

# Lyme carditis presenting with an incessant atrioventricular nodal reentrant tachycardia masking a variable atrioventricular block



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## Introduction

Lyme disease is a common tick-borne illness with a high prevalence in the Northeast region of the United States.<sup>1</sup> It is an infection caused by *Borrelia burgdorferi*, a gram-negative spirochetal bacterium carried by infected ticks such as *Ixodes scapularis*. They are found outdoors, latched onto blades of grass until they can attach to a host. If not treated early, serious complications can develop. Since Lyme antibodies can take several weeks to develop, false-negatives can arise in about 50% of cases when testing is done too early.<sup>1-3</sup> False-negatives can delay treatment and lead to complications such as Lyme carditis. The most common cardiac complications of Lyme disease are conduction disorders. A variable heart block with associated bradycardia is the most common conduction disorder seen.<sup>1</sup> Tachyarrhythmias are uncommon, and there have been few documented cases (Table 1).

We present a case of a young male patient with untreated Lyme disease who developed Lyme carditis. He presented with an incessant atrioventricular nodal reentrant tachycardia (AVNRT) masking a variable heart block. This report details how we managed this rare manifestation of Lyme carditis.

## Case report

A fit and well 32-year-old male patient with no significant past medical history presented to the emergency room with complaints of a persistently elevated pulse rate of up to 140 beats per minute (bpm). He denied any associated symptoms except intermittent bilateral knee pain and swelling. He denied any prior history of palpitations, elevated pulse rate, or arrhythmia. On examination, he was not in acute distress. He was afebrile, tachycardic at 143 bpm, and normotensive.

**KEYWORDS** Electrophysiology; Lyme carditis; Atrioventricular block; Propafenone; Lyme-associated tachyarrhythmia (Heart Rhythm Case Reports 2022;8:829-835)

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## KEY TEACHING POINTS

- Though much less common than bradycardias, tachyarrhythmias can occur in Lyme disease. This is because a minority of patients have preexisting dormant abnormal conduction pathways, possibly activated by inflammatory changes induced by *Borrelia* spirochetes in the conducting system, leading to a tachyarrhythmia instead of the typical bradycardia.
- Lyme carditis with severe atrioventricular block, defined by a PR interval of 300 ms or more, is associated with a higher risk of progression to complete heart block and cardiac arrest. Therefore, it should be managed in the inpatient setting.
- Lyme-associated tachyarrhythmia is a severe presentation of Lyme carditis, as there is a high risk of the tachyarrhythmia leading to sudden cardiac arrest if it is not treated appropriately with antiarrhythmics until antibiotics can take effect.
- In the case of persistent tachyarrhythmia in the setting of Lyme carditis, propafenone has shown benefit as a good antiarrhythmic choice for achieving rhythm control.

Physical examination was only significant for a small area of hyperpigmentation to the posterior aspect of the left knee. No rashes or ticks were found on examination.

Three months prior, he presented to his primary care physician after finding an engorged tick on the back of his left knee. Lyme serology testing was done and was negative at that time. Therefore, no treatment or further testing was done. One week later, he noted a bull's-eye rash to the posterior left knee with associated pain and swelling. Symptoms improved with ibuprofen.

Throughout admission, complete blood counts with differential and complete metabolic panels remained within normal limits. The urine toxicology screen was negative. Thyroid function was normal. Lyme serology was repeated, and titers were positive at 11.3 (normal range < 0.90). This was confirmed by Western blot testing. Testing for coinfections with *Ehrlichia chaffensis*, *Babesia microti*, *Babesia duncuni*, and *Babesia divergens* were all negative. However, he tested positive for *Anaplasma phagocytophilum* with titers of 1:64 (normal range < 1:64).

An echocardiogram revealed a normal left ventricular function, a normal ejection fraction, and no valvular disease or cardiomyopathy. The chest radiograph was also unremarkable. He was placed on telemetry monitoring, and serial electrocardiograms (ECGs) were also done (Figure 1).

