Renal and hemorrhagic complications following bee sting envenomation: a case report from the hospital of Zinder

ABSTRACT

Renal and hemorrhagic complications following envenomation by bee stings are rare, posing significant problems for early diagnosis and treatment in the clinical setting. Bee venom is generally well tolerated. A 44-year-old patient was admitted to the infectious and tropical diseases department of the Zinder National Hospital with cervicofacial edema following bee stings. Initially, the patient lost consciousness. Respiratory symptoms included dyspnea and 91% desaturation on room air. The patient was put on oxygen to alleviate hypoxia, doxycycline to prevent secondary bacterial infections, and prednisone to reduce inflammation. The clinical picture was complicated by a worsening of the patient's condition due to a deterioration in renal function, and the subsequent appearance of hematemesis. A renal biopsy revealed acute tubulointerstitial nephropathy, due to direct venom toxicity on the tubules. He was discharged after 35 days. We report a rare case of bee sting complicated by hemorrhagic syndrome and renal involvement.

Keywords: bees, envenomation, kidney complications, hemorrhages, Zinder, Niger Republic

1. INTRODUCTION

Renal and hemorrhagic complications following bee stings with envenomation are most commonly observed due to venom inoculation[1,2]. They constitute a medical emergency, necessitating prompt intervention. Bee venom injection can lead to pain, fever, swelling, and multi-visceral involvement in affected individuals. The injected venoms are harmful either due to their quantity (multiple stings) or because the stung individual is allergic[3,4]. Bee stings can induce various clinical manifestations that jeopardize the patient's life. Localized pain, tissue necrosis, and anaphylactic reactions followed by wasp stings are well-documented[5]. Besides these, they can produce systemic reactions and organ dysfunction[1,2]. Among these, rhabdomyolysis has been identified as one of the main mechanisms. Mellitin is believed to be the primary molecule responsible for rhabdomyolysis, as well as hemolysis[6]. Phospholipase A2, to a lesser extent, may play a role in the occurrence of rhabdomyolysis[2]. Acute renal failure results from tubular obstruction caused by myoglobin and the direct nephrotoxic effect of the venom[3]. Different cases may present varying severity levels, including mild cases (local edema with acute pain and skin reaction, sometimes accompanied by fever), moderate cases (syncope with dizziness, nausea, severe headaches, palpitations, skin reaction, and allergic reactions in certain organs), and fatal cases that can occur following anaphylactic shock [1–3].

Regarding hymenopterans, particularly bee venom, the presence of histamine, a polypeptide called apitoxin, and riboflavin have been identified. Several properties of bee venom have been elucidated, such as its hemolytic action, decreased blood coagulability, hormonal balance alterations, histamine effects, and the potency of apitoxin responsible for allergic reactions. Feldberg and Kellaway also demonstrated in 1937 that bee venom causes a significant release of histamine in the lungs and perfused livers of guinea pigs[6,7].
As we mentioned below, several authors have described cases of envenomation by bee stings resulting in renal and hemorrhagic complications, hence the need for this case report to highlight the situation.

2. METHODS

This study examines a case of renal and hemorrhagic complications following bee stings at the Department of Infectious and Tropical Diseases of the National Hospital of Zinder. The aim was to describe the occurrence of complications in bee sting envenomation. The methodology used allowed for the collection of interview data. Diagnostic methods included physical examination and laboratory tests.

3. CASE PRESENTATION

The patient is a 44-year-old individual admitted to the Department of Infectious and Tropical Diseases of the National Hospital of Zinder for cervicofacial edema following bee stings.
The patient reported no pathological history.
Initially, the patient lost consciousness, and thenupon interrogation, fever and vomiting were reported after the sting. Physical examination revealed multiple painful sting sites with embedded stingers, cervicofacial edema, and pale mucous membranes and conjunctivae.

