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Pediatric acute kidney injury following bee sting-induced anaphylaxis: a case series

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Abstract

Background Bee stings account for approximately 14% of anaphylactic reactions and are the second most common trigger after food allergies. The body's response to bee venom can range from allergic reactions to systemic toxicity, with a mortality rate of 15–25%. Survivors might develop complications such as acute kidney injury, hypertension, anemia, rhabdomyolysis, liver damage, heart attack, and breathing difficulties, depending on the number of stings.

In this case series, we discuss two pediatric patients who experienced acute kidney injury following a bee sting, emphasizing the risk of severe systemic complications in children.

Case presentation We report two Black African boys with bee sting-induced acute kidney injury: an 11-year-old and a 7-year-old, both with swelling and reduced urine output. Both underwent three hemodialysis sessions and recovered renal function. A year later, the 11-year-old's kidneys were normal, but the 7-year-old was lost to follow-up.

Conclusion Bee stings, the second leading cause of fatal anaphylaxis after food allergies, can cause serious complications. This case series emphasizes the importance of early detection and swift treatment to improve outcomes.

Keywords Bee sting, Anaphylaxis, Renal failure, Hymenoptera venom, Acute kidney injury (AKI)

Introduction

Bees, belonging to the order Hymenoptera, alongside wasps, are ecologically essential but also medically significant owing to their ability to inflict venomous stings. Among the various species, Africanized bees, commonly known as "killer bees," are highly aggressive and tend to attack in large numbers, delivering substantial amounts of venom and posing a significant risk to humans. The risk of developing severe diseases following bee stings has not been extensively documented in children, particularly in Africa [1].

Epidemiologically, bee stings are a notable cause of morbidity, accounting for approximately 14% of anaphylactic reactions, surpassed only by food-induced anaphylaxis, which constitutes about 33% of cases [1, 2], In cases of multiple stings, mortality rates range from 15% to 25%, with survivors at risk of severe complications, including acute kidney injury (AKI), hypertension, transfusion-dependent anemia, rhabdomyolysis, hepatic dysfunction, myocardial infarction, and respiratory distress. These adverse outcomes are largely driven by the venom's principal components, melittin and phospholipase A2, which together comprise 62% of the venom's composition and are key mediators of its toxicity [3].

Acute kidney injury (AKI) stands out as one of the most critical complications of bee venom toxicity. The pathogenesis of AKI following bee stings is multifaceted,

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likely involving direct nephrotoxic effects of venom components, acute tubular necrosis, pigment nephropathy secondary to hemolysis and rhabdomyolysis, and hypoperfusion from anaphylactic shock.

This case series presents two pediatric patients, aged 11 and 7 years, who developed profound systemic complications following bee stings. Over a 1-year follow-up period, the 11-year-old demonstrated clinical stability, whereas the 7-year-old was lost to follow-up after the initial year. This report underscores the complexities of managing bee venom toxicity and offers contemporary insights into the clinical care of patients with severe systemic reactions to bee stings, contributing to the broader understanding of this medical challenge within the scientific community.

Case history

Case 1

An 11-year-old Black African boy presented with a 1-week history of generalized body swelling. The swelling was acute in onset, starting with the face and neck, and progressively involved the abdomen, scrotum, and lower limbs following multiple bee stings sustained while returning home from school. He also reported a 4-day history of reduced urine output, which was scant, without blood, and not associated with painful urination.

Additionally, he experienced postprandial vomiting for 1 day, with approximately four episodes, with no abdominal pain, early satiety, difficulty breathing, chest pain, palpitations, or itching. He had no known food or drug allergies. Initially evaluated at a local healthcare facility and discharged the same day, his condition worsened with persistent vomiting, necessitating readmission and referral to the National Hospital for further evaluation and management.

On examination, the patient was alert and afebrile. He was not pale, jaundiced, dyspneic, or cyanosed and had no palpable peripheral lymph nodes. A popular rash was present on both upper limbs, face, and legs, as shown in Fig. 1, with no lower limb edema. Vital signs included blood pressure (BP) of 121/84 mmHg (above the 95th percentile), respiratory rate of 20 breaths per minute, oxygen saturation (SpO2) of 98% on room air, pulse rate (PR) of 98 beats per minute, weight of 36 kg, height of 140 cm, and body mass index (BMI) of 18.4 kg/m^2 (normal). Abdominal examination revealed significant bilateral scrotal swelling without ascites. Other systemic examinations were unremarkable.

