Axillary Abscess Due to *Yersinia enterocolitica*\(^\dagger\)

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*Yersinia enterocolitica* rarely causes extraintestinal disease. A 54-year-old construction worker with chronic hepatitis C developed an axillary abscess following an injury to his finger. An aspirate from the axillary mass grew *Y. enterocolitica*. Direct inoculation is proposed as the mode of transmission of this classically enteric pathogen.

CASE REPORT

A 54-year-old African-American construction worker presented to an outpatient clinic associated with our academic tertiary referral center in early January 2009 with a painful “lump” at the right axilla of 2 days’ duration. His medical history was significant for chronic hepatitis C without clinical decompensation. At the initial visit, the patient also pointed out a healing pustule on a finger of his right hand which he had recently self-debrided with a razor. The pustule had developed from a poorly healing wound from splinter trauma during a visit to a landfill 2 months earlier. He felt otherwise well, and he had no fever. Application of warm compresses to the axilla was recommended. One week later, he returned to his primary care provider with complaints of increasing pain and size of the right axillary mass as well as fever and night sweats. The temperature was 100.3°F. On examination, there was a palpable tender mass the size of approximately one-half of a tennis ball with mild overlying erythema but no fluctuance. The remainder of his examination was normal. The peripheral leukocyte count was 15,400 cells/mm\(^3\). Ultrasound imaging indicated an encapsulated mass of 8.0 cm by 5.5 cm by 5.4 cm with a largely cystic necrotic center. Gram stain of an aspirated specimen indicated 4+ white blood cells and no organisms. After 24 h of incubation, the culture yielded pure culture of a non-lactose-fermenting Gram-negative bacillus that was identified as *Yersinia enterocolitica*, using the Vitek GNI identification system (bioMérieux, Hazelwood, MO), with 99.00% probability, and motility tests (motile at room temperature but not at 35°C). Identity was confirmed with 16S rRNA gene sequencing. There were three to five colonies each on sheep blood and MacConkey agar plates in addition to a positive broth culture. No blood or stool culture was ordered or performed. Sensitivity studies using the Vitek 2 instrument showed susceptibility to ceftazidime, ceftriaxone, imipenem, amingoglycosides, ciprofloxacin, levofloxacin, and trimethoprim-sulfamethoxazole; the isolate was resistant to ampicillin, ampicillin-sulbactam, and cefazolin. Upon further interview, the patient denied any abdominal pain or diarrhea. He also denied consumption or exposure to pork chitterlings or other raw pork products in recent weeks. The mass resolved on a 4-week oral regimen of trimethoprim-sulfamethoxazole, and he made a full recovery.

*Y. enterocolitica* is a Gram-negative bacillus that causes sporadic illness and occasional food-borne outbreaks in the United States, as compared to many European countries, where the incidence appears to be higher (1, 3). Although many domestic animals may harbor this organism, swine are a major reservoir of pathogenic *Y. enterocolitica*. Human yersinia is primarily acquired through the gastrointestinal tract as a result of ingestion of contaminated food—usually raw or inadequately cooked pork. Recent *Y. enterocolitica* outbreaks in the United States have involved young children exposed indirectly during the cleaning and preparation of raw or undercooked pork chitterlings, a traditional winter-holiday food in some African-American households (10). Other outbreaks have been associated with water, contaminated milk, bean sprouts, and tofu (3). In affected infants and young children, *enterocolitis* with an inflammatory diarrhea occurs; in older children and young adults, acute terminal ileitis and mesenteric lymphadenitis mimicking appendicitis are common clinical syndromes (1).

Extraintestinal manifestations of *Y. enterocolitica* infection are rare and when reported are usually described as a consequence of septicemia in immunocompromised hosts or in those with iron overload (1). Increased host susceptibility to *Y. enterocolitica* infection from an iron-overloaded state occurs in individuals with conditions such as hemochromatosis, thalassemia, transfusion-dependent blood dyscrasis, and iron chelating therapy. An iron-overloaded state promotes systemic spread by facilitating growth of these organisms and thwarting phagocytic killing and other immune system functions. The clinical course of *Y. enterocolitica* septicemia may include liver and spleen abscesses (12), empyema (2), endocarditis (8), and septic arthritis (4). Occasional cases of bacteremia have been linked to transfusion of contaminated blood products (13). Transient occult bacteremia during a mild self-limited gastrointestinal infection in donors may account for cases of transfusion-associated yersiniosis.

Case reports of nonmesenteric supplicative adenitis due to
Y. enterocolitica and Yersinia pseudotuberculosis in the absence of gastrointestinal symptoms or focus have been described in addition to primary cutaneous infections (7, 9, 11). These suggest an alternative non-food-borne route for Y. enterocolitica transmission. Our patient did not have gastrointestinal symptoms or a febrile course prior to the development of his axillary abscess. The recent history of the finger pustule arising as a consequence of traumatic puncture presents the possibility that direct inoculation from an environmental source may have been the mode of transmission followed by regional spread to the axillary lymph nodes. A similar route of transmission was proposed in a patient with Y. enterocolitica axillary abscess whose employment as a butcher subjected him to frequent cut wounds to the hand (6). Our patient was otherwise healthy, except for chronic hepatitis C infection, although one may speculate that subclinical liver cirrhosis could contribute to impaired reticuloendothelial clearance of this organism or could result in increased susceptibility to infection from impaired iron metabolism (14).

Most intestinal illness caused by Y. enterocolitica is self-limited and generally does not require antimicrobial treatment. However, therapy should be considered in patients with septi-cemia, focal extraintestinal infection, and an immunocompro-mised state with enterocolitis (1, 3). Y. enterocolitica is typically resistant to aminopenicillins and narrow-spectrum cephalosporins; therefore, empirical therapy with commonly used agents such as amoxicillin-clavulanic acid or cephalexin would not provide effective antimicrobial activity in complicated Y. enterocolitica intestinal infection or in extraintestinal infection (5). For serious infections, broad-spectrum cephalosporins or fluoroquinolones are recommended. Trimethoprim-sulfameth-oxazole is also effective for less serious infections.

This case lends further support to the observation that Y. enterocolitica, a classically enteric pathogen, can occasionally present as a primary skin and soft tissue abscess from direct inoculation and extend to cause regional supplicative adenitis.

REFERENCES