After AVNRT was identified on the initial ECG (Figure 1A), the patient was given adenosine ( $\times 2$ ), causing conversion to sinus rhythm but with a quick return of the AVNRT each time. Intravenous (IV) pushes of diltiazem and metoprolol were not effective. He was then started on a diltiazem infusion, which was also ineffective. As a result, it was discontinued, and he was started on oral metoprolol and given 1 dose of flecainide. This resulted in brief periods of conversion of the AVNRT to sinus rhythm (Figure 1B). An underlying atrioventricular (AV) block (both first- and second-degree) with a PR interval of 240–320 ms was noted on telemetry (Figure 1C and 1D). The patient was then started on propafenone, and metoprolol was continued. This antiarrhythmic regimen achieved significantly better rate and rhythm control before starting antibiotics. At this point, he tested positive for Lyme disease and *Anaplasma*.

He was then started on ceftriaxone and oral doxycycline. Antiarrhythmic treatment was continued with propafenone and metoprolol. His heart rate continued to improve, and he maintained sinus rhythm with rates of 90–110 bpm. The PR interval remained prolonged at 320 ms, but he remained hemodynamically stable, and there was no evidence of progression to a higher-grade block.

In addition to having a normal echocardiogram, the patient displayed no clinical signs or symptoms of heart failure throughout admission, and he had a good clinical response to antibiotics and antiarrhythmics. Therefore, further investigation with cardiac magnetic resonance imaging or myocardial biopsy was deferred. He was discharged with a cardiac event monitor, was prescribed a 21-day course of oral doxycycline, and continued propafenone and metoprolol.

Six days, cardiac event recorder results revealed resolution of the AVNRT and maintenance of sinus rhythm but the persistence of the first-degree AV block. The PR interval decreased from 320 ms to 220 ms (Figure 2). He was responding adequately to treatment. Antiarrhythmics were continued for 1 extra week, and he completed the rest of his 21-day course of oral doxycycline. His second follow-up was done 2 weeks later, after completing antibiotics and antiarrhythmics 1 week prior. There was no evidence of AVNRT, the average heart rate was 70–80 bpm, and the heart block resolved.

