

Sinus Pause in Association with Lyme Carditis

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*Lyme disease is the most prevalent tick-borne disease in the United States. It is caused by the spirochete *Borrelia burgdorferi*. Cardiac involvement is seen in 4% to 10% of patients with Lyme disease. The principal manifestation of Lyme carditis is self-limited conduction system disease, with predominant involvement of the atrioventricular node. On rare occasions, Lyme carditis patients present with other conduction system disorders such as bundle branch block, intraventricular conduction delay, and supraventricular or ventricular tachycardia. We report the unusual case of a 59-year-old man who presented with new-onset symptomatic sinus pauses one month after hiking in upstate New York. To our knowledge, this is the first case report from North America that describes the relationship between symptomatic sinus pause and Lyme carditis. (Tex Heart Inst J 2015;42(3):248-50)*

Lyme disease is a tick-borne systemic disease caused by the spirochete *Borrelia burgdorferi*. Cardiac involvement is seen in 4% to 10% of patients with Lyme disease. The cardinal manifestation of Lyme carditis is a self-limited conduction system disease, most frequently involving the atrioventricular (AV) node and leading to varying degrees of AV nodal block. Less frequently, endocarditis, myocarditis, pericarditis, coronary artery aneurysm, QT prolongation, tachyarrhythmias, myocardial infarction, and congestive heart failure have been reported.¹ We describe the unusual case of a man who presented with symptomatic sinus pauses due to Lyme carditis.

Key words: *Borrelia burgdorferi*; heart block/etiology; Lyme disease/diagnosis; sinoatrial block/diagnosis; sinus node dysfunction; sinus pause; tick-borne diseases

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Case Report

In October 2013, a 59-year-old man presented at Advocate Condell Medical Center's emergency department because of new-onset dizzy spells and near-syncope, which he had experienced multiple times a day for the past 3 weeks. His symptoms were brief (<30 s), paroxysmal, and not associated with any particular activity. He reported no chest pain, shortness of breath, palpitations, or fainting spells. He had a history of hypertension, for which he had taken lisinopril (5 mg/d). Lisinopril had been discontinued 3 weeks before, because of his new symptoms. He also took the dietary supplements fish oil, coenzyme Q, and flax seed. He had no known allergies and his family history was noncontributory. He lived with his family in Chicago, drank approximately one bottle of wine every week, and had no history of cigarette smoking or substance abuse. He reported having gone on a hiking trip in the Catskill Mountain region of New York State a month before his presentation. He also reported that, upon his return from this trip, he had developed an erythematous rash on both arms, which lasted for a few days and was accompanied by generalized fatigue, subjective fever, and arthralgia in multiple joints.

On examination, his temperature was 36.9 °C, his blood pressure was 156/87 mmHg, and his heart rate was 64 beats/min. No other abnormalities were present upon physical examination, except for an irregular heart rhythm. His electrocardiogram (ECG) on admission showed bifascicular block pattern (right bundle branch block [RBBB] with left posterior fascicular block),² sinus bradycardia (ventricular rate, 56 beats/min) with 1st-degree AV block and a sinus pause (3 s) with a junctional escape beat (Fig. 1). According to his medical records, a previous ECG, performed prior to the current illness, was reported to be normal, except for an RBBB pattern. We were not able to verify this, because no previous ECG was available for review. His initial laboratory results—including complete blood count, electrolytes, thyroid-

