Case Review

Pericarditis as a manifestation of Lyme disease

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A 69-year-old man arrived at the emergency department at 5 AM after two episodes of syncope at home. The paramedics reported that the patient was found on the kitchen floor conscious, alert, and oriented to time, person, and place. His wife reported that he had "passed out" twice since midnight, both episodes lasting 20 to 30 seconds. No seizure activity, incontinence, or respiratory distress occurred during these episodes.

The patient did not recall the incidents; he denied chest pain and shortness of breath. His only symptom was "pain in his esophagus," which he had been having for several weeks. He denied nausea, vomiting, diarrhea, or dark, tarry stools.

Before he arrived at the hospital an IV infusion line was inserted with a large-bore needle and he received high-flow oxygen by nonrebreather mask at 10 L/min. No baseline pulse oximetry reading was obtained. The patient had hypotension with a blood pressure of 80/30 mm Hg and he had a heart rate of 56 to 60 beats/min. A fluid bolus of 250 ml normal saline solution was given en route to the hospital. The patient remained alert and pain free during transport.

When the patient arrived at the hospital, he was alert and oriented to time, person, and place, and had good skin color; he denied pain. His vital signs were as follows: tympanic temperature 98.8° F; pulse 59 beats/min; respirations 22 breaths/min; blood pressure was 112/62 mm Hg in the left arm and 109/57 mm Hg in the right arm. The cardiac monitor showed sinus bradycardia with short episodes of junctional rhythm (Figure 1). Pulse oximetry was 100% on 100% oxygen by nonrebreather mask.

The patient stated that tonight his "esophagus pain" had become unbearable, occurring with each pulsation of his heart; the pain increased at rest. He took two aspirin and shortly afterward he became nauseated and diaphoretic, lost consciousness, and fell to the floor.

His medical history included a heart murmur, recently diagnosed left lower lobe pneumonia, and Lyme disease (LD); there was no prior history of other heart disease or hypertension. Lyme disease had been diagnosed 1 week earlier; the patient was currently taking clarithromycin 500 mg every 12 hours; he denied having any medication allergies. He also denied use of tobacco products and reported only occasional alcohol use, none within the last month.

Physical examination showed an alert, elderly man. His head was normocephalic and showed no signs of trauma. Pupils were equal, round, and reactive to light. No jugular vein distention was noted with the head elevated to 45 degrees. Heart rate was regular with a grade II/VI systolic murmur; the heartbeat was heard best at the apex. The lungs were clear to auscultation except for rales at the base of the left lung. The abdomen was soft, nontender, and nondistended with positive bowel sounds. Extremities were not cyanotic or edematous and all peripheral pulses were good.

The patient received aggressive antibiotic therapy with ceftriaxone sodium. The ST elevations in his EKG returned to normal with treatment.

Nursing diagnoses of alteration in comfort related to esophageal pain, and alteration of safety related to syncopal episodes were assigned to the patient. The plan of care included elevation of both siderails for safety, and oxygen delivery by nasal cannula at 4 L/min. This was tolerated well with subsequent pulse oximetry readings of 100%.

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Table 1
Clinical manifestations of Lyme disease*

	Early localized disease	Early disseminated disease*	Late disease*
Occurrence	Few days to a month after tick bite	Days to 10 months after tick bite	Months to years after tick bite
Signs and symptoms	Erythema migrans Fatigue/malaise/lethargy Headache Myalgia Arthralgias Regional/generalized lymphadenopathy	Carditis (8 to 10% of patients who are not treated) Conduction defects Mild cardiomyopathy Neurologic disease (10% to 12% of patients who are not treated) Meningitis Encephalitis Cranial neuropathy, usually facial Peripheral neuropathy/radiculopathy Myelitis Musculoskeletal (50% of patients who are not treated) Migratory polyarthritis and/or polyarthralgias Fibromyalgia Skin — lymphocytoma, erythema nodosum Lymphadenopathy — regional and/or generalized Eye — conjunctivitis, iritis, choroiditis, vitritis, retinitis Liver — liver function test abnormalities, hepatitis Kidney — microhematuria, proteinuria	Musculoskeletal Migratory polyarthritis will develop in 50% of patients who are not treated Chronic monoarthritis, usually of the knee, will develop in 10% of patients who are not treated Fibromyalgia Neurologic disease Chronic, often subtle, encephalopathy and/or peripheral neuropathy Ataxia Dementia Sleep disorder Cutaneous Acrodermatitis chronica atrophans (Europe)

These entities may develop without previous symptoms of Lyme disease; alternate explanations for the symptoms should be ruled out.
*Data from Siegel LH, Academy of Medicine of New Jersey. Lyme disease in New Jersey: a practical guide for New Jersey clinicians. (Sect. X) 1993. p. 2.

