Pigs as Source for Toxigenic Corynebacterium ulcerans

To the Editor: Toxigenic Corynebacterium ulcerans may cause a zoonotic infection similar to diphtheria caused by C. diphtheriae. Previously, dairy cattle were considered to be the main reservoir for C. ulcerans (1), but recent publications suggest pet dogs and pet cats as carriers (cats often show bilateral nasal discharge) (2). We report a case of severe C. ulcerans diphtheria-like disease in a person who had had contact with pigs.

In December 2007, a previously healthy 56-year-old female farmer was admitted to the Ear, Nose and Throat Department of the University Hospital Erlangen with a 1-week history of sore throat and progressive dysphagia. She did not report fever and had not received prior treatment with antimicrobial drugs. She had thick, whitish pseudomembranes on her uvula, pharynx, and both tonsils. Endoscopic examination of her larynx and hypopharynx showed that both vocal cords were mobile and the mucosa was erythematous. Enlarged cervical lymph nodes were palpable on both sides of her neck. She had no signs of cranial nerve palsies. Her temperature was 36.5°C. Because the barn doors were reportedly closed at all times. Because handling of C. ulcerans–infected pigs may lead to diphtheria-like illnesses, studies of toxigenic C. ulcerans carriage among pigs are needed. Similar to our case, diphtheria-like disease caused by an erythromycin- and clindamycin-resistant toxigenic C. ulcerans strain in a livestock animal and a human, as well as harboring of toxigenic C. ulcerans in pigs. Introduction of C. ulcerans from wild animals seems unlikely because the barn doors were reportedly closed at all times. Because current recommendations based on C. diphtheriae–caused disease consider erythromycin as the second-line option for treatment or postexposure prophylaxis, these findings highlight the importance of antimicrobial-drug susceptibility testing of toxigenic C. ulcerans strains.

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References


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Campylobacter jejuni HS:23 and Guillain-Barré Syndrome, Bangladesh

To the Editor: Guillain-Barré syndrome (GBS) is an acute peripheral neuropathy triggered by a preceding infectious illness. Gastroenteritis caused by Campylobacter jejuni is the most frequently reported antecedent event (1). In Japan, South Africa, China, and Mexico, Campylobacter strains with certain Penner heat-stable (HS) serotypes, including HS:19 and HS:41, are overrepresented among isolates from GBS case-patients, compared with isolates from enteritis case-patients (2,3). Several studies indicate that C. jejuni HS:19 and HS:41 have a clonal population structure and suggest that these serotypes might have unique virulence properties that are intricately linked to development of GBS (4). However, data from the United Kingdom and the Netherlands suggest that such virulence properties may not be restricted to specific HS serotypes because many other serotypes can be cultured from patients with GBS (5). We report a non-HS:19 and non-HS:41 C. jejuni serotype and sequence type (ST)–3219 that are overrepresented among isolates from GBS patients in Bangladesh.

We conducted a prospective case-control study of the serotype and genotype of C. jejuni associated with GBS in Bangladesh. Case-patients were 97 persons with GBS admitted to Dhaka Medical College Hospital, Bangabandhu Sheikh Mujib Medical University, and Dhaka Central Hospital during July 2006–June 2007. All fulfilled the diagnostic criteria for GBS of the National Institute of Neurological Disorders and Stroke of the US National Institutes of Health (Bethesda, MD, USA) (6). The control group comprised 97 patients with other neurologic diseases, matched with