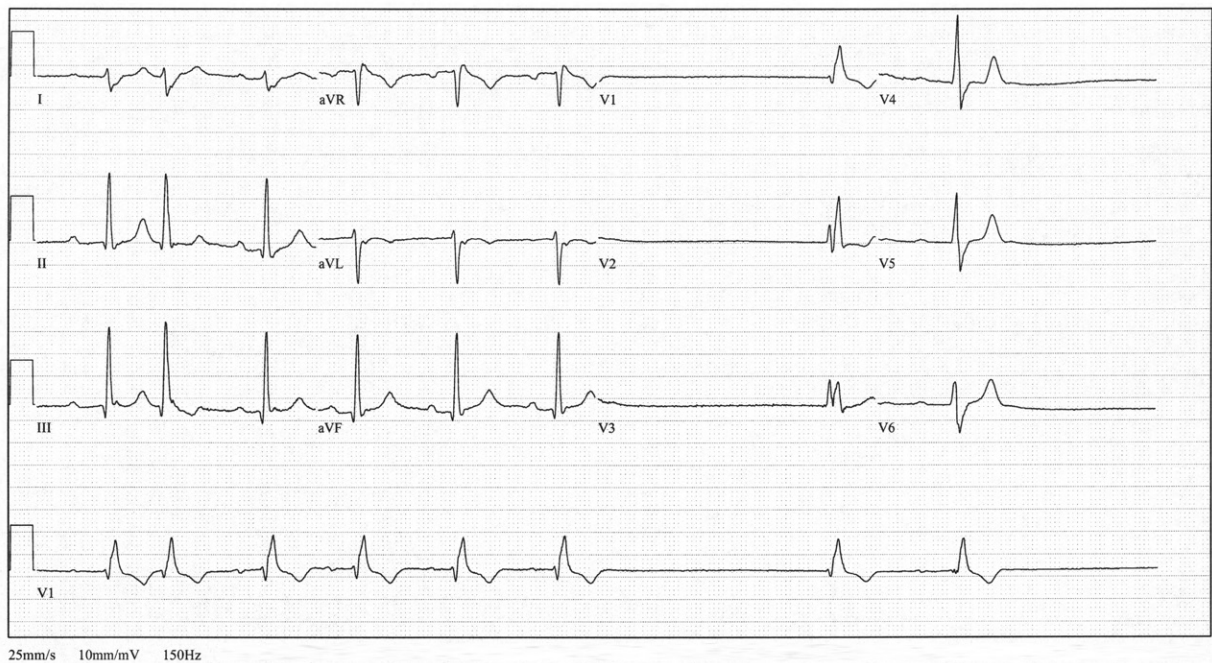


Fig. 1 Twelve-lead electrocardiogram from the emergency department shows sinus bradycardia, a ventricular rate of 56 beats/min, sinus pause (3 s), a junctional escape beat, premature atrial complex, 1st-degree atrioventricular block (PR interval, 218 ms), and bifascicular block (right bundle branch block with left posterior fascicular block).

stimulating hormone, and troponin ($\times 3$)—were within normal limits. After admission to the hospital, the patient continued to experience frequent paroxysmal episodes of lightheadedness that were accompanied by sinus pauses (3–8 s) on telemetry (Fig. 2). Infrequent episodes of 2nd-degree type I (Wenckebach) AV block were also recorded on telemetry (Fig. 3). He did not need external or transvenous pacing during his hospital stay. His echocardiograms showed mild mitral and aortic valvular regurgitation, but no other abnormalities.

The patient had no recollection of a tick bite; however, the following findings led to the diagnosis of Lyme carditis as the cause of his symptomatic sinus pause: his history of skin rash, his constitutional symptoms and arthralgia after hiking in a region where Lyme disease is endemic, the presence of 1st-degree AV block, and the absence of any structural or ischemic heart disease. A positive enzyme-linked immunosorbent assay serologic test and Western blot protocol (immunoglobulins M and G) later supported that diagnosis.

On the day of admission, our patient was started on a regimen of intravenous ceftriaxone for suspected Lyme carditis. He improved slowly but progressively throughout his 14-day hospital stay. The sinus pauses became shorter and less frequent, and his symptoms resolved. However, the 1st-degree AV block persisted. He was discharged from the hospital with instructions to take oral doxycycline. The results of 48-hour Holter monitoring approximately 7 weeks after his hospital discharge con-

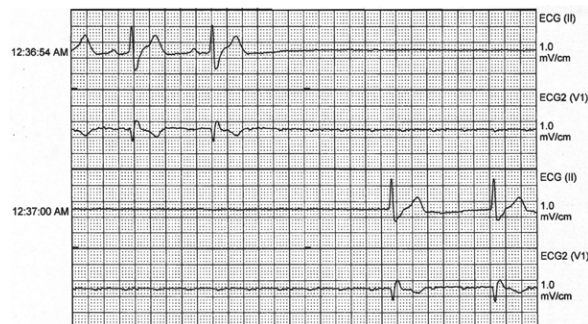


Fig. 2 Telemetry recording from the medical ward shows sinus bradycardia with 1st-degree atrioventricular block, sinus pause (8.3 s), and junctional escape beats.

