Case Report

Stingray Envenomation and Subsequent Skin and Soft-Tissue Infection Due to *Vibrio parahaemolyticus* and *Aeromonas hydrophila*

Urgent message: Failure to recognize and treat the early development of skin and soft-tissue infection from a stingray envenomation may result in significant tissue necrosis and systemic inflammatory response syndrome.

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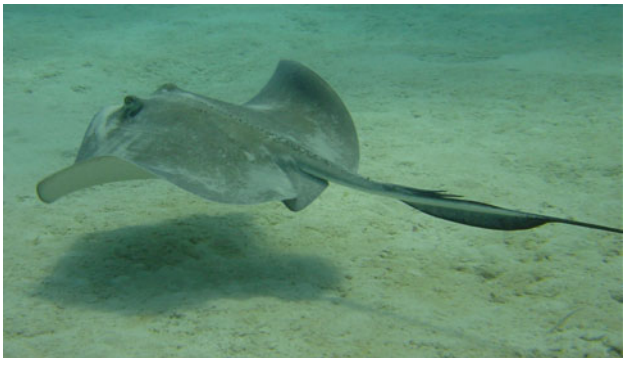
Introduction

Stingray injuries to humans are very common but rarely fatal. Approximately 2000 cases are reported annually in the United States.[1–3] At my rural hospital on the west coast of Florida, we see an average of 45 such injuries each year. The majority of these occur between April and October. Most patients with stingray injuries will present to a local urgent care center for treatment. My rural emergency department (ED) functions in this capacity.

I report here the only known case of a stingray envenomation producing a skin and soft-tissue infection (SSTI) with the causative agents being *Vibrio parahaemolyticus* and *Aeromonas hydrophila*. This is a unique finding and alters the routine antibiotic treatment normally used in these patients. It is this finding that is important to urgent care clinicians providing treatment. The urgent care clinician must be aware that penicillin and its derivatives may not provide adequate antibiotic coverage in all stingray envenomations.



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Case Presentation

A 42-year-old man was stung by a stingray on the dorsal

aspect of his right foot approximately 16 hours before presenting to my ED for evaluation and treatment. He called his primary-care physician and received a prescription for levofloxacin (500 mg). He had taken one dose before presenting to the ED. Although this individual presented to the local ED, many patients with envenomation will seek initial care at an urgent care clinic.

Observations and Findings

Upon initial evaluation the patient was febrile (temperature: 100.0°F), had a resting pulse rate of 112 beats/min, was normotensive, and had a toxic appearance. He reported intermittent nausea and mild abdominal cramping. Physical examination of the right foot revealed a 1.0-cm puncture wound over the midportion of the third metatarsophalangeal joint. There was a 3.0-cm-circumference area of erythema around the puncture wound site. There was nonpitting edema of the dorsal aspect of the right foot, and the wound had a small amount of serosanguineous drainage. Pain was out of proportion to physical findings. Local wound exploration, which is a routine part of the physical examination, and minimal debridement were performed. The patient had no identifiable risk factors for the development of systemic inflammatory response syndrome.

Diagnostic Studies

Initial laboratory values demonstrated a leukocytosis with a white blood cell count of 18,000/µL, with a neutrophilic shift (86%). There was no radiographic evidence of a retained foreign body or free air in the soft tissue.

Initial Treatment

The patient was given doxycycline (100 mg intravenously [IV]), cefazolin (1 g IV), and ciprofloxacin (400 mg IV) in the ED. Tetanus, toxoid-reduced diphtheria toxoid, and acellular pertussis vaccine were administered to ensure tetanus immunity.

The patient was admitted to the hospital. Shortly after admission, his temperature began rising to >101.6°F. Blood cultures showed no growth, but wound cultures demonstrated growth of *V. parahaemolyticus* and *A. hydrophila*. The patient continued to receive doxycycline, cefazolin, and ciprofloxacin IV. Metronidazole (500 mg IV every 8 hours) was added to the antibiotic regimen. Symptomatically the patient's febrile episodes resolved within 24 hours. Pain and erythema of the right foot lessened, and serosanguineous drainage from the wound decreased significantly.

