

Lyme Carditis: A Case Involving the Conduction System and Mitral Valve

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ABSTRACT

Lyme disease is the most common tick-borne infection in the Northern hemisphere. Cardiac manifestations of Lyme disease typically include variable atrioventricular nodal block and rarely structural heart pathology. The incidence of Lyme carditis may be underestimated based on current reporting practices of confirmed cases. This case of a 59-year-old man with Lyme carditis demonstrates the unique presentation of widespread conduction system disease, mitral regurgitation, and suspected ischemic disease. Through clinical data, electrocardiograms, and cardiac imaging, we show the progression, and resolution, of a variety of cardiac symptoms attributable to infection with Lyme.

KEYWORDS: Lyme, echocardiography, mitral regurgitation, atrial arrhythmia, conduction

CASE REPORT

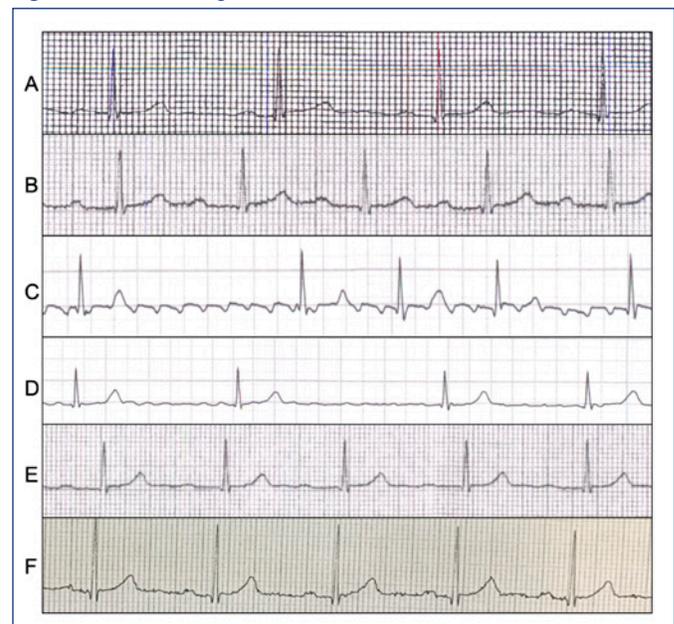
A 59-year-old man without significant past medical history was referred for evaluation of syncope. Six weeks prior to presentation, while vacationing in Cape Cod, the patient noticed an oval-shaped rash below his left knee. At a local clinic he was diagnosed with cellulitis and given a short course of oral cephalexin. Three weeks after the vacation, he awoke in the night to use the bathroom. Thirty minutes later, he found himself on the bathroom floor, not recalling any premonitory symptoms for syncope. In the following weeks, he experienced a sharp decrement in exercise tolerance. An avid marathon runner, this patient was experiencing exertional dyspnea after only a hundred yards of running and, at times, jaw discomfort.

Physical exam was remarkable for a temperature of 98.4 F, heart rate 58 bpm, blood pressure 126/68 mmHg, and 99 percent oxygen saturation. A 2/6 systolic murmur was audible at the cardiac apex. The initial electrocardiogram (ECG) demonstrated first-degree atrioventricular (AV) nodal block with a PR interval of 229 milliseconds (ms) (Figure 1). In light of the exertional dyspnea and jaw discomfort, an exercise ECG stress test was performed using the Bruce protocol. After eight minutes of total exercise, stage three intensity reproduced symptoms of dyspnea and induced ECG changes notable for 3mm ST depressions in leads V3-V6 (Figure 3).

