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CASE REPORT Brief, recurrent, and spontaneous episodes of loss of consciousness in a healthy young male

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Introduction: Lyme disease is caused by bacterial spirochete Borrelia burgdorferi and is transmitted by Ixodes scapularis and Ixodes pacificus ticks, which get infected while feeding on the reservoir host of the bacteria.¹ About 248,074 cases of Lyme disease were reported by the US Centers for Disease Control and Prevention from 1992-2006.² Over 95% of these cases are reported from the Northeastern and upper Midwestern United States.³ Carditis is usually a clinical manifestation/complication of Lyme disease and is seen in approximately 5% of untreated cases.4

Case presentation: A 32-year-old male Hispanic from Chile presented with brief episodes of loss of consciousness and awareness of irregular heart beat, and denied any history of tick bite. The patient was found to have a heart rate of 40 beats per minute and fluctuating variable atrioventricular blocks. A transvenous pacemaker was placed with good capture. The diagnosis was made with serological testing and gallium scanning. Treatment with antibiotics and continuous cardiac monitoring resulted in remarkable symptomatic improvement of the patient.

Conclusion: Absence of history or evidence of tick bite must not rule out the possibility of Lyme carditis in a patient with a transient heart block. Prompt recognition of this reversible cause of heart block is essential for avoiding implantation of an unnecessary, permanent pacemaker. Keywords: Lyme carditis, transient heart block

Introduction

This is an interesting presentation of an otherwise healthy male admitted for brief episodes of loss of consciousness. Lyme disease is a multisystem disease caused by infection with B. burgdorferi.¹ Cardiac involvement occurs during the early disseminated phase of the disease, usually within weeks to a few months after infection.^{1,5} Lyme carditis is seen in about 5% of untreated cases with a male predominance of 3:1.67 The clinical features of Lyme carditis include heart block related to dysfunction of the conduction system and decreased cardiac contractility due to pericarditis.8

Case presentation

A 32-year-old otherwise healthy Hispanic man from Chile was brought in by Emergency Medical Services for multiple and brief episodes of spontaneous loss of consciousness, lasting 20–40 seconds each over the previous 2 days prior to admission. He denied shortness of breath, impaired exercise tolerance, diaphoresis, orthopnea, paroxysmal nocturnal dyspnea, headache, nausea, fever, chills, cough, sore throat, flu-like symptoms, skin rash, arthralgias, leg swelling, trauma, or seizures. However, he reported

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feeling that his heart skipped beats occasionally (irregular heartbeat) but denied chest pain and dizziness.

On further questioning, the patient admitted to have gone on a camping trip in a wooded area, but denied any tick or spider bite. Incidentally, he also recalled having been bitten by a fish on his right leg during swimming about a month ago and was treated with an antibiotic.

Social history: Patient was a furniture deliverer. Besides smoking ¹/₂ pack of cigarettes per day for 10 years, he also admitted to recent use of marijuana, cocaine, and alcohol. He was sexually active with one female partner and practiced safe sex.

Family history: Mother had hypertension, but denied any sudden cardiac death in the family.

Pertinent findings on physical examination

Vital signs: Oral temperature, 97.7°F; pulse, 45 beats per minute (bpm); blood pressure, 139/88 (sitting and standing); respiratory rate, 16 breaths per minute; and oxygen saturation by pulse oximetry was 98% while breathing room air.

Head: Normocephalic and no trauma noted. Examination of the head, eyes, ears, nose, and throat were normal.

Neck: No jugular venous distention, rigidity, or lymphadenopathy was noted.

Lungs: Bilateral fair air entry without rales or wheezes.

Heart: Bradycardia with variable first heart sound and normal S2, but no murmurs, rubs or S3 were noted.

Abdomen: Bowel sounds present, soft and nontender. No hepatomegaly or abdominal bruits noted.

Extremities: There was no edema or clubbing. No effusion or joint tenderness noted. A residual old healed scar behind the right leg was noted. Peripheral pulses were full.

Skin: No rash or ecchymoses noted.