The patient's first 12-lead EKG showed diffuse ST elevations in all leads (Figure 1). The second, performed 1 hour later, showed the same ST elevations but with a shortened PR interval.

LD is a multisystem, inflammatory illness caused by infection with the spirochete *Borrelia burgdorferi* and transmitted by the bite of an infected tick.

Because of the diffuse ST elevations and the patient's history of two syncopal episodes preceded by nausea and diaphoresis, IV nitroglycerin 50 mg/250 ml 5% dextrose and water was started at 3 ml/hr. An IV bolus of heparin, 5600 units, was given.

Laboratory studies ordered included complete blood cell count with differential, chemistry 12 profile, prothrombin and partial thromboplastin times, cardiac enzyme levels, and isoenzyme concentrations. All results were within normal limits including a creatine phosphokinase <20 IU/L. Portable radiography showed a left lower lobe pleural effusion, without evidence of pericardial effusion. After the patient was seen by the cardiologist, nitroglycerin therapy was discontinued and he was admitted to the ICU with a diagnosis of pericarditis and a left pleural effusion. It was suspected that the diagnosis was related to his known LD.

Discussion

The patient's stay in the emergency department, which was just over 2 hours, was uneventful. Early interventions were consistent with treatment for myocardial infarction because of the history of two syncopal episodes preceded by nausea and diaphoresis, and because he had hypotension and bradycardia. His esophageal pain was suspected to be referred pain in relation to a cardiac condition. When results of the patient's cardiac enzyme studies

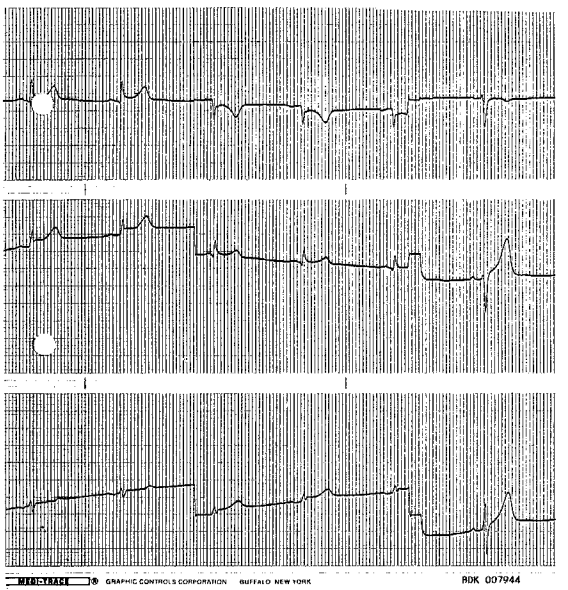

Figure 1
Sinus bradycardia, normal PR interval, diffuse ST segment elevation suggestive of pericarditis. Previous electrocardiogram is within normal limits with no evidence of early repolarization.

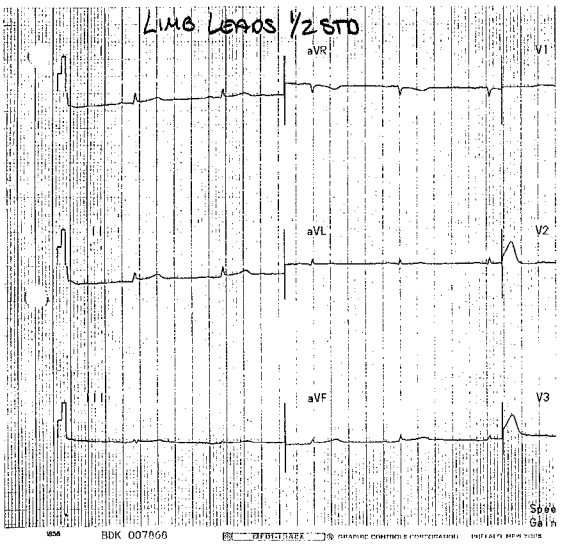

Figure 2
Sinus rhythm, normal axis, low voltage over limb leads, nonspecific ST wave abnormalities. Compared with Figure 1, voltage over limb leads is lower.

were negative but he still had ST elevations in all EKG leads, the focus changed to pericarditis.

