Outbreak of Necrotizing Fasciitis Due to Clostridium sordellii among Black-Tar Heroin Users

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In California, black tar heroin (BTH) use among injection drug users (IDUs) has resulted in an increased number of cases of wound botulism due to Clostridium botulinum, tetanus due to Clostridium tetani, and necrotizing soft-tissue infections due to a variety of clostridia. From December 1999 to April 2000, nine IDUs in Ventura County, California, developed necrotizing fasciitis; 4 died. Cultures of wound specimens from 6 case patients yielded Clostridium sordellii. Some of the patients appeared to have the toxic shock syndrome previously reported to be characteristic of toxin-mediated C. sordellii infection, which is characterized by hypotension, marked leukocytosis, and hemoconcentration. The suspected source of this outbreak was contaminated BTH that was injected subcutaneously or intramuscularly (“skin popped”). This outbreak of C. sordellii infection serves as another example of how BTH can potentially serve as a vehicle for transmitting severe and often deadly clostridial infections, and reinforces the need to educate IDUs and clinicians about the risks associated with skin popping of BTH.

Necrotizing soft-tissue infections, including necrotizing fasciitis, are among the most severe and life-threatening infections to affect injection drug users (IDUs) [1–3]. In recent reports of necrotizing soft-tissue infections in IDUs, the anaerobic bacteria recovered have often included clostridial species, most commonly Clostridium perfringens and occasionally others, such as Clostridium sordellii [2–4]. C. sordellii has been reported to cause rapidly progressive myonecrosis with a fulminant shock syndrome, particularly in obstetric patients [5–7], but its role in infections among IDUs has not been well documented.

In California, the widespread illicit use of black tar heroin (BTH) among IDUs since the 1990s has led to an epidemic of wound botulism caused by Clostridium botulinum [8, 9], an increase in cases of tetanus caused by Clostridium tetani [10], and an apparent increase in the number of necrotizing soft-tissue infections caused by Clostridium species [2, 3, 10]. BTH is a dark and gummy form of heroin manufactured in Mexico that is less refined and cheaper than the white powder variety of heroin. BTH is frequently mixed with a variety of diluents, such as dextrose, burned cornstarch, instant coffee, and sometimes even dirt [8]; during this process, bacterial spores can be introduced into the final product.

In February 2000, a local surgery group in Ventura County (VC), California, reported to Ventura County Public Health (VCPH) that there were 7 patients with necrotizing fasciitis who required surgical debridement from December 1999 to February 2000. All 7 patients had histories of injecting BTH subcutaneously or intramuscularly (“skin popping”). At the time of this initial report, 2 patients had laboratory-confirmed Clostridium infections, and 1 patient had died. Here we describe the results of our investigation into this unusual outbreak of necrotizing fasciitis among IDUs, which was likely a result of a contaminated batch of BTH.
**PATIENTS AND METHODS**

**Case finding and epidemiologic review.** A case was defined as illness in a person who (1) was admitted to a VC hospital with a deep-tissue infection that required extensive surgery or was examined by the VC coroner and had deep-tissue infection listed as a cause of death, (2) had a history of heroin or BTH use, and (3) had an onset of illness during the period 1 December 1999 to 1 May 2000.

VCPH requested all VC hospitals and the VC coroner’s office to review their records and report all patients who received a diagnosis of necrotizing fasciitis during the 5-month period from 1 December 1999 to 1 May 2000. Once a case patient was verified as meeting the case definition, hospital records were reviewed by means of a standardized chart-abstraction form to collect demographic information, clinical history, hospital course, medications administered, laboratory tests performed, and hospital costs charged. Attempts were also made to locate and interview surviving patients regarding their medical history and drug use behaviors. For the 1 case reported by the coroner, the autopsy report and death certificate were reviewed.

**Laboratory investigation.** Wound specimens from hospitalized patients were cultured by the clinical laboratories at the admitting hospitals with standard aerobic and anaerobic techniques. Wound specimens from the coroner’s case patient were sent to the VCPH laboratory for performance of aerobic and anaerobic cultures. In addition, the VC Sheriff’s Department confiscated BTH from a heroin dealer during a drug raid in February 2000. A sample of this BTH was sent to the Centers for Disease Control and Prevention in Atlanta, Georgia, to culture for anaerobic pathogens.

**Data analysis.** Chart abstraction data were entered into a Microsoft Access database and analyzed with SAS, version 8.00 (SAS Institute). Because of the small number of cases, the Wilcoxon rank-sum exact test was used to compare continuous variables for patients who survived with those who died.

