Hemolysis and Acute Renal Failure Following a Portuguese Man-of-War Sting

Harry A. Guess, MD, PhD, Peter L. Saviteer, MD, and C. Richard Morris, MD

From the Department of Pediatrics, University of North Carolina Chapel Hill

ABSTRACT. Portuguese man-of-war stings occur frequently at North American beaches. Most produce only local pain and pruritis. A 4-year-old girl who developed a hemolytic reaction and acute renal failure following a severe sting is described. As neither sequela has been previously reported, it is speculated that either the large dose of venom or chance intravenous inoculation may have been responsible. Evidence of hemolysis should be sought in small children with extensive Portuguese man-of-war stings. Pediatrics 70:979—981, 1982; acute renal failure, hemolysis, bites and stings, coelenterate venom, Portuguese man of war.

Coelenterates are members of a phylum of aquatic invertebrates that includes such stinging marine animals as the Portuguese man-of-war (Physalia physalis) and numerous species of jellyfish. The most toxic coelenterates are the cuboidal jellyfish of the order Cubomedusae. Two species, Chironex fleckeri and Chiropsalmus quadrigatus, are found in northern Australian waters and are responsible for numerous fatalities.1 Cubomedusae are rare in North American waters, but the Portuguese man-of-war is common in the Atlantic Ocean and is found as far north as Nova Scotia.2,3 Although the stings of this species may produce relatively severe local cutaneous reactions, life-threatening systemic responses have not been reported. Fatalities previously attributed to the Portuguese man-of-war remain unsubstantiated and are now considered to have been due to C. fleckeri in northern Australia and to C. quadrigatus in the Philippines and elsewhere in Malaysia.4 We report the experience of a small child who developed a severe hemolytic reaction and acute renal failure following a severe sting. Physicians caring for victims of stings should be aware of the potential for severe systemic reactions.

CASE REPORT

A 4½-year-old girl, while wading in waist-deep water at a North Carolina beach, was stung by red and purple tentacles which, by an observer's description to a Professor of Marine Biology, were identified as characteristic of a Portuguese man-of-war. Minutes after their removal, painful, red, vesicular skin lesions, estimated to cover 10% of her body surface, appeared along the tentacle tracks. She was treated immediately with dexamethasone, epinephrine, and diphenhydramine. Persistent, severe pain and progressive edema of the involved extremities and absence of urination for ten hours after her sting prompted reevaluation. Blood pressure was 108/62 mm Hg and heart rate 132 beats per minute. Bladder catheterization revealed only 40 ml of burgundy, Hemastix positive urine. The sediment contained no red blood cells or casts. Serum was grossly red, and the hematocrit was 28.9%. Total and conjugated bilirubin were 5.1 and 3.7 mg/100 ml, respectively. Potassium was 6.9 mEq/liter, creatinine was 1.8 mg/100 ml and BUN 50 mg/100 ml. Hyperkalemia was controlled, and packed red blood cells were given. No reticulocyte count, smear description, or additional hematocrit level was obtained prior to transfusion. Oliguria persisted (130 ml in 24 hours), and the patient was referred to North Carolina Memorial Hospital.

On admission 24 hours following the sting, the patient appeared acutely ill. Blood pressure was 120/80 mm Hg, pulse rate was 104 beats per minute, and respirations were 28/min. Tender, purple, indurated, linear lesions, with adjacent, superficial, hemorrhagic bullae and edema were present on the left arm and both thighs. Peripheral pulses were absent in the left arm but were present in both feet. There was scleral icterus. The remainder of the examination disclosed no abnormalities. Catheter urine was burgundy and contained 1,000 mg/100 ml of protein. The sediment revealed numerous red blood cells per high power field (HPF). The serum was grossly red. Lactic
dehydrogenase (LDH) was 7,371 IU, SGOT 455 IU, and creatine phosphokinase (CPK) 554 units/liter. Total bilirubin was 7.4 mg/100 ml. Measurements of serum-free hemoglobin, obtained 48 and 96 hours after the sting, were 12.3 and 11.0 mg/100 ml, respectively. Hematocrit values did not change after the initial transfusion. Serum creatinine concentration was 3.0 and BUN 82 mg/100 ml. Injection of 131I iodohippurate sodium (Hipppuran I 131) nucleotide was followed by the appearance of minimal, symmetrical radioactivity in both kidneys.