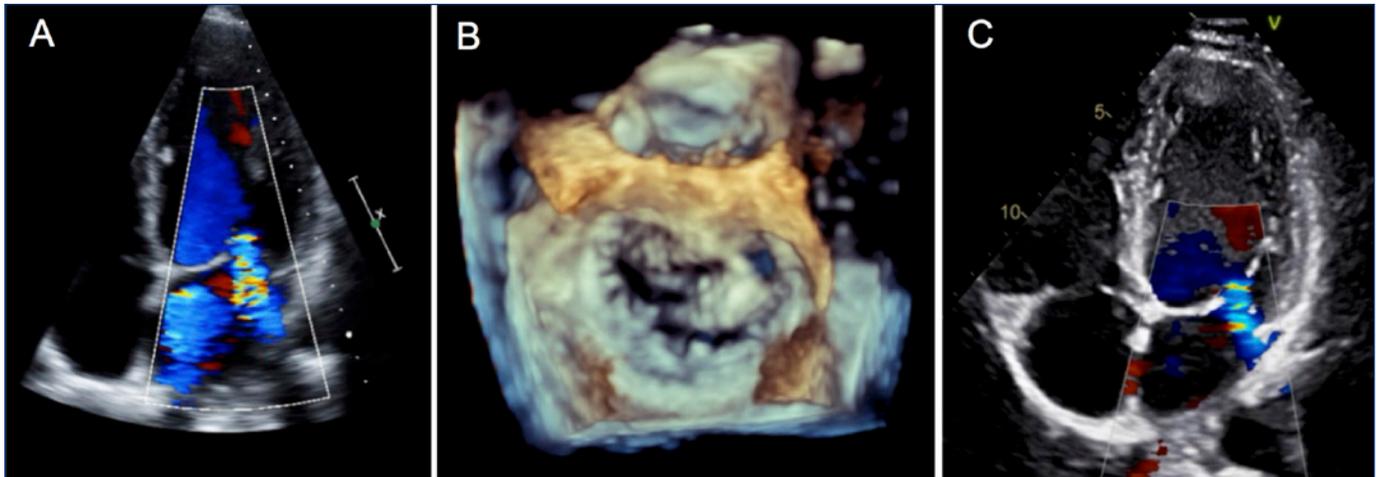
Transthoracic echocardiogram (TTE) revealed preserved left ventricular function, severe mitral regurgitation (3+) and a dilated left atrium (Figure 2). Laboratory studies revealed anemia (hgb 10.7gm/dL, hematocrit 32.0%), iron deficiency (iron 24µg/dL, saturation 10%) without any history of melena or hematochezia, and an elevated ESR (80mm/h). The patient was sent for cardiac catheterization to evaluate for coronary disease.

Right and left cardiac catheterization demonstrated no flow-limiting, significant, epicardial coronary disease (Figure 3). Left ventriculography confirmed moderately severe mitral regurgitation; right heart pressure recordings showed a prominent V wave (25mm Hg) with a normal wedge pressure. Over the next few days the patient experienced shaking chills and fevers to 101.2 F. Blood cultures in triplicate and a tick panel were ordered, and the patient was scheduled for transesophageal echocardiogram (TEE) to evaluate for

Figure 1. Electrocardiograms, Lead II.



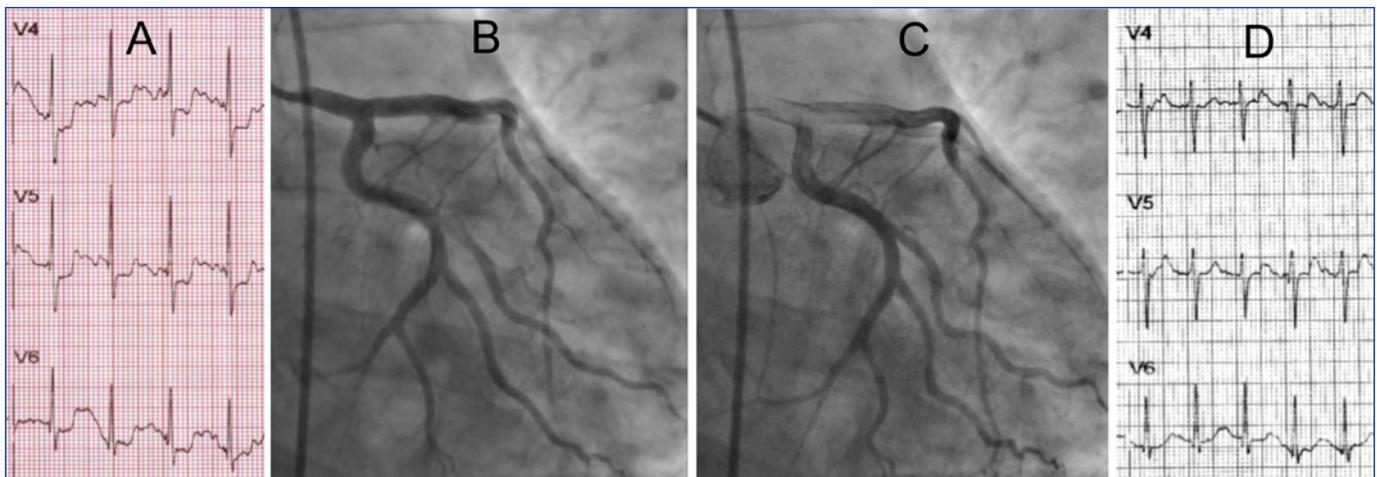
- A. Initial ECG showing 1st degree AV block (PR=229ms).
- B. Lengthening of PR interval to 298ms.
- C. Atrial flutter with variable ventricular response on hospital day 3.
- D. Atrial fibrillation on hospital day 4.
- E. Return to sinus rhythm at discharge.
- F. Significantly shortened PR interval at follow-up (PR=204ms).

