Case Report

MODS and *Enterobacter*-related cellulitis following multiple wasp stings

Stalin Viswanathan[1], Bhavith Remalayam[2], Vivekanandan Muthu[3], Jolly Anil[1]

[1]Department of General Medicine, Indira Gandhi Medical College and Research Institute, Kathirkamam, Pondicherry, India.
[2]Department of Internal Medicine, Pondicherry Institute of Medical Sciences, Kalapet, Pondicherry, India.
[3]Department of General Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Gorimedu, Pondicherry, India.
Correspondence to: Stalin Viswanathan, E-mail: stalinviswanathan@ymail.com

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Abstract

Skin infections following insect bites and stings are unusual. Apart from the local and systemic reactions that have been generally described, wasp stings also cause significant morbidity due to skin infections. We report the case of a 75-year-old lady with multiple wasp stings who developed rhabdomyolysis, renal failure, hepatitis, and cellulitis. During her hospital stay, the sites of wasp stings formed pustules and grew *Enterobacter cloacae*. She improved with antibiotics and multiple sessions of hemodialysis.

KEY WORDS: Wasp sting cellulitis, skin infections, hymenoptera, insects

Introduction

Insects cause injuries by their bites and stings, allergic reactions to secretions, systemic toxicity, and by transmitting diseases.[1] Skin infections may follow trauma such as injections, insect bites, burns and postoperative injuries, and can lead on to necrotizing fasciitis.[2] Among the insects, centipedes, spiders, and hymenoptera have been reported to cause skin lesions.[2,3] Among the hymenoptera, only bee stings-related skin infections have been described. We report a rare *Enterobacter cloacae*-related cellulitis following multiple wasp stings.

Case Report

A 75-year-old nondiabetic lady was admitted with multiple wasp stings (>50) to her scalp, neck, and chest, which were received while working in a banana plantation. She had been administered intravenous fluids, hydrocortisone, and tramadol at a community health center; she was referred for persisting pain at the sting sites even after the extraction of seven stingers. At admission, 1 h after the stinging incident, she was in pain, conscious and oriented with a pulse of 98 beats/min, blood pressure 130/90 mmHg, and respiratory rate of 25/min. She had multiple sting marks on her anterior chest, neck and forearms, and the systemic examination was normal. Investigations revealed mild leukocytosis, blood sugar of 19.44 mmol/L, deranged liver and renal functions [Table 1], elevated creatine kinase [CK], and lactate dehydrogenase [LDH] enzymes. Viral markers and ceruloplasmin levels were noncontributory. Urine hemoglobin and myoglobin were positive; she had a normal chest radiograph and abdominal ultrasonogram.

Central venous access (internal jugular) was secured and alkaline diuresis was initiated for rhabdomyolysis under central venous pressure [CVP] monitoring. On day 3, she developed skin induration and pustules with surrounding necrosis [Figure 1] at the site of stings and was associated with increase in heart and respiratory rates. On the sixth and eighth days, her pustule fluid and urine cultures grew *E. cloacae* and *Proteus vulgaris*, respectively, which responded well to imipenem. Her renal functions continued to deteriorate from the time of admission and were accompanied by mild dyspnea. She did not have recorded fever

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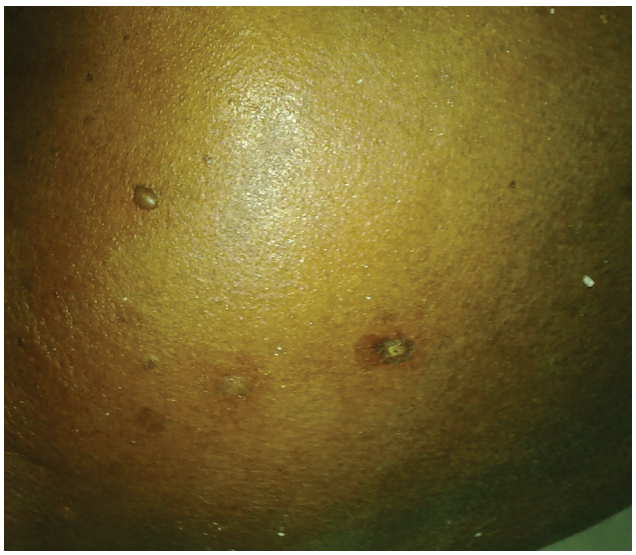
Table 1: Investigations of patient from day 1 to day 20 of hospitalization

Tests	Normal	Day 1	Day 2	Day 4	Day 5	Day 9	Day 14	Day 17	Day 20
Hb	12–14 g/dL	13.3	12.8	9.7	9.2	8.0	8.6	8.5	8.2
TLC	4–9.5 × 10^9/L	10.7	16.9	13.5	12.5	16.8	20.5	10	8.1
Platelets	150–450 × 10^9/L	320	220	340					
PT	Seconds	1.7			1.2				
aPTT	Seconds	41.6			3.4				
Urea	2.5–7 mmol/L	19.28	20	26	50.7	32.8			
Creatinine	44–80 µmol/L	105.6	184	272.8	536.8	413.6	554	440	290.4
SGOT	0.2–0.65 µkat/L	92	64	51.2	19.35	1.02	0.8	0.2	
SGPT	0.12–0.7 µkat/L	167	28.4	10.56	6.3	3.7	1.58	0.96	
ALP	0.56–1.6 µkat/L	1.90	2.10	1.10		3.4	2.9	2.2	
Bilirubin	5.1–22 µmol/L	52.7	28.9						
Direct	1.7–6.8 µmol/L	10.2	6.8						
Protein	7–10 g/dL	7.1			5.4				
Albumin	4–5.5 g/dL	4.0			3.0				
CK	0.66–40 µkat/L	205							
LDH	225–410 U/L	810							