## Discussion

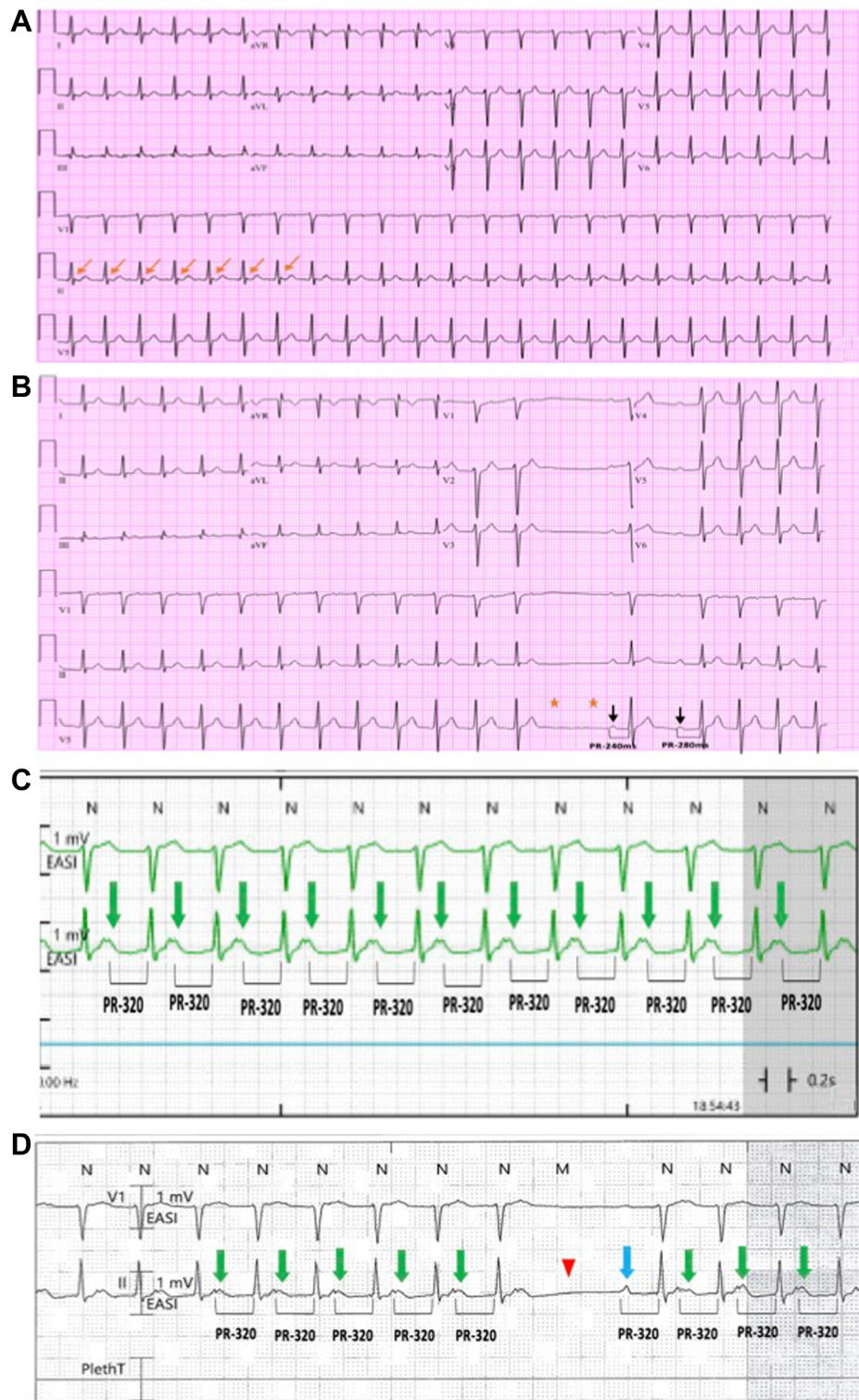
Lyme carditis is a rare complication of Lyme disease, accounting for <10% of cases in the United States.<sup>2–5</sup> It occurs when *Borrelia* spirochetes enter and infect the myocardium, producing a local inflammatory response that most commonly affects the conduction system.<sup>1–3</sup> For reasons unknown, the AV node and bundle of His are the most common parts affected. The most common cardiac manifestation is a transient AV block, which can vary in degree.<sup>2,4</sup> In addition, patients often present with bradycardia. However, few cases of tachycardia are documented<sup>6–12</sup> (Table 1).

Extensive research has been done on the anatomy and electrophysiology of the AV node, which suggests the presence of a “dual” AV nodal system thought to involve 2 pathways: “fast” and “slow” pathways.<sup>5</sup> The slow pathway is present in a minority of patients.<sup>5</sup> This explains why few patients with Lyme disease are prone to developing reentrant tachycardia while most develop bradycardia.<sup>8</sup> We postulate that Lyme-induced tachyarrhythmias result from a spirochete-induced local inflammatory response in the conduction system, causing activation of dormant abnormal conduction pathways in some individuals, such as this patient.

