Cutaneous

*Corynebacterium diphtheriae*: A traveller’s disease?

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CASE REPORT

A BERIH. *Corynebacterium diphtheriae*: A traveller’s disease? Can J Infect Dis 1995;6(3):150-152. A Canadian soldier incurred a nonhealing traumatic skin ulcer while on duty in Somalia. The diagnosis of localized cutaneous diphtheria was confirmed by isolation of a toxigenic strain of *Corynebacterium diphtheriae* from the ulcer. The patient was placed in isolation and treated with erythromycin and penicillin for 10 days without antitoxin. He was released when two consecutive daily cultures were negative. Public health officials evaluated his wife, two children and close contacts for carriage, but no carriers or secondary cases were identified. Cutaneous diphtheria as a diagnostic and management patient problem and potential public health problem are discussed.

Key Words: *Corynebacteremia, Cutaneous diphtheria, Diphtheria, Somalia, Travel*

*Corynebacterium diphtheriae* cutané : maladie du voyageur?

RÉSUMÉ : Un soldat canadien a subi un ulcère cutané traumatique non cicatrisant lors d’une mission en Somalie. Le diagnostic de diphtérie cutanée localisée a été confirmé lors de l’identification d’une souche toxigène de *Corynebacterium diphtheriae* à partir de l’ulcère. Le patient a été placé en isolement et traité par érythromycine et pénicilline pendant 10 jours sans antitoxines. Il a reçu son congé après avoir obtenu des résultats négatifs à deux cultures quotidiennes consécutives. Les autorités sanitaires ont soumis son épouse, ses deux enfants et ses proches à des analyses pour vérifier s’ils étaient porteurs, mais aucun cas de porteur ni d’infection secondaire n’a été identifié. Le présent article décrit la diphtérie cutanée comme un problème diagnostique et thérapeutique chez le patient et comme un problème potentiel de santé publique.

Cutaneous diphtheria is commonly seen in tropical countries (1). In the 1940s ulcerative cutaneous diphtheria was a serious problem among American troops stationed in the Mediterranean, South Pacific and Asian regions (2,3). In North America cutaneous diphtheria has become a rare event and a medical curiosity. A case of toxigenic *Corynebacterium diphtheriae* imported from Somalia that had the potential to become a major public health hazard is reported.
CASE PRESENTATION

A 35-year-old previously healthy infantryman presented with a history of having been cut on his right shin by a garbage can in Somalia. The patient was seen by the medical staff in Somalia and treated with cloxacillin prophylactically. On presentation in Canada two weeks later, he had a red, inflamed, punched out wound of the right shin approximately 1 cm in diameter, with a yellow exudate. The wound was swabbed and patient started on oral trimethoprimsulfamethoxazole. Toxinogenic C diphtheriae, biotype mitis and Staphylococcus aureus were isolated from the wound culture.

The patient was recalled and placed on contact isolation in accordance with public health and in-hospital protocol (4). Throat and nose swabs were taken and wound swabs were repeated. The patient was given penicillin G 1.2 MU intramuscularly and then erythromycin 500 mg four times a day by mouth for 10 days. He was considered fully immunized repeated. The patient was given penicillin G 1.2 MU intramuscularly and then erythromycin 500 mg four times a day by mouth for 10 days. He was considered fully immunized against diphtheria toxin. Wound dressings were changed daily, and no topical antibiotics were applied. The wound was crusty but not healed when the course of antibiotics was completed. The patient remained on contact isolation until cultures from the nose, throat and wound, taken on two occasions 24 h after cessation of antimicrobial therapy, were reported as negative (4). Signs and symptoms of myocarditis or neuritis did not appear. No diphtheria antitoxin was administered. Cultures were repeated 14 days after discontinuation of antibiotics to rule out microbiological relapse; no C diphtheriae bacilli were found.

The patient and his five-year-old daughter were up to date for diphtheria immunization, but his wife and four-month-old child were given diphtheria toxoid boosters. Throat swabs were taken from the immediate family and they were started on prophylactic antibiotics. Contact tracing was conducted. Cultures from all family and other contacts were negative.

