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CASE STUDY

Massive Envenomation Syndrome or Multiple Honey Bee Stings: A case series of Multiple Organ Dysfunctions

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Abstract

Background Multiple organ dysfunction can occur after a large number of honey bee stings, typically developing 24 to 48 hours after the stings.

Case presentation This report describes three cases of multiorgan dysfunction following multiple honeybee stings. Case 1 presented with abnormal renal and liver function. Laboratory investigations revealed leukocytosis (17,000 cells/µl), elevated serum lactate dehydrogenase (925 U/L), and creatine kinase-MB (283 U/L), suggestive of rhabdomyolysis. Urinalysis showed dark-colored urine with marked albuminuria (3+ or > 3 g/24 h). Serum urea (101.8 mg/dl) and creatinine (5.01 mg/dl) were elevated, consistent with acute kidney injury. Liver function tests demonstrated elevated total bilirubin (3.39 mg/dL), indirect bilirubin, and transaminases, consistent with acute liver injury. The patient underwent three sessions of hemodialysis and showed continuous clinical improvement over the following two days. Case 2 involved cerebrovascular stroke secondary to multiple honeybee stings, and Magnetic resonance imaging of the brain demonstrated multiple acute infarcts, and Laboratory investigations were within normal limits. Case 3 presented with severe generalized pain and tachycardia, and Laboratory investigations were within normal limits. Pain was managed effectively using a combination of analgesics, including non-steroidal anti-inflammatory drugs and opioids such as fentanyl and buprenorphine.

Conclusion When an individual sustains more than 20 stings, the volume of venom injected can lead to multiorgan dysfunction. Such patients require urgent medical attention and may necessitate intensive care unit management to minimize the risk of severe anaphylactic reactions.

Keywords Anaphylaxis, Bee Stings, Dialysis, Multiple Organ Dysfunction Syndrome, Venoms

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1 Introduction

Honeybee venom possesses strong allergenic properties, with reactions ranging from mild local responses to life-threatening systemic anaphylaxis and even death. According to the World Allergy Organization, honeybee venom allergy is among the most common causes of adult-onset allergies leading to anaphylaxis.[1] Approximately 14% of anaphylactic reactions are attributed to bee venom. A higher number of stings can cause direct organ injury, and exposure to 50–500 stings may be fatal.[2]
Organs commonly affected include the brain, lungs, muscles, hematological system, and kidneys. Acute kidney injury (AKI) is a well-recognized and potentially fatal complication following multiple bee stings. Although AKI is treatable, its most severe presentation occurs in the context of shock, multiorgan dysfunction, and massive envenomation. Clinical manifestations range from minor local inflammation, with or without systemic effects, to severe anaphylaxis.[3] Prompt and aggressive management, including effective source control, is crucial for improving patient outcomes.
In certain circumstances, individuals may be subjected to numerous stings from a swarm of honeybees, resulting in the injection of large amounts of venom and subsequent multiorgan dysfunction. This report presents three such cases of patients who sustained multiple honeybee stings.

Case Report 1

A 29-year-old male presented to the Emergency Department one day after sustaining multiple honeybee stings. He reported several episodes of dark, non-bilious, non-projectile vomiting, accompanied by 2–3 episodes of diarrhea. He also complained of diffuse abdominal pain, facial swelling, jaundice, and reduced urine output.

Primary Survey

On arrival at the Emergency Department, the patient was conscious. The airway was clear, and both the rate and depth of respiration were normal. Oxygen saturation was 100% on room air. Blood pressure was recorded at 160/100 mmHg, with a heart rate of 79 bpm. Peripheral pulses were regular in rate and rhythm with adequate volume, and capillary refill time was within normal limits (Figure 1).

