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Toxic shock syndrome after a human bite to the hand

The first known case of toxic shock syndrome resulting from a human bite is reported. An awareness of the presenting features and clinical manifestations are needed in the early recognition and intensive medical management of this life-threatening condition. (J HAND SURG 1988;13A:957-9.)

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Toxic shock syndrome (TSS), first described in 1978 by Todd et al.[1] is an acute illness affecting three or more organ systems and is characterized by hypotension, fever, hyperemia of the mucous membranes, and erythroderma with subsequent desquamation of skin. TSS has been reported after use of highly absorbent tampons during menstruation,[2] as a complication after elective surgical procedures,[3-7] and in association with cutaneous and subcutaneous infections. To our knowledge, this is the first report of TSS in a patient after a human bite.

Case report

A 21-year-old Hispanic man, in good health, lacerated the skin over the fifth metacarpophalangeal joint of his right hand when he struck another man in the mouth. Two days later he consulted a physician because of pain and swelling of the hand, fever, chills, nausea, diarrhea, and vomiting. He received an injection of an antibiotic and oral antibiotics were prescribed. The following day he could not retain medication, food, or water, and had generalized myalgia and dizziness. He came to the hospital emergency room the fifth day after injury. He was lethargic and confused. His vital signs were as follows: blood pressure, 50/0 mm Hg; oral temperature, 103° F; pulse, 135/min; and respiratory rate, 24/min. There was a diffuse macular erythrodermatous rash of the entire body. His lips were parched and the mucous membranes were dry. The chest was clear and the abdomen was soft and nontender. A 6 mm open wound was present over the metacarpophalangeal joint of the small finger and although the hand was swollen, there was minimal drainage (Fig. 1). The extremities were not edematous and there were no palpable epitrochlear or axillary nodes. X-ray films of the right hand were normal. Chest x-ray films showed mild, diffuse, fluffy interstitial alveolar infiltrates.

Normal saline solution was administered intravenously until the patient had received 8 liters. In addition, he received oxygen, diphenhydramine hydrochloride (Benadryl) 50 mg, intramuscularly, epinephrine, 0.4 mg, subcutaneously, cefoxitin, 2 gm intravenously, acetaminophen, 10 grains by rectal suppository, methyl prednisolone sodium succiuate, 250 mg, intravenously, hyperimmune globulin 250 Units intramuscularly, and tetanus toxiod 0.1 ml subcutaneously. Wound, blood, and throat cultures were obtained and Swan-Ganz catheter monitoring was instituted. Twelve hours after admission, systolic blood pressure was 90 mm Hg. He was taken to the operating room and with the patient under local anesthesia, the wound and metacarpophalangeal joint of the small finger were explored. There was a small amount of purulent exudate in the metacarpophalangeal joint and a tiny defect in the articular cartilage of the metacarpal head. The soft tissues were not necrotic. The wound was irrigated and packed open, and he returned to the surgical intensive care unit.

Blood cultures and throat cultures showed no growth at 72 hours, and a repeat chest x-ray film was negative. The initial wound culture grew *Staphylococcus aureus* coagulase positive. Initial laboratory studies included a white blood cell count of 8.5 ($\times 10^3$ per cubic millimeter), with 58.1% neutrophils. Total platelet count was 37,000/mm^3. Electrolyte studies revealed a blood urea nitrogen (BUN) value of 68 mcg/L and a creatinine level of 8.4 mg/dL, a total calcium level of 5.0 mmol/L, a blood level of 88 U/L, and a creatinine phosphokinase of 4280 U/L. Coagulation factors were prolonged with a partial thromboplastin time of 51 seconds (control, 30) and a prothrombin time of 12.5 seconds (control 10). The patients urine toxicology screen was normal.

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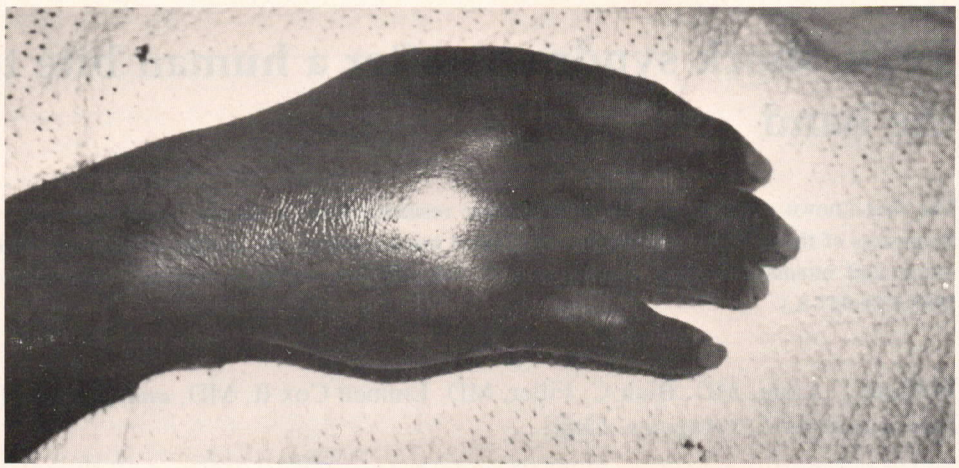

Fig. 1. In the emergency room the dorsum of the right hand was swollen and erythematous. A laceration at the fifth metacarpalphalangeal joint caused bleeding and a minimal amount of purulent exudate was noted.