EKG abnormalities consistent with pericarditis are diffuse ST segment elevations; they are often confused with myocardial ischemia. This rhythm may be accompanied by PR interval depressions. Clinical manifestations can include a biphasic or triphasic pericardial rub, which this patient did not have. Cardiac tamponade was ruled out by echocardiogram. The patient did have hypotension and bradycardia but did not have any of the other associated symptoms such as jugular vein distention, paradoxical pulse, or an enlarged heart.

The patient received aggressive antibiotic therapy with ceftriaxone sodium. The ST elevations in his EKG returned to normal with treatment (Figure 2). His esophageal pain occurred when the patient had a junctional rhythm with retrograde conduction into the atrium causing pressure on the esophagus. His condition steadily improved, and he was discharged home with outpatient follow-up.

Lyme disease: An overview

Hunterdon County is a rural county in west central New Jersey with a population of approximately 115,000 people. A 1996 unpublished report from the Centers for Disease Control and Prevention ranked Hunterdon County third in the United States for LD cases, with Nantucket County, Massachusettes, ranked first and Dutchess County, New York second. Hunterdon's case rate was 530 cases per 100,000 population (Figure 3). LD has been reported in 47 of the 50 states but it occurs in the greatest numbers in the Northeastern, North Central, and Mid-Atlantic regions. In this area, 80% of the cases occur between May and August, with the highest incidence in July. Although LD is now the most common arthropod-borne illness in the United States with approximately 12,000 to 16,000 new cases each year,[2] diagnosis can be challenging because of the variability of its manifestations and the lack of identified marker proteins to define the patient's stage of disease.

> These patients appear to be more ill, with high fever, severe headache, stiff neck, significant arthralgias, and fatigue.

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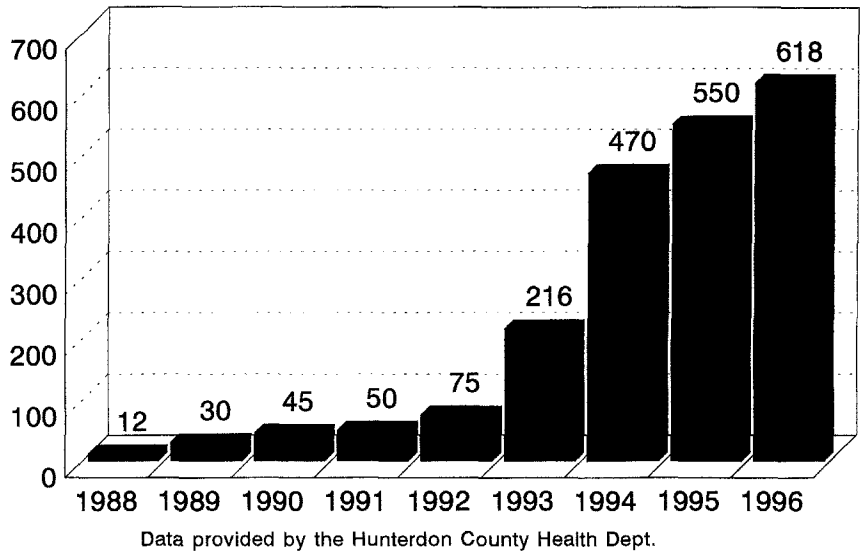

Figure 3
Reported Lyme disease cases in Hunterdon County, 1988-1996.

and transmitted by the bite of an infected tick. It frequently appears as a localized skin lesion but in its disseminated form may affect the joints, cardiovascular, and nervous systems. Person-to-person transmission does not occur. Although transplacental transmission has been documented, adverse fetal effects are thought to occur rarely, if ever.

Clinical illness can be categorized as early localized disease, early disseminated disease, and late disease, although considerable overlap exists among categories. Early localized disease is characterized by the appearance of the typical LD rash, erythema migrans, an expanding erythematous rash that often occurs at the tick bite site 2 to 30 days after exposure. Early localized disease is usually cured within 3 to 4 weeks of oral antibiotic therapy.

