CLINICAL CASE REPORT

Toxic inoculation associated with a presumptive stingray injury

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ABSTRACT

Introduction: Stingray spine injuries are among the most common marine animal injuries in humans. While most resolve with immersion in warm water, a few become infected and require antibiotics. We present a case report of a presumptive stingray injury that evolved to a major slough and which required prolonged healing in a patient with diabetes mellitus. Our literature review was unable to find a similarly reported case.

Materials: A co-author was asked to evaluate and manage an ominous-appearing wound on the right foot of a diabetic. The problem developed after the individual had been wading in shallow ocean beach water. The patient's diabetic sensory neuropathy obscured the immediate association of the problem with a stingray injury, but this became the presumptive diagnosis when pain developed and necessitated that he seek medical care.

Findings/Clinical Course: After an initial urgent care visit, increasing pain and worsening appearance of the patient's foot necessitated a visit to our emergency department. The patient was admitted the next day due to symptoms of systemic sepsis. On the fourth hospital day, a large bulla on the lateral side of the right foot was excised. This unroofed a full-thickness slough to the periosteum level of the underlying bones. Not until the 16th hospital day had enough improvement occurred to discharge the patient. Over the next 16 weeks, the wound improved, developed a vascular base and epithelialized.

Conclusions: With a dearth of literature about stingray injuries in patients with diabetes mellitus reported, our case is unique: The patient's wound course more closely resembled a toxic inoculation than the typical puncture wound-cellulitis presentations associated with stingray injuries.

INTRODUCTION

From 1950 to 2006, the incidences of reported stingray injury in the United States ranged from 750 to 2,000 each year [1]. Most of these injuries affect the feet and/or lower extremities, typically when a victim inadvertently steps on the animal in shallow waters. A common sequela of stingray injuries is intense pain, minor soft tissue injury, and infection. Generally, puncture injuries from stingrays are resolved with hot-water immersion, local wound care, and antibiotics [2]. On rare occasions fatalities have resulted from stingray injuries that penetrate a major vessel or organ. One such case occurred with well-known naturalist Steve Irwin in 2006 when an Australian bull ray's barb penetrated his heart. Given recent reports of warmer water temperatures that enable stingrays to migrate closer to shores, the number of injuries from these animals is expected to rise [3].

Stingrays are members of the shark family. They have flat bodies that allow them to rest or hide under sand in shallow water at the beach. Stingrays are generally docile animals; they do not attack or even defend themselves, swimming away from danger when they can. However, they have tails with dorsally located spines that they use to whip over their bodies when they are disturbed. These spines are sharp and serrated, and can tear skin and soft tissues, as well as lacerate tendons and ligaments. In addition, the spines are encased in an integumentary sheath that contains proteinaceous material. When this sheath ruptures and the material is released into the wound, it causes intense pain and possible tissue necrosis [4].

We describe a presumptive stingray injury to a diabetic patient's foot with an unusual presentation and an atypical clinical course. Because of the massive slough, healing challenges, and prolonged healing, we feel this case report deserves sharing with those care providers who need to evaluate and manage injuries from marine animals.

KEYWORDS: clinical toxicology; diabetes; diving incidents; incidents; marine animals; stingray; toxins; wounds

CASE REPORT

A 36-year-old male was at an ocean beach wading in shallow, warm summer waters when he felt an intense sharp pain to his right foot. He came out of the water and noted his foot was bleeding actively from a puncture site. The patient went to an urgent care clinic, where he was given a tetanus shot, his wound was cleansed, and he was discharged home. Later that day, due to increasing pain, he presented to our hospital emergency department, where he was given insulin for a blood glucose (BG) level over 400 mg%. Of note, the patient had been diagnosed with diabetes mellitus five years prior, but had never taken medication for it nor monitored his BG levels.

His foot was soaked in warm water, which markedly improved his pain symptoms. As his foot had only a small scab with no signs of infection, he was discharged home with the medications doxycycline and metformin.

The patient returned to the hospital the next day due to increased pain, fever, and chills. He was admitted with a white blood cell count (WBC) of 26.7, tachycardia and signs of erythema, edema, and ecchymosis in the right foot consistent with infection. Piperacillin/tazobactam (Zosyn®) was administered intravenously at this time. The patient had a small linear scab about 1 cm in length embedded with small barbs consistent with a stingray spine. The patient said that he never saw what caused the wound.

While hospitalized, this wound quickly evolved to bulla formation and surrounding erythema (Figure 1). The initial culture and sensitivity report obtained from the wound showed rare growth of *Streptococcus salivarius* and *Streptococcus viridans* groups. The intravenous antibiotics were continued and the wound was covered with dry gauze.