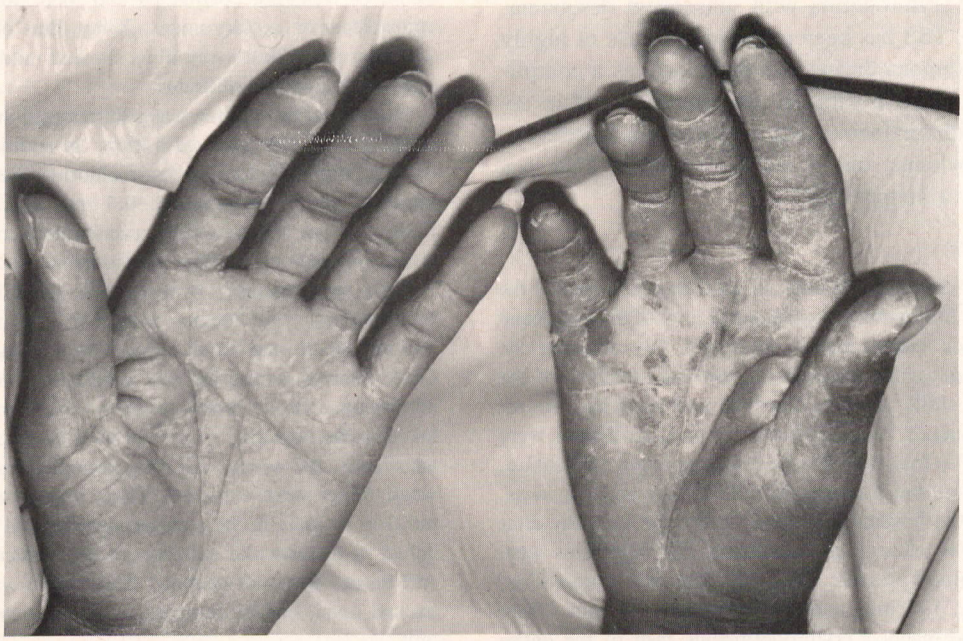

Fig. 2. Desquamation of the hands occurred 16 days after the onset of symptoms.

On the second postoperative day, the patient's temperature and blood pressure were normal. He still had myalgia, diarrhea, and dizziness, but the erythematous rash began to resolve. By the fourth postoperative day all symptoms had cleared except for desquamation of the skin on the neck and upper chest. Desquamation of the hands commenced on the sixteenth day (Fig. 2). He was discharged from the intensive care unit on the fifth postoperative day. Nafcillin and penicillin, administered intravenously, were continued. Results of all laboratory tests were normal by the sixth postoperative day. He was discharged from the hospital 14 days after admission. The wound of injury healed by secondary intention.

Discussion

Systemic toxicity associated with staphylococcus scarlet fever was described in 1927,[8] but the syndrome now termed "toxic shock" was seldom recognized or diagnosed until 1978.[1, 9, 10] TSS has been reported as a postoperative orthopedic complication after arthros-

copy,[14] osteoplasty of the hip,[4] skin grafts,[6] and arthrodesis of the wrist,[5] and after other orthopedic procedures. In 1986, Kreiswirth et al.[11] stated that approximately 200 cases of TSS had been reported after operations. A fatality rate of 7% to 10% has also been reported.[3-12] TSS can be transmitted to patients by surgeons.[11]

Staphylococcus aureus has been cultured from 91% of wounds associated with toxic shock, and recent studies have implicated nonsuppurative *Staphylococcus aureus* phage I, types 29 and 52.[11, 14] One hundred percent of the *Staphylococcus aureus* organisms isolated and cultured from patients with TSS have produced exotoxin. Exotoxin C and enterotoxin F have also been identified and have been labeled TSST-I by Bergdoll et al.[12, 13] These toxins are thought to cause massive vasodilatation and a shift of fluid and red blood cells from the intravascular to the extracellular compartment. *Staphylococcus aureus* producing TSST-I is collagenase positive and produces β-lactamase and often penicillinase,[2] and a wound contaminated with this particular staphylococcus usually appears benign.

The diagnosis of TSS is usually made from clinical findings, and by exclusion, because there is no conclusive laboratory test for this syndrome. An abrupt onset of fever greater than 38.9 degrees C, hypotension associated with a diffuse erythematous blanching rash should suggest this diagnosis. Involvement of the gastrointestinal system causes nausea, vomiting, and diarrhea, involvement of the muscular system causes myalgia and arthralgia, involvement of the central nervous system causes confusion and agitation, and involvement of the renal system causes an elevated blood urea nitrogen (BUN) and creatinine level, When the hepatic system is involved, results of liver enzyme studies will be abnormal. The mucous membranes, especially the throat, are often affected; the patient is dehydrated, and the platelet count is diminished. Blood cultures and throat cultures are almost always negative in contrast to septic shock syndrome, when they are usually positive.[7, 14]

The differential diagnosis includes acute viral syndrome, acute rheumatic fever, acute pyelonephritis with hemolytic uremic syndrome, gastroenteritis, Legionnaire's disease, Kawasaki's disease, leptospirosis, pelvic inflammatory disease, Rocky Mountain spotted fever, septic shock, streptococcal scarlet fever, cystic lupus erythematosus, and tic typhus; diagnosis can usually be confirmed by a specific laboratory test for most of these conditions.

Once the diagnosis is made, immediate treatment is mandatory. Quantities of normal saline solution should be given, electrolytes should be monitored by a Swan-Ganz catheter, and oxygen is administered. There is some evidence that steroids may ameliorate the effect of the toxin.[15] An anti-β-lactamase antibiotic, such as nafcillin, cephalosporins, methicillin, or vancomycin may be of some benefit.[2] The toxin rather than the bacteria seems to be the offending agent, so antibiotics are of limited benefit. However, they may decrease the number of bacteria producing the toxin, and this may be helpful.

Local incision of the wound with debridement and irrigation may decrease the number of bacteria and limit the production of toxin.

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