Acute Heart Failure From Lyme Carditis

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Carditis can complicate Lyme disease in an estimated <5% of cases, but cardiogenic shock is rare. We report a case of severe biventricular heart failure as a manifestation of a Jarisch-Herxheimer reaction in a patient with early Lyme disease following treatment with ceftriaxone.

Case Report
A 47-year-old woman presented with subacute fever, erythema migrans-like rash, shortness of breath, and spells of dizziness that had progressed to “blackouts.” At admission, complete atrioventricular (AV) block was present (Figure 1A), and ceftriaxone 1g was given initially for suspected Lyme disease. Within 6 hours of antimicrobial treatment, she developed polymorphic ventricular tachycardia (Figure 1B). She was resuscitated with intravenous magnesium, a 150 mg bolus of amiodarone, and defibrillation. Once stabilized, echocardiography revealed severe biventricular heart failure with left ventricular ejection fraction (LVEF) of 10%. Inotrope support was initiated. On hospital day 2, coronary angiography demonstrated patent epicardial coronary arteries, and endomyocardial biopsy revealed diffuse lymphocytic myocarditis (Figure 2). On day 3, methylprednisolone 1000 mg was administered daily for 3 days, followed by a 12-day taper of oral prednisone. An underlying sinus rhythm was present, but third-degree AV block persisted with intermittent 3 to 4 second sinus pauses, requiring placement of a temporary transvenous pacing wire. In a few instances, these had transformed into ventricular tachycardia and ventricular flutter (Figure 1C). Intermittent failure of the temporary pacemaker to sense and capture was evident and requiring dilated cardiomyopathy. Among these patients, earlier ceftriaxone treatment may have been associated with complete recovery or improved LVEF, but the role of steroids remains unclear.

This patient’s clinical course and treatment brought to question what caused her disease; specifically, what is the contribution of foreign bacteria versus the immune response? Her sudden decompensation within hours of antimicrobial therapy, followed by 2 to 3 days of ongoing fevers, increasing CRP, and difficulty with transvenous pacing, all suggested a Jarisch-Herxheimer reaction after initi-
Antigen release triggered the inflammatory response, the putative pathological entity driving the “disease” state of the patient’s arrhythmias and cardiogenic shock. Corticosteroids temporally were associated with substantial clinical improvement, cessation of life-threatening arrhythmias, marked decline in CRP, and restoration of near-normal LVEF.

**Conclusion**

Severe biventricular failure is a rarely described manifestation of Lyme infection, which in this case was presumably precipitated by a Jarisch-Herxheimer reaction. Implementation of steroids should be considered as adjunctive therapy if CRP acutely rises after initiation of antimicrobial treatment of Lyme carditis. The use of CRP as a diagnostic therapeutic

**Figure 1.** A, ECG at admission showing complete heart block. B, Telemetry demonstrating regression from third-degree atrioventricular block to polymorphic ventricular tachycardia (VT) as the patient began reporting presyncopal symptoms. C, During the first 5 hospital days, the patient had several transformations into polymorphic VT that required pacing. This rhythm strip demonstrates ventricular flutter that occurred on hospital day 3.

**Figure 2.** Endomyocardial biopsy. The tissue sample demonstrates active lymphocytic myocarditis without evidence of giant cell myocarditis or sarcoidosis (hematoxylin-eosin stain, original magnification 200×). Additional stains for fungus and acid-fast bacilli were negative.
Figure 3. Treatment time course in relation to serum C-reactive protein: 10% (echo), 45% (echo), and 61% (cardiac magnetic resonance).

marker for indicating the role of corticosteroids has not been described and should be investigated further in myocarditis.

Disclosures
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References

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