Intravenous administration of isotonic saline, 150 ml, followed by furosemide, 15 mg, was associated with no change in urine volume. Oliguria persisted, and dialysis was required on five occasions. Resolution of oliguria began on the tenth day following the sting and urine volume returned to normal in progressive increments over the next ten days.

When seen two months later, the patient was normotensive, serum creatinine was 0.5 mg/100 ml, and urinalysis and serum electrolytes were normal.

**DISCUSSION**

Coelenterate venom is injected by microscopic “stinging organoids” called nematocysts that act as independent effectors capable of skin penetration and venom release in response to a variety of chemical and mechanical stimuli. Nematocyst discharge continues to occur as long as the tentacle fragments which they are located remain in contact with the victim.

The response to coelenterate stings depends primarily upon the species. The length of time the tentacles are in contact with the victim, the extent of skin surface area involved, and the sensitivity of the victim to the venom are additional factors. Based on animal experiments using intravenous and subcutaneous injections of venom from one of the most highly toxic coelenterates, Keen and Crone speculated that fatal human stings could be explained on the basis of the chance inoculation of venom directly into the circulation through a superficial skin vessel.

Despite differences in severity, the sequelae range from transient, local irritation to nearly instant death. Local effects may be severe. Excruciating pain is a constant finding. Subcutaneous edema and vascular spasm without thrombosis may be sufficiently severe to produce necrosis of digits. Systemic reactions include headache, backache, myalgias, abdominal rigidity, arthralgias, nausea, vomiting, chills and fever, pallor, hysteria, throat constriction, respiratory distress, collapse and coma. The stings of certain coelenterates are frequently fatal within minutes to hours and produce a sensation of chest constriction followed by vasomotor and respiratory failure, pulmonary edema, and death. Pulmonary congestion is a nearly constant finding in fatal stings, and morphologic evidence of visceral and cerebral congestion, pulmonary edema, minute hemorrhages into the cerebral hemispheres, lymphoid hyperplasia, and acute toxic nephritis have been described.

Venoms from several coelenterate species have been shown to produce hemolysis in experimental animals and in human red blood cells in vitro. Portuguese man-of-war venom is mildly hemolytic, and venoms of *C fleckeri* and *C quadriratus* are strongly hemolytic. This property is shared by the venom of other animals, and severe hemolysis and acute renal failure have been described in humans following the stings of brown recluse spiders and honeybees and the bites of certain species of snakes. Scorpion stings in southern Iran have been followed by a course strikingly similar to that of our patient. Of 15 patients described, most developed hyperbilirubinemia and dark urine within 24 hours, and 11 developed oliguria or anuria which persisted from six to 21 days.

The pathogenesis of acute renal failure (ARF) following coelenterate stings is unknown. Hemolysis is the most apparent source of injury in this child, and heme pigments have been associated with ARF in humans. Although myoglobin is regularly associated with ARF, other factors, such as preexistent dehydrogenase and severe acidosis, seem important in the genesis of hemoglobin-induced ARF. Neither were documented in our patient, and volume expansion failed to induce a diuresis. This observation suggests the possibility that some other factor contributed to the genesis of this child’s ARF.

Four general mechanisms have been implicated in the pathogenesis of oliguria in ARF: intratubular obstruction, “backleak” of tubular fluid into peritubular capillaries, reduction in renal blood flow and reduction in glomerular capillary permeability. Each has been investigated in experimental models of heme pigment ARF, and there is good evidence for a pronounced, initially reversible decrease in renal blood flow. This decrease in renal blood flow is sustained by a subsequent, irreversible increase in renal vascular resistance, presumably mediated by endogenous vasoconstrictors or by the inhibition of vasodilators. Analogous to the clinical situation, however, this series of events depends on preexistent dehydration. As no other predisposing factor was apparent in this child, it was conceivable that vasoactive substances in coelenterate venom could initiate irreversible vasoconstriction. Malhotra et al similarly speculated that serotonin in scorpion venom produced afferent arteriolar constriction in the patients they studied.

Although certain coelenterate venoms are known to contain serotonin, histamines, and prostaglandins, Portuguese man-of-war venom does not, and the kinen-like substances known to be present...
would be expected to produce vasodilation. Unless previously undetected vasoactive substances are present, a direct role for venom seems unlikely.

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