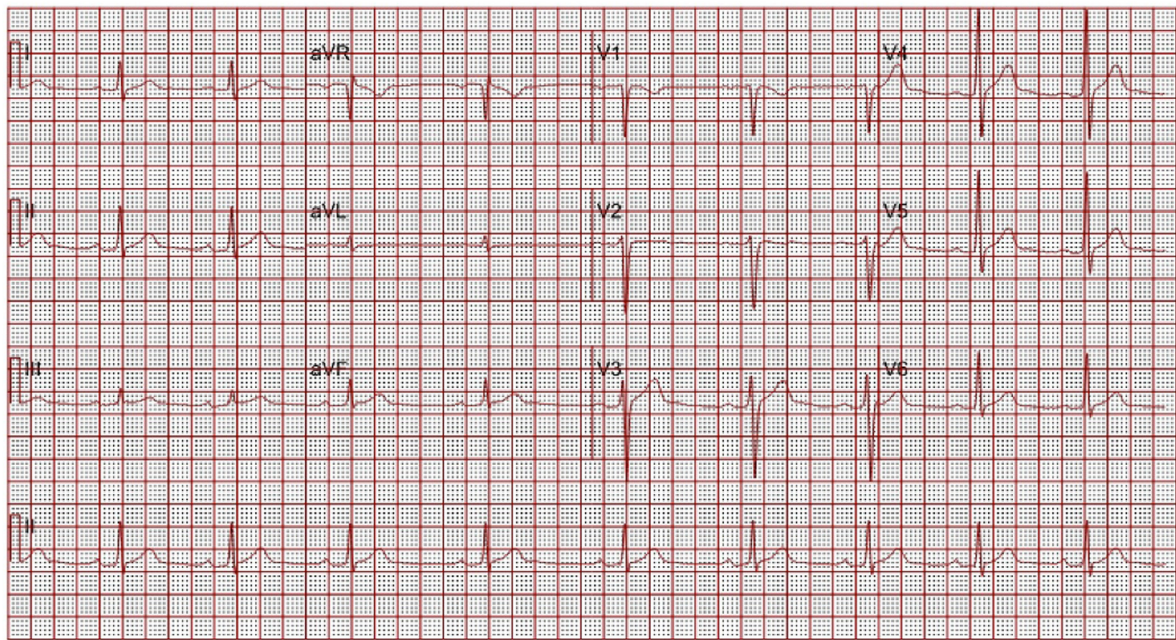
Typically impulses travel from the sinus node to the AV node, down the fast pathway, and into the bundle of His.<sup>5</sup> In patients with a slow pathway, the impulse travels down the slow pathway at a much slower conduction velocity, only to be canceled by retrograde impulses from the fast pathway where the 2 pathways meet.<sup>5</sup> However, in situations where the anterograde flow of the fast pathway is affected, atrial impulses preferentially take the slow pathway. In these cases, retrograde impulses from the slow pathway can travel up the fast pathway and back down the slow pathway in a continuous loop, leading to AVNRT.<sup>5</sup>

We postulate that this patient had both a fast and slow pathway, and inflammation in the conduction system caused hyperexcitability in both pathways. This likely led to depolarization and increased conduction velocity in both pathways. Because the slow pathway typically has a shorter refractory period than the fast pathway,<sup>5</sup> the increased conduction velocity from the inflammation likely led to impulses traveling preferentially down the slow pathway, while the fast pathway was experiencing a longer refractory period. This effect likely allowed the fast pathway to be much more susceptible to receiving retrograde impulses from the now hyperexcitable and quickly recovering slow pathway, leading to travel of the retrograde impulses back down the slow pathway again, causing the formation of a reentrant loop. The increase in the conduction velocity from inflammation likely led to recurrent propagation of this reentrant loop, which would explain why our patient’s AVNRT was resistant to commonly effective medications like adenosine.

One may also argue that the concomitant *Anaplasma* infection may have caused the tachyarrhythmia. However, despite rare cases of myocarditis, *Anaplasma* is not known to commonly affect the cardiac conduction system.<sup>13</sup> Second, this patient had a very mild infection of *Anaplasma*,



