported this hypothesis by reporting adult patients with autoimmune disease and complete heart block in whom anti-Ro antibodies were present (Jobling & Rajabally, 2018).

In the present case, anti-Ro antibodies were not measured due to limited diagnostic resources. Nevertheless, the presence of a positive ANA test with a speckled pattern supports an autoimmune etiology, and the patient's clinical response suggests that immune-mediated mechanisms likely played a role.

Immune Complex Deposition and Myocardial Inflammation

Beyond direct autoantibody effects, immune complex deposition represents another important mechanism contributing to cardiac involvement in SLE. Circulating immune complexes may deposit within myocardial interstitial spaces and small intramyocardial vessels, activating complement pathways and promoting the release of pro-inflammatory cytokines. Chronic inflammation may lead to myocardial edema, cellular infiltration, and ultimately fibrotic remodeling, impairing both mechanical and electrical cardiac function (Pan et al., 2022; Miner & Kim, 2014).

Lupus myocarditis, although rare, represents a severe manifestation of SLE-related cardiac involvement and may directly affect the conduction system. Inflammatory infiltration may extend to perinodal tissue, disrupting atrioventricular conduction. Importantly, myocarditis may occur in the absence of overt systolic dysfunction, making diagnosis challenging without advanced imaging modalities such as cardiac magnetic resonance imaging (CMR).

Diagnostic Challenges in Resource-Limited Settings

The diagnosis of SLE-related myocardial and conduction system involvement often requires advanced imaging and serological testing, which may not be readily available in all healthcare settings. CMR and endomyocardial biopsy are considered gold standard modalities for diagnosing myocarditis and inflammatory cardiomyopathy, allowing detailed assessment of myocardial edema, fibrosis, and inflammatory infiltration (Du Toit et al., 2023). However, access to these investigations may be limited, particularly in low- and middle-income countries. In the present case, CMR and comprehensive autoantibody profiling could not be performed due to resource constraints. Despite these limitations, a presumptive diagnosis was made based on clinical presentation, ECG findings, echocardiographic results, and positive ANA serology. The patient's favorable response to immunosuppressive therapy further supports an inflammatory autoimmune etiology. This highlights the importance of integrating clinical judgment with available diagnostic tools when evaluating unexplained conduction abnormalities, particularly in settings where advanced investigations are not feasible.

Therapeutic Implications and Reversibility of Conduction Abnormalities

Management of SLE-related CHB requires a dual approach addressing both the acute hemodynamic consequences of conduction block and the underlying autoimmune process. Temporary or permanent pacing remains the cornerstone of acute management in patients with symptomatic bradycardia or hemodynamic instability. In many reported cases, including the present one, permanent pacemaker implantation was required due to persistent conduction block and high risk of recurrent syncope or sudden cardiac death.

Immunosuppressive therapy plays a critical role in treating underlying inflammatory myocardial involvement. High-dose corticosteroids are the mainstay of treatment for lupus myocarditis, often combined with additional immunosuppressive agents such as azathioprine, mycophenolate mofetil, or cyclophosphamide in severe cases (Tanwani et al., 2018). Mohanty

et al. reported a patient with lupus myocarditis who responded favorably to intensive immunosuppressive therapy, demonstrating the potential reversibility of inflammatory myocardial injury (Mohanty et al., 2020).

Interestingly, some patients with SLE-related CHB demonstrate partial or complete recovery of intrinsic conduction following immunosuppressive treatment, suggesting that inflammatory edema or reversible cellular dysfunction may contribute to conduction impairment in certain cases. In the present case, outpatient ECG follow-up revealed reduced pacing dependency, supporting the hypothesis of at least partial reversibility of conduction system injury.

Clinical Implications and Lessons Learned

This case underscores several important clinical lessons. First, SLE should be considered in the differential diagnosis of unexplained complete heart block, particularly in younger patients and women without structural heart disease. Second, conduction abnormalities may precede or occur independently of classic systemic manifestations of lupus, necessitating a high index of suspicion. Third, early initiation of immunosuppressive therapy may improve outcomes and potentially reverse inflammatory conduction system injury. Finally, multidisciplinary collaboration between cardiology and rheumatology is essential for optimal management.

CONCLUSION

Complete heart block should be recognized as a rare but serious manifestation of systemic lupus erythematosus. Increased awareness among clinicians is essential to ensure early diagnosis, appropriate immunosuppressive treatment, and timely pacing therapy. Reporting additional cases and accumulating clinical experience will be crucial to improving understanding of the underlying mechanisms, optimizing management strategies, and ultimately enhancing outcomes for patients with this uncommon but life-threatening complication.

REFERENCES

Bharati, S., De La Fuente, D. J., Kallen, R. J., Freij, Y., & Lev, M. (1975). Conduction system in systemic lupus erythematosus with atrioventricular block. The American Journal of Cardiology, 35(2), 299–304. https://doi.org/10.1016/0002-9149(75)90017-X

Buch, M. H., Mallat, Z., Dweck, M. R., Tarkin, J. M., O'Regan, D. P., Ferreira, V., Youngstein, T., & Plein, S. (2024). Current understanding and management of cardiovascular involvement in rheumatic immune-mediated inflammatory diseases. *Nature Reviews Rheumatology*, *20*(10), 614–634.