Secondary Survey

On assessment, the patient was conscious, coherent, and responsive to commands, oriented to time, place, and person. Multiple honeybee sting marks were observed on the face, forehead, chest, and thorax. Icterus was noted, but there was no evidence of pallor, clubbing, cyanosis, lymphadenopathy, or pedal edema. Electrocardiography

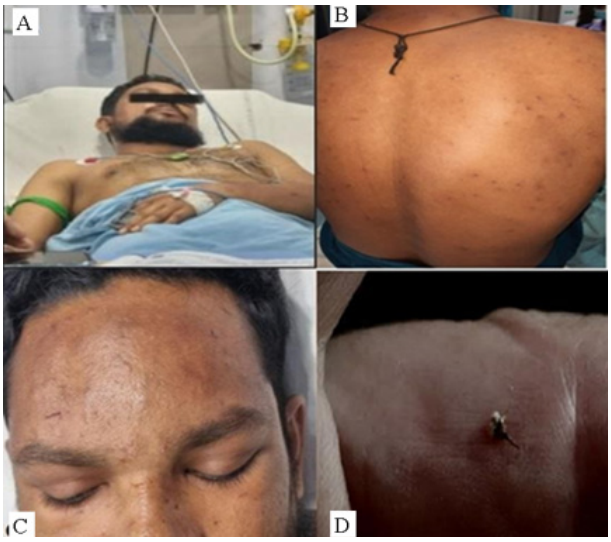

Figure 1 Patient characteristics of Case 1: (A) Patient arrival at the Emergency Department, (B) Multiple stings on the back, (C) Multiple stings on the forehead and face, (D) Honeybee stinger

(ECG) demonstrated a normal sinus rhythm with a heart rate of 80 beats per minute. Arterial blood gas (ABG) analysis on room air revealed metabolic acidosis. Imaging studies, including chest X-ray, abdominal and pelvic ultrasound, and 2D echocardiogram, were unremarkable. Routine blood investigations showed leukocytosis with neutrophilic predominance. Elevated serum lactate dehydrogenase (LDH) and creatine kinase-MB (CK-MB) levels suggested rhabdomyolysis. Urinalysis revealed dark-colored urine with marked albuminuria (3+ or > 3 g/24 h). Serum urea and creatinine levels were raised, consistent with AKI. Liver function tests show elevated total and indirect bilirubin with increased transaminases, indicating acute liver injury with transaminitis (Table 1).

Table 1 Laboratory diagnosis of Case 1

S No	Test	Result
1	Serum total bilirubin (mg/dL)	3.39 mg/dL
2	Serum bilirubin direct (mg/dL)	0.67
3	Total protein (g/dL)	5.76
4	Serum albumin (g/dL)	3.43
5	Aspartate aminotransferase (U/L)	434.39
6	Alanine transferase (U/L)	82.7
7	Alkaline phosphatase (ALP)	106.13
8	albumin to globulin (A/G) ratio	1.4
9	C-reactive protein (mg/L)	57
10	LDH (U/L)	925
11	Urine albumin (mg/g)	+++
12	Urine red blood cell (cells per microliter)	6-8/HPF
13	CK-MB (U/L)	283 IU/L

The patient was admitted to the Intensive Care Unit with a provisional diagnosis of anaphylaxis, presenting with features of multiorgan dysfunction. This included AKI secondary to rhabdomyolysis, along with acute liver injury evidenced by elevated transaminase levels.

Course of treatment during hospital stay

Patients were treated with IV fluids, Antibiotics, Antianalgesics, Antihistaminics, and Antiemetics. On admission, the patient remained hemodynamically stable. By the second day, however, the condition progressed to septic shock, requiring noradrenaline infusion for inotropic support to maintain a blood pressure of 110/80 mmHg. On the third day, laboratory investigations revealed worsening renal function, with serum creatinine rising to 7.12 mg/dL and serum urea rising to 111.29 mg/dL, along with a decline in urine output to 20 mL/min (Table 2).

Ultrasound findings demonstrated bilateral grade 1 renal parenchymal changes and bilateral pleural effusions. The patient subsequently underwent three sessions of hemodialysis. By the fifth day, hemodynamic stability was restored, with blood pressure maintained at 130/70 mmHg without inotropic support. Urine output had normalized, and ABG parameters were satisfactory. The patient was transferred out of the ICU. Over the next two days, continued clinical improvement was observed, and the patient was discharged with instructions for outpatient follow-up in 1 week.

Case Report 2

Cerebrovascular strokes secondary to multiple honeybee stings are exceedingly rare. We report the case of a 48-year-old male smoker who presented to the Emergency Department four days after sustaining numerous honeybee stings. Six hours post-exposure, he developed weakness in the right upper and lower limbs, accompanied by slurred speech and multiple episodes of vomiting. Notably, the patient had no prior history of cerebrovascular or cardiovascular disease (Figure 2).