**Figure 1** Rhythm changes throughout admission. **A:** Initial electrocardiogram demonstrating a narrow complex tachycardia with retrograde p waves (red arrows) and short RP intervals (atrioventricular nodal reentrant tachycardia [AVNRT]). **B:** Showing a brief conversion of the AVNRT to sinus rhythm with a variable heart block followed by return of the AVNRT. Note there is a pause for 2 beats (indicated by the red stars) prior to the start of sinus rhythm (black arrows indicating p waves) before the AVNRT starts again. Although PR intervals are noted to increase from the first sinus beat to the next, the AVNRT rhythm re-emerges before any further prolongation and subsequent dropped beats can be seen. This may represent a prematurely terminated Mobitz type 1 heart block. **C:** Telemetry strip showing a decrease in the patient’s heart rate after a trial of flecainide and metoprolol. There is an appearance of the t waves (green arrows). These may represent retrograde p waves from the AVNRT with an unusually prolonged RP interval or sinus tachycardia with a first-degree heart block containing a PR interval, causing p waves to be partially hidden in the preceding t waves. **D:** Telemetry strip showing a brief progression of a first-degree heart block to a second-degree Mobitz type 2, as evidenced by the constant PR interval of 320 ms followed by an abrupt pause (red inverted triangle). Note how the p wave after the pause (blue arrow) is clearly seen when there is no preceding t wave to partially hide it.



**Figure 2** Showing electrocardiogram on the first follow-up visit demonstrating resolution of atrioventricular nodal reentrant tachycardia. Sinus bradycardia with first-degree heart block with an improvement in the appearance of length (220 ms) is seen.

evidenced by a lack of transaminitis, thrombocytopenia, and hemolytic anemia typically found in clinically significant *Anaplasma* infections. Therefore, this factor was less likely to cause his tachyarrhythmia.

### Understanding the rhythm changes seen in this case

Figure 1A shows AVNRT on this patient's first ECG with notable retrograde p waves with short RP intervals. These features are characteristic of slow-fast AVNRT.<sup>5</sup> This rhythm was incessant, which suggested that there was an underlying pathologic process causing this rhythm to persist despite the use of agents that usually terminate the arrhythmia. The use of class 1C antiarrhythmics led to a slowing of rate and changes in the rhythm, which unmasked a variable heart block. Changes are first noted in Figure 1B, where the AVNRT is followed by a conversion pause and emergence of sinus with a gradually prolonged PR interval for 2 beats before the AVNRT starts again. This was the point where the underlying AV block was unmasked.

Figure 1C reveals an interesting rhythm change that can be interpreted in 1 of 2 ways. On one hand, the double peaks of the t waves may be due to retrograde p waves partially hidden in the t waves. This could represent a slow-fast AVNRT. However, it is also reasonable to consider that the double notch in the t wave may represent the p wave from sinus tachycardia partially hidden in the t wave owing to an extremely long PR interval >300 ms. The patient did have an underlying systemic infection, which could explain why he would be in sinus tachycardia after having a supraventricular tachycardia. Furthermore, the ECG in Figure 1D shows intermittent dropped beats followed by p waves in the exact

distance from the following QRS, where the double-notched t wave would be expected to be present had there not been a dropped beat. The underlying p wave was completely exposed when there was no T wave from a preceding QRS complex to partially obscure it. This would mean that the PR interval was prolonged, constant, and then followed by sudden dropped beats, providing evidence of a progression to Mobitz type 2 captured in Figure 1D.

### Role of antiarrhythmics in Lyme-induced tachyarrhythmias

While antibiotics are effective as monotherapy in the treatment of Lyme carditis, a literature review revealed that Lyme-associated tachyarrhythmias have a propensity to become quickly unstable and result in unfavorable outcomes (Table 1). Therefore, in this case, it was essential to achieve and maintain rhythm control with antiarrhythmics until antibiotics could take effect and eliminate spirochetes from the conduction system.

Propafenone and flecainide are class 1C antiarrhythmics.<sup>14</sup> Unlike flecainide, propafenone is known to have calcium channel and beta antagonist effects as well,<sup>14,15</sup> which also provides suppression of automaticity in nodal tissues. Better rate control was achieved in this case when it was added to metoprolol before antibiotics were initiated. These features made it an ideal choice for rhythm and additional rate control in an incessant supraventricular tachycardia resulting from hyperexcitable AV nodal reentrant inflammatory pathways. Table 1 describes some cases supporting propafenone's effectiveness in managing incessant Lyme-associated tachyarrhythmias, resistant to other classes of antiarrhythmics.

