BEE ENVENOMATION INDUCED ACUTE RENAL FAILURE IN AN 8 YEAR OLD CHILD

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Abstract

Massive envenomations by bees are capable of causing multiorgan dysfunction as a result of direct toxic effects of the large venom load received. Although all varieties of honey bee have the potential for these attacks, the Africanized honey bee (*Apis mellifera scutellata*) is the most commonly implicated subspecies. In the United States, the Africanized strain is found primarily in the southwestern states and is known for its highly defensive behavior if disturbed. Mechanisms behind the multiorgan dysfunction produced by these mass envenomations are not clearly understood. We present a case of an 8-year-old boy who was stung by multiple bees and developed progressive upper-body swelling and systemic manifestations of mass envenomation including rhabdomyolysis, renal insufficiency, and a transient transaminase elevation.

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Key Words: environmental risk, renal failure, rhabdomyolysis, bee envenomation.

Introduction

Stinging events involving honeybees and wasps are rare; most deaths or clinically important incidents involve very few stings (< 10) and anaphylactic shock. However mass stinging events can prove life threatening via toxic action of the venom when injected in large amounts.[1] Several types of uncommon reactions have been described including serum sickness, renal diseases, respiratory and neurological manifestations, hepatic dysfunction and delayed hypersensitivity phenomena.[2]

Case Report

An 8 year old boy was stung by a swarm of bees (forest bees, Apis dorsata) two days before reporting while he was collecting fodder under a tree. He was initially treated at a district hospital and referred because he developed hematuria and decreased urine output. On examination, the patient was conscious and oriented. He had multiple sting marks all over the body (~ 200), skin was infiltrated by edema and stung areas were edematous and erythematous. There was swelling of the face with pallor and dehydration. He was hemodynamically stable. No abnormality was found on systemic examination. His investigations revealed: Hemoglobin 17.5g/dL, total WBC count 17,000/cmm, platelets $390x10^9$/L, peripheral smear showed reticulocytosis and polychromatic RBCs. Blood urea was 139 mg/dL, S. creatinine 7.8 mg/dL (normal 0.5-1.5), sodium 125 meq/L, potassium 5.12 meq/L, calcium 8.3 mg/dL, phosphorous 8 mg/dL, uric acid 7.5 mg/dL, protein 5.2 gm/dL, albumin 3.5 gm/dL, ASO titre -554IU/ml, C3-1.04g/l, HBs Ag-negative, bilirubin 1.5 mg/dL (conjugated 0), aspartate aminotransferase (AST) 121 IU/L, alanine aminotransferase (ALT) 149 IU/L, alkaline phosphatase 112 U/L, lactate dehydrogenase (LDH) 2740 U/L (normal 240-480), creatine phosphokinase (CPK) 2888 U/L (normal < 167).

Arterial blood gas (ABG) revealed pH 7.35, paO_2 80 mmHg, $paCO_2$ 34, HCO_3 18 meq/L. Urine examination: color was reddish, appearance was initially clear then hazy, albumin + +, pus cells 2-6 /

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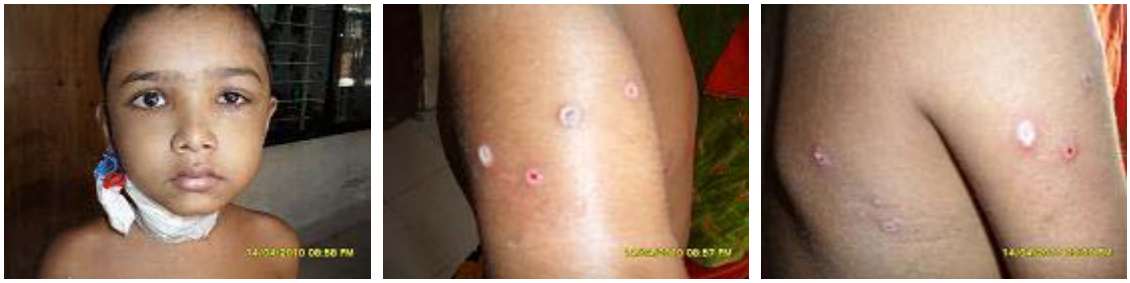