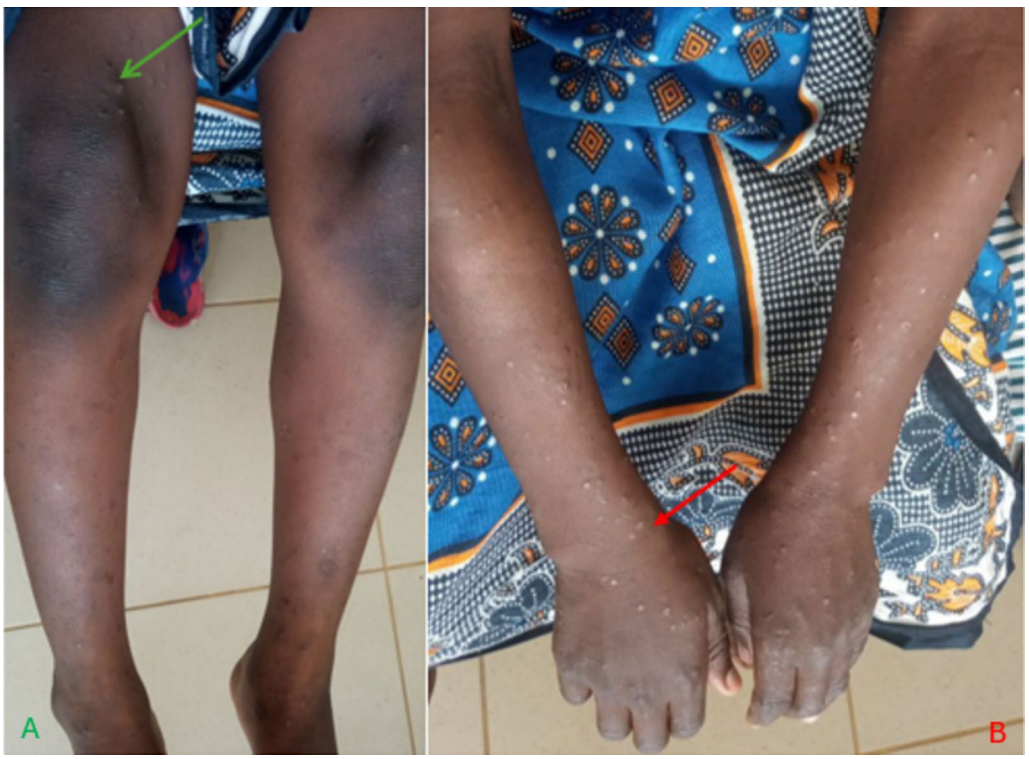

Fig. 1 **A,B** Images showing areas of bee stings with resultant rashes on the upper limbs and parts of the lower limbs; bee stings leaving rashes around the body on the lower limbs around the knee area (**A**), as shown by the green arrow, and some parts of the upper limbs, as shown by the red arrow, on the wrist (**B**)

Laboratory investigations revealed leukocytosis of 22.13×10^9/L with a neutrophil predominance of 19.5×10^9/L (86.5%), hemoglobin of 9.3 g/dL (normal ≥ 11.5 g/dL), normal platelet counts, and markedly elevated creatinine levels of 1540 µmol/L (estimated glomerular filtration rate [eGFR] 3 mL/minute/1.73 m^2). Blood urea was 44.6 mmol/L, and the potassium level was elevated at 5.8 mmol/L, with normal bleeding indices. A kidney, ureter, and bladder (KUB) ultrasound showed normal parenchymal echogenicity, while echocardiography was normal.

The patient was managed for acute kidney failure and hypertension with intravenous furosemide 20 mg twice daily and oral amlodipine 5 mg once daily. Subsequently, the patient underwent three sessions of hemodialysis, each lasting 3 hours, with the second and third sessions adhering to dietary restrictions for high salt and potassium intake. Monitoring of fluid intake and urine output was maintained. He recovered well, and at the 6-month follow-up, his creatinine was 55 µmol/L (eGFR 93 mL/minute/1.73 m^2), with a normal routine KUB ultrasound. The patient is doing well and has resumed school.

Case 2

A 7-year-old Black African boy presented with a 1-week history of generalized body swelling affecting the face, arms, and legs, accompanied by reduced urine output, characterized by anuria, without blood or painful micturition. He also reported a 3-day history of confusion, marked by inappropriate speech, though without convulsions, aggressive behavior, or blurred vision.