**Table 1** Documented cases of reported Lyme-associated tachyarrhythmias and how they were managed

Case #	Article title	Year	Short case description
1	Ventricular tachycardia associated with Lyme carditis <sup>6</sup>	1991	<ul style="list-style-type: none"> <li>• 67-year-old male patient with a history of tick bite presented with multiple episodes of nonsustained ventricular tachycardia</li> <li>• Found to have idiopathic cardiomyopathy on echo with reduced EF</li> <li>• Failed rhythm control with lidocaine and procainamide</li> <li>• Trial of propafenone completely suppressed both sustained and nonsustained VT in this patient</li> <li>• Treated with IV ceftriaxone for 6 months, which resulted in improvement of cardiac function</li> </ul>
2	Lyme myocarditis presenting as fascicular tachycardia with underlying complete heart block <sup>7</sup>	1997	<ul style="list-style-type: none"> <li>• 42-year-old male patient presented with 2 weeks of headaches, malaise, and loose stools and episodes of syncope 2 days prior to presentation</li> <li>• Noted to have fascicular tachycardia with RBBB and left posterior hemiblock with AV dissociation</li> <li>• Use of adenosine and lidocaine failed to control rhythm</li> <li>• Cardioversion attempts were complicated by induction of ventricular fibrillation, which required defibrillation</li> <li>• Postdefibrillation rhythm returned to fascicular tachycardia, which then improved on infusion of procainamide</li> <li>• Tachycardia slowly resolved, revealing sinus rhythm and complete heart block, for which temporary pacing had to be initiated</li> <li>• He tested positive for Lyme disease</li> <li>• IV ceftriaxone was started, which resulted in gradual resolution of fascicular tachycardia and heart block after 9 days</li> </ul>
3	Junctional tachycardia in a child with Lyme carditis <sup>8</sup>	2011	<ul style="list-style-type: none"> <li>• 3-year-old female patient presented with 1-week history of malaise</li> <li>• Found to be febrile and tachycardic on admission</li> <li>• Noted to have erythema migrans rash</li> <li>• Electrocardiogram showed junctional tachycardia</li> <li>• At this time patient was not given antiarrhythmics but was started on IV ceftriaxone, since she was hemodynamically stable despite tachycardia</li> <li>• Tachycardia resolved and on conversion to sinus rhythm there was no AV block</li> </ul>
4	[Cardiac arrest due to torsades de pointes ventricular tachycardia in a patient with Lyme carditis] <sup>9</sup>	2014	<ul style="list-style-type: none"> <li>• A 45-year-old female patient presented with third-degree AV block and was diagnosed with Lyme carditis</li> <li>• Patient went into torsade de pointes VT and cardiac arrest despite being on appropriate antibiotics and despite appropriate antibiotic treatment and continuous ventricular pacing</li> </ul>

(Continued)

