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## Epidemic *Clostridium difficile* Strain in Hospital Visitation Dog

**To the Editor:** Rates of illness and death from *Clostridium difficile*-associated disease (CDAD) and reports of CDAD in persons without traditional risk factors (1) have been increasing. One particular strain of *C. difficile* has been implicated in outbreaks of CDAD in hospitals in North America and Europe and appears to be spreading internationally at an alarming rate. This strain is classified as ribotype 027, toxinotype III, and possesses genes encoding toxins A, B, and CDT (binary toxin) as well as a deletion in the *tcdC* gene, which is believed to increase virulence (2).

We report this toxin-variant strain of *C. difficile* in a healthy, 4-year-old

toy poodle that visits persons in hospitals and long-term care facilities in Ontario on a weekly basis. *C. difficile* was isolated from a fecal sample collected in the summer of 2004 as part of a cross-sectional study evaluating pathogen carriage by visitation dogs (3). The isolate was subsequently characterized by ribotyping (4) and by polymerase chain reaction (PCR) detection of genes that encode production of toxins A and B (5). Toxin CDT was confirmed by amplifying the portion of the gene (*cdtB*) that encodes for the receptor-binding component of the toxin, according to a previously reported protocol (6). As a result, the isolate was classified as ribotype 027, toxinotype III (7), and was found to possess all 3 toxin genes. The *tcdC* gene deletion was also confirmed with PCR (8).

These results indicate that this canine isolate is indistinguishable from the major strain implicated in outbreaks of highly virulent CDAD around the world. According to the infection control practitioner at the hospital the dog visited, CDAD cases were occurring at increased frequency in the facility around the time the dog's fecal specimen was collected. However, patient diagnosis was made solely through fecal toxin testing, and strains were not characterized. The facility has reported only sporadic cases of CDAD in the past few years.

This is the first report of this human, epidemic strain of *C. difficile* in a dog. Many *C. difficile* strains isolated from animals, including dogs, are indistinguishable from strains associated with disease in humans (9). To date, no study, including this one, has shown that interspecies transmission occurs; however, that possibility exists, as is becoming apparent with other pathogens, such as methicillin-resistant *Staphylococcus aureus*. The recurrent exposure of this dog to human healthcare settings suggests that the animal acquired this strain during visits to the hospital or long-

term care facility, either from the healthcare environment or contaminated hands of human contacts. We recommend that future studies evaluating the dissemination of this strain and investigations of the movement of *C. difficile* into the community consider the role of animals.

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## *Streptobacillus moniliformis* Endocarditis

**To the Editor:** *Streptobacillus moniliformis* is a facultatively anaerobic, pleomorphic, gram-variable bacillus often seen in chains and as long unbranched filaments. It is found in the nasopharynx and oropharynx of wild and laboratory rats. Human infections result either from rodent bites (rat bite fever) or contaminated milk or other foods (Haverhill fever). The most common manifestations of infection are arthralgia, fever, and rash; endocarditis occurs as a rare complication (1). We report a case of *S. moniliformis* endocarditis in India in a patient with congenital heart disease.

An 18-year-old man was admitted to the Department of Cardiology at the Government General Hospital in Chennai, India, in November 2005, with a fever of 2 months' duration with cough, epistaxis, palpitations, and persistent joint pain. His medical history indicated congenital heart disease with a ventricular septal defect.

On physical examination, his blood pressure was 100/70 mm Hg, pulse rate was 100 beats/min, and temperature was 38.5°C. Laboratory tests showed a leukocyte count of 7,600/ $\mu$ L, a platelet count of 127,000/ $\mu$ L, and an erythrocyte sedimentation rate of 70 mm/h. An electrocardiogram showed normal sinus rhythm. A transthoracic echocardiogram demonstrated a ventricular septal defect and vegetations on the septal leaflet of the tricuspid valve.

Three blood cultures were prepared, and treatment with antimicrobial drugs (intravenous penicillin G, 3  $\times$  10<sup>6</sup> U every 6 h, and gentamicin, 50 mg every 8 h for 4 weeks) was initiated. The blood cultures were incubated at 37°C in an atmosphere of 5%–10% CO<sub>2</sub>. Characteristic white, downy, crumblike granules were observed on the surface of the erythrocytes in all 3 cultures within 18–24 h of incubation. Characteristic puff balls were seen after 48 h of incubation. Gram-stained smears showed gram-negative bacilli in long chains. Cultures were subcultured onto 5% sheep blood agar plates and MacConkey agar plates. The plates were incubated at 37°C in an atmosphere of 5%–10% CO<sub>2</sub>. After 18–24 h of incubation, growth was seen on the sheep blood agar plates. Colonies were 1–2 mm in diameter, gray, smooth, and butyrous. A Gram stain of these colonies identified gram-variable, pleomorphic coccobacilli that were negative for catalase, oxidase, urease, and citrate, and did not produce indole or reduce nitrate.

Antimicrobial susceptibility testing was performed by using the Kirby-Bauer disk diffusion method according to recommendations of the National Committee for Clinical Laboratory Standards (2). The isolate was sensitive to penicillin G, ceftriaxone, cephalexin, amoxicillin, gentamicin, and erythromycin. The patient responded well to treatment and became afebrile within 48 h after initiation of therapy. Treatment with

antimicrobial drugs was continued for 4 weeks. The blood cultures were negative when repeated after 2 weeks. The patient had an uneventful recovery and was discharged from the hospital.

Rat bite fever is a zoonosis caused by either *Streptobacillus moniliformis* or *Spirillum minus* (1,3). *S. moniliformis* is found in the nasopharynx of small rodents, especially rats. Rats that are carriers have no symptoms but can effectively transmit the infection by bite or through infected body fluids such as urine.

This patient had a history of living in a rat-infested area, and admitted having been bitten by a rat several months before the onset of symptoms. However, we considered it unlikely that disease contracted by a rat bite would take months to be manifested. Thus, it is more likely that he contracted the infection from food or water contaminated with rat excreta. Endocarditis is a rare complication of *S. moniliformis* infection, and cardiac valvular abnormalities have been reported in 50% of cases (4). This patient, however, had only a small ventricular septal defect. This is the first report of *S. moniliformis* endocarditis from India.

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