Fatal Infection after a Bee Sting

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Life-threatening or even fatal infections can rarely develop after bee stings.

Deaths resulting from bee stings are uncommon [1, 2]. When they do occur, the deaths are usually due to anaphylactic shock, suffocation after stings in the airways, or preexisting diseases, such as atherosclerotic heart disease [2]. Deaths due to massive envenomation have also been noted, especially among individuals stung by so-called Africanized honeybees, which attack in great numbers [3]. There are rare reports of local [4] and disseminated [5–7] infections after bee stings, none of which proved fatal. We report a case of fatal disseminated infection after a bee sting; to our knowledge, it is the first such case ever reported in the English-language medical literature.

The left hand of a 71-year-old man was stung by a bee. The man had undergone aortic valve replacement 12 years before presentation, and he had undergone placement of a cardiac pacemaker 2 years before presentation. Less than 2 weeks after the bee sting, intense pain in the left hand, malaise, and chills developed. Within 24 h of the onset of these symptoms, the patient developed severe pain in the right foot, which led to an inability to bear weight on the right leg. The patient became increasingly lethargic, was moaning, and eventually fell out of bed, which prompted his admission to a hospital. At the time of admission, his temperature was 41.4°C, and his systolic blood pressure was 70 mm Hg.

The patient was transferred to our institution, The Johns Hopkins Hospital, Baltimore, for evaluation of presumptive prosthetic valve endocarditis. Our initial evaluation revealed marked edema of the left hand and right foot, with purpura and break-down of the skin. The patient was treated with broad-spectrum antibiotics for suspected sepsis. An echocardiogram showed poor left ventricular function with global hypokinesis, but no evidence of aortic valve vegetations. The patient underwent intubation. Meanwhile, at the first hospital where the patient was treated, the results of culture of blood samples became positive for group A β-hemolytic Streptococcus species (Streptococcus pyogenes). The patient became progressively hypotensive, ventilator dependent, and deeply comatose. His left arm and right leg were described as “necrotic.” In view of the grim prognosis, the attending physicians and the patient’s family decided to institute a “do not resuscitate” order. The patient died 4 days after the onset of symptoms.

An autopsy was performed (excluding examination of the brain). There were multiple bullous lesions of the skin that involved the left hand and distal forearm, right distal lower extremity, scrotum, and left earlobe; these lesions were identified as toxic epidermal necrolysis associated with bacterial exotoxin. There was erythema and induration of the entire left upper extremity and the distal right lower extremity to the level of the midcalf. Histological examination of the lower extremity lesions revealed acute inflammation and frequent gram-positive cocci within the subcutaneous soft tissue. The heart had several microscopic abscesses within the right ventricular free wall, in the region of the pacemaker wire anchoring, and these contained numerous gram-positive cocci (figure 1). There was no evidence of aortic valve vegetations. The left ventricular myocardium showed patchy replacement fibrosis but no distinct infarcts. There was no significant narrowing or thrombosis of the coronary arteries. In addition, there was histological evidence of disseminated intravascular coagulation and ischemia of multiple organs and tissues.

Infections resulting from bee stings are rare and have not been studied as a medical phenomenon. Klug et al. reported a case of toxic shock syndrome in a 25-year-old man whose lower back had been stung by a bee [5]. Their patient had a pustular lesion with peripheral induration at the site of the sting; culture of a specimen from the lesion yielded Staphylococcus aureus. The patient exhibited classic signs of toxic shock syndrome, but he recovered relatively quickly. Richardson and Schmitz [6] reported a case of chronic relapsing cervicofacial necrotizing fasciitis caused by a bee sting on the eyelid of a 61-year-old patient with diabetes. The patient had group A β-hemolytic Streptococcus sepsis and severe necrotizing deep fascial infection of the neck and face, which assumed a relapsing course. This led to temporary multisystem organ failure that required intensive medical care and multiple surgical procedures that excised a major amount of facial tissue. Ultimately, however, the disease was not fatal. Anderson et al. [7] reported a case of...
disseminated infection with numerous microorganisms, including *Pseudomonas aeruginosa*, *Enterococcus faecalis*, *Xanthomonas maltophilia*, and coagulase-negative *Staphylococcus* species, in a 4-year-old girl who had massive breakdown of the skin as a result of eosinophilic cellulitis (Wells' syndrome) after a bee sting to the left foot. Finally, Shahar and Frand [4] reported a case of *Pseudomonas aeruginosa* arthritis and osteomyelitis of the foot in a 10-year-old boy whose foot had been stung by a bee [4]. In that case, the infection appears to have remained localized.

Insect bites can lead to severe infections, including necrotizing fasciitis [8]. However, the extent to which bees contribute to this problem is virtually unknown. No field studies have been performed to determine the degree to which bees are contaminated by pathogenic microorganisms, and the exact sequence of events that lead to infection of bee stings is unknown. Several mechanisms can be suggested.

The abdomen of the honeybee is covered with numerous hairs, most of them branched and plumelike [9], to which pathogenic bacteria could attach. It is known that honeybees are occasionally attracted by garbage [3], which may further contaminate them with pathogens.

The sting of the honeybee is a complex organ, consisting of a stylet, 2 barbed lancets, and a venom sac. When the bee uses its sting, the sting becomes deeply embedded in the skin and briskly advances by alternating the thrusts of both lancets, which, because of the direction of their barbs, can only move forward. Meanwhile, the venom from the sac is injected into the victim. Eventually, the entire stinging mechanism detaches from the body of the bee (which kills the bee); however, because of the automatism of the intrinsic muscles, the lancets continue to advance, and the venom is continuously pumped [3, 9]. In this manner, any bacteria on either the insect's body or its sting, or on the surface of the victim's skin, can be inoculated under the epidermis. Unless carefully removed, the sting remains in the wound and may facilitate introduction of the infection.

The venom of the honeybee contains numerous enzymes and biologically active substances [3], which produce, among other effects, extensive local swelling and degranulation of mast cells. Itching is usually associated with the sting [10], and scratching can cause further epidermal injury and intradermal implantation of pathogenic bacteria. Furthermore, the edema surrounding the sting site may temporarily impair lymphatic drainage and reduce clearance of the infection by the immune system.

In brief, there are numerous mechanisms by which bee stings can cause local and systemic infection. The case we describe has several important features. First, the infection progressed remarkably rapidly and caused the patient to die. There was no evidence to suggest that our patient was immunocompromised. The reasons for his cardiac surgery, unfortunately, were unavailable. His cardiac function may have been impaired before the infection, although there were no specific myocardial changes or coronary artery lesions, which could have led to a more precipitous circulatory collapse and death. Second, it is unknown whether establishment of a local infection in the patient's heart was related to the presence of the pacemaker electrodes. Finally, although the subcutaneous inflammation in the patient's extremities was diagnosed as cellulitis at the time.

**Figure 1.** Microabscess in the right ventricular myocardium of a patient in whom group A β-hemolytic *Streptococcus* infection developed after he was stung by a bee. Left, Hematoxylin and eosin stain showing focal cardiomyocyte necrosis, hemorrhage, and numerous polymorphonuclear leukocytes (original magnification, ×100). Right, Gram-Weigert stain showing numerous gram-positive cocci surrounding cardiomyocytes (original magnification, ×630).
of autopsy, it is possible that the development of necrotizing fasciitis in this patient was either imminent or ongoing. This is particularly possible because sepsis was severe and because group A β-hemolytic Streptococcus species have a propensity to cause necrotizing fasciitis. Further research is needed to determine the relationship between honeybee stings and infection.

References