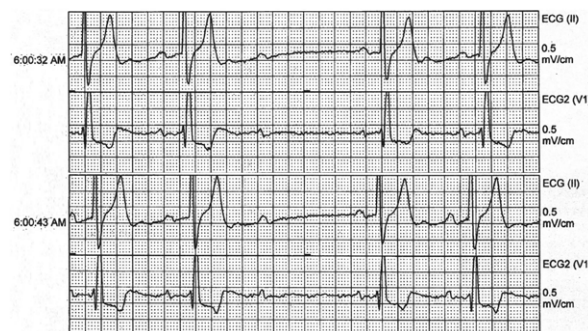


Fig. 3 Telemetry recording from the medical ward shows sinus bradycardia with 2nd-degree type I (Wenckebach) atrioventricular block.

firmed the complete resolution of his sinus pauses. At that time, the patient reported that he had been symptom free. The only relevant abnormality recorded on Holter monitoring was the very occasional episode of “dropped beat” due to 2nd-degree type I (Wenckebach) AV block.

Discussion

Lyme disease is the most prevalent vector-borne disease in the United States. Lyme carditis can manifest itself as early as 4 days or as late as 7 months after a tick bite. The diagnosis of Lyme carditis is challenging: it is usually made in the presence of other manifestations of Lyme disease—concomitant erythema migrans, arthritis, or neurologic disease, or such cardiac manifestations as ECG findings and congestive heart failure—together with positive serologic testing for *B. burgdorferi* infection. One should note that positive serologic testing is not by itself diagnostic, nor does negative serologic testing exclude the diagnosis.

The association of Lyme carditis with conduction system disturbances (predominantly AV nodal) is well known. In a retrospective study, 86% of patients with Lyme carditis had some degree of AV block.³ On rare occasions, Lyme carditis has been reported to cause bundle branch block, intraventricular conduction delay, and tachycardia (supraventricular or ventricular). Despite the extensive medical literature supporting a strong association between Lyme disease and AV-node dysfunction, the effect of Lyme disease on sinus node function is still unclear. Our literature search revealed only 2 reports of cases, both from Europe, describing sinus node dysfunction caused by *B. burgdorferi* infection.^{4,5}

Ours appears to be the first report from North America to describe a case of Lyme carditis in which the patient presented with sinus node dysfunction. The presence of 1st-degree and 2nd-degree type I (Wenckebach) AV blocks show that sinus node dysfunction was accompanied by AV-node involvement. The patient did not really need an electrophysiologic study, which would have helped to identify the underlying mechanisms of his bradyarrhythmias. Because Lyme carditis manifests itself mainly with conduction abnormalities, it could be hypothesized that the mechanism of pauses was sinus exit block, rather than the effect on automaticity of the sinus node. Our patient did not have the characteristic ECG findings of sinus exit block: group beating patterns and progressive shortening of P-P intervals (seen with 2nd-degree type 1 sinus exit block) or pauses lasting exact multiples of immediately preceding P-P intervals (seen with 2nd-degree type 2 sinus exit block).⁶ The irregular sinus arrhythmia at baseline and the junctional escape beats prevented our making a clear distinction between sinus pause and sinoatrial exit block on the basis of ECG and telemetry recordings.

No randomized controlled trials have specifically studied the efficacy of antibiotics in the treatment of Lyme carditis. Despite this lack of strong evidence, oral or intravenous antibiotic therapy is recommended in patients with Lyme carditis.⁷ Antibiotic therapy started during the early stages of Lyme disease has been shown to prevent sequelae. Our patient showed improvement of sinus node dysfunction within 2 weeks of antibiotic initiation. However, it is not possible to attribute this improvement solely to antibiotic therapy, because Lyme carditis is a self-limiting disease; conduction disturbances usually resolve spontaneously within one to 6 weeks.²

Physicians should be aware that, on rare occasions, Lyme carditis might present with sinus node dysfunction. Timely diagnosis and appropriate management of Lyme carditis might prevent unnecessary invasive procedures, such as pacemaker placement.

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