Necrotizing Fasciitis Complicating Intravenous Drug Abuse

Jeffrey M. Jacobson, MD, Shalom Z. Hirschman, MD

- Two cases of necrotizing fasciitis in intravenous cocaine abusers are presented to alert the medical community to the possibility of these severe infections in such addicts. Antimicrobial therapy should include agents effective against *Bacteroides* species, streptococci, and Gram-negative aerobes; therapy directed only against staphylococci and Gram-negative aerobes is not sufficient.

(Arch Intern Med 1982;142:634-635)

Intravenous (IV) narcotic abuse is often complicated by serious infections, including localized cellulitis or abscess,\(^1\) bacteremia with septic arthritis,\(^2\) osteomyelitis or metastatic abscesses,\(^3\) bacterial or fungal endocarditis,\(^4\) pneumonia,\(^5\) viral hepatitis,\(^6\) and malaria.\(^6\) We describe two patients in whom necrotizing fasciitis developed following IV injection of cocaine.

REPORT OF CASES

CASE 1.—A 52-year-old man who was an IV drug abuser and an insulin-dependent diabetic was admitted to The Mount Sinai Hospital, New York, for further treatment of necrotizing fasciitis of his left upper extremity. The patient had abused heroin for 25 years, until three years prior to admission, when he enrolled in a methadone hydrochloride maintenance program. Nine months before admission he began to use IV cocaine.

The patient was admitted to a local hospital on April 30, 1981, with a one-week history of progressive pain, swelling, and erythema of his left forearm, the site where cocaine had been injected just before the onset of symptoms. On admission there, he had a rectal temperature of 38.3 °C, and swelling and erythema of the left forearm. The WBC count was 20,200/cu mm, with 65 neutrophils, 19 band forms, eight lymphocytes, seven monocytes, and one eosinophil. A diagnosis of superficial cellulitis was made and cefazolin sodium, 1 g IV every seven hours, was administered. On May 1, 1981, roentgenograms revealed air in the soft tissues. The wound was incised and 100 mL of pus was evacuated; gentamicin sulfate therapy, 60 mg IV every eight hours, was added. Over the next few days the cellulitis spread, drainage increased, and crepitation formed. On May 4, 1981, the wound was incised and necrotic muscle, fascia, and fat tissue were removed; clindamycin phosphate therapy, 600 mg IV every six hours, was added. Further debridement of necrotic tissue was carried out the next day. Culture of the wound yielded *Bacteroides* species and viridans streptococci.

On May 6, 1981, the patient signed out of the local hospital against medical advice and came to The Mount Sinai Hospital. His rectal temperature was 39.4 °C and his left upper extremity was exposed anteriorly from the wrist to the elbow. There was a minimal amount of necrotic tissue. The venous hematocrit level was 31.5% and the WBC count was 22,000/cu mm, with 81 neutrophils, eight band forms, seven lymphocytes, and three monocytes. The blood glucose level was 254 mg/dL and blood electrolyte levels were normal. Roentgenograms showed gas in the soft tissues. In the operating room, multiple pockets of pus were found tracking along muscle groups proximally and distally, with necrosis of all flexor muscles of the arm and forearm. The periosteum of the humerus and extensive areas of subcutaneous tissue were also necrotic. The patient had given penicillin G potassium, 6 million units IV every four hours, clindamycin phosphate, 600 mg IV every six hours, and tobramycin sulfate, 80 mg IV every eight hours, as well as daily hyperbaric oxygen treatments. However, on May 8, 1981, because the necrotizing fasciitis had extended onto the pectoral major muscle as well as...
widely and deeply into the flexor compartment of the hands, a left shoulder disarticulation was performed. On May 20, 1981, further debridement and drilling of the left glenoid fossa were carried out. Penicillin G therapy was discontinued on May 13, but clindamycin and tobramycin therapy were continued for four weeks. The wounds had healed well by the time the patient was discharged.

Case 2
A 39-year-old man who was an IV drug abuser was admitted to The Mount Sinai Hospital on May 9, 1981, because of swelling and pain in the left forearm.

The patient had been an IV heroin abuser over the last five years. Despite recent enrollment in a methadone maintenance program, he indulged in IV drug abuse, including both heroin and, for the first time, cocaine in the week before hospitalization. Four days prior to admission an injection of cocaine into his left forearm caused a burning sensation and, the next day, the patient noted the onset of stiffness of the left wrist and lower part of the forearm. Two days later the lower part of his forearm began to swell, with gradual spread both distally to his fingers and proximally above his elbow. The arm became increasingly painful, and the day prior to admission, black discoloration and bullae formed at the site of the cocaine injection. He inserted a pin into a bulla and foul-smelling, grayish pus began to ooze. He became nauseated and vomited, but did not note fever or chills.