**Table 1** (Continued)

Case #	Article title	Year	Short case description
5	Junctional ectopic tachycardia secondary to myocarditis associated with sudden cardiac arrest <sup>10</sup>	2016	<ul style="list-style-type: none"> <li>• 12-year-old patient presented with sudden-onset dyspnea respiratory failure</li> <li>• Went into cardiac arrest from pulseless ventricular fibrillation</li> <li>• Patient received cardiopulmonary resuscitation, was intubated and vented</li> <li>• Noted to have persistent junctional ectopic tachycardia with periods of fascicular tachycardia</li> <li>• Treated with procainamide and amiodarone</li> <li>• Later found to have underlying Lyme disease and Lyme carditis</li> <li>• Cardiac function improved after a 21-day course of IV ceftriaxone</li> </ul>
6	A 15-year-old male with wide complex tachyarrhythmia <sup>11</sup>	2018	<ul style="list-style-type: none"> <li>• 15-year-old male patient presented with exertional syncope</li> <li>• Found to have unstable wide complex tachycardia</li> <li>• Required synchronized cardioversion, which provided symptomatic improvement, but then was found to be in complete heart block requiring pacing</li> <li>• Later found to have Lyme disease</li> <li>• Treated with IV ceftriaxone</li> </ul>
7	Lyme disease: A cause of polymorphic ventricular tachycardia <sup>12</sup>	2020	<ul style="list-style-type: none"> <li>• 38-year-old female patient presented with complaints of fever and presyncope for 2 weeks prior to presentation</li> <li>• Noted to have first-degree heart block and atrial bigeminy</li> <li>• Later developed sinus rhythm with second-degree heart block with paroxysms of PMVT</li> <li>• Transvenous pacing was also done but she continued to have PMVT</li> <li>• This persisted despite IV magnesium, amiodarone, and a combination of amiodarone and lidocaine</li> <li>• Patient was found to be positive for Lyme disease and started on IV ceftriaxone and underwent dual-chamber defibrillator implantation</li> </ul>

AV = atrioventricular; EF = ejection fraction; IV = intravenous; PMVT = polymorphic ventricular tachycardia; RBBB = right bundle branch block; VT = ventricular tachycardia.

### Prognosis and current guidelines for management of Lyme carditis and associated AV block

There is a high level of variability in Lyme AV block.<sup>2</sup> Furthermore, there is an increased chance of progression from first- to third-degree block when the PR interval is >300 ms in untreated Lyme disease.<sup>3</sup> When the patient presents with tachycardia, an underlying severe AV block can be masked and become evident only after progression to a higher grade. In addition, the active treatment of tachycardia may exacerbate bradycardia.

In severe cases of Lyme carditis, 2 weeks of IV ceftriaxone is recommended. However, this is due to its recommended use in Lyme neuroborreliosis. No data currently suggest that IV ceftriaxone is superior to oral medications such as doxycycline in the rate of elimination of spirochetes from the myocardium or the resolution rate of AV block.<sup>1</sup> Although the Centers for Disease Control and Prevention does recommend 2 weeks of IV ceftriaxone in severe symptomatic cases, there is a consideration (which was applied in this case) for transition to oral doxycycline when symptoms

resolve, there is no progression to higher-grade AV block, and the patient remains hemodynamically stable.<sup>1</sup>

## Limitations

### Multiple antiarrhythmic drugs were used in this case. Could this better explain the finding of the AV block?

This patient was kept on propafenone and metoprolol for weeks after discharge, and repeat ECGs confirmed gradual closure of the PR interval (Figure 2) despite continued use of both antiarrhythmic drugs. Therefore, it is doubtful that the antiarrhythmics caused the AV block. Instead, we maintain that it was due to conduction abnormalities induced by the patient's untreated Lyme disease.

### Since antibiotics, in addition to metoprolol and other antiarrhythmics, were used, did propafenone truly play a significant role?

All other antiarrhythmics used alone or in combination with each other failed to terminate this patient's AVNRT. Sinus rhythm was achieved and maintained only after the introduction of propafenone. Furthermore, it is essential to note that this was achieved before antibiotics were introduced. Hence propafenone played a significant role in stabilizing the incessant tachyarrhythmia, which could have easily led to hemodynamic instability if it was not terminated early enough.

## Conclusion

Tachyarrhythmias are very rare in Lyme carditis. We present a unique case of an incessant AVNRT masking an underlying severe AV block with a PR interval >300 ms in a patient with Lyme carditis. This case highlighted the need for extra vigilance in patients with tachyarrhythmias who test positive for Lyme disease. Furthermore, our case is one of very few where propafenone proved to be more effective than other

antiarrhythmic classes in managing Lyme-associated tachyarrhythmias. Given the high risk of decompensation, early initiation of antiarrhythmics in Lyme-associated tachyarrhythmias is recommended to achieve early rhythm control until antibiotics take effect.

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