CASE REPORT

A Case of Massive Envenomation with Bee Sting: A Case Report

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Abstract

Envenomation from Hymenoptera stings, including honeybees, wasps, and hornets, usually results in local erythema and edema. Envenomation with bee stings can have varied presentations, from local erythema and systemic reactions (intravascular hemolysis, rhabdomyolysis, acute kidney injury, and liver dysfunction) to anaphylaxis, depending on the number of stings, comorbidities of the patient, and prior sensitization to bee stings. We report a case of massive bee sting envenomation with over 200 bee stings on a patient who presented to our hospital and developed rhabdomyolysis, liver dysfunction, and an elevated troponin I. Early interventions with adequate fluid resuscitation, urine alkalinization, and hepatoprotective measures resulted in a favorable outcome.

Keywords: Anaphylaxis, Bee sting, Case report, Envenomation, Rhabdomyolysis, Urine alkalinization.

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Introduction

Hymenoptera stings include insects from Apidae (bees), Vespidae (wasps and hornets), and Formicidae (ants). The stings typically result in localized erythema, edema, and pain. Complications like acute kidney injury, rhabdomyolysis, thrombocytopenia, liver dysfunction, myocardial infarction, seizures, and acute stroke occur with massive envenomation. Atopic patients, for example, those with allergic rhinitis, bronchial asthma, or prior sensitization to bee venom, can present with anaphylactic reactions like bronchoconstriction. We present a case of a 57-year-old female with massive envenomation from bee stings, who presented to our hospital within hours of the event.

Case Description

A 57-year-old female presented to the ER after an unprovoked attack by a large swarm of bees near her farm, complaining of rashes all over her face, back, chest, and both upper limbs. She had diabetes mellitus and hypothyroidism as comorbidities. She was initially taken to a nearby government hospital within an hour of being stung, where she was treated with injectable pheniramine maleate and hydrocortisone. Since she was found to have elevated troponin I levels, she was referred to our hospital for further management.

On presentation to the emergency room, she was found to have multiple erythematous rash-like lesions with numerous bee stingers *in situ* all over her upper torso, back, and both upper limbs, along with facial puffiness, eyelid edema, and swollen lips. Her respiratory rate was 28/minute, oxygenation saturation was 98%, and she was able to maintain her airway. Her heart rate was 73/minute, blood pressure was 130/70 mm Hg. Her Glasgow Coma Scale was E4V5M6, and pupils were bilaterally reactive to light. On physical examination, she was found to have numerous erythematous lesions over the face, bilateral upper limbs, chest, and upper back. She also had swelling of both upper and lower lips. She was shifted to the critical care unit after initial stabilization. In the intensive care unit, she was reevaluated and found to have numerous visible stingers, which were removed. She also had angioedema of both lips without involvement of the soft palate and uvula. The rest of the physical examination was unremarkable. Her initial blood sugar levels were elevated, with a random blood sugar of 409 mg/dL and arterial blood gas suggestive of metabolic acidosis. Her urine dipstick assay was positive for ketones (++). She was started on intravenous (IV) crystalloids with Ringer's lactate at 125 mL/hour and insulin at 0.1 units/kg body weight as diabetic ketoacidosis treatment. Her presentation blood investigations showed a total count of 18,000/cc; serum creatinine was 0.7 mg/dL, aspartate aminotransferase (AST) was 117 U/L, alanine transaminase (ALT) was 37 U/L, an international normalized ratio of 1.22, creatine phosphokinase (CPK) was >8000 U/L, and high sensitivity (HS) troponin I 4700 pg/mL (normal <9 pg/mL). She denied any difficulty in breathing, lightheadedness, or swallowing difficulty. She did not have anaphylaxis or anaphylactic shock. She was administered IV hydrocortisone 100 mg three times a day, IV chlorpheniramine maleate three times a day, IV ranitidine 50 mg three times a day, and IV fentanyl boluses for pain relief. Within the next 4 hours of presentation, dark cola-colored urine was noticed. Urine alkalinization was initiated with 150 mEq of sodium bicarbonate in 1 L of Ringer's lactate, infused at