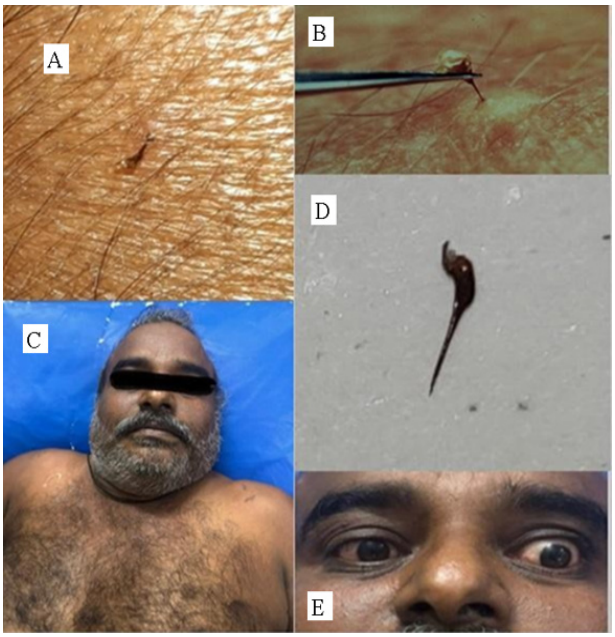

Figure 2 Multiple bee stings on Case 2: (A) Beesting site, (B) Beesting removal, (C) Conscious patient came with right-sided weakness and left gaze preference, (D) Beestinger intact, and (E) Eye characteristics

Primary Survey

On arrival at the Emergency Department, the patient was conscious and alert. The airway was clear, and oxygen saturation was 100% on room air. No tachypnea or tachycardia was observed, and blood pressure was within normal limits.

Table 2 Laboratory parameters at treatment intervals

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Total leukocyte count (cells per microliter)	17000	13600	10900	7400	9200	11400	12200	13700
Serum creatinine (mg/dL)	5.01	4.56	7.12	4.96	5.73	3.58	3.8	3.19
Serum urea (mg/dL)	101.8	73.06	111.2	63.94	79.37	45.77	43.63	49.0
Serum bilirubin (mg/dL)	3.39	-	0.38	-	-	0.26	0.26	-
Direct bilirubin (mg/dL)	0.67	-	0.21	-	-	0.45	0.45	-
Aspartate aminotransferase (U/L)	434.3	-	101	-	-	28.97	-	-
Alanine transferase (U/L)	82.7	-	63	-	-	38.36	-	-
LDH (U/L)	925	-	-	-	-		-	-
CK-MB (U/L)	283	-	-	-	-		-	-
CUE	ALB3+ RBC 6-8 hpf	-	-	-	-		-	-

Secondary Survey

The patient was conscious, coherent, and responsive to commands, oriented to time, place, and person. Multiple honeybee sting marks were observed on the face, forehead, and lower limbs. There was no evidence of pallor, jaundice, clubbing, cyanosis, lymphadenopathy, or pedal edema. A detailed examination was performed to identify residual stingers, and all visible stingers were successfully removed. In addition, all necessary diagnostic investigations and neuroimaging studies were undertaken.

ECG findings were consistent with an evolved inferior wall myocardial infarction. Magnetic resonance imaging of the brain demonstrated multiple acute infarcts, likely embolic in origin, involving the bilateral fronto-parieto-temporo-occipital lobes, the body and splenium of the corpus callosum, and the parafalcine region of the left frontal lobe. SWAN imaging revealed blooming in the parafalcine area of the left frontal lobe, suggestive of possible hemorrhagic transformation (Figure 3).