Figure1 Patient stung by bees

Upon arrival, the farmer is found to be covered with numerous stings on the face, skull, neck, upper limbs, and thorax. The most affected areas are the face, neck, and thorax. In the impossibility of counting each sting, Wallace's rule is applied, which divides the body surface into proportional zones:

- 9% for the head and neck
- 9% for each upper limb
- 18% for the anterior trunk
- 18% for the posterior trunk
- 18% for each lower limb
- 1% for the genitals

Based on this estimation, we can calculate that the total surface area affected by stings for this farmer is 27% (head/face = 9% and anterior trunk = 18%). According to the formula used to calculate body surface area taking into account height (in cm) and weight (in kg): X=1+(height+weight-160)/100
Patient constants: Weight=83 kg, Height=178 cm. Area=X/100
This amounts to 20,100 cm2, with 27% receiving at least one sting per cm2, the total number of stings can thus be estimated between 6000 – 7000 or 70 stings per Kg of body weight. This number is comparable to that of bees in a hive (about 50,000 insects). It corresponds to inoculation of 2.1 to 4.9 mg of venom in total (one sting releases 0.03-0.07 mg of venom and the reservoir contains 3-4 mg of liquid).
The patient was fully conscious, with a Glasgow score of 15/15. Additionally, he had a fever with a temperature of 38.6°C, his heart rate was within the normal range (97 beats/min), SPO2 at 97% in ambient air, and his blood pressure (BP) was (143/70 mmHg).
On the second day, the patient was dyspneic and desaturated to 91% in ambient air.
On the third day, the clinical picture was complicated by worsening the patient's condition with the appearance of hematemesis and sleep disturbances as well as deterioration of renal function.
On the tenth day, the patient experienced a low-grade fever (38.5°C), moderate hypoxia with SPO2 at 93% in ambient air, and spontaneous hematemesis.

The hematological assessment showed leukocytosis at 42,900 cells/mm3, anemia at 9.6 g/dl, and thrombocytopenia at 126,000 cells/mm3. The biochemistry revealed a blood glucose level of 5.63 mmol/l, uremia at 3.30 mmol/l, and a creatinine level of 82.54 µmol/l.

The hemostasis assessment included a prothrombin time of 84.9%, an activated partial thromboplastin time of 30 sec, and C-reactive protein at 384 g/l. SRV serology and HbSantigenemia were negative. Transaminases and creatine kinase could not be performed due to technical limitations.
On the third day, deterioration of renal function occurred [Uremia at 49.97 mmol/l, creatinine at 1265.63 µmol/l (GFR at 4.22 ml/min)]. On the urine dipstick, proteinuria at one cross and hematuria at 4 crosses were noted. Faced with this severe uremia, the indication for emergency dialysis was raised. A renal biopsy revealed acute tubulointerstitial nephropathy.
On the thirteenth day, follow-up blood tests showed normalization of various parameters.
By the twenty-fourth day, esophagogastroduodenal endoscopy revealed pangastropathy.
On the thirty-second day, hematological and renal function tests were all normal.

He received paracetamol intravenously at a dose of 60 mg/kg per day, divided into four doses, methylprednisolone 120mg in 250cc of solution, Saline serum 40mg/kg/d, Ceftriaxone 50 mg/kg/d.Oxygen, doxycycline, and prednisone were administered.
The patient's condition improved after four dialysis sessions but he developed a catheter infection during hospitalization which required catheter removal. Culture of the catheter tip isolated Staphylococcus aureus sensitive to vancomycin. The patient was treated with vancomycin and gentamicin.
Hematological assessment revealed anemia at 6.7 g/dl, requiring a blood transfusion.
Treatment involved administering oxygen at 5 l/min, followed by the transfusion of 2 units of packed red blood cells (450 cc each) matched for ABO and RhD using delta Hb, along with gastric dressing with aluminum hydroxide/magnesium hydroxide2 sachets three times a day.
A treatment protocol was initiated, including ampicillin 2g IV twice daily, metronidazole 500 mg three times a day, gentamicin 160mg IM, intravenous omeprazole 2 vials as a bolus followed by 1 vial twice daily, saline serum 500mg twice daily, and paracetamol intravenously 1g.
The patient was discharged cured after 35 days.