Three days after hospital admission, a consultation for wound evaluation and management was requested. At that time the patient's WBC was 13.6 with hemoglobin A1c (HgA1c) 12.3. A bulla, partly blood and partly serous fluid, measured about 3 x 8 square cm and was located over the dorsal, lateral aspect of the right foot. Sharp debridement of the bullae and its amorphous base to the tenosynovium and periosteum of the underlying bones was completed. Silver sulfadiazine (Silvadene®) was used as the wound-dressing agent. Pain was associated with dressing changes and was managed with hydrocodone/paracetamol (Norco®).

After the debridement and eight days since the injury occurred, the patient's WBC decreased to 11.3. However, the wound base remained worrisome, with minimal development of vascular tissue (timeline, Figure 1). Negative pressure wound therapy (NPWT) was initiated on the 10th post-injury day. On Day 12, the WBC had normalized to 7.5. The wound vacuum dressing was discontinued at this time because the wound base appeared more avascular and the periwound skin had become macerated with new areas of necrosis along the proximal medial edge of the wound. Management of the wound was switched to daily moist saline dressings; then on Day 13 it was changed to acetic acid solution (AAS) moist dressings.

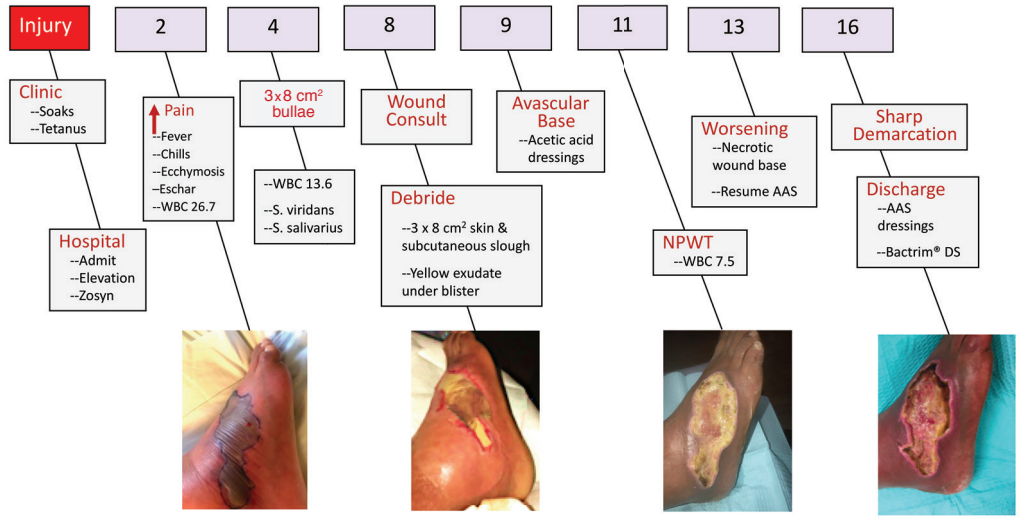
On hospital day 15 the slough areas around the periphery began to demarcate, faint signs of vascularity were appearing in the wound base, and the wound margins were starting to contract. With these improvements, the patient was discharged on the 16th hospital day on sulfamethoxazole/trimethoprim (Bactrim DS®). Daily AAS dressing changes were to continue at the patient's home. Arrangements were made for weekly follow-up appointments at our Wound Healing Center.

After hospital discharge, the wound initially stagnated (Figure 2). Management was changed from AAS dressings to the collagenase, Santyl®, and finally, again to moist saline dressings. Each wound dressing agent was used for two to three weeks. Another culture and sensitivity study obtained in the clinic showed a light growth of *Pseudomonas aeruginosa* and a heavy growth of group B Streptococcus. The patient was continued on sulfamethoxazole/trimethoprim (Bactrim DS®). After two months healthy granulation tissue began appearing in the wound base, and the wound improved at an accelerated rate (time line in Figure 2). By the time the wound base was ready for split-thickness skin grafting, enough marginal epithelialization had occurred that the decision was made to allow for coverage by secondary intention. By the 16th post-injury week, the wound site had epithelialized completely and the patient was cleared to return to work without restrictions.