DISCUSSION

C diphtheriae is a nonsporulating, unencapsulated, pleomorphic Gram-positive bacillus (5). In the tropics and subtropical climates cutaneous diphtheria infections prevail over respiratory infections. Endemic skin diphtheria appears to be responsible for acquisition of immunity in the tropics (6). From 1921 to 1924 infections due to C diphtheriae have been described from indigent urban residents in British Columbia (7).

Cutaneous diphtheria is highly contagious, more so than respiratory diphtheria (8). Cutaneous sites of C diphtheriae have been shown to contaminate the environment and induce human infections more efficiently than pharyngeal infections (9,10). It has been shown that C diphtheriae can survive in floor dust for at least 14 weeks (11). Complications of C diphtheriae infections may affect nerves (principally motor), the heart and kidneys (12).

Clinical suspicion of cutaneous diphtheria depends on epidemiological and morphological features. Definitive diagnosis of Corynebacterium infections depend on culturing of organisms. When any case of diphtheria is diagnosed, control measures should include a search for cutaneous C diphtheriae (10). Besides isolation and strict bed rest, the only specific treatment of Corynebacterium infections is the administration of antitoxin to C diphtheriae (13).

Antibiotics do not alter the course, incidence of complications or outcome of the infection (12). However, antibiotics are beneficial because they eliminate the carrier state, terminate toxin production and ameliorate local infections (5). In light of the absence of toxic symptoms such as myocarditis and polyneuritis, and because the patient was up to date for immunization, he was not given antitoxin after a local infectious disease specialist was consulted. The risk of complications is much lower in immunized patients. Potential systemic complications of cutaneous diphtheria must be weighed against potential adverse effects of antitoxin treatment (14).

Spontaneous healing of the wound takes six to 12 weeks, but cases lasting one year have been observed (8).

Although cutaneous diphtheria is only a medical curiosity in North America, primary care physicians and specialists should place C diphtheriae on their differential diagnosis of a patient who presents with a nonhealing punched out ulcer with slightly elevated margin and a history of recent travel to the tropics. Swabbing of the lesion is essential. If diagnosis is confirmed by culture, the patient should be placed on contact isolation and given antibiotics; oral erythromycin or penicillin G intramuscularly are the antibiotics of choice (4). The patient should be monitored for toxic complications, especially myocarditis and polyneuritis. Electrocardiogram and neurological examinations should be performed. Toxinogenic C diphtheriae is a major public health hazard, and the patient with C diphtheriae can be a reservoir for an epidemic (10). Therefore, notification of public health authorities is essential.

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**CLINICAL VIGNETTE**

**Diagnosis?**

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**CASE PRESENTATION**

A 28-year-old Ugandan-born male was known to be human immunodeficiency virus-positive for three years. He had been followed for ileal tuberculosis (treated for 18 months with isoniazid, rifampin, ethambutol and pyrazinamide), perianal herpes and mild chronic diarrhea due to cryptosporidia. His wife had died several years earlier of cryptococcal meningitis while still in Uganda. He lived alone in Montreal, did not travel and had no animal exposure.

He presented with sudden onset of right knee pain and swelling. A radiograph was normal and the knee was tapped for 50 mL of slightly turbid yellow fluid. The fluid contained 15,000 leukocytes/µL and 600 erythrocytes/µL and no crystals, and cultures for bacteria, fungi and mycobacteria were all negative. He was treated with a nonsteroidal anti-inflammatory medication and all symptoms and signs resolved. Three months later, he presented with sudden onset of low back pain. A radiograph showed a destructive process in the spinous process of L2, confirmed by a computed tomography scan (Figure 1). Bone and gallium scanning showed uptake of both tracers (consistent with osteomyelitis) at L2 and in the right knee at the distal femur, despite a nominal repeat radiograph of the right knee. He underwent an open biopsy of the spinous process of L2. What is the diagnosis?

*For diagnosis, see page 176*

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**Figure 1** Computed tomography scan (inset). Detail shows soft tissue mass invading and destroying the pedicle and spinous process of the vertebral body.
