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Vibrio furnissii: an Unusual Cause of Bacteremia and Skin Lesions after Ingestion of Seafood▽†

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Vibrio furnissii **in the blood is rarely reported, which may explain why clinical features of bloodstream infections with this organism have not been described. We describe a patient who developed skin lesions and** ***V. furnissii*** **bacteremia and was successfully treated with fluoroquinolones.** ***V. furnissii*** **may be a serious pathogen in patients with underlying comorbidities who are exposed to seafood.**

CASE REPORT

A 62-year-old man from central Virginia presented in July 2009 with a 2-day history of bilateral lower extremity swelling and erythema associated with burning, weeping lesions. He had a history of cardiovascular disease, chronic kidney disease, peripheral arterial disease, and uncontrolled type II diabetes mellitus complicated by peripheral neuropathy. Although initially afebrile, his temperature subsequently increased to 38.2°C. His white blood cell count was 15,100 cells/mm^3 with 88% neutrophils. He was tachycardic but otherwise hemodynamically stable, with a blood pressure of 101/57. On exam, he had changes consistent with chronic venous stasis, tinea pedis, and onychomycosis. Both legs had multiple grayish sloughing lesions suggestive of prior bullous lesions, some with fresh blood at the base. He also had a tender fluid-filled blister with a black-gray center on one foot.

He was initially started on intravenous vancomycin (2.5 g total) and oral moxifloxacin at 400 mg daily for broad-spectrum antimicrobial coverage. Blood cultures drawn at his initial presentation grew Gram-negative rods in two sets. Repeat blood cultures drawn the following day were negative. The organism was initially identified by the Vitek Legacy as a possible *Vibrio* species and was susceptible to ceftazidime, cefepime, trimethoprim-sulfamethoxazole, gentamicin, amikacin, ampicillin-sulbactam, imipenem, meropenem, piperacillin, piperacillin-tazobactam, chloramphenicol, tetracycline, ciprofloxacin, and levofloxacin by microdilution. Wound cultures were not obtained. He rapidly defervesced, and his white blood cell count normalized after 1 day of systemic antibiotics.

The organism was later identified as *Vibrio furnissii* by phenotypical and biochemical characteristics and by *rpoB* sequencing at the Centers for Disease Control and Prevention (CDC). After further questioning, the patient recalled eating cooked shrimp at a local restaurant approximately 1 week prior to his hospital admission. He had no nausea, abdominal pain, or diarrhea. His antibiotic regimen was changed to oral ciprofloxacin at 500 mg twice daily on the fifth hospital day.

Although the patient's leg wound improved somewhat after 2 weeks of antibiotics, he continued to have necrotic material at the base of some leg ulcers, with surrounding erythema and a small amount of yellowish discharge. He required no surgical debridement but was given oral moxifloxacin at 400 mg daily. After 3 weeks of moxifloxacin therapy, the necrotic material and surrounding erythema had significantly decreased. The patient continued to receive local wound care as needed.

V. furnissii is an oxidase-positive, motile Gram-negative rod that was originally categorized as an aerogenic strain of *V. fluvialis* until 1983, when researchers demonstrated that both species were phenotypically and genetically distinct (1). The two species are highly similar in their phenotypic characteristics, and *V. furnissii* is differentiated from *V. fluvialis* by its production of gas from the fermentation of carbohydrates (1). It is 1 of at least 11 noncholera *Vibrio* species pathogenic in humans (6). Disease typically occurs after ingesting contaminated raw or undercooked seafood or after contact with warm marine environments (6). *V. furnissii* has been cultured from the intestines of healthy brown shrimp (4).

Although *V. furnissii* has been associated with outbreaks and isolated cases of gastroenteritis (1, 2, 5, 8, 9), the relative pathogenicity of *V. furnissii* in most of these instances was unclear, in that other pathogens may have contributed to the disease or that the individuals were asymptomatic at the time of stool collection. The pathology of *V. furnissii* in gastroenteritis is potentially related to cytolysin and hemolysin production (9).

From 1970 through 1985, approximately 1,230 *Vibrio* isolates were studied at the *Vibrio* Reference Laboratory at the CDC

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(3). Of these, 16 were *V. furnissii* strains, and all 16 were associated with intestinal infections. In 1988, the CDC, the U.S. Food and Drug Administration (FDA), and the Gulf Coast states (Alabama, Florida, Louisiana, Mississippi, and Texas) established the Cholera and Other *Vibrio* Illness Surveillance System (COVIS), with most of the United States reporting cases of human disease from *Vibrio* species by 1997. The Council of State and Territorial Epidemiologists (CSTE) has recommended reporting all *Vibrio* species since January 2007. The CDC reported 10 isolates of *V. furnissii* from 1997 to 2008, 3 from blood, 2 from a wound, and 5 from stool. *V. fluvialis* was also isolated from one wound and from one blood culture. Four patients required hospitalization, and one patient died, but neither the specimen types nor additional epidemiologic data for these sources were provided. At the time of this writing, data have not been made publically available for the years after 2008 (http://www.cdc.gov/nationalsurveillance/cholera_vibrio_surveillance.html).

In addition to the isolate associated with our case report (2009V-1068), a second blood isolate (AM40833) was sent to the CDC from Virginia in 2009 through COVIS. Because *V. furnissii* is rarely implicated in blood infections, we were interested in whether the two cases from Virginia were related and perhaps caused by a single bacterial clone. We first examined the relationship between *V. furnissii* strains by sequencing the *rpoB* genes by established methodology (10). The two blood isolates clustered with the other *V. furnissii* strains in the phylogenetic tree, as shown in Fig. S1 in the supplemental material, but differed at 6 of 855 nucleotide positions. The genetic divergence in the *rpoB* genes indicated that the two strains represented separate lineages and were not representatives of a single clone. The phenotype of the two strains also differed as follows: the case study isolate showed atypical results for two tests (negative for Simmons citrate utilization and negative for mannose fermentation), whereas the phenotype of the other isolate was typical for *V. furnissii.* The full phenotypic profiles of the strains are provided in Table S1 in the supplemental material. Overall, the two strains differed by five test results. The conclusions from the phenotypic and genotypic testing support the notion that multiple *V. furnissii* lineages are capable of causing blood infections and that any apparent increase in bacteremia associated with *V. furnissii* is not caused by a single emerging clone.

Although *V. furnissii* has previously been isolated from the bloodstream, the clinical features associated with bacteremia have not been described. To our knowledge, we report the first case of *V. furnissii* bacteremia with associated bilateral lower extremity lesions. The clinical presentation was similar to those previously described in *V. vulnificus* infections although less severe (7). The patient's only known exposure was consumption of fried seafood 1 week prior to developing symptoms. He had no gastroenteritis symptoms and no history of a preceding wound. His poorly controlled diabetes may have contributed to the increased virulence of his disease, although his fever and leukocytosis decreased promptly with fluoroquinolones. Peripheral arterial disease may have also played a role in the delayed wound healing. Although uncommon, *V. furnissii* may be an important source of morbidity in patients with chronic underlying illnesses. More data are necessary to define risk factors and to determine the best course of prevention and treatment.

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