**RESULTS**

**Descriptive epidemiology.** A total of 9 persons met the case definition; 8 were reported by VC hospitals and 1 by the VC coroner’s office. The dates of illness onset ranged from 1 December 1999 to 8 April 2000. The median age of patients was 45 years (range, 25–57 years) (table 1); 6 (67%) were men. Six patients were Hispanic and 3 were white. None were known to have HIV infection, hepatitis B infection, or diabetes; however, 3 (33%) were known to have hepatitis C infection. Four patients (44%) died, 3 in hospitals and 1 (the coroner’s case patient) at a friend’s home. Six (67%) were known to have used BTH; the specific type of heroin used was unknown for 3 of the patients. All 8 hospitalized patients had histories of skin popping heroin before admission.

**Hospitalized patients.** Seven cases were reported by hospital A (a county facility) and 1 by hospital B (a community facility). Table 1 shows the vital signs at presentation for the hospitalized patients. The median time between symptom onset and hospital admission was 3.5 days (range, 2–7 days). It is notable that all patients were afebrile (median temperature, 36.4°C; range, 34.4°C–37.1°C). Blood pressures ranged from 81/49 to 142/90 mm Hg. Four patients had infection consistent with necrotizing fasciitis in the upper arm/shoulder region and 4 in the hip/buttocks/thigh region. For all 8 patients, surgical debridement was performed after admission; no patients required amputation. Two patients died within 24 h after surgery. Five patients from hospital A were transferred after surgery for

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**Table 1. Hospital admission characteristics of black-tar heroin users with necrotizing fasciitis, Ventura County, California, December 1999–April 2000.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Median value (range)</th>
<th>Median value (range)</th>
<th>Median value (range)</th>
<th>Median value (range)</th>
<th>Median value (range)</th>
<th>Median value (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All patients (n = 8)</td>
<td>Patients who survived (n = 5)</td>
<td>Patients who died (n = 3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>42.5 (25–57)</td>
<td>34 (25–45)</td>
<td>55 (47–57)</td>
<td>.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of days from onset of symptoms to admission</td>
<td>3.5 (2–7)</td>
<td>4 (2–5)</td>
<td>3 (3–7)</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>111.5 (81–142)</td>
<td>113 (102–128)</td>
<td>110 (81–142)</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>69.0 (49–90)</td>
<td>68 (49–86)</td>
<td>90 (49–90)</td>
<td>.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature, °C</td>
<td>36.4 (34.4–37.1)</td>
<td>36.4 (36.3–37.1)</td>
<td>35.6 (34.4–36.1)</td>
<td>.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse, beats/min</td>
<td>102.5 (83–142)</td>
<td>98 (83–140)</td>
<td>110 (98–142)</td>
<td>.29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiration rate, breaths/min</td>
<td>17 (12–40)</td>
<td>18 (16–20)</td>
<td>16 (12–40)</td>
<td>.66</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBC count, ×1000 cells/mm³</td>
<td>28.3 (9.8–61.6)</td>
<td>23.2 (9.8–32.3)</td>
<td>54.3 (54.3–61.6)</td>
<td>.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemoglobin level, g/dL</td>
<td>14.6 (11.7–22.0)</td>
<td>14.0 (11.7–15.1)</td>
<td>21.6 (19.5–22.0)</td>
<td>.04</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Wilcoxon rank-sum exact test comparing patients who survived with those who died.
hyperbaric oxygen treatment to hospital C, where 1 patient died 3 days after surgery.

The 3 hospitalized patients who died and the 5 who survived differed in some characteristics (table 1). Patients who died were older than those that survived (P = .03). Furthermore, at presentation, those who died had higher hemoglobin levels (median of 21.6 g/dL, compared with 14.0 g/dL for survivors; P = .04), and all 3 had extensive involvement of the hip/buttock/thigh region. The patients who died had refractory hypotension despite administration of aggressive fluid resuscitation before death. The cause of death for all 3 hospitalized patients was overwhelming sepsis secondary to necrotizing fasciitis. However, the patients who died did not wait longer after the onset of symptoms before seeking medical treatment than did the patients who survived.

For the 3 hospitalized patients who died, the median hospital charge was $31,763 (range, $9398–$55,597), and the median length of stay before death was 3 days (range, 1–4 days). For the 5 surviving patients, the median hospital charge was $74,179 (range, $7861–$130,439), and the median length of stay was 14 days (range, 5–47 days). Total charge for all 8 hospitalized patients was $398,341. Two patients had private insurance, 1 had Medicaid coverage, 4 were uninsured, and 1 had unknown insurance status.

Coroner’s case patient. The VC coroner’s office reported 1 case patient who died at a friend’s home ~4 days after onset of pain and redness in the left arm. The coroner’s report listed “acute morphine intoxication” as the cause of death and “chronic parenteral drug abuse with acute and chronic necrotizing fasciitis of left arm” as the underlying cause of death.

