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DOI:  
10.4103/tjem.tjem\_138\_22

# Severe pulmonary-renal syndrome in honeybee sting envenomation – A case report

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## Abstract:

Honeybee stings with features of envenomation (either local or allergic and rarely systemic toxicity) are often seen in agriculture workers. An initial presentation with severe diffuse alveolar hemorrhage or pulmonary-renal syndrome is rare and only confined to a few case reports. Herein, we describe a case of a 45-year-old male who presented with multiple bee stings and subsequently developed acute kidney injury and pulmonary hemorrhage. He was managed with hemodialysis, invasive mechanical ventilation, red-cell transfusion, and pulse methylprednisolone. However, he developed cardiac arrhythmias with ventricular tachycardia and died.

## Keywords:

Bee sting, case report, diffuse alveolar hemorrhage, envenomation, honeybee, pulmonary-renal syndrome, renal failure

## Introduction

Bee sting and subsequent hypersensitivity reactions constitute frequent emergency department visits globally. Severe adverse events or life-threatening organ failure may occur, especially after multiple stings.<sup>[1]</sup> Renal dysfunction is not uncommon and usually has a good prognosis.<sup>[2]</sup> Diffuse alveolar hemorrhage (DAH) is rarely described with bee sting and is limited to only case reports.<sup>[3-5]</sup> Management is primarily intensive supportive care. Corticosteroid therapy has been tried. Herein, we describe a case of multiple bee stings complicated by renal dysfunction, pulmonary hemorrhage, and cardiac arrhythmias.

## Case Report

A 45-year-old male, farmer by occupation, had multiple bee stings while working on

his farm. He applied homemade herbal preparations over the bite sites. Two days later, he started having a decreased urine output and facial puffiness. He had one episode of hemoptysis and presented to our institute's emergency department. He did not have other bleeding tendencies. There was no history of fever, dysuria, myalgia, or arthralgia. His past medical history was unremarkable, without any comorbidities. On physical examination, the pulse was 110 beats per min, blood pressure was 136 / 90 mm Hg, respiratory rate was 26 breaths per minute, and the oxygen saturation was 92% while the patient was breathing high-flow oxygen through a venturi mask. He had multiple sting marks on his arms, face, and trunk with surrounding erythema [Figure 1]. Pallor, periorbital puffiness, and edema over both shins were noted. Lung auscultation

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**How to cite this article:** Shaji A, Parvez M, Chirumamilla NK, Sharma N, Pannu AK. Severe pulmonary-renal syndrome in honeybee sting envenomation – A case report. Turk J Emerg Med 2023;23:246-9.

Submitted: 01-05-2022  
Revised 1: 03-06-2022  
Revised 2: 04-07-2022  
Accepted: 05-07-2022  
Published: 28-09-2022

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revealed inspiratory crackles diffusely scattered over both lung fields.

Laboratory evaluation showed anemia with a hemoglobin drop from 10.2 g/dL to 7.5 g/dL within 24 h, consistent with hemoptysis. His total leukocyte count was  $13 \times 10^9/L$  and platelet count was  $178 \times 10^9/L$  at presentation and did not show any falling trend. The coagulation profile was normal with an international normalized ratio of 1.1. He had acute kidney injury (AKI) with blood urea of 434.0 mg/dL, creatinine of 16.0 mg/dL, and metabolic acidosis (blood pH, 7.2; bicarbonate, 9 mmol/L). Serum bilirubin, liver enzymes, and creatinine kinase were grossly normal. Urine routine analysis showed albuminuria (2+ on dipstick) and no red cell or cast. A 24-h urine examination detected 1.4 g of protein. A chest X-ray revealed perihilar infiltrates in both lungs with peripheral sparing [Figure 2]. Hemodialysis was started for AKI; however, the dyspnea and oxygen saturation did not improve. The hemoglobin further decreased to 6 g/dL. A thoracic computed tomography revealed diffuse alveolar infiltrates sparing periphery in both lungs [Figure 3].

The patient had systemic envenomation with AKI. His shortness of breath was initially attributed to fluid overload secondary to AKI. However, due to hemoptysis, progressive hemoglobin drop, and no improvement after multiple hemodialysis sessions, a possibility of DAH was kept. Chest imaging was also consistent with DAH. Testing for common etiologies for the pulmonary-renal syndrome, including antineutrophil cytoplasmic antibodies, antinuclear antibodies, complements (C3 and C4) levels, double-stranded DNA antibodies, and anti-glomerular basement membrane antibodies was noncontributory. Serologies for the human immunodeficiency virus, hepatitis B, and hepatitis C were negative. Workup for hemolysis (i.e. plasma hemoglobin, urine hemoglobin, G6PD enzyme deficiency, and schistocytes on the peripheral blood smear) was negative. After ruling out common etiologies for the immune-mediated pulmonary-renal syndrome, bee sting envenomation-related DAH was diagnosed.

For envenomation-related DAH, the patient was treated with pulse methylprednisolone pulse 1 g/day for 5 days. Hemodialysis was given along with packed red cell transfusions. For progressive respiratory failure and DAH, he required invasive mechanical ventilation. Topical mupirocin was applied over the skin lesions, and intravenous piperacillin-tazobactam was started. However, he developed ventricular tachyarrhythmia on Day 7 of hospitalization and died.



