
This version is available at https://strathprints.strath.ac.uk/48935/

Strathprints is designed to allow users to access the research output of the University of Strathclyde. Unless otherwise explicitly stated on the manuscript, Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Please check the manuscript for details of any other licences that may have been applied. You may not engage in further distribution of the material for any profitmaking activities or any commercial gain. You may freely distribute both the url (https://strathprints.strath.ac.uk/) and the content of this paper for research or private study, educational, or not-for-profit purposes without prior permission or charge.

Any correspondence concerning this service should be sent to the Strathprints administrator: strathprints@strath.ac.uk

The Strathprints institutional repository (https://strathprints.strath.ac.uk) is a digital archive of University of Strathclyde research outputs. It has been developed to disseminate open access research outputs, expose data about those outputs, and enable the management and persistent access to Strathclyde's intellectual output.
**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 (bioMérieux, Marcy l’Etoile, France) to evaluate the swab samples suggested that the infection was caused by either *C. ulcerans* or *C. pseudotuberculosis* (50% CI). The isolate detected from this process was sent to the *Streptococcus* and Diphtheria Reference Unit, Public 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*. The family dog was not tested for presence of the organism, although it is known that *C. ulcerans* infections are often of a zoonotic nature ([1,2]). After treatment, the patient made a full recovery.

Toxigenic *C. ulcerans* can produce both diphtheria-like and Shiga-like toxins ([3]); to identify the genetic basis of toxin production and other potential virulence factors in this strain, a whole genome sequencing approach was applied to the isolate. The genome was sequenced by using an Ion PGM System (Thermo Fischer Scientific, Loughborough, Leicestershire, UK) and resulting reads (2,965,044 reads, >90x coverage). Data are available on GenBank SRA: high-throughput DNA and RNA sequence read archive (http://www.ncbi.nlm.nih.gov/Traces/sra/sra.cgi?view=search_obj, accession no.: SRR1145126) and were mapped onto the published genome sequences of a Shiga-like toxin–producing clinical isolate 809, asymptomatic canine strain BR-AD22 ([3]), and diphtheria-like toxin–producing strain 0102 ([4]). Most of the previously identified virulence genes ([3,4]) were present in the patient isolate (Table). The *tox* gene, encoding diphtheria toxin, was present, which verified the diphtheria-like disease in the patient. The *rpb* gene, responsible for Shiga toxin–like ribosome-binding protein, was absent. However, strain RAH1 also possessed the venom serine protease gene (*vsp2*), which, in *C. ulcerans* strain 809, has been implicated in the increased virulence in humans. The *tox* gene was present in a prophage that showed similarities to *ΦCULC809I* ([3]) and *ΦCULC0102-I* ([4]). Genome-based phylogenetic analysis of the RAH1 strain (ClonalFrame analysis [5]) and strains 809, BR-AD22, and 0102 indicates a much wider phylogenetic diversity of *C. ulcerans* strains than previously appreciated (data not shown).

This case raises the issue of waning vaccine protection in older patients and suggests that toxin-mediated corynebacterial disease remains a threat to public health. The declining costs of next-generation sequencing and availability

---

**Address for correspondence:** Jonas Schmidt-Chanasit, Bernhard Nocht Institute for Tropical Medicine, World Health Organization Collaborating Centre for Arbovirus and Haemorrhagic Fever Research and Reference, Bernhard-Nocht-Strasse 74, 20359 Hamburg, Germany; email: jonassi@gmx.de

---


---

**EMERGING INFECTIOUS DISEASES**

**Conference summaries and other reports available online**
http://www.cdc.gov/eid/articles/conferencesummaries/volume-20

---

Emerging Infectious Diseases • www.cdc.gov/eid • Vol. 20, No. 7, July 2014
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.

Vartul Sangal, Leena Nieminen, Barbara Weinhardt, Jane Raeside, Nicholas P. Tucker, Catalina-Diana Florea, Kevin G. Pollock, and Paul A. Hoskisson

Author affiliations: Strathclyde Institute of Pharmacy and Biomedical Sciences, University of Strathclyde, Glasgow, Scotland UK (V. Sangal, L. Nieminen, N.P. Tucker, P.A. Hoskisson); Faculty of Health and Life Sciences, Northumbria University, Newcastle upon Tyne, UK (V. Sangal); Health Protection Scotland, Glasgow (K.G. Pollock); and Royal Alexandra Hospital, Paisley, UK (B. Weinhardt, J. Raeside, C.-D. Florea)

Address for correspondence: Paul A. Hoskisson, Strathclyde Institute of Pharmacy and Biomedical Sciences, University of Strathclyde, 161 Cathedral St, Glasgow, G4 0RE, Scotland, UK; email: paul.hoskisson@strath.ac.uk

DOI: http://dx.doi.org/10.3201/eid2007.140216

References


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 co-infection 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 nasopharyngeal swab sample, however, grew a methicillin-resistant S. aureus strain (PVL-MRSA) pneumonia strain.

Address for correspondence: Paul A. Hoskisson, Strathclyde Institute of Pharmacy and Biomedical Sciences, University of Strathclyde, 161 Cathedral St, Glasgow, G4 0RE, Scotland, UK; email: paul.hoskisson@ strath.ac.uk

Find emerging infectious disease information on

http://www.facebook.com

DOI: http://dx.doi.org/10.3201/eid2007.140216

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>
</tr>
</thead>
<tbody>
<tr>
<td>tox</td>
<td>0102</td>
<td>P</td>
<td>Diphtheria-like toxin</td>
</tr>
<tr>
<td>rbp</td>
<td>809</td>
<td>A</td>
<td>Shiga toxin–like ribosome binding protein</td>
</tr>
<tr>
<td>opp</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Corynebacterial protease CP40, protective antigen against caseous lymphadenitis</td>
</tr>
<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>
</tr>
<tr>
<td>spaE</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Surface-anchored protein, minor pilin subunit</td>
</tr>
<tr>
<td>spaD</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Surface-anchored protein, major pilin subunit</td>
</tr>
<tr>
<td>spaC</td>
<td>809, BR-AD22, 0102</td>
<td>P†</td>
<td>Surface-anchored protein, pilus tip protein</td>
</tr>
<tr>
<td>spaB</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Surface-anchored protein, minor pilin subunit</td>
</tr>
<tr>
<td>pfl</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Resuscitation-promoting factor interacting protein</td>
</tr>
<tr>
<td>cwI H</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Cell wall–associated hydrolase</td>
</tr>
<tr>
<td>nanH</td>
<td>809, BR-AD22, 0102</td>
<td>P</td>
<td>Neuraminidase, glycosyl hydrolases</td>
</tr>
<tr>
<td>vspl</td>
<td>809, BR-AD22</td>
<td>P</td>
<td>Venom serine protease</td>
</tr>
<tr>
<td>vspl2</td>
<td>809</td>
<td>P</td>
<td>Venom serine protease</td>
</tr>
<tr>
<td>tspA</td>
<td>809, BR-AD22</td>
<td>P</td>
<td>Tryptase-like serine protease</td>
</tr>
</tbody>
</table>

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