Disposition and Discharge Instructions

After 72 hours, the patient was discharged to home after being prescribed cephalexin (500 mg by mouth four times a day), doxycycline (100 mg by mouth twice a day), and levofloxacin (500 mg by mouth daily) for an additional 10 days.

Follow-Up

A follow-up examination at 30 days showed a healed puncture wound on the dorsal aspect of the right foot with no significant sequelae.

Discussion

This case report is unique in several aspects. It is the first reported stingray envenomation with resulting SSTI from the microorganisms *V. parahaemolyticus* and *A. hydrophila*. Also, the patient had systemic toxigenic symptoms without the development of fulminating necrotizing fasciitis or without any underlying risk factors.

Venom Delivery and Structure

Injuries are inflicted by the stingray's spine. This apparatus has a unique histologic and anatomic architecture and venom delivery system. The venom apparatus consists of bilateral retroserrate spines with an integumentary sheath. The vasodentin spine has two ventrolateral grooves that contain the venom glands. The integumentary sheath tears open when traumatically introduced into an unsuspecting victim, unroofing glandular tissue to diffuse venom release. Often barbs, integumentary sheath, and venom-secreting glandular cells are left behind in the wound.[2] This constellation of animal products increases the risk of SSTI infections secondary to prolonged envenomation and foreign-body reaction.

Stingray venom is composed of enzymatically active proteins that are heat-labile and can be cardiotoxic. The venom contains the neurotransmitter serotonin and two enzymes, 5-nucleotidase and phosphodiesterase. Serotonin is responsible for the intense local pain reaction, and the other enzymes can cause significant tissue necrosis.

Effects of Envenomation

V. parahaemolyticus causes three major syndromes of clinical illness: gastroenteritis (most common), wound infections, and septicemia.[4,5] Since 1970 there have been only 2 reported deaths from SSTIs due to *V. parahaemolyticus*. Only 10 cases of necrotizing fasciitis have been reported.[5–8] Wound infections from *V. parahaemolyticus* are generally minor infections and comprise approximately one-third of all *V. parahaemolyticus*

infections. However, these infections can be life-threatening because of the rapid invasion and destruction of the tissue planes, accompanied by the release of several cytotoxins. Eighty-eight percent of these individuals have underlying risk factors, including cirrhosis, diabetes, hepatitis C, and chronic renal failure. Diabetes and liver disease present the greatest risk.[5]

A. hydrophila infections typically occur on the extremities after a traumatic aquatic injury. They occur more frequently in brackish waters during the summer months.[7,8] This microorganism produces a cytotoxic enterotoxin and multiple exotoxins that can cause reactions ranging from mild skin infections to necrotizing fasciitis. The quintessential invasive disease is septicemia, with a mortality rate of 33%. Common risk factors include an immunocompromised state, chronic liver disease, diabetes, and chronic renal failure. *Aeromonas* is uniformly resistant to treatment with penicillin and ampicillin.[5]

Toxic systemic effects of envenomation can produce nausea, vomiting, diarrhea, abdominal cramps, seizure, respiratory difficulties, hypotension, and cardiac dysrhythmias. After envenomation, intense localized pain peaks at 30 to 90 minutes and may last up to 48 hours if left untreated.[1,9] Immediate treatment consists of immersing the injured appendage in water, heated to >115°F, for 30 to 90 minutes. Repeated heat-immersion therapy may be required for up to 4 hours after injury. Generally the wound must be explored for any retained spine products. The patient's tetanus status must be up to date.

Conclusion

Because of the potential of penicillin-resistant bacteria causing SSTIs from stingray envenomations, the urgent care clinician should prescribe treatment with oral doxycycline and levofloxacin for at least 10 days. Close follow-up within 72 hours of treatment is required. ■

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