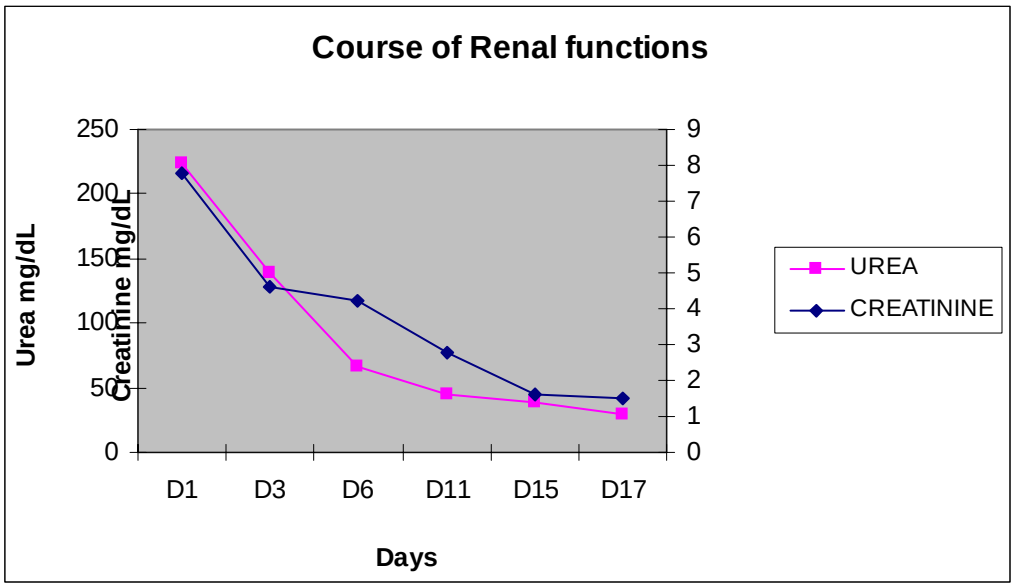
hpf, RBCs 35-45/hpf, urine hemoglobin +, urine culture - sterile.

Laboratory findings were consistent with intravascular hemolysis, rhabdomyolysis, acute renal failure and hepatic dysfunction. Patient was treated with fluid restriction, diuretics, antibiotics, steroids, antihistamins and sodium bicarbonate. Ultimately he needed four sessions of hemodialysis after which he gradually improved and renal function returned to near normal by day fifteen.

Discussion

This case demonstrates that multiple bee stings may cause rhabdomyolysis and hemolysis with consequent ATN. Components of venom include toxic surface-active polypeptides (mellitin and apamin), enzymes (phospholipase A_2 and hyaluronidase) and low molecular weight agents (histamine and aminoacids). Mellitin and phospholipase are important components causing rhabdomyolysis following a toxic action on striated muscles which also acts on the red cell membrane and provokes hemolysis.[3] The elevated levels of enzymes CPK and aspartate-aminotransferase suggest the existence of rhabdomyolysis and hemolysis is suggested by anemia, unconjugated hyperbilirubinemia, reticulocytosis, increased serum LDH and hemoglobinuria.[3]

Rhabdomyolysis and hemolysis can induce ARF, particularly in hypovolemic or acidotic individuals. It has been postulated that myoglobin and hemoglobin released from muscle or red blood cells cause ARF by toxic effects on tubule epithelial cells or by inducing intratubular cast formation. Hypovolemia or acidosis may contribute to pathogenesis of ARF in this setting by promoting intratubular cast formation. In addition, both hemoglobin and myoglobin are potent inhibitors of nitric oxide bioactivity and may trigger intrarenal vasoconstriction and ischemia in patients with borderline renal hypoperfusion.[4]



The mortality associated with Africanized honeybee attacks is primarily the result only of the number of the number of stings.[5] A number of about 500 stings have been considered necessary to cause death by direct toxicity, but as few as 30-50 stings have proved fatal in children.[3] Our patient had about 200 stings and survived with complete renal recovery. The primary therapeutic goal is to prevent the factors that cause ARF, i.e. volume depletion, tubular obstruction, aciduria and free radical release. Patients are administered saline for intravascular volume expansion and sodium bicarbonate for urine alkalization (to urine pH level above 7). The ideal fluid regimen for patients with rhabdomyolysis consists of half isotonic saline (0.45%, or 77 mmol/L sodium), to which 75 mmol/L of sodium bicarbonate is added. Once overt renal failure has developed, the only reliable therapeutic modality is extracorporeal blood purification.[4] Exchange transfusion or plasmaphresis has been found useful because it acts through a direct effect of reduction of the massive circulating venom or removal of the circulating mediators of inflammation caused by the venom itself.[3,6]

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