These symptoms began after he was stung multiple times by bees while throwing stones at a hive during play, an incident that led to the death of another child a few hours later. The bee stings caused severe pain and significant periorbital swelling that prevented him from opening his eyes and difficulty walking.

As his condition worsened, he experienced dyspnea, chest tightness, generalized weakness, and awareness of his heartbeat, but no cough or cyanosis (bluish discoloration of the limbs or lips) was noted. He had no known allergies to food or medication. Initially treated at a local facility, he was later referred to the National Hospital, a 12-hour bus journey away, for further evaluation and management.

Upon arrival at the emergency department, the patient appeared alert but ill-looking. There were no danger signs or evidence of dehydration; he was severely pale, with no jaundice or cyanosis. Examination revealed bilateral periorbital edema, tender lip, sores, and multiple bee sting marks on the scalp, face, arms, and legs. Bilateral pitting edema was present in the lower limbs, upper limbs, sacral area, and face, as shown in Fig. 2. No peripheral lymph nodes were palpable.

Vital signs included a blood pressure of 97/53 mmHg (between the 50th and 90th percentile), a pulse rate of 136 beats per minute, a respiratory rate of 20 breaths per minute, an oxygen saturation of 99% on room air, and a temperature of 36.5 °C. Anthropometric measurements were as follows: body weight 27 kg, height 115 cm, and body mass index 21.7 kg/m^2. The remainder of the systemic examination was unremarkable.

Laboratory investigations showed an elevated serum creatinine of 4969 µmol/L (eGFR of 7 mL/minute/1.73 m^2), severe anemia with a hemoglobin level of 5.8 g/dL (normal ≥ 11.5 g/dL), leukocytosis with a white blood cell count of 24×10^9/L (predominantly neutrophils), thrombocytopenia at 78×10^3/µL (normal $150–450\times10^3$/µL), an alanine aminotransferase level of 431 U/L (normal 0–40 U/L), and hyperkalemia at 5.3 mmol/L, with other serum electrolytes within normal limits.

The patient was transfused and admitted to the pediatric unit and received intravenous furosemide 20 mg twice daily, spironolactone 25 mg once daily, and paracetamol 375 mg every 8 hours. His fluid intake was restricted to 500 mL per 24 hours, and he was placed on a low-salt diet. Daily monitoring of body weight, urine output, and blood pressure was performed, along with urine dipstick tests. The patient was managed for acute kidney failure and underwent three sessions of hemodialysis and recovered well.

During treatment, he reported left calf pain, but a Doppler ultrasound ruled out deep vein thrombosis. He also experienced an episode of fever, managed as sepsis with renal-adjusted doses of intravenous meropenem to avoid its toxicity due to slow clearance, leading to increased half-life of meropenem, hence accumulation. The patient

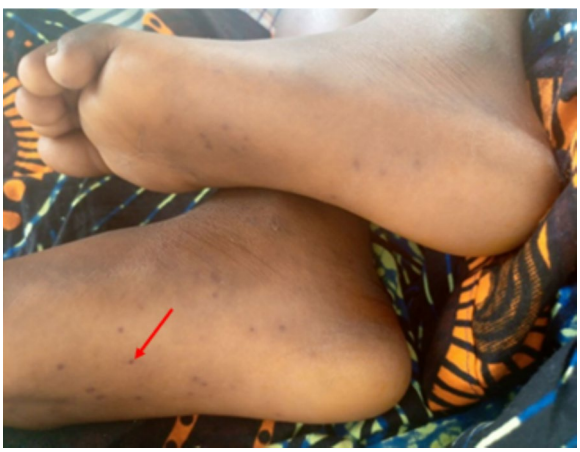

Fig. 2 Image showing part of the soles of the feet with a blackish lesion after a bee sting, as shown by the red arrow

was discharged with a creatinine level of 140 µmol/L (eGFR 30 mL/minute/1.73 m^2) and a follow-up plan scheduled at 2, 6, 12, 24, and 52 weeks, as shown in Fig. 3.

Discussion

This case series illustrates anaphylactic reactions resulting from bee stings, a rare occurrence in literature, which are often fatal owing to the toxicity of bee venom. Bee venom, which contains 62% phospholipase A2 and melittin with toxic components, causes erythema, swelling, and localized pain, followed by immunoglobulin-mediated allergic reactions such as hives, angioedema, and respiratory issues [4, 5]. In both cases, patients showed periorbital swelling, oliguria, and elevated blood urea, indicating renal dysfunction likely caused by venom toxins, including melittin, phospholipase A2, and hyaluronidase [6].