Figure 1: Bee sting marks on patient's back



Figure 2: Chest X-ray showing perihilar infiltrates in both lungs with peripheral sparing

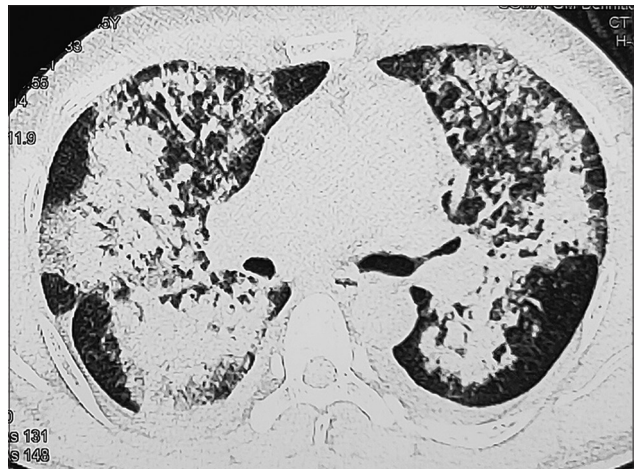


Figure 3: Thoracic computed tomography showing diffuse alveolar infiltrates sparing periphery in both lungs

Written informed consent is present. The patient's brother was explained about the confidentiality, and the case information will be used for education purposes only.

## Discussion

Hymenoptera insects are characterized by having two pairs of wings. Within the hymenoptera order, the three families of stinging insects are the Apidae (honeybees and bumblebees), Vespidae (hornets, yellowjackets, and wasps), and Formicidae (ants). The honeybee and the bumblebee are the only two bees of medical importance. Bee sting envenomation can range from mild-to-severe clinical features and depends on the number of stings, atopy, age, and associated medical comorbidities.<sup>[1,6]</sup> Most patients have local manifestations, which are usually self-limiting. Allergic features such as pruritus, angioedema, and diarrhea and anaphylactic reactions such as bronchoconstriction and shock are attributed to biogenic amines in the venom and result from immunoglobulin E-mediated hypersensitivity.<sup>[1]</sup> Systemic features include vomiting, diarrhea, myalgia, hemolysis, rhabdomyolysis, AKI, hypotension, vasculitis, and rarely, pulmonary hemorrhage.<sup>[7]</sup> Systemic envenomation is thought to be mediated by hyaluronidase in the venom, resulting in the cleavage of hyaluronic acid from the extracellular matrix.<sup>[6]</sup>

The pathogenesis of AKI is usually multifactorial. It includes ischemic acute tubular necrosis secondary to hypotension, toxin-induced direct tubular injury, pigment nephropathy due to hemolysis or rhabdomyolysis, vasculitis, glomerulonephritis, and thrombotic microangiopathy.<sup>[8-10]</sup> In the index patient, the AKI was likely due to tubular injury with or without glomerular involvement, as evident by the nonnephrotic range proteinuria; however, the histopathological examination could not be performed.

Bee sting-related DAH is rare. The proposed mechanisms include hypersensitivity to the bee venom and direct toxicity to alveolar capillaries manifesting as alveolocapillaritis or as a part of systemic vasculitis.<sup>[3-5]</sup> The onset is usually abrupt, i.e. within a week of the bee sting. Clinical presentation is similar to other autoimmune causes, such as cough, breathlessness, fever, and hemoptysis.<sup>[11]</sup> Chest imaging reveals diffuse bilateral alveolar opacities with peripheral sparing. Because fluid overload may mimic the clinic-radiological picture, the presence of associated advanced renal failure may delay the diagnosis. Management is usually supportive with ventilation and blood transfusion. Because prospective data are lacking, the role of steroids is limited to the case reports. A therapeutic benefit of plasma exchange is not described. Cardiac manifestations include myocardial ischemia, myocarditis, and arrhythmias (including conduction blocks and tachyarrhythmia).<sup>[12]</sup> These

manifestations can occur up to a week after the sting, thus requiring vigilance, appropriate treatment, patient education, and follow-up. Unlike ours, the patients with systemic envenomation who present within 24–48 h of bee sting had favorable outcomes.<sup>[3-5]</sup>

## Conclusion

Life-threatening adverse events after bee sting envenomation are infrequent; however, it is imperative to recognize severe features early, particularly if multiple stings are noted. DAH is rarely described as a bee sting. Management is primarily intensive supportive care and a delay in treatment results in a fatal outcome. Education of at-risk populations and prompt medical care are essential. Further studies on the role of steroids, immune modulators, and potential antivenom therapy are mandated.

### Author contributions

<sup>1</sup>AS: collected patient data, drafted the manuscript

<sup>2</sup>MMP, <sup>3</sup>NKC, <sup>4</sup>NS: collected patient data

<sup>5</sup>AKP: collected patient data, drafted and revised the manuscript

The corresponding author is responsible for ensuring that the descriptions are accurate and agreed by all authors

### Conflicts of interest

None declared.

### Patient's consent

Written informed consent is present. The patient's brother was explained about the confidentiality, and the case information will be used for education purposes only.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

### Funding

None.

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