Neurological exam: The neurological examination was nonfocal. There was no asterexis.

On arrival in the emergency department, the patient was found to have a heart rate of 40 bpm and the initial electrocardiogram showed a bifascicular block (Figure 1). The patient continued to have transient, witnessed episodes of loss of consciousness with normal and stable blood pressure. He was hospitalized for continuous cardiac monitoring that showed progression to bifascicular block and subsequently to complete heart block with ventricular escape within the first hour. Subsequently, the patient had a brief episode of asystole, lasting for 30 seconds. He was given atropine 1 mg by IV push and continued to have asystolic episodes briefly without hemodynamic compromise.



Figure I Showing bifascicular heart block.

In the first few hours of cardiac monitoring, the patient had fluctuating variable atrioventricular (AV) blocks: first degree AV block with prolonged PR interval, Wenckebach type-2, a higher degree block with 5:1 block, and a complete heart block with ventricular escape (Figure 2).

As the patient had persistent complete heart block with recurrent loss of consciousness without hemodynamic compromise, a transcutaneous pacemaker was placed and subsequently a transvenous pacemaker was placed with good capture (Figure 3).

Laboratory and other tests

Results of a complete blood cell count, serum chemistry panel, liver function profile, lipid profile, urine analysis, serum alcohol level, emergency toxicology, drugs of abuse urine, and cardiac enzymes for all normal. Erythrocyte sedimentation rate was 15, but C-reactive protein was elevated (Table 1).

Chest radiograph showed no active disease, infiltrates or effusion. A transthoracic echocardiogram showed concentric left ventricular hypertrophy with normal left ventricular systolic function without any pericardial effusion, possibly due to the strenuous nature of his work. However, it showed right ventricular enlargement and abnormal septal motion consistent with conduction abnormality. A gallium scan was performed that showed increased uptake, suggestive of myocarditis.

Of the various diagnostic markers in this patient (Table 2), screening for Lyme disease was positive with Lyme titer



Figure 2 Complete heart block with junctional escape rhythm.



Figure 3 On transvenous pacemaker.

Ab elevated at 2.25. The diagnosis of Lyme carditis was confirmed on the 5th day when the Western blot results were found to be positive for Lyme disease.

The patient was started on intravenous ceftriaxone 2 gm/day with continuous cardiac monitoring. Subsequently, patient symptoms improved from complete block to 2:1 AV block, and eventually to sinus bradycardia 1st degree block. However, within 3 days of treatment with ceftriaxone, the patient had a remarkable improvement in symptoms and remained completely asymptomatic

After 14 days of intravenous ceftriaxone, continuous cardiac monitoring of the patient showed remarkable improvement with sinus bradycardia at 50 bpm with 1st degree AV block without any further episodes of asystole or complete heart block, or any symptoms of loss of consciousness (Figure 4).

The patient was discharged home on amoxicillin 500 mg PO tid for another 4 weeks. During subsequent followups at 1 month and 3 months after the antibiotic treatment, the patient showed normal sinus rhythm with 1st degree AV block.

Discussion

In this patient, the absence of any risk factors and the absence of any previous history of symptoms for coronary artery disease together with an active lifestyle made myocardial infarction unlikely. Neither did his history suggest the viral illness. The negative antistreptolysin O titers and the absence of any other associated symptoms ruled out acute rheumatic



Figure 4 Post treatment electrocardiograph on discharge with first degree AV block.

fever. As the patient was from Chile, Chagas disease was considered and was ruled out with negative *Trypanosoma cruzi* immunoglobin G antibody. Further serological markers for other microorganisms and diseases were all negative, including antinuclear antibody and rapid plasma reagin (Table 2). The patient had no other findings consistent with infiltrative heart disease although the patient admitted to using alcohol.