On admission, the patient was a young man in notable pain, appearing acutely ill. The rectal temperature was 38.1 °C. The entire left upper extremity was edematous to the shoulder. There was a 9 × 5-cm necrotic ulceration of the volar aspect of the left forearm with extensive subcutaneous erythema; grayish-black, foul-smelling pus oozed from the ulcer. The radial pulse was diminished, but there was no neurological deficit.

The venous hematocrit level was 41% and the WBC count was 15,300/µl, with 56 neutrophils, 32 band forms, eight lymphocytes, and four monocytes. A roentgenogram of the left arm showed gas in the deep tissues extending to the elbow. Gram’s stain of the pus was reported by the house staff as showing only Gram-positive cocci.

A diagnosis of necrotizing fasciitis was made and the patient began receiving IV penicillin G potassium, 6 million units every four hours. The next morning he was taken to the operating room, where a wide incision and extensive debridement of necrotic fascia, muscle, and tendon were performed; multiple microabcesses were noted. Further debridement was performed on the third and fifth hospital days. By then the cellulitis had spread up the inner aspect of the left upper arm and onto the anterior chest wall. More necrotic muscle and pockets of pus were found and an infectious disease consultant was called. Review of the original Gram’s stain revealed many WBCs and a mixture of Gram-positive cocci and anaerobic Gram-negative bacilli. Cultures of the wound yielded *Aerococcus* species, *Peptostreptococcus* species, *Propionibacterium acnes*, *B melaninigenicus*, viridans streptococci, nonhemolytic streptococci, *Escherichia coli*, *Klebsiella pneumoniae*, and *Actinobacter calcucae*.

Antimicrobial therapy was changed to penicillin G potassium, 4 million units IV every six hours, metronidazole, 500 mg IV every eight hours, and tobramycin sulfate, 90 mg (1 mg/kg) IV every eight hours, and continued for two weeks. Over the ensuing 24 hours, the advancing edge of cellulitis reeded and now necrotic tissue formed. The patient has regained almost full use of the involved extremity.

**COMMENT**

Necrotizing fasciitis, an unusual infection of the subcutaneous and fascial tissues, causes dramatic local disease as well as systemic toxic effects. It begins similarly to a cellulitis with localized edema, erythema, and tenderness. As the area of erythema spreads with a variable amount of edema, the central zone may turn purple and then become necrotic with eventual ulceration. Vesicles and bullae may also form. The presence of subcutaneous crepitation and gas in soft-tissue roentgenograms is variable. On incising the lesion, serosanguineous fluid is obtained and gray or frankly necrotic fascia is readily visualized. Thus, subcutaneous gas, central ulceration, and inordinate systemic toxic effects are important diagnostic clues.

Although group A β-hemolytic streptococci were initially implicated in this infection, it is now generally believed that a mixed infection of both aerobes and anaerobes is usually present. Eighty percent of cases are believed to be related to minor trauma and insect bites, with the remainder following a surgical procedure. The extremities are the most common sites of infection, with greater incidence in the lower than upper extremities. Infections occurring on the abdomen are usually subsequent to surgery.

Cellulitis frequently develops in narcotic addicts at the site of IV injection. As already stated, the earliest manifestations of necrotizing fasciitis are indistinguishable from cellulitis. The two cases described herein demonstrate the potential for necrotizing developing fasciitis from contaminated IV injection. Under such circumstances, it should be realized that localized erythema and tenderness might indicate more than mere cellulitis. A careful evaluation and close observation should rule out the potentially more devastating possibilities of necrotizing fasciitis or clostridial anaerobic cellulitis and myonecrosis, the treatment of which involves much more aggressive surgical debridement and a different choice of antimicrobial agents. Most cellulitides in drug addicts are treated with an isoxazolyl penicillin or a cephalosporin, to cover the β-lactamase-producing staphylococci, often in combination with an aminoglycoside to include coverage of Gram-negative aerobes. However, anaerobes, including *Bacteroides* species, are frequent pathogens in necrotizing fasciitis. We suggest that an agent active against *Bacteroides* species, such as clindamycin, cefoxitin sodium, or metronidazole, be given in combination with IV penicillin G and an aminoglycoside, to IV cocaine abusers who have signs of cellulitis at injection sites. Clindamycin and cefoxitin have the added advantage of activity against staphylococci and may be used before the true nature of the infection is established.

The pathogenesis of necrotizing fasciitis in IV cocaine abusers can only be speculated on. Cocaine constricts blood vessels. Both of our patients reported that the cocaine injections preceding their infections missed the veins. Such subcutaneous intramuscular infiltration of a vasoconstrictor, with contaminated material, could provide the appropriate ischemic and anaerobic environment for necrotizing fasciitis to develop. More experience with this disease, and studies in experimental animals, will be necessary to support this hypothesis.

**References**


Arch Intern Med—Vol 142, March 1982

Necrotizing Fasciitis—Jacobson & Hirschman 635

Downloaded From: https://jamanetwork.com/ by a Non-Human Traffic (NHT) User on 03/29/2020