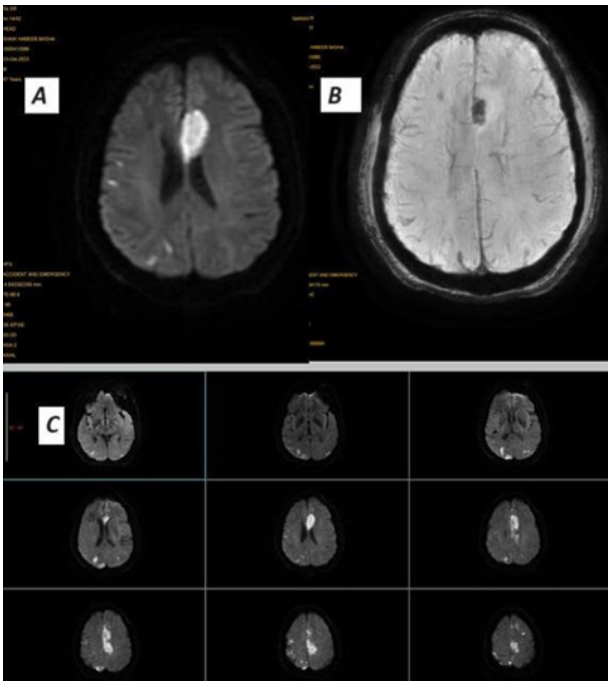

Figure 3 Magnetic resonance imaging. (A) and (C) Multiple acute infarcts, probably embolic origin, involving bilateral fronto-parieto-temporo-occipital lobes, (B) SWAN image showing blooming in the left frontal lobe, indicating haemorrhagic transformation

A 2D echocardiogram showed a hypokinetic inferior regional wall motion abnormality with mild left ventricular dysfunction (ejection fraction 48%), but no thrombus was detected in the left atrium or ventricle. Cardiac Troponin I was negative (< 0.10). Routine laboratory investigations, including complete blood count, serum electrolytes, renal and liver function tests, and coagulation profile, were within normal limits. Carotid Doppler imaging revealed a nearly occlusive plaque in the right common carotid artery.

The patient received intravenous fluids, antiemetics, antiplatelet agents, antihistamines, intravenous steroids, antiepileptics, and analgesics. Clinical improvement was noted, and the patient was discharged with advice to follow up in 10 days. Bee stings are known to cause transient electrocardiographic changes, including inferior wall myocardial infarction and inferior regional wall motion abnormalities, mediated through the local vasoactive, cardiotoxic, or anaphylactic properties of the venom. In some instances, bee venom may induce arrhythmias or destabilize atherosclerotic plaques, potentially resulting in embolic strokes. Case Report 2 highlights an instance of bilateral cardioembolic infarcts occurring six hours after multiple honeybee stings.

Case Report 3

Not all cases of multiple bee stings result in organ dysfunction; in some instances, the manifestations are limited to dermatological reactions such as flushing, itching, and pain. We present the case of a 37-year-old male smoker who arrived at the Emergency Department eight hours after sustaining multiple honeybee stings, with the primary complaint of severe generalized pain.

Primary Survey

On arrival at the Emergency Department, the patient was conscious but restless. The airway was clear, and oxygen saturation was 100% on room air. Tachycardia was observed, which resolved following administration of analgesics. Blood pressure remained within normal limits.

Secondary Survey

The patient was conscious, coherent, and responsive to commands, oriented to time, place, and person. Multiple honeybee sting marks were observed on the face, forehead, and lower limbs. There was no evidence of pallor, jaundice, clubbing, cyanosis, lymphadenopathy, or pedal edema. A detailed examination was conducted to identify retained stingers, and all visible stingers were successfully removed. Despite stinger removal, the patient continued to experience severe pain lasting 12–24 hours. Pain was managed with appropriate analgesics, ranging from non-steroidal anti-inflammatory drugs (NSAIDs) to opioids such as fentanyl and buprenorphine. Blood investigations, electrocardiogram (ECG), echocardiography, and chest X-ray were all within normal limits. Over the next two days, the patient showed significant clinical improvement, with reduced pain, and was subsequently discharged with advice for outpatient follow-up.

Ethics statement

Because the study was retrospective, informed consent was not required. Patient data were collected from the medical records of the Emergency Medicine department. The Institutional Ethics Committee provided an Ethical waiver for this study.

2 Discussion

A bee sting incident involving more than fifty stings is classified as massive envenomation. Local reactions commonly include redness, swelling, and pain, while IgE-mediated allergic responses may result in urticaria, angioedema, respiratory distress, AKI, or life-threatening anaphylactic shock. Bee venom is composed of various proteins, such as phospholipase A2 and hyaluronidase, as well as peptides including melittin and apamin, which constitute its principal toxic components. Additional bioactive substances present include peptide 401, histamine, dopamine, and norepinephrine.[4,5] Among these, melittin is particularly noted for its lethal potential. Furthermore, bee venom can trigger the release of vasoactive mediators such as histamine, bradykinin, serotonin, and prostaglandins, leading to complications including hypotension