ACUTE RENAL FAILURE COMPLICATED BY MULTIPLE BEE STINGS: A CASE REPORT AND LITERATURE REVIEW

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Attacks by bees are not rare. Multiple bee stings may be associated with severe complications including cardiovascular insufficiency (shock, myocardial damage), respiratory failure (adult respiratory distress syndrome, bronchospasm), disseminated intravascular coagulation (DIC), renal and hepatobiliary disorders. We describe a patient who developed anaphylactic shock followed by acute renal failure and hepatic dysfunction after numerous bees attack. More than one hundred sting wounds were found over his face, head and extremities. No evidence of hemolysis with hemoglobulinuria or rhabdomyolysis with myoglobinuria was detected. Initial anaphylactic shock or direct nephrotoxicity of bee venom was presumed to be the pathophysiologic mechanism of acute renal failure. He was treated with a series of managements including hemodialysis. Fortunately, he recovered completely and was without renal and hepatic sequelae two months after the bee attack. (Acta Nephrologica 2000; 15: 20-22)

Key words: acute renal failure, bee stings, hemodialysis

INTRODUCTION

Bee sting is frightening, especially when masses of bee attack, not only because of the severe pain but also the possibility of fatal reaction. In the literature, there have been many reports of serious complications of multiple bee stings. Three major reactions occur after bee envenomation. The first is local swelling and irritation, which is produced by vasoactive components of bee venom. The second, less common reaction, is a generalized anaphylactic responses with urticaria, angioedema, dyspnea and hypotension. These more extensive reactions are caused by an immediate hypersensitivity reaction. The third reaction is serum-sickness-like symptoms including thrombocytopenia, DIC, hemolysis, rhabdomyolysis and acute renal failure. It is possibly mediated through circulating immune complex or delayed hypersensitivity reaction.

We report the case of a patient who survived a massive bees attack but developed anaphylactic shock, acute renal failure and hepatic dysfunction.

CASE REPORT

A 60-year-old male farmer was attacked by a swarm of hornet bees (Vespa) while working in the farmland. He very rapidly developed erythematous swelling and steady burning pain at the sting sites. He also felt nausea, dizziness and general weakness. He was taken to a local clinic where he was found to be obtunded and hypotensive. His blood pressure was 80/50 mmHg. Emergent treatment included intravenous fluid supplement, administration of parental hydrocortisone and antihistamine. He went home with some improvement several hours later. Over the following two days, he became to aware of poor appetite, general malaise and decreased urinary volume and he was sent to our emergency room. A physical examination revealed him to be drowsy and dehydrated. Numerous bee-sting lesions were noted on his body especially the head, face and extremities. (Fig. 1 and 2) The number of stings was estimated to be more than one hundred. His blood pressure was 92/60 mmHg and pulse rate was 102/min. EKG and chest X-rays were normal. Abnormal laboratory test results confirmed that the patient had multiple systems organ dysfunction, with a predominance of renal and hepatic involvement. Laboratory data during admission are listed in Table 1. Renal echogram
show normal size and contour of bilateral kidneys. Normal renal function test was documented in the record at our hospital three months earlier and thus acute renal failure was diagnosed. After admission, initial management consisted of administration of low dose dopamine and intravenous fluid replenishment. Three-times-weekly hemodialysis was initiated because of the high level of blood urea nitrogen, creatinine and potassium. His clinical and laboratory parameters improved gradually. Urine volume was restored 12 days after admission and hemodialysis was discontinued. Twenty-five days after the bee attack, he was discharged in good health. No renal, hepatic or neurologic sequelae were observed after one month of OPD follow-up.

**DISCUSSION**

We describe a man, without previous renal disease, who developed anaphylactic shock, acute renal failure and hepatic dysfunction after multiple bee stings. He underwent a short time of hemodialysis therapy. Our patient presented with local and systemic reactions to
the bee envenomation. Just after the bee attack, he experienced local erythematous swelling over the sting sites. He was found to be obtunded in state with shock later in a local clinic. The symptoms and signs mimicked minor anaphylactic shock which is mediated by vasoactive substances in bee venom. He subsequently developed acute renal failure with oliguria. Many authors have reported acute renal failure following multiple bee stings. The pathogenetic mechanism involved in the development of acute renal failure after bee stings is not clear. Intravascular hemolysis with hemoglobinuria, rhabdomyolysis with myoglobinuria, anaphylactic shock or direct nephrotoxicity of bee venom are all possible causes. Bee venom contains toxic polypeptides (mellitin), enzymes (phospholipase A2, hyaluronidase) and low-molecular-weight agents (histamine and amino acids). Mellitin and phospholipase A2 have hemolytic properties and might result in rhabdomyolysis due to a toxic effect on striated muscles. In our patient, tests for myoglobinuria were repeatedly negative. He denied passage of dark urine.

No evidence of hemolysis was noted either. Therefore, it is probable that his acute renal failure was not related to pigment tubulopathy. But initial hypotension following bee attacks does occur. We presume that his acute renal failure was attributable to shock due to anaphylaxis or to direct nephrotoxicity of the bee venom. Acute renal failure with acute tubular necrosis (ATN) is a well known pathological finding after multiple bee stings. Acute tubulointerstitial nephritis was also cited by one author. Unfortunately, our patient refused to undergo percutaneous renal biopsy, so we were unable to determine the exact pathology in this case. Analysis of his urine revealed microscopic hematuria, pyuria and granular cast, which favors the presence of ATN more than prerenal azotemia.

Thrombocytopenia has been observed in victims of bee sting and it occurred in our patient. No fibrinogen degradation products were detected in the serum and DIC was thus excluded. It is possible that thrombocytopenia resulted from a direct toxicity of the bee venom. Hepatocellular dysfunction may also be an adverse reaction to bee sting. Our patient had increased serum level of aspartate aminotransferase (AST) and alanine aminotransferase (ALT). He denied any previous liver disease and no evidence of hepatitis B or C was found during this admission. An abdomen echogram did not reveal any hepatic abnormality. Anaphylactic shock or hepatotoxicity of bee venom may be implicated in hepatocellular necrosis.

Most fatalities related to bee stings have been attributed to shock and respiratory failure. Anaphylactic shock is a medical emergency that requires the prompt use of epinephrine (0.3 to 0.5mL of 1:1000) and antihistamine (eg. 50-100mg of diphenhydramine). Airway management and assurance of adequate ventilation and oxygenation are very important. Aggressive fluid resuscitation should be initiated especially for dehydrated patients. Vasopressors with α-adrenergic agonist properties (eg. dopamine, norepinephrine) may be necessary to support blood pressure unresponsive to fluids and epinephrine. Fortunately, though our patient developed anaphylactic shock, acute renal failure and hepatic dysfunction, he recovered completely eventually. At present, there is no specific treatment such as antivenom for bee stings. Appropriate management for multiple bee stings requires careful attention to the possible severe complications.

REFERENCE