Diphtheria-like Disease Caused by Toxigenic Corynebacterium ulcerans Strain

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Diphtheria-like Disease Caused by Toxigenic Corynebacterium ulcerans Strain

To the Editor: Toxigenic Corynebacterium ulcerans is an increasingly reported cause of diphtheria in the United Kingdom and is often associated with a zoonotic origin (1,2). Here, we report a case of diphtheria caused by toxigenic C. ulcerans in a woman, 51 years of age, from Scotland, UK, who was admitted to a hospital in August 2013 with a swollen, sore throat and a gray-white membrane over the pharyngeal surface. The patient had returned from a 2-week family holiday in the state of Florida, United States, before the admission and also reported recent treatment of a pet dog for pharyngitis. The patient was believed to have been vaccinated against diphtheria during childhood. She was immediately admitted to an isolation ward and treated with a combination of clindamycin, penicillin, and metronidazole.

Microscopic examination of the throat biofilm (collected by using a swab) showed gram-positive bacilli; swab samples from the exudative membrane and throat produced small, black colonies indicative of Corynebacterium spp. on Hoyle medium. Further efforts to identify the strain by using VITEK MS and VITEK2 ANC card systems were negative for Staphylococcus aureus, Streptococcus spp., and Brucella spp. This specimen was sent to the Health England, Colindale, UK, and was confirmed to be a toxigenic C. ulcerans strain that we designated RAH1. Throat swab samples were collected from family members of the patient and were negative for C. ulcerans.

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EMERGING INFECTIOUS DISEASES
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of easy-to-handle bioinformatics tools emphasize the suitability of deep-sequencing technology for rapid diagnostics and for the development of high-resolution genotyping. It is time for the wider introduction of this technology into public health investigations.

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References


Table. Virulence genes associated with Corynebacterium ulcerans present in strain RAH1 isolated from patient with diphtheria-like disease, 2013, United Kingdom*

<table>
<thead>
<tr>
<th>Gene</th>
<th>Strains</th>
<th>Strain RAH1</th>
<th>Potential function</th>
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<tbody>
<tr>
<td>tox</td>
<td>0102</td>
<td>P</td>
<td>Diphtheria-like toxin</td>
</tr>
<tr>
<td>rbp</td>
<td>089</td>
<td>A</td>
<td>Shiga toxin–like ribosome binding protein</td>
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<tr>
<td>cpp</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Corynembacterial protease CP40, protective antigen against caseous lymphadenitis</td>
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<tr>
<td>pld</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Toxic phospholipase D</td>
</tr>
<tr>
<td>spaF</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Surface-anchored protein, pilus tip protein</td>
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<tr>
<td>spaE</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Surface-anchored protein, minor pilin subunit</td>
</tr>
<tr>
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<td>P</td>
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<td>P†</td>
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<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Resuscitation-promoting factor interacting protein</td>
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<tr>
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<td>P</td>
<td>Cell wall–associated hydrolase</td>
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<td>nanH</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Neuraminidase, glycosyl hydrolases</td>
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<td>809, BR-AD22</td>
<td>P</td>
<td>Venom serine protease</td>
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<tr>
<td>vsp2</td>
<td>809</td>
<td>P</td>
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<tr>
<td>tspA</td>
<td>809, BR-AD22</td>
<td>P</td>
<td>Tryptic-like serine protease</td>
</tr>
</tbody>
</table>

*P, present; A, absent. †p>700 bp deletion.

Death of Woman with Peripartum Influenza B Virus Infection and Necrotizing Pneumonia

To the Editor: Pregnant women are at increased risk for severe influenza-related complications (1). Bacterial pneumonia with Panton-Valentine leukocidin-producing (PVL) Staphylococcus aureus is infrequently described in the literature as occurring concurrently with influenza B virus infection (2–4). Additionally, only 2 occurrences of peripartum PVL-methicillin-resistant S. aureus (MRSA) pneumonia have been described (5,6). We report a case of influenza B virus and PVL-MRSA coinfection during pregnancy.

In December 2012, a previously healthy pregnant woman, 38 years of age, at 37 weeks’ gestation and in active labor, sought treatment in a New York hospital reporting 2 days of fever, productive cough, shortness of breath, and pleuritic chest pain. Household contacts included children with influenza-like illness. The patient had declined influenza vaccination while receiving prenatal care. On arrival, examination showed that her temporal temperature was 101.6°F, blood pressure was 122/71 mm Hg, pulse was 121 beats per minute, respiratory rate was 40 breaths per minute, and oxygen saturation was 89% on room air; bilateral inspiratory crackles were heard on lung auscultation. Rapid influenza screening of a nasopharyngeal swab sample by using ELISA was negative for influenza A and B viruses. Culture of the patient’s nasopharynx was positive for MRSA colonization. Laboratory evaluation showed leukopenia of 1500/mL, and although imaging was limited by the patient’s lead apron, a chest radiograph demonstrated bibasilar opacities (Figure, panel A).