Du Toit, R., Karamchand, S., Doubell, A. F., Reuter, H., & Herbst, P. G. (2023). Lupus myocarditis: Review of current diagnostic modalities and their application in clinical practice. Rheumatology, 62(2), 523–534. https://doi.org/10.1093/rheumatology/keac409

Fan, Z., Han, Y., Sun, G., & Dong, Z. (2025). Immunosuppressant adherence after heart transplantation: a review on detection, prevention, and intervention strategies in a multidisciplinary. *Frontiers in Cardiovascular Medicine*, *12*, 1558082.

Gómez-Barrado, J. J., García-Rubira, J. C., Polo Ostáriz, M. A., & Turégano Albarrán, S. (2002). Complete atrioventricular block in a woman with systemic lupus erythematosus. International Journal of Cardiology, 82(3), 289–292. https://doi.org/10.1016/S0167-5273(01)00590-3

Jobling, K., Rajabally, H., & Ng, W.-F. (2018). Anti-Ro antibodies and complete heart block in adults with Sjögren's syndrome. European Journal of Rheumatology, 5(3), 194–196. https://doi.org/10.5152/eurjrheum.2018.18019

Karnabi, E., & Boutjdir, M. (2010). Role of calcium channels in congenital heart block. Scandinavian Journal of Immunology, 72(3), 226–234. https://doi.org/10.1111/j.1365-3083.2010.02439.x

Lazzerini, P. E., Murthy Ginjupalli, V. K., Srivastava, U., et al. (2023). Anti-Ro/SSA antibodies blocking calcium channels as a potentially reversible cause of atrioventricular block in adults. JACC: Clinical Electrophysiology, 9(12), 1631–1648. https://doi.org/10.1016/j.jacep.2023.03.007

Lo, C. H., Wei, J. C. C., Tsai, C. F., Li, L. C., Huang, S. W., & Su, C. H. (2018). Syncope caused by complete heart block and ventricular arrhythmia as early manifestation of systemic lupus erythematosus in a pregnant patient: A case report. Lupus, 27(10), 1729–1731. https://doi.org/10.1177/0961203318782425

Maier, W. P., Ramirez, H. E., & Miller, S. B. (1987). Complete heart block as the initial manifestation of systemic lupus erythematosus. Archives of Internal Medicine, 147(1), 170–171. https://doi.org/10.1001/archinte.1987.00370010168034

Miner, J. J., & Kim, A. H. J. (2014). Cardiac manifestations of systemic lupus erythematosus. Rheumatic Disease Clinics of North America, 40(1), 51–60. https://doi.org/10.1016/j.rdc.2013.10.003

Mishra, V., Chatterjee, T., & M. C. (2023). Complete heart block: An atypical presentation with an atypical diagnosis. International Journal of Research in Medical Sciences, 11(10), 3855–3858. https://doi.org/10.18203/2320-6012.ijrms20233045

Mohanty, B., & Sunder, A. (2020). Lupus myocarditis: A rare case. Journal of Family Medicine and Primary Care, 9(8), 4441. https://doi.org/10.4103/jfmpc.jfmpc_716_20

Natsheh, A., Shimony, D., Bogot, N., Nesher, G., & Breuer, G. S. (2019). Complete heart block in lupus. Lupus, 28(13), 1589–1593. https://doi.org/10.1177/0961203319881198

Opałka, B., Żołnierczuk, M., & Grabowska, M. (2023). Immunosuppressive agents—effects on the cardiovascular system and selected metabolic aspects: a review. *Journal of Clinical Medicine*, *12*(21), 6935.

Pan, S.-Y., Tian, H.-M., Zhu, Y., et al. (2022). Cardiac damage in autoimmune diseases: Target organ involvement that cannot be ignored. Frontiers in Immunology, 13, 1056400. https://doi.org/10.3389/fimmu.2022.1056400

Popescu, M. R., Dudu, A., Jurcut, C., Ciobanu, A. M., Zagrean, A.-M., & Panaitescu, A. M. (2020). A broader perspective on anti-Ro antibodies and their fetal consequences: A case report and literature review. Diagnostics, 10(7), 478. https://doi.org/10.3390/diagnostics10070478

Shariff, R., Lim, C., & Kasim, S. (2020). Complete heart block in a 40-year-old man with anti-SSA/Ro autoantibodies. Proceedings of Singapore Healthcare, 30(4). https://doi.org/10.1177/2010105820978668

Tana, C., Kouranos, V., Bernardinello, N., Mantini, C., Scarpa, R., Cinetto, F., Israël-Biet, D., & Spagnolo, P. (2025). Redefining cardiac sarcoidosis with advanced imaging and therapeutic strategies. *American Journal of Cardiovascular Drugs*, 1–22.

Tanwani, J., Tselios, K., Gladman, D. D., Su, J., & Urowitz, M. B. (2018). Lupus myocarditis: A single center experience and a comparative analysis of observational cohort studies. Lupus, 27(8), 1296–1302. https://doi.org/10.1177/0961203318770018

Wahren-Herlenius, M., & Sonesson, S.-E. (2006). Specificity and effector mechanisms of autoantibodies in congenital heart block. Current Opinion in Immunology, 18(6), 690–696. https://doi.org/10.1016/j.coi.2006.09.012