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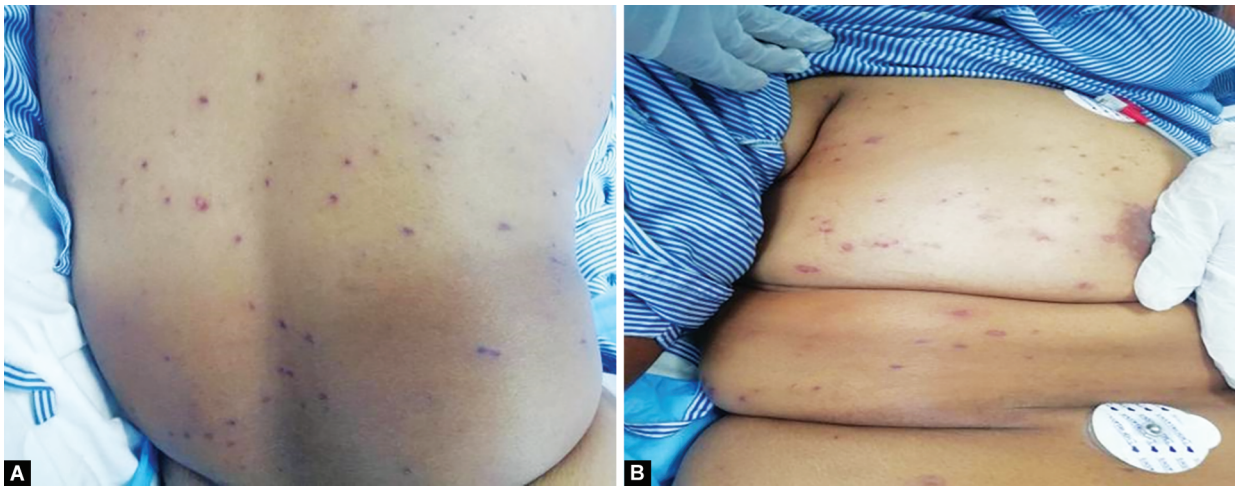
125 mL/hour. Fluids were titrated to achieve a urine output of 1–2 mL/kg/hour. The patient's CPK levels started to downtrend from day 5. The patient was initiated on injectable ceftriaxone as an empirical antibiotic cover in view of multiple skin lesions due to the stings (Table 1).

Her kidney functions remained within normal limits till discharge, probably due to early intervention. Subsequent liver function tests on the following day showed elevation in AST and ALT. N-acetylcysteine infusion for 72 hours was initiated as a hepatoprotective measure as per a nonparacetamol protocol of 150 mg/kg in 1 hour, followed by 12.5 mg/kg/hour for 4 hours, which was followed by 6.25 mg/kg/hour for 67 hours. Liver function tests started to normalize from day 5. The patient did not show any signs of encephalopathy. She was discharged on day 8 in good health (Figs 1 and 2).[1–15]

Discussion

Bee sting venom is composed of several components, including proteins, peptides, amino acids, phospholipids, sugars, and biogenic amines like melittin, phospholipase A, apamin, hyaluronidase, and mast cell degranulating peptide. Melittin, a major component of the venom, can cause pain and is involved in hemolysis as a lytic peptide. Phospholipase A, the second-largest component of bee venom, acts synergistically with melittin to cleave membrane phospholipids and cause hemolysis. Clinical manifestations of bee envenoming can present in the following ways: (1) local inflammatory reactions; (2) allergic manifestations; (3) anaphylactic shock; and (4) systemic toxic reactions. Local inflammatory reactions like pain, swelling, and pruritus are the most common manifestations. Venom-related systemic reactions are less common, and anaphylaxis is the least common. Allergic reactions are immunoglobulin E-mediated type I hypersensitivity reactions, largely mediated through phospholipase A. Anaphylactic shock occurs more often in patients who have had prior sensitization. Systemic reactions are mediated by the toxic effects of the venom itself and are seen with envenomation of >50 stings during one attack, characterized by nausea, vomiting, diarrhea, intravascular hemolysis, rhabdomyolysis, acute renal failure, and myocardial injury.

Fig. 2: The above image shows the stingers which were removed from the patient



Figs 1A and B: The above images show the honeybee sting marks

Table 1: Table showing outcomes of the Investigation

Investigation	*Day 1*	*Day 2*	*Day 3*	*Day 4*	*Day 5*	*Day 6*	*Day 7*
Total count (cc)	18,000	15,500	15,600	16,400	13,300	Not done	12,700
Serum creatinine (mg/dL)	0.71	0.67	Not done	0.57	0.53	0.57	0.61
Blood urea nitrogen (mg/dL)	12		Not done		6	6	3
AST (U/L)	117	666	Not done	493	175	156	
ALT (U/L)	37	101		144	106	123	
Urine ketones	++	+		Nil			
CPK (U/L)		>8000		18687	9094	7989	5184

Massive envenomation is defined as a minimum of 50 stings, causing systemic toxicity due to the amount of venom inoculated, with an estimated lethal dose of 500–1,000 stings. Our patient had over 200 stings and exhibited features of systemic toxicity, including intravascular hemolysis and rhabdomyolysis, with the appearance of cola-colored urine and elevated serum creatine phosphokinase levels. She also had elevated transaminases and cardiac troponin I (HS troponin I). Early identification of intravascular hemolysis and/or rhabdomyolysis and early initiation of treatment are of paramount importance. In our patient, although the initial HS troponin I level was 4700 U/L, no changes were seen in electrocardiogram or two-dimensional echocardiogram; hence, further HS troponin I levels were not pursued. Liver dysfunction, indicated by elevated transaminases, was conservatively managed with N-acetylcysteine following a nonparacetamol poisoning protocol. Our patient was discharged home in a stable condition. It should be borne in mind that there can be potential fatality when management is delayed or insufficiently administered following bee stings.

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