Figure 2. Echocardiography images

A. TTE apical four-chamber view with doppler flow demonstrating mitral regurgitation. Graded as 3+ due to the presence of two regurgitant jets extending to the pulmonary veins.

B. 3D TEE view of the mitral valve showing two areas of incomplete valve leaflet coaptation.

C. Stress TEE, apical four-chamber view with doppler flow showing significantly improved mitral regurgitation after antibiotic treatment, graded as mild MR.

Figure 3. Possible ischemic disease

A. ECG stress test upon initial evaluation showing 3mm ST depressions in lateral precordial leads recorded after eight minutes of exercise.

B, C. Positive stress test prompted left heart catheterization with coronary angiography which demonstrated no flow-limiting, significant, epicardial coronary disease.

Angiograms were reviewed independently by several interventionalists and confirmed no spontaneous coronary artery dissection.

D. Repeat ECG stress test after completion of antibiotics shows no ST segment changes after fourteen minutes of exercise.

endocarditis. 3D TEE showed moderate MR with poor coaptation of the mitral valve leaflets and anterior leaflet prolapse, without vegetations (**Figure 2**). A repeat ECG showed lengthening of the PR interval to 298ms (**Figure 1**).

Lyme antibody enzyme immunoassay (EIA) detected significant levels of IgG (24.3; ref range <1), IgM (9.6; ref range <1), and IgA (>9.9; ref range <1) to *B. burgdorferi*. The patient was admitted to the hospital for intravenous ceftriaxone and telemetry monitoring. On admission, Troponin I measurement and electrolytes were in the normal range. CRP (87.5mg/l) and ESR (73mm/h) were elevated without associated leukocytosis.

During the admission, the patient's heart rhythm alternated between sinus bradycardia with first-degree block (352ms) and atrial flutter with slow, variable ventricular response (**Figure 1**). Occasional periods of atrial fibrillation and three-second sinus pauses were also noted, and he was started on oral anticoagulation. The patient developed symptoms of heart failure with an elevated NT-proBNP of 2212 ng/l and was treated with oral furosemide. Atrial arrhythmias resolved with continued therapy for presumed Lyme carditis. At the time of discharge, the patient was asymptomatic and had been in sinus rhythm for over thirty-six hours with improving first-degree AV block. He was discharged to

complete oral doxycycline for a total treatment course of twenty-one days.

After discharge, the patient remained asymptomatic and resumed exercise without dyspnea. The systolic murmur of MR was less apparent (grade 1/6) and ECGs showed progressive shortening of the PR interval (**Figure 1**). Three weeks after discharge, he tolerated, without dyspnea, Bruce protocol stage-five exercise. Total exercise time was fourteen minutes. No ST changes or atrial arrhythmias were noted. Both rest and stress echocardiography revealed reduced mitral regurgitation (**Figure 2**). Given the lack of arrhythmia and symptoms during the test, the patient was instructed to stop anticoagulation and furosemide. He was not referred to cardiac surgery for valve repair. A few weeks later, the patient successfully completed a local five-kilometer race.

DISCUSSION

Cardiac symptomology associated with Lyme disease includes syncope, lightheadedness, dyspnea, palpitations, and chest pain. Such manifestations usually present two to five weeks after the erythema migrans rash, and typically involve the electrical conduction system. Only 1.1 percent of Lyme cases reported to the CDC include cardiac manifestations; however, surveillance guidelines have changed such that only cases of second- or third-degree AV block are frequently reported (1). Thus, the true incidence of Lyme carditis may be underestimated as case reports have documented abnormalities ranging from first-degree heart block to myocarditis to acute heart failure (1-3). The case described here is unique in that infection with *Borrelia burgdorferi* likely involved both the electrical conduction system and the mitral valve.