Early disseminated disease is associated with hematogenous spread to other body sites. These patients appear to be more ill, with high fever, severe headache, stiff neck, significant arthralgias, and fatigue. In approximately 4% to 10% of patients who are not treated, Lyme carditis develops[3]; this may be the first symptom of the disease. Patients often have shortness of breath, lightheadedness, palpitations, or chest pain. Symptoms generally reflect a mild myocarditis with variable degrees of atrioventricular node conduction defects; 50% of patients have reversible complete heart block. Intravenous antibiotics are indicated and a temporary cardiac pacemaker is sometimes required.

Approximately 10% to 15% of patients who are not treated have signs of neurologic involvement with LD[4]; in those who are treated this is a rare event. Symptoms usually occur 2 to 3 months after onset of the Lyme rash and may include facial nerve palsy, headache, mild neck stiffness, photophobia, and a mild encephalopathy characterized by memory and concentration problems. Radiculopathies are sometimes seen. Treatment consists of IV antibiotic therapy for 3 to 4 weeks unless the only symptom of neurologic involvement is facial nerve palsy and the cerebrospinal fluid reveals no inflammatory changes; then oral antibiotics may be used.

A small percentage of people with LD may experience a clinical syndrome called tertiary neuroborreliosis; its symptoms include progressive encephalomyelopathy, polyneuritis, and mental or psychiatric changes.[5] Others report continued articular disease months or years after treatment of the disease. Ophthalmologic disease has been described with conjunctivitis, corneal infiltrations, and acute visual impairment or loss.[6] Because LD is a clinical diagnosis, with laboratory testing used for confirmation only,[7] it is important that the diagnosis is accurate and that other causes of these conditions are ruled out.

Conclusion

LD is currently classified by the Centers for Disease Control and Prevention as one of several "emerging" infectious diseases. In some areas it has *emerged* more fully than in others. In some areas, LD

has only recently been recognized; in others, LD is already endemic and so are fears reflecting common misconceptions.

Points to remember about LD:

- All but a very small percentage of LD cases are cured within 3 to 4 weeks by use of oral antibiotic therapy. Reassure your patients that they can expect cure without recurrence.
- Diagnostic laboratory tests should never be used for screening purposes. When used in this setting, currently available Lyme tests are both sensitive and specific. Help your patients understand this when they want a "Lyme test" for tick bites or without specific symptoms.
- Prevention techniques are simple and effective. Teach your patients to wear light-colored clothing, consider using tick repellents on clothes and skin, and check themselves and their children for ticks and the Lyme rash after exposure to high-risk areas.

LD is most often a recognizable, treatable disease. Emergency nurses can make a difference by providing education, which is important in this emerging infection.

References

1. Sigal LH, Academy of Medicine of New Jersey. Lyme disease in New Jersey: a practical guide for New Jersey clinicians. (Sect. X). Lawrenceville (NJ): Academy of Medicine of New Jersey; 1993. p. 2.
2. Massachusetts Medical Society. Lyme disease—United States, 1995. MMWR 1996;45:481-4.
3. Sigal LH. Early disseminated Lyme disease: cardiac manifestations. Am J Med 1995;98(Suppl 4A):25-9.
4. Sigal LH, Academy of Medicine of New Jersey. Early neurologic manifestations—clinical features. In: Lyme disease in New Jersey: a practical guide for New Jersey clinicians. (Sect. IV) Lawrenceville (NJ): Academy of Medicine of New Jersey; 1995. p. 5.
5. Sigal LH, Academy of Medicine of New Jersey. Tertiary neuroborreliosis—clinical features. Lyme disease in New Jersey: a practical guide for New Jersey clinicians. (Sect. IV) Lawrenceville (NJ): Academy of Medicine of New Jersey; 1993. p. 9.
6. Sigel LH, Academy of Medicine of New Jersey. Other clinical problems thought to be part of Lyme disease. In: Lyme disease in New Jersey: a practical guide for New Jersey clinicians. (Sect. IV) Lawrenceville (NJ): Academy of Medicine of New Jersey; 1993. p. 10.
7. Schoen RT. Identification of Lyme disease. Diagnostic Issues 1994;20:361, 363, 365, 367, 369.

This section features actual emergency situations with particular educational value for the emergency nurse. Contributions (four to six typed, double-spaced pages) should include a case summary focused on the emergency care phase, accompanied by pertinent case commentary. Submit to ***Patty Campbell,*** *Section Editor, c/o Managing Editor, 216 Higgins Rd., Park Ridge, IL 60068-5736; phone (847) 698-9400; E-mail: khalm@ena.org.*