Bee Stings and Acute Tubular Necrosis

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Dear Sir,

Animal venoms have occasionally been incriminated in the genesis of acute renal failure. Most case reports concern snake venoms but exceptionally venom from insects of the Hymenoptera species has been implicated [1,2]. We report 1 patient in whom rhabdomyolysis and hemolysis with consequent acute renal failure developed after innumerable bee stings.

A 31-year-old European man without history of renal disease was attacked in Zaire (Africa) by a swarm of bees and sustained over 1,000 stings over the head, the thorax and the abdomen. The exact species of the bees was not identified. He was admitted to the local hospital, where he received over the first few hours 750 mg hydrocortisone, 90 mg calcium, 100 mg promethazine and solutes (macro-molecular solution and physiologic saline). No blood transfusion was given. In spite of the treatment, the patient developed 10 h after the attack, a hypovolemic shock lasting 3 h (systolic blood pressure below 70 mm Hg). During the subsequent 12 h he produced only twice 20 ml of dark urine. He was subsequently transferred to Belgium.

On admission he was subcomatose. The skin was diffusely infiltrated by edema. Vital signs were normal, but anuria persisted. Laboratory data are given in table I. Serum bilirubin reached 56 $\mu\text{mol/l}$; serum haptoglobin was decreased to 33% of control values; haptoglobin-hemoglobin complexes were visible on gel agarosis electrophoresis; no urine was available for analysis; EKG and chest X-rays were normal. On the plain film of the abdomen, both kidneys appeared enlarged (bipolar axis 14 and 14.5 cm). Total anuria persisted 12 days and required 7 hemodialyses. Renal function improved gradually there-

Table I. Laboratory findings and evolution

The first and last hemodialyses were performed on days 3 and 17, respectively.

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after and 27 days after admission the patient was discharged. Two months later, renal function and urinalysis were normal (table I) and, on IVP, kidney size had returned to normal.

This case demonstrates that multiple bee stings may cause rhabdomyolysis and hemolysis with consequent acute tubular necrosis. The elevated levels of muscle enzymes, i.e. MM-CPK and aldolase and the acute rise of serum creatinine to 867 $\mu\text{mol/l}$ within less than 48 h, strongly suggest the existence of rhabdomyolysis. The subsequent development of severe anemia within 9 days, the presence of haptoglobin-hemoglobin complexes and the low serum haptoglobin level

on admission point to an associated hemolysis. These two disorders together with a transient hypotension were probably responsible for the development of acute tubular necrosis. The chemical composition of bee venom is not completely described. Among others, it contains mediators and neurotransmitters, enzymes such as phospholipase A and surface-active agents such as apamine and mellitine [3,4]. Some of these substances act on muscles: PínLim et al. [1] observed increased serum levels of muscle enzymes in 14 of 17 patients admitted for wasp or bee stings; the patient whose serum enzyme level was most elevated, developed acute tubular necrosis. Two similar cases were reported by Shilkin et al. [2]. Surface-active agents and phospholipase A also act on red cell membranes and provoke hemolysis which may contribute to the development of acute tubular necrosis. The explanation of the severe thrombocytopenia present on admission is not clear: the lack of fibrinogen degradation products in the serum argues against intravascular coagulation; it is possible that it resulted from a direct toxic action of bee venom, as it has been observed in dogs injected with venom from oriental hornet [5].

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