Disseminated Lyme disease with a herpetiform center

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INTRODUCTION

Every year, nearly half a million Americans are diagnosed and treated for Lyme disease,[1] with the overwhelming majority of cases from the Northeast. Lyme disease is the most common tick-borne disease and is caused by the spirochete *Borrelia* (*B. burgdorferi*, *B. afzelii,* or *B. garinii*) and transmitted by the *Ixodes* tick. Clinically the disease is subdivided into the following 3 phases: early localized, early disseminated, and late disease. Early localized disease occurs within days to weeks after exposure and is characterized by erythema migrans (EM) at the site of tick bite with or without systemic symptoms. Similarly, early disseminated disease occurs in the same timeframe but with multiple EM lesions and associated neurologic (eg, facial nerve palsy) and cardiac features (eg, atrioventricular block). Late Lyme disease often manifests months to years after the initial untreated infection with chronic arthritis as a defining feature.[2]

EM is pathognomonic for Lyme disease and is classically a targetoid, "bull's eye", red macule or plaque at the site of the tick bite. EM may not always present with an area of central clearing, but as the rash expands, the area of central pallor may become more visible. Although EM is usually painless, painful and burning lesions have also been described.[3] Atypical variants of EM include hemorrhagic, vesiculobullous, and necrotic subtypes,[4] which are rare but important to recognize to initiate prompt treatment. Here we describe a patient with a rare variant of Lyme disease with a herpetiform center that should be distinguished from other vesicular skin conditions.

Abbreviation used:

EM: erythema migrans

CASE REPORT

A woman in her 30s with no significant medical history presented to her primary care provider with a small red "bump" and surrounding swelling on her back. She also noted having a rash in her left axilla with an ipsilateral swollen lymph node. Due to concern for cellulitis, she was started on cephalexin. Her symptoms progressed the next day despite 3 doses of cephalexin, with larger, warm, and more painful bumps on her back with increased swelling, prompting her to visit the emergency department. Additional symptoms included weeping and crusting of the bumps with the worst pain in the center of the targetoid lesion. Exposure history revealed that she had been hiking in the Northeast region a few days before symptom onset, but she denied seeing any ticks. She also denied exposure to poison ivy, chemical agents, or other irritants. She had never had a similar rash in the past but did tend to have exuberant reactions to bug bites. Additional systemic symptoms included mild cough, bilateral hip pain, and headache. Review of symptoms was otherwise negative.

Physical examination revealed normal vital signs and a 2-cm soft, tender, mobile lymph node in the left anterior axillary fold with no other lymphadenopathy. Skin examination showed a large, circular, well-demarcated erythematous plaque on the back with a central cluster of herpetiform vesicles, bullae, and purple necrotic puncta (Fig 1), as well as erythematous patches on the anterior aspect of the chest and left lateral aspect of the breast. Initial laboratory evaluation revealed an elevated erythrocyte sedimentation rate (25 mm/hr) and C-reactive protein

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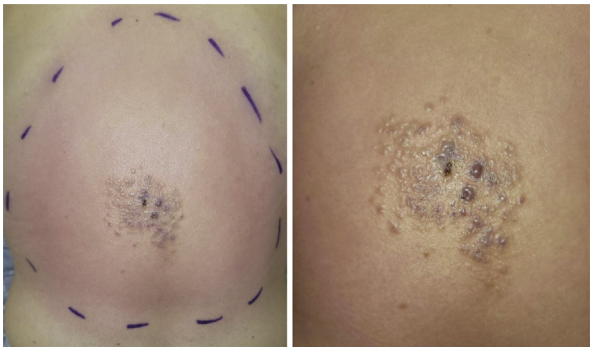

Fig 1. Initial presentation.

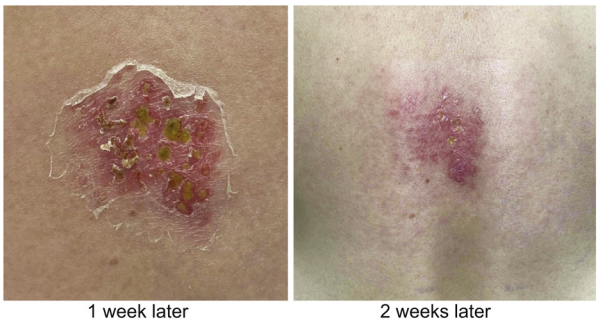

Fig 2. Post-doxycycline treatment.

(174 mg/L), while the complete blood count with differential was unremarkable. Bacterial wound cultures and blood cultures showed no growth. Herpes simplex virus, varicella-zoster virus, and *Ehrlichia/Anaplasma* polymerase chain reactions were negative. There was no evidence of malaria or babesiosis on Giemsa blood smears. Lyme immunoglobulin M and G antibodies were not detected.

In the emergency department, she was initially treated with cefazolin and doxycycline but was given vancomycin and clindamycin after developing a fever of 101 °F with tachycardia and hypotension. After consultation and evaluation by Dermatology, her cutaneous presentation was felt to be consistent with vesiculobullous EM, with her systemic symptoms following doxycycline attributed to a Jarisch-Herxheimer reaction[5] in the setting of early disseminated Lyme disease. Once she became hemodynamically stable, she was discharged on a 14-day course of doxycycline (100 mg twice daily) with outpatient follow-up. After 1 week, the center of the rash on her back had largely crusted over, and the surrounding erythema had largely subsided (Fig 2). She denied any local pain or pruritus, and her fever, lymphadenopathy, and joint pain had resolved. She ultimately completed the full course of doxycycline with complete resolution. Notably, a repeat Lyme antibody screen 6 weeks after the onset of symptoms was positive and confirmed by Western blot.

DISCUSSION

This case showcases early disseminated Lyme disease with a herpetiform center, a rare cutaneous presentation, as well as an example of Jarisch-Herxheimer reaction in response to initiating treatment of Lyme. Vesiculobullous Lyme may be more common than previously thought, representing 8% of Lyme cases in a study of one diagnostic center in New York.[6] Given the low sensitivity of serologic testing during early phases of disease manifestation, Lyme disease is often diagnosed clinically by history and skin examination. While classic EM with central clearing is more recognizable, it is important to keep Lyme on the differential for atypical presentations of EM with central necrosis, hemorrhagic crusting, or vesiculobullous changes as observed in this case.[7] Early diagnosis and treatment of Lyme are key to preventing more severe late stage neurologic and cardiac complications, and hence it is important that clinicians remain aware of this rare variant.

Conflicts of interest

None disclosed.

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