DISCUSSION

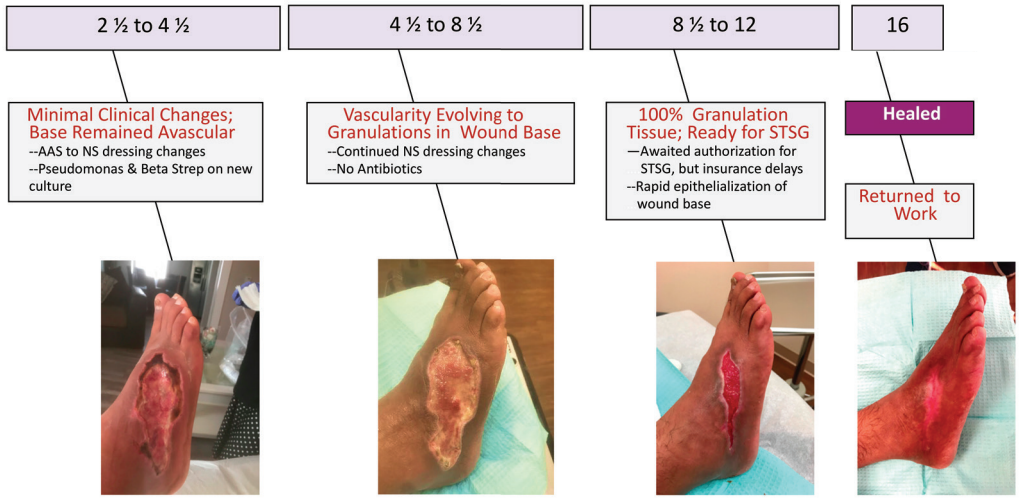
Stingray injuries can cause cutaneous necrosis of varying severities. Freshwater stingray injuries are reported to more likely cause severe skin necrosis than their saltwater counterparts [5]. Tissue necrosis has been reported in association with necrotizing fasciitis caused by *Photobacterium damselae* [6,7]. Tetanus has been reported from stingray injuries [8]. However, we were unable to find any reports of a stingray injury with extensive necrosis and prolonged healing that more closely resembled

Figure 1: Timeline of stingray injury events in hospital days



Key: **AAS** = Acetic acid solution dressing changes, **cm** = centimeters, **DS** = Double strength, **NPWT** = Negative pressure wound therapy (i.e. "wound vac"), **S.** = Streptococcus, **WBC** = White blood cell count

Figure 2: Timeline of stingray injury events in outpatient weeks



Legend: After a latency period (i.e. no improvement in the appearance of the wound base) of over a month, vascularity began to develop in the wound base. After 2 ½ months post injury, the wound was ready for skin grafting. The injury resulted in 4 months of disability

Key: **AAS** = Acetic acid solution dressing changes, **NS** = Normal Saline, **Strep** = Streptococcus, **STSG** = Split thickness skin graft

a toxic inoculation than an injury from a bacterial infection. How much the patient's poorly controlled diabetes at the time of injury contributed to the wound's course is open to question. Even though immersing the foot in hot water at our emergency department helped with pain control, as suggested by Clark, et al., it apparently did not inactivate the toxins [2]. We postulate that toxins contributed to the patient's prolonged convalescence.

In this report, the extent of tissue necrosis was surprisingly large in a wound that initially cultured only a rare growth of *Streptococcus salivarius* and *Streptococcus viridans*. Both were likely to be skin contaminants only. We hypothesize that the major skin slough and ensuing clinical course most likely conformed to a toxic reaction from the stingray injury. Eventually a satisfactory clinical outcome was achieved by focusing on making the wound

environment (i.e., selection of wound dressing agents) as physiological as possible rather than debridements to bleeding tissues, which have involved removing tendons, periosteum and decorticating the outer surfaces of the underlying bones. The delayed evaluation and management by a wound care provider (i.e., post-injury day 4) may have contributed to the patient's protracted wound healing course by allowing toxic substances to remain in contact with underlying tissues for this interval.

CONCLUSION

The consequences of diabetes mellitus probably played an important role in the prolonged healing time of the injury. The patient was initially admitted with a blood glucose level of over 400 and an HbA1c of 12.3 due to neglect of his diabetes. Two months after discharge from the hospital, with proper medication and diet management a repeat HgA1c improved to 8. This coincided with the improving clinical picture of the wound.

Any stingray puncture injury in a person with diabetes requires not only management of the injury but attention to diabetes management as well. The patient's diabetic condition most likely contributed to his wound morbidity. However, the clinical course more closely resembled a toxic reaction in tissues than infection from the stingray injury itself. Thus, the patient's diabetes status at the time of injury probably contributed to but was not the overriding consideration in the patient's prolonged wound healing course.

Conflict of interest statement
The authors have declared that no conflict of interest exists with this submission.

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