US Centers for Disease Control and Prevention (CDC) recommends a two step testing process for the diagnosis of Lyme disease, with the first step being the enzyme linked immunosorbent assay test (ELISA), which is highly sensitive, followed by Western blot to confirm the positive titres.^{7,9,10} Diffuse, intense uptake on gallium myocardial scan is suggestive of myocarditis.^{11,12}

Key features of Lyme carditis

In the United States, Lyme disease, caused by the spirochete *B. burgdorferi*, is transmitted mainly by *Ixodes scapularis* (also called *Ixodes dammini*) ticks.^{1,4,13} It is a multisystemic process with dermatologic annular rash of *Erythema chronicum migrans*, asymmetric oligoarthritis involving large joints, neurological, and cardiac manifestations (Table 4).^{1,12} About 5% of patients develop cardiac manifestations within several weeks to three months of spirochete transmission.³ The signs and symptoms are syncope or dizziness, shortness of breath, substernal chest pain, or palpitations.^{12,14} It also rarely presents with the symptoms of seizures with bradycardia.^{1,15,16}

Lyme disease is associated with serious cardiac rhythms and all degrees of AV blocks (Table 3).^{17,18} However, heart block may be the first manifestation of Lyme carditis and commonly presents with marked sinus bradycardia with 1st degree AV block in untreated patients.^{7,8} The other cardiac manifestations are transient complete heart block with ventricular escape, sinus bradycardia with 2:1 AV conduction, bifascicular block with tachycardia, and rarely with irreversible complete heart block.^{19,20} In this case, we report Lyme carditis presenting as recurrent syncope with sinus bradycardia that progressed bifascicular block and subsequently complete heart block with ventricular escape, within minutes.

Though cardiac conduction abnormalities associated with Lyme disease are generally self-limited, treatment choice depends on the severity of the block and symptoms. While patients with minor first-degree AV block but with PR interval less than 0.30 seconds can usually be treated with doxycycline or amoxicillin, those with severe cardiac Table I Laboratory values on the day of admission

White cell count	10.8	(4.8–10.8 k/cmm)		
Neutrophils	82.4% (42.2–75.2%)			
Lymphocytes	10.7%	(20.0–51.0%)		
Basophils	0.2%	(0.0–0.8%)		
Monocytes	6.2%	(1.7–9.3%)		
Eosinophils	0.5%	(0–5%)		
Hematocrit	39	(34–44%)		
Platelets	257	(130–400 k/cmm)		
Mean corpuscular volume	88	(79–96 fl)		
Prothrombin time (sec)	12	(9.1–11.6 sec)		
Partial-thromboplastin time (sec)	28.2	(24.3-33.8 sec)		
Sodium	146	(136–145 mmol/L)		
Potassium	3.8	(3.5–5.1 mmol/L)		
Chloride	110	(98–107 mmol/L)		
Bicarbonate	22	(22–29 mmol/L)		
Urea nitrogen	10	(7–26 mg/dL)		
Creatinine	I	(0.6–1.1 mg/dL)		
Glucose	127	(60–140 mg/dL)		
Calcium	9.2	(8.4–10.2 mg/dL)		
Magnesium	1.9	(1.6–2.6 mg/dL)		
Phosphorus	2.7	(2.3–4.7 mg/dL)		
Cholesterol (mg/dL)				
Total	125	(<200 mg/dL)		
Triglyceride	68	(45–155 mg/dL)		
High-density lipoprotein	46	(30–70 mg/dL)		
Low-density lipoprotein	66	(65–180 mg/dL)		
Aspartate aminotransferase (U/liter)	40	(8–34 U/L)		
Alanine aminotransferase	62	(6–55 U/L)		
Alkaline phosphatase	219	(40–150 U/L)		
Albumin	4.8	(3.5–5.0 g/dL)		
Total protein	71	(6.4–8.3 g/dL)		
Bilirubin, total (conjugated)	0.6	(0.2–1.2 mg/dL)		
Lactate dehydrogenase	563	(56–194 IU/L)		
Thyroid stimulating hormone	0.9	(0.25–4.30)		
Creatinine kinase	145; 123; 127	(29–168 U/L)		
CKMB/Interpretation	1.7/-; 1.8/-; 1.4/- (<10.4)			
Emergency toxicology	Negative			
Drugs of abuse urine	Negative			
Erythrocyte sedimentation rate	15	(I–I3 mm/hr)		
C-reactive protein	Reactive (1:2)	· /		
Urinalysis	Normal			