Laboratory investigation. Wound specimens were collected from all 9 patients. All patients had at least 1 culture that yielded ≥1 species of bacteria. C. sordellii was isolated from 6 of the patients; Staphylococcus species (at least 2 isolates were Staphylococcus aureus) from 4 patients; α-hemolytic Streptococcus species from 3 patients; and Bacillus species from 2 patients (table 2). Blood cultures for all patients, including patients who died, did not yield pathogens. No organisms were recovered from the BTH sample obtained by the VC sheriff’s department.

Patient interviews. VC case workers were able to locate and interview 3 of the 5 surviving patients. Two patients independently mentioned the same park in VC as their source of drugs. The third patient would not reveal his drug source. All 3 reported that they used BTH.

DISCUSSION

We suspect that a batch of BTH contaminated with C. sordellii was sold within VC from the end of 1999 through the early months of 2000, resulting in an outbreak of necrotizing fasciitis among IDUs. During this time, 9 IDUs in VC developed necrotizing fasciitis; 4 died. C. sordellii was the most commonly isolated organism, present in 67% of the patients.

C. sordellii is an anaerobic, gram-positive, spore-forming bacillus that can be isolated from environmental sources and from normal human and animal gastrointestinal contents [5]; however, C. sordellii is occasionally reported as the cause of fulminant infection, most commonly in obstetric patients [6, 7]. C. sordellii deep-tissue infection has also been associated with injection drug use. This organism was the most commonly isolated anaerobic organism (4 of 9 anaerobic isolates) in a case series of IDUs with necrotizing soft-tissue infections at a San Francisco hospital during 1992–1997 [2]. In 1999, another cluster of clostridial myonecrosis occurred among San Francisco IDUs; 2 of 5 patients had wound specimens that yielded C. sordellii [11]. Nonetheless, to our knowledge, our cluster of C. sordellii infections is the largest reported to date, and it serves as another example of how BTH can potentially serve as a

Table 2. Isolates from culture of wound specimens from black-tar heroin users with necrotizing fasciitis, Ventura County, California, December 1999–April 2000.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Date of onset of symptoms</th>
<th>Pathogen(s) isolated</th>
<th>Organism 1</th>
<th>Organism 2</th>
<th>Organism 3</th>
<th>Organism 4</th>
<th>Disposition at discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>14 Dec 1999</td>
<td>C. sordellii</td>
<td></td>
<td>Staphylococcus species</td>
<td>...</td>
<td>...</td>
<td>Alive</td>
</tr>
<tr>
<td>2</td>
<td>28 Jan 2000</td>
<td>Bacillus species</td>
<td></td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Alive</td>
</tr>
<tr>
<td>3</td>
<td>30 Jan 2000</td>
<td>α-Hemolytic Streptococcus species</td>
<td></td>
<td>Staphylococcus species</td>
<td>...</td>
<td>...</td>
<td>Alive</td>
</tr>
<tr>
<td>4</td>
<td>1 Feb 2000</td>
<td>C. sordellii</td>
<td></td>
<td>Clostridium perfringens</td>
<td>...</td>
<td>...</td>
<td>Alive</td>
</tr>
<tr>
<td>5</td>
<td>4 Feb 2000</td>
<td>C. sordellii</td>
<td></td>
<td>Staphylococcus species</td>
<td>...</td>
<td>...</td>
<td>Alive</td>
</tr>
<tr>
<td>6</td>
<td>6 Feb 2000</td>
<td>α-Hemolytic Streptococcus species</td>
<td></td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Alive</td>
</tr>
<tr>
<td>7</td>
<td>21 Feb 2000</td>
<td>C. sordellii</td>
<td></td>
<td>C. perfringens</td>
<td>...</td>
<td>Bacillus species</td>
<td>Dead</td>
</tr>
<tr>
<td>8</td>
<td>1 April 2000</td>
<td>C. sordellii</td>
<td></td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Dead</td>
</tr>
<tr>
<td>9</td>
<td>8 April 2000</td>
<td>C. sordellii</td>
<td></td>
<td>α-Hemolytic Streptococcus species</td>
<td>Staphylococcus species</td>
<td>...</td>
<td>Dead</td>
</tr>
</tbody>
</table>

NOTE. Patients 1–8 were reported by hospitals; patient 9 was reported by the coroner.
vehicle for transmitting severe and often deadly clostridial infections.

Some strains of *C. sordellii* are toxigenic under certain circumstances [7]. *C. sordellii* has been described as having the potential to cause a fulminant toxic shock syndrome characterized by absence of fever, progressive refractory hypotension, tissue edema, marked leukocytosis, and hemoconcentration [7, 12]. This syndrome is thought to be mediated by 2 toxins, lethal and hemorrhagic, that are released by some strains of *C. sordellii* [5–7]. Intradermal injection of these toxins into animals results in necrosis, progressive edema due to local and systemic vascular permeability, and death, a process similar to that seen in some *C. sordellii* infections in humans [13, 14]. It is plausible that some of our patients may have skin-popped BTH contaminated with *C. sordellii*, which led to toxin production, which in turn resulted in severe necrotizing disease, toxic shock syndrome, and death.