Hb, hemoglobin; TLC, total leukocyte count; PT, prothrombin time prolongation in seconds; aPTT, activated partial thromboplastin time prolongation in seconds; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; ALP, alkaline phosphatase; CK, creatine kinase; LDH, lactate dehydrogenase



Q1 **Figure 1:**

or hemodynamic instability during her hospital stay. Her hemoglobin level continued to fall until day 9 and was probably related to intravascular hemolysis, rhabdomyolysis, and hemodialyses. Tachypnea stabilized after five sessions of hemodialysis, bicarbonate infusions, packed cell transfusion, and use of a Venturi mask (FiO_2 35%). By the 17th day her liver functions had normalized, while creatinine levels had stabilized by the 18th day. She was discharged on day 21 to complete her antibiotics at home. Two weeks on a follow-up visit, her creatinine level was 106 µmol/L. She was lost to follow-up thereafter.

Discussion

Insect bites can lead to various skin lesions such as pustules, vesicles, papules, nodules, erosions and urticaria.[4] These cutaneous lesions vary from person to person. Histologically, epidermal spongiosis, eosinophilic panniculitis, eosinophilic hidradenitis, crusts with eosinophils and neutrophils, spongiotic infundibulitis, perivascular and periadnexal infiltrate are seen.[4] Hymenoptera sting lesions can present as focal spongiosis, dyskeratosis, or hyperplasia.[4] Papillary dermis edema is suggestive of hymenoptera sting.[5]

Severity of hymenoptera sting depends on the site of sting, volume of venom injected, and the patient's allergic status.[6] The exact sequence of transmission of infection from hymenoptera is not known. Breakdown of skin may be the portal of entry of various organisms to cause disseminated infection. Retained stingers, surrounding edema, allergic venom, pruritus, and inoculation of surface organisms lead to infections[5]—the first two factors were contributory in our patient. Apamin, histamine, and melittin in bee venom contribute to hemolysis and an inflammatory reaction.[5] Venom of wasps contain serotonins, amines, peptides, proteases,[2] acetylcholine, mastparan, phospholipase A, and hyaluronidase, which causes proteolysis and rhabdomyolysis.[7] Both IgE-mediated anaphylaxis and direct toxic effect of wasp venom occur.[7]

E. cloacae infections are seen in a wide variety of hospital settings such as infusions/parenteral feeds, catheters/grafts, and even in operation suites.[8] These infections are mostly urinary and respiratory and have been generally observed in immunocompromised patients and in victims of burn injuries.[8] Apart from old age, our patient did not have any other reasons for impaired immunity. Blood and fluid cultures

were sent only on the third day after she developed pustules—blood cultures were sterile. Disruption in the skin barrier due to stings/retained stingers may have predisposed toward entry of these commensal organisms, leading to cellulitis.

Nine reports of infection following bee stings have been reported. Five cases of necrotizing fasciitis and one case each of eosinophilic cellulitis, labial abscess, staphylococcal toxic shock syndrome, and osteomyelitis have been reported in the literature.[7,8] One *Streptococcus pyogenes*-related necrotizing fasciitis had developed in a 71-year-old man within 2 weeks of bee sting and had ended fatally.[7] The other four patients had improved with surgical debridement, antibiotics, and skin grafting.[6,7] The patient with the labial abscess had been stung on the floor of the nose, which healed 4 weeks after surgical drainage and intravenous antibiotics.[5] Cellulitis and necrotizing fasciitis have not been previously reported following wasp stings. A Japanese study of wasp stings-related cutaneous findings reported the ability of skin necrosis/hemorrhage to predict multiorgan dysfunction similar to our patient.[9] Number of stings >50 generally predicted multiple organ injury. Our patient had comparable skin necrosis at the site of sting but also had induration of skin of neck, anterior and posterior chest, and both forearms. Immune deficiencies due to diabetes, alcoholism, and chronic kidney disease can lead to cellulitis and necrotizing fasciitis arising from insect bites.[3] Disseminated intravascular coagulation was not suspected in this patient since the platelet counts were persistently normal and the initially abnormal aPTT and PT values had also normalized by fifth day [Table 1].

Conclusion

Apart from the local and large local skin reactions, wasp stings can cause infective cellulitis, which may lead to morbidity. Early and aggressive therapy with antibiotics, fluid management and, on occasion, debridement leads to recovery. Skin hemorrhages/necrosis as in our patient may predict multiorgan dysfunction in wasp sting victims.

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