MODS and Enterobacter-related cellulitis following multiple wasp stings

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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. Skin infections may follow trauma such as injections, insect bites, burns and postoperative injuries, and can lead on to necrotizing fasciitis. Among the insects, centipedes, spiders, and hymenoptera have been reported to cause skin lesions. 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, elevated creatine kinase and lactate dehydrogenase 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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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 hemodialysis. Tachypnea stabilized after five sessions of hemodialysis, bicarbonate infusions, packed cell transfusion, and use of a Venturi mask (FiO₂ 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.

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

<table>
<thead>
<tr>
<th>Tests</th>
<th>Normal</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 9</th>
<th>Day 14</th>
<th>Day 17</th>
<th>Day 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>12–14 g/dL</td>
<td>13.3</td>
<td>12.8</td>
<td>9.7</td>
<td>9.2</td>
<td>8.0</td>
<td>8.6</td>
<td>8.5</td>
<td>8.2</td>
</tr>
<tr>
<td>TLC</td>
<td>4–9.5 x 10⁹/L</td>
<td>10.7</td>
<td>16.9</td>
<td>13.5</td>
<td>12.5</td>
<td>16.8</td>
<td>20.5</td>
<td>10</td>
<td>8.1</td>
</tr>
<tr>
<td>Platelets</td>
<td>150–450 x 10⁹/L</td>
<td>320</td>
<td>220</td>
<td>340</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PT</td>
<td>Seconds</td>
<td>1.7</td>
<td></td>
<td>1.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aPTT</td>
<td>Seconds</td>
<td>41.6</td>
<td></td>
<td>3.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urea</td>
<td>2.5–7 mmol/L</td>
<td>19.28</td>
<td>20</td>
<td>26</td>
<td>50.7</td>
<td>32.8</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Creatinine</td>
<td>44–80 µmol/L</td>
<td>105.6</td>
<td>184</td>
<td>272.8</td>
<td>536.8</td>
<td>413.6</td>
<td>554</td>
<td>440</td>
<td>290.4</td>
</tr>
<tr>
<td>SGOT</td>
<td>0.2–0.65 µkat/L</td>
<td>92</td>
<td>64</td>
<td>51.2</td>
<td>19.35</td>
<td>1.02</td>
<td>0.8</td>
<td>0.2</td>
<td></td>
</tr>
<tr>
<td>SGPT</td>
<td>0.12–0.7 µkat/L</td>
<td>167</td>
<td>28.4</td>
<td>10.56</td>
<td>6.3</td>
<td>3.7</td>
<td>1.58</td>
<td>0.96</td>
<td></td>
</tr>
<tr>
<td>ALP</td>
<td>0.56–1.6 µkat/L</td>
<td>1.90</td>
<td>2.10</td>
<td>1.10</td>
<td>3.4</td>
<td>2.9</td>
<td>2.2</td>
<td></td>
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<tr>
<td>Bilirubin</td>
<td>5.1–22 µmol/L</td>
<td>52.7</td>
<td>28.9</td>
<td></td>
<td></td>
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<tr>
<td>Direct</td>
<td>1.7–6.8 µmol/L</td>
<td>10.2</td>
<td>6.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>7–10 g/dL</td>
<td>7.1</td>
<td>5.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>4–5.5 g/dL</td>
<td>4.0</td>
<td>3.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td>0.66–40 µkat/L</td>
<td>205</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>LDH</td>
<td>225–410 U/L</td>
<td>810</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

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.

**Discussion**

Insect bites can lead to various skin lesions such as pustules, vesicles, papules, nodules, erosions and urticaria. These cutaneous lesions vary from person to person. Histologically, epidermal spongiosis, eosinophilic panniculitis, eosinophilic hidradenitis, crusts with eosinophils and neutrophils, spongiform infundibulitis, perivascular and perinodal infiltrate are seen. Hymenoptera sting lesions can present as focal spongiosis, dyskeratosis, or hyperplasia. Papillary dermis edema is suggestive of hymenoptera sting. Severity of hymenoptera sting depends on the site of sting, volume of venom injected, and the patient’s allergic status. 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, pruritis, and inoculation of surface organisms lead to infections—the first two factors were contributory in our patient. Apamin, histamine, and melittin in bee venom contribute to hemolysis and an inflammatory reaction. Venom of wasps contain serotonin, amines, peptides, proteases, acetylcholine, mastparan, phospholipase A, and hyaluronidase, which causes proteolysis and rhabdomyolysis. Both IgE-mediated anaphylaxis and direct toxic effect of wasp venom occur.

*E. cloacae* infections are seen in a wide variety of hospital settings such as infusions/parenteral feeds, catheters/grafts, and even in operation suites. These infections are mostly urinary and respiratory and have been generally observed in immunocompromised patients and in victims of burn injuries. 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—

to stings/retained stingers may have predisposed toward

erve of these commensal organisms, leading to cellulitis.

Nine reports of infection following bee stings have been

were 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 necrotiz-
ing 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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