agents to avoid treatment until doctors better understand the disease.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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References


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Reply to Phillips et al.

Sir—Contrary to the comments made by Phillips et al. [1], much progress has been made in our understanding of southern tick–associated rash illness (STARI). It is not Lyme disease; bacterial causes seem unlikely on the basis of advanced microbiological and molecular studies, including eubacterial PCR, and no evidence of infection with other agents has emerged. Its principal epidemiological and clinical features have been described [2–6].

That my commentary [7] indicates a “regression” in the understanding of Lyme disease ecology and epidemiology, as claimed by Phillips et al. [1], is refuted by other articles written by myself and my colleagues [8, 9]. In their comparison of patients with erythema migrans–like rashes in New York and Missouri, Wormser et al. [3] found no differences in the duration of rash prior to treatment. This doesn’t support the argument of Phillips et al. [1] that earlier identification of illness in patients with STARI in the series of Wormser et al. [2] may account for the milder clinical presentation of STARI, compared with the erythema migans of Lyme disease. Observational studies consistently describe STARI as a mild illness, and, in contrast to Lyme disease, the appearance of sequelae after the acute illness is not a documented feature of this condition. Importantly, there is no evidence that antibiotics have any therapeutic effect on STARI. Such a conclusion could only be made after analyzing placebo-controlled trials. Prescribing intravenously administered or prolonged courses of antibiotics to patients with STARI or to patients with suspected but unsubstantiated Lyme disease is unjustified, unsafe, costly, and poor practice [10, 11].

In North America, birds play a minor role as reservoirs for Borrelia burgdorferi [9] and have not been shown to establish permanent geographical foci of infection in the absence of reservoir–competent, commingling tick and rodent populations. The Lone Star tick (Amblyomma americanum) is not a vector of B. burgdorferi [12], and the report by Clark [13] that describes chromosomal flagellin (flaB) gene and 16S rDNA amplification products of this organism in the midguts of these ticks in Florida was not substantiated by PCR amplification of outer surface protein A or other genes, or by culture. Even where enzootic cycles of B. burgdorferi exist in the southern United States (and these cycles can be described as scattered and tenuous), the risk of infection to humans is minimized by the preferential and prophylactic feeding by vector ticks there on lizards, instead of rodents or humans [9].

True Lyme disease cases cluster in 20 or so states in the northeastern, mid-Atlantic, upper midwestern and Pacific coastal regions [14]; Lyme disease reported from states outside these regions is likely the result of exposures elsewhere or misdiagnosis.

Without supporting scientific evidence, some medical professionals have promulgated the notion that Lyme disease exists throughout the United States, and that even those cases lacking evidence of complicated infection may sometimes require prolonged and intravenously administered courses of antibiotics in their treatment. To protect patients, governmental agencies and professional societies should be encouraged to aggressively investigate and regulate laboratories and clinical practices that ignore accepted scientific standards in the diagnosis and management of Lyme disease and STARI.

Acknowledgments

Table 1. Human papillomavirus (HPV) type-specific prevalence and concordance in paired self-collected and clinician-collected anorectal swab specimens obtained from 63 young men who have sex with men.

<table>
<thead>
<tr>
<th>HPV type</th>
<th>Either swab</th>
<th>Clinician-collected swab</th>
<th>Self-collected swab</th>
<th>Agreement, % (95% CI)</th>
<th>( \kappa ) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>30</td>
<td>30</td>
<td>22</td>
<td>92 (82–97)</td>
<td>0.80 (0.56–1.00)</td>
</tr>
<tr>
<td>18</td>
<td>13</td>
<td>11</td>
<td>8</td>
<td>94 (84–98)</td>
<td>0.63 (0.39–0.88)</td>
</tr>
<tr>
<td>45</td>
<td>14</td>
<td>13</td>
<td>10</td>
<td>94 (84–98)</td>
<td>0.68 (0.44–0.92)</td>
</tr>
<tr>
<td>51</td>
<td>14</td>
<td>13</td>
<td>11</td>
<td>95 (87–99)</td>
<td>0.77 (0.52–1.00)</td>
</tr>
<tr>
<td>Any high-risk type*</td>
<td>68</td>
<td>62</td>
<td>67</td>
<td>92 (82–97)</td>
<td>0.83 (0.58–1.00)</td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>11</td>
<td>13</td>
<td>92 (82–97)</td>
<td>0.62 (0.38–0.87)</td>
</tr>
<tr>
<td>42</td>
<td>11</td>
<td>10</td>
<td>8</td>
<td>95 (87–99)</td>
<td>0.70 (0.46–0.95)</td>
</tr>
<tr>
<td>q62</td>
<td>14</td>
<td>11</td>
<td>11</td>
<td>94 (84–98)</td>
<td>0.68 (0.43–0.93)</td>
</tr>
</tbody>
</table>

* High-risk HPV types include 16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 69, 70, 73, 82, and 1339.