3. DISCUSSION

Venomous bee stings are rare but often serious due to the occurrence of various complications[8]. According to literature data, Africanized bee species are the most aggressive[9]. Bee stings are considered a public health problem in Latin America, where they are frequent and responsible for a significant number of deaths[10]. Even in Africa, cases of bee stings have been reported, notably in Morocco[4]. The lethal threshold generally ranges between 300 and 500 stings; however, in some cases, simultaneous stings can be deadly, and for this patient, the number was estimated at 70. In the United States, bee stings are the second leading cause of anaphylactic reactions after penicillin[11]. The extreme toxicity of massive bee envenomation is due to the direct toxic effect of large quantities of injected venom. Bee venom is a complex combination of enzymes and amines. Mellitin, a component that initiates pain, constitutes 50% of the venom's weight and acts in conjunction with phospholipase A2 as a cytolytic agent. Hyaluronidase increases capillary permeability and facilitates toxin diffusion[12,13].
Immediate reactions following numerous stings typically include localized pain, sweating, and erythema at the sting site, this patient had all these reactions. Within 24 hours, hemolysis, hemoglobinuria, rhabdomyolysis, and hepatic injury may develop[7]. A hemorrhagic syndrome was observed 24 hours after admission in this patient.Acute renal involvement is often linked to the direct toxicity of the venom[3,14]. Hemostasis and coagulation disorders have been reported by Przybilla and Ruëff[9]. Rhabdomyolysis, which can lead to acute renal failure, is a complication of massive envenomation[3]. Creatine phosphokinase, lactate dehydrogenase, and aspartate aminotransferase levels would be elevated[15]. In this case, therefore, muscle enzyme assays could not be performed. Nevertheless, given the clinical picture (myalgia, dark urine), it is highly likely that the patient suffered from massive rhabdomyolysis, complicated by renal failure. Although renal involvement may go unnoticed[16], cases of renal failure requiring dialysis have been reported, and this patient had developed severe uremia requiring dialysis sessions[1,2]. Tissue necrosis appears to result from the cytolytic effects of three venom components, namely phospholipase A2, hyaluronidase, and serine proteases[11]. The location of the stings is an important prognostic factor, particularly stings to the head and neck as well as those in the pharynx and larynx, which can lead to death due to airway obstruction[17],and in this case, respiratory symptoms were present. Ocular lesions from bee stings can occur[13,18], but this patient did not present with direct ocular injuries.Neurological and cardiac involvement is much rarer[16].
Urgent anti-anaphylactic measures should be instituted, such as fluid resuscitation, corticosteroid therapy, and subcutaneous administration of adrenaline, due to the similarity of clinical manifestations with acute anaphylactic reactions[19], this patient was treated effectively and urgently. Early management is essential, with prompt patient admission, massive fluid resuscitation, high-dose corticosteroid therapy, and removal of stingers, our patient received a similar treatment.Local treatment involves gentle scraping to remove stingers, followed by local disinfection with an antiseptic such as hydrogen peroxide or bleach[6]. The use of analgesics such as paracetamol is recommended. The prognosis of envenomation is related to venom plasma concentrations[20], and in severe cases, early plasmapheresis

may be useful to reduce circulating venom concentrations and eliminate venom-mediated mediators[19].This method was not used in this case, due to its unavailability. This patient experienced a massive envenomation resulting in multi-organ failure, particularly acute renal failure. Furthermore, this patient benefited from effective management despite the absence of a specific protocol for managing envenomations in general and bee stings in particular. Specific antivenom immunotherapy is not yet available.

4. CONCLUSION

The renal and hemorrhagic complications found in this patient provide evidence of the seriousness of bee stings with envenomations. The prognosis depends on the location and number of stings. In this case, appropriate treatment is essential.

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THIS STUDY WAS CONDUCTED BY ETHICAL PRINCIPLES AND RECEIVED APPROVAL FROM THE INSTITUTIONAL REVIEW BOARD (IRB) OF THE FACULTY OF MEDICAL SCIENCES UNIVERSITY OF ZINDER (FSS-UAS), NIGER. THE IRB REVIEWED THE RESEARCH PLAN, ENSURING THAT IT ADHERED TO ETHICAL STANDARDS AND GUIDELINES FOR CONDUCTING RESEARCH INVOLVING HUMAN SUBJECTS. THE APPROVAL FROM THE IRB SIGNIFIES COMPLIANCE WITH THE PRINCIPLES OF CONFIDENTIALITY, INFORMED CONSENT, AND THE PROTECTION OF PARTICIPANTS' RIGHTS.

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