The periorbital edema was probably caused by melittin and phospholipase A2, which compromised vascular integrity by releasing histamine and bradykinin, which increased capillary permeability, while hyaluronidase facilitated venom spread into tissues, causing severe swelling, as observed in our second case [7].

Oliguria, a common presentation of acute kidney injury (AKI), likely results from direct tubular toxicity and tubular obstruction by pigment casts formed from myoglobin released during rhabdomyolysis or hemoglobin released during hemolysis, a frequently documented pathology in literature [1, 2, 7].

In our second case (case 2), the patient presented with severe anemia (hemoglobin 5.8 g/dL), underscoring the hemolytic potency of bee venom, primarily driven by melittin and phospholipase A2, which disrupts the red blood cell membranes and releases hemoglobin into the circulation, potentially requiring blood transfusion [6, 7]. This intravascular hemolysis aligns with the documented systemic effects of bee envenomation, emphasizing its hematological impact.

In our first presented patient (case 1), episodes of confusion may have resulted from the accumulation of uremia toxins due to AKI, or from direct neurotoxic effects of venom components such as apamin, whose exact mechanism remains unknown [6]. It is well documented in literature that bee stings can cause a wide range of toxin-mediated effects, from mild to life-threatening, including hemolysis, pancreatitis, laryngospasm, respiratory distress, myocardial infarction, and stroke. [5, 8, 9].

Palpitations reported by our first patient likely arose from an allergic reaction to bee venom [2], with symptom severity tied to the significant, though unquantified, number of stings; studies note that 30–50 stings in children can be grave, while a single sting may trigger fatal anaphylaxis in hypersensitive individuals [10].

Acute kidney injury (AKI), the most frequent severe complication following bee stings, was diagnosed in both cases on the basis of Kidney Disease Improving Global Outcomes (KDIGO) criteria, which defines AKI as urine output below 0.5 mL/kg/hour for 6 hours or a 1.5-fold increase in serum creatinine within 7 days [11]. Both patients underwent three hemodialysis sessions, restoring normal renal function.

Laboratory investigation for two cases when Inpatient and follow-up

CASE 1	Day	1	4	3week	17 week	
	eGFR	3	13	85	93	
	SCR	1540.9	397.7	60.5	55	
	BUN	44.6	8.5	1.7		
CASE 2	Day	1	7	10	13	16
	SCR	817	4969	645	270	140
	eGFR	5	9	7	16	30
	BUN	45.7	22.9	30	23	14

NB At Discharge
On Followup

Fig. 3 Laboratory investigation for both patients during the inpatient and follow-up period—chart indicating laboratory investigation for estimated glomerular filtration rate (milliliter/minute/1.73 m^2), serum creatinine (micromole/liter), and blood urea for both case 1 and case 2

Both patients were treated for AKI with hemodialysis and achieved complete recovery of renal function. Follow-up data were available only for Patient 1, who showed normal renal function and unremarkable kidney, ureter, and bladder (KUB) ultrasound findings at 3- and 6-month visits. Patient 2 was lost to follow-up, so long-term outcomes remain unknown.

This case series is limited by incomplete follow-up data for the second patient. Adherence to the plans as highlighted in literature remains a challenge [1]; this underscores the need for robust patient tracking systems within the context of emerging technologies.

Conclusion

Bee stings can precipitate severe complications, primarily from toxin exposure, and rank as the second leading cause of anaphylaxis-related mortality after food allergies. This case series highlights the crucial role of early detection and timely management in enhancing outcomes for patients with severe bee sting reactions.

Abbreviations

AKI	Acute kidney injury
BUN	Blood urea
eGFR	Estimated glomerular filtration rate
KDIGO	Kidney Disease Improving Global Outcomes
KUB	Kidney ureter and bladder
HB	Hemoglobin
SGR	Serum creatinine
PRBCs	Packed red blood cells

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Author contributions

AK drafted the first manuscript. AK, AM, EW, EM, PS, GS, and FF reviewed and edited the manuscript. All authors approved the final version of the manuscript.

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Data availability

Will be available upon reasonable request to the corresponding author.

Declarations

Ethics approval and consent to participate

Publication of the case details does not necessitate institutional approval.

Consent for publication

Written informed consent was obtained from the patients' legal guardians to publish this case series and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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