involvement with first degree AV block with PR interval greater than 0.30 seconds, or higher degree blocks including third degree block, should be treated with intravenous ceftriaxone of 2 gm/day for 14–21 days.^{7,12,21–23} We started our patient on intravenous ceftriaxone 2 gm/day with continuous cardiac monitoring. Subsequently, patient symptoms improved from complete block to 2:1 AV block and eventually to sinus bradycardia 1st degree block. However, within 3 days of treatment with ceftriaxone, the patient had remarkable improvement in symptoms and remained completely asymptomatic.

Though there are cases of irreversible complete heart block despite treatment with appropriate antibiotics and steroids, most patients have complete symptom recovery with the common electrocardiographic (EKG) manifestation of bradycardia with 1st degree AV block.^{7,14}

Conclusions

The patient's syndrome of recurrent episodes of syncope was a complication of Lyme carditis. Complete block accompanying syncope was likely exacerbated by myocarditis as evidenced by gallium scan. The astute clinician must consider Lyme carditis in patients with higher degrees of AV blocks and/or complete block, as it is associated with serious rhythm disturbances, which may otherwise lead to unnecessary procedures such as placement of a permanent pacemaker.

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Table 2 Serology evaluation

	Negative
	Negative
	Negative
	Negative (<1.1)
	Positive
	2.25 (0.00-0.99)
18 KD	Reactive
23 KD	Reactive
39 KD	Reactive
41 KD	Reactive
45 KD	Reactive
58 KD	Reactive
	18 KD 23 KD 39 KD 41 KD 45 KD 58 KD

Though the diagnosis is based on history, symptoms, exposure to ticks, physical findings, and serological testing, it is worthwhile for patients such as this man, who was otherwise healthy except for a bite of unknown source, to start intravenous ceftriaxone or penicillin empirically, even if their history is not strongly suggestive of Lyme disease.

Lyme disease should be confirmed by a positive result on a 2-step antibody test of an ELISA and Western blotting for *B. burgdorferi* antibodies. Though systemic steroids were

Table 3 Causes of	acquired	atrioventricular	bloc
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Coronary artery disease	
Coronary vasospasm	
Drugs	
Amiodarone	
Beta-blockers	
Calcium channel blockers	
Class IC antiarrrhythmic agents	
Digoxin	
Procainamide	
Infiltrative myocardial diseases	
Amyloidosis	
Hemochromatosis	
Sarcoidosis	
Tumors (primary or metastatic to heart)	
Arrhythomogenic right ventricular dysplasia	
Collagen vascular diseases	
Systemic Lupus Erythomatosis (SLE)	
Rheumatoid arthritis	
Scleroderma	
Ankylosing spondylitis	
Infectious disease	
Acute rheumatic fever	
Toxoplasmosis	
Trypanosomiasis (Chagas disease)	
Bacterial endocarditis	
Viral myocarditis	
Syphilis	
Lyme disease	

Table 4 Cardiac manifestations of Lyme disease

lachyarrhythmias	
Atrial: probably due to pericarditis	
Ventricular: uncommon and usually as an escape rhythm	
Myopericarditis	
Mild myocardial dysfunction	
Chronic congestive cardiomyopathy	
Conduction defect	
First degree heart block	
Second degree heart block	
Complete heart block	
Bundle branch block	
Fascicular block	
Asystole (rare)	
Nonspecific S-T and T-wave abnormalities	
Pericarditis	
Fulminant congestive heart failure	
	-

also used for symptomatic high-grade heart block in Lyme disease, intravenous antibiotic therapy with penicillin or ceftriaxone is recommended and should be monitored closely for irreversible complete heart block as it may occasionally occur. When confronted with an unexplained higher AV degree blocks or complete block in a healthy person without hemodynamic instability, Lyme carditis should be strongly considered before embarking upon permanent pacemakers as asymptomatic disease is more common than symptomatic disease.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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