The most common clinical feature of Lyme carditis is transient atrioventricular nodal block. As the degree of AV block can fluctuate rapidly, current guidelines recommend inpatient telemetry monitoring for any patient with cardiac symptoms, a PR interval longer than 300ms, or second- or third-degree heart block (4). Although this case initially presented with relatively benign ECG findings, telemetry admission because of cardiac symptoms and an increasing PR interval allowed for prompt diagnosis and management of unexpected atrial arrhythmias. Conduction involvement outside the AV node is rare (5-7). In this case, the presence of first-degree AV block, atrial flutter, and atrial fibrillation suggested simultaneous conduction abnormalities of various etiology. AV nodal block and slow ventricular response to atrial arrhythmias suggest conduction disease within the AV junction. Atrial flutter and fibrillation, however, being macro- and micro-reentrant arrhythmias of the atrium, could represent more widespread conduction disease or more likely developed due to left atrial enlargement and mitral regurgitation. Complete conduction recovery with antibiotic treatment occurs in more than ninety percent of Lyme carditis cases (5, 8, 9).

Conduction system disease alone did not explain this

patient's acute decrement in exercise tolerance. Mitral regurgitation was diagnosed, given the murmur on exam, regurgitant flow on 2D echocardiography, right heart catheterization, cardiac ventriculography, and leaflet prolapse on 3D TEE. It is highly unlikely that such severe mitral regurgitation, graded 3+ by echocardiography (**Figure 2**), was chronic because the patient had been asymptomatic, engaging in high levels of aerobic exercise without limitations. Initial echocardiography imaging showed dilatation of the left atrium, typically a sign of chronic mitral regurgitation. In this case, six weeks of untreated Lyme carditis, characterized by exacerbated mitral regurgitation with paroxysmal atrial flutter and fibrillation, better explains the left atrial dilation. Stress echocardiography after completed treatment of Lyme carditis demonstrated reduced mitral regurgitation (**Figure 2**). Temporally, this correlated with the patient resuming exercise without dyspnea. Based on these echocardiography findings, we conclude that this case of Lyme carditis involved the mitral valve, contributing to symptoms of dyspnea. No vegetation was detected on TEE, but it is possible local myocardial inflammation worsened regurgitation across the prolapsed mitral valve. Lyme disease is generally not considered a cause of valvular pathology. One case report of Lyme endocarditis, however, described histopathologic detection of *Borrelia afzelli* DNA in a replaced mitral valve with inflammatory infiltrates and fibrin deposition (10). Autopsy samples from fatal Lyme infections have demonstrated spirochete and macrophage-predominant infiltrates particularly near connective tissues of the AV junction but also in endocardium and myocardium (11). The AV junction lies within the fibrous trigone near the mid-septal mitral orifice (12). In this case, local spirochete invasion and macrophage infiltration from the AV junction to the contiguous mitral valve may have caused significant leaflet edema. Inflamed and edematous leaflets likely explain the poor valve coaptation and mitral regurgitation seen on echocardiography.

The progression of this case started with concern for ischemic disease: the patient experienced exertional dyspnea and jaw discomfort with 3mm horizontal ST depressions during stress testing. ST segment depressions are the most reliable indicator of exercise-induced ischemia, but can also be false positive data (13). Cardiac catheterization ruled out significant epicardial coronary disease. Additionally, full review of the angiography films revealed no evidence of non-occlusive plaque rupture or spontaneous coronary artery dissection within the coronary tree. Alternatively, if the ST changes were true for ischemic disease, sub-endocardial vessels may have been affected. Autopsy samples of Lyme carditis have shown the presence of perivascular inflammation (11). The same macrophage-predominant infiltrate that affected the AV node and mitral valve likely extended locally to small sub-endocardial vessels. Reduced oxygen delivery because of this sub-endocardial vasculitis may have elicited ST depressions during exercise. Repeat exercise stress testing after treatment for Lyme did not produce the same ECG changes,

thus, it is possible that oxygen delivery to the endocardium was restored with resolution of the inflammatory response. Coronary vasculitis from Lyme has not been described in the literature, and ST depressions are rare ECG changes in Lyme carditis (14).

CONCLUSION

This case is one of the first to describe and illustrate mitral valve involvement and widespread conduction disease in a single case of Lyme carditis. Additionally, the possibility of transient ischemic disease in the case may be explained by a microvasculitis manifestation of Lyme carditis. Although cardiac involvement of Lyme disease is uncommon, clinical implications such as complete heart block and acute heart failure, if left untreated, could be severe and fatal. Additionally, the incidence of Lyme carditis may be underestimated based on current surveillance guidelines and practices. In areas endemic for Lyme, the evaluation of new onset cardiac symptoms, with evidence of conduction disease or valvular pathology, should warrant a work-up for Lyme carditis. Prompt diagnosis and treatment of Lyme carditis may prevent unnecessary and invasive electrophysiological testing or surgical intervention for conduction system and valvular manifestations, respectively.

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