Injecting BTH into soft tissue rather than the bloodstream increases the risk of abscess formation, and the resultant devitalized tissue sets up an anaerobic environment conducive to clostridial toxin formation [9]. Wound botulism occurs after spores of *C. botulinum* germinate in a wound and produce botulinic toxin, resulting in flaccid paralysis [8, 9, 15]. In California, the primary risk factor for wound botulism is skin popping of BTH [8]. Similarly, tetanus develops when *C. tetani* introduced into a wound germinates and releases toxin [16]. Nearly one-half of the cases of tetanus reported in California between 1988 and 2000 were associated with injection drug use; of these, at least 60% were associated with reported heroin or BTH use [10]. We suspect that the conditions that lead to toxin production by *C. sordellii* are similar to those that lead to toxin production by *C. botulinum* and *C. tetani* [8–10].

Clostridial contamination of heroin has been documented. In Scotland, Ireland, and England in 2000, an outbreak of serious soft-tissue infections, mainly due to *Clostridium novyi*, occurred among IDUs who skin-popped heroin [17, 18]. A contaminated supply of heroin was suspected to be the source; 2 species, *C. perfringens* and *Clostridium saccharolyticum*, were isolated from heroin belonging to the patients [17]. In the 1999 San Francisco cluster, 3 of the 5 patients were roommates who shared a supply of BTH [11]. One roommate had confirmed *C. perfringens* infection, and another had *C. sordellii* infection. BTH confiscated from their apartment yielded *C. perfringens*, demonstrating that it was likely the common source of their necrotizing soft-tissue infections [11].

In our outbreak investigation, no anaerobic organisms were isolated from the sample of BTH confiscated during the drug raid; however, the BTH was not necessarily from the suspected supply that made the VC patients ill. Nonetheless, evidence suggests that BTH can be contaminated by clostridia before purchase by the user, most likely when being mixed with diluents [8, 9]. Although we cannot rule out the possibility that the patients in our study became infected through contaminated needles or other drug-injection paraphernalia, it is most likely that the contaminated BTH was the source of the necrotizing infections. This hypothesis is supported by the following evidence: (1) interviews with 3 surviving patients confirmed that they did not know each other and did not share paraphernalia but did use BTH; (2) of the interviewed patients reported that they had purchased BTH at the same VC park; (3) *Clostridium* species have previously been isolated from BTH [11], so the hypothesis is biologically plausible; and (4) there is an ongoing California epidemic of infections due to 2 other toxin-producing *Clostridium* species, *C. botulinum* and *C. tetani*, also due to skin popping of contaminated BTH [8–10].

Necrotizing fasciitis requires aggressive and timely surgical debridement to prevent rapid progression of infection [19]. However, necrotizing fasciitis can sometimes be difficult to distinguish from simple cellulitis or abscess, creating a diagnostic challenge [2]. Physicians should evaluate for necrotizing fasciitis whenever an IDU presents with an infection at an injection site. It is notable that, among the cases in our cluster, marked leukocytosis, high hemoglobin count, and lower body temperature at presentation were associated with death. Although only 2 of the 3 hospitalized patients who died had cultures that yielded *C. sordellii*, all 3 seemed to develop the toxic shock syndrome reported to be characteristic of toxin-mediated *C. sordellii* infection [5–7]. Physicians should be alerted to the presence of this syndrome; survival may be improved by prompt recognition and aggressive treatment. Antitoxin treatment of this toxic shock syndrome due to *C. sordellii*, similar to the treatment given for botulism and tetanus, has been suggested [5–7]. Further research into the effectiveness of this treatment is needed.

Treatment of necrotizing fasciitis is costly because of the need for wound debridement, treatment with multiple antibiotics, hyperbaric treatments, and lengthy hospital stays. The total charge for the 8 hospitalized patients in this investigation was ∼$400,000; only 2 patients had private health insurance. This imposes a large financial burden on the health care system that could potentially be reduced through aggressive education of IDUs and physicians.

Although many IDUs understand the risks of contracting hepatitis and AIDS from injecting drugs, most may be unaware of the additional and potentially lethal health risks associated with skin popping of BTH, such as the development of necrotizing fasciitis. IDUs should be instructed to seek medical attention at the first sign of infection at an injection site because of the rapid progression and high fatality rate of necrotizing fasciitis. Correctional facilities, halfway homes, methadone clinics, and other agencies caring for IDUs should be used as venues to educate IDUs about the dangers of skin popping of BTH.
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References