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Unusual Presentation of Chikungunya Virus Infection With Concomitant Erysipelas in a Returning Traveler From the Caribbean: A Case Report

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Chikungunya fever is a mosquito-borne febrile illness caused by Chikungunya virus (CHIKV), an alphavirus from the Togaviridae family. It is transmitted by primarily Aedes aegypti and Aedes albopictus mosquitoes [1]. Once of little importance in the Americas, local transmission was identified in the Caribbean in late 2013. More than 1000 travelers returning to the continental United States have been diagnosed with CHIKV. More importantly, there have been 9 documented cases of autochthonous disease in Florida as of September 16, 2014 [2].

CASE

A 19-year-old previously healthy female presented to our hospital in early June 2014 for evaluation of fever and a rash that started 4 days before admission during a mission trip to Haiti. Her symptoms were fever and a painful erythematous lesion on her right arm. An aspiration was attempted at a medical facility in Haiti, and antibiotics were prescribed without improvement. There is no documentation that blood cultures were obtained in Haiti. She returned to the United States 4 days after the onset of her fever and described right upper quadrant pain and intermittent right-sided pleurisy. She did not have myalgias or arthralgias. She was taking chloroquine for the prevention of malaria.

Initial vital signs were all within normal range. Her exam was remarkable for erysipelas of the right arm (Figure 1A) and associated tender axillary adenopathy. The right arm erysipelas improved within 48 hours of receiving cefazolin. However, on hospital day 2, she developed recurrent fevers with shortness of breath and hypoxia. Contrast-enhanced pulmonary embolus protocol computerized tomography was negative for embolus but showed evidence of small bilateral pleural effusions and

Figure 1. A, Erysipelas of right arm. B, Maculopapular rash over anterior chest.
Table 1. Patient’s Laboratory Results During Hospital Course

<table>
<thead>
<tr>
<th></th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>White cell count (per mm³)</td>
<td>19.1</td>
<td>11.1</td>
<td>9.8</td>
</tr>
<tr>
<td>Absolute neutrophil count (per mm³)</td>
<td>16.7</td>
<td>Not available</td>
<td>6.9</td>
</tr>
<tr>
<td>Lymphocyte count (per mm³)</td>
<td>1.01</td>
<td>Not available</td>
<td>1.79</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>10.0</td>
<td>8.6</td>
<td>9.0</td>
</tr>
<tr>
<td>Platelets (per mm³)</td>
<td>199</td>
<td>165</td>
<td>199</td>
</tr>
<tr>
<td>Aspartate aminotransferase (U/L)</td>
<td>23</td>
<td>Not available</td>
<td>53</td>
</tr>
<tr>
<td>Alanine aminotransferase (U/L)</td>
<td>13</td>
<td>Not available</td>
<td>19</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>81</td>
<td>Not available</td>
<td>108</td>
</tr>
<tr>
<td>Serum albumin (g/dL)</td>
<td>3.3</td>
<td>Not available</td>
<td>2.4</td>
</tr>
<tr>
<td>Iron (μg/dL)</td>
<td>19</td>
<td>Not available</td>
<td>Not available</td>
</tr>
<tr>
<td>Urine protein/creatinine (g/g)</td>
<td>0.14</td>
<td></td>
<td></td>
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</tbody>
</table>

pulmonary edema. Serum samples were sent to the Centers for Disease Control and Prevention for arbovirus testing on hospital day 4 (and 8 days into illness) after she developed a diffuse maculopapular blanching rash (Figure 1B). Chikungunya virus polymerase chain reaction (PCR) was positive and dengue immunoglobulin (Ig)M was negative. A reverse transcription (RT)-PCR for dengue fever was not performed. Routine laboratory tests are summarized in Table 1.

DISCUSSION

Chikungunya virus has been known to cause human disease since the 1950s, although it did not reach international attention until a 2004 outbreak in Kenya. This outbreak was followed by several outbreaks in several islands in the Indian Ocean as well as India [3]. Of note, autochthonous transmission of CHIKV has been reported in Europe, which raises concern for the potential for geographic dispersion of CHIKV to temperate climates [4, 5].

As of May 30, 2014, a total of 103,018 suspected and 4,406 laboratory-confirmed CHIKV infections were reported due to autochthonous transmission of CHIKV in the Americas [6]. It is estimated that more than 1 million travelers returned to the continental United States (CONUS) from areas with ongoing transmission between May and July of 2014, many to cities where Aedes spp are present [7]. Subsequently, 9 cases of autochthonous transmission of CHIKV have been documented in Florida [2]. Thus, it is paramount for infectious diseases physicians to become acquainted with varied presentations of Chikungunya fever.

The incubation time for CHIKV is 1 to 12 days. Borgherini et al [8] evaluated the clinical features of 157 patients with laboratory-confirmed disease: arthralgia was present in 96.1% of patients, fever was present in 89% of patients, and rash was present in 40.1% of patients. The rash is usually maculopapular, transient, and tends to affect torso, limbs, and face [9]. In almost all cases, multiple joints are involved and arthralgias tend to be symmetric. Although joint edema has been reported in up to 50% of the cases, other inflammatory signs are usually absent [8, 9]. Chronic arthralgia is the most disabling consequence of infection. The frequency and predictive factors for this complication are subject to debate because studies have substantial differences in methodology. However, older age seems to be predictive for developing this complication [10, 11]. Several atypical manifestations of CHIKV, including end-organ damage, have been reported. The direct contribution of CHIKV to these acute complications is unclear. Most of these patients have underlying medical conditions, whereas our patient had no pre-morbid disease [12]. To our knowledge, capillary leak has not been reported with acute CHIKV, but it is a common manifestation of dengue fever. Hypoalbuminemia and proteinuria are hallmarks of endothelial dysfunction in dengue fever [13]. Our patient developed hypoalbuminemia but did not develop proteinuria. However, given the fact that the patient had a negative fluid balance of 0.5 L after the first 2 days of hospitalization (2.9 L given in total), iatrogenic fluid overload was an unlikely cause of her pulmonary complications. This result suggests that she did have an element of capillary leak. Lastly, lymphopenia is typical with CHIKV, whereas thrombocytopenia is less common and more typical for dengue fever [13]. The patient’s erysipelas likely masked lymphopenia due to the left shift caused by the bacterial infection.

Reverse transcription-PCR and enzyme-linked immunosorbent assay are used to diagnose CHIKV. Reverse transcription-PCR can be positive up to 7 days after onset of disease, whereas IgM is detectable 2 to 7 days after onset of disease [14]. Treatment is supportive, although in vitro studies suggest that chloroquine has activity against CHIKV [15]. Clinical studies have produced mixed results, and chloroquine is not currently recommended for treatment of CHIKV infection [15]. Although dengue RT-PCR was not performed, the negative IgM 8 days into the patient’s illness supports that this was a monoinfection with CHIKV.

In summary, this patient had an atypical presentation given lack of joint pain, concomitant erysipelas, and leukocytosis. Her clinical course was complicated by capillary leak, which is also unusual for CHIKV. Furthermore, she was taking chloroquine for malaria prophylaxis, which may have masked arthralgias. It, therefore, highlights the need for physicians to have a high index of suspicion in returning travelers with a febrile illness to prevent further spread of CHIKV in CONUS and other areas with the Aedes vectors Testing for CHIKV, which can be arranged through state health departments (available at: http://www.cdc.gov/chikungunya/hc/diagnostic.html).
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Potential conflicts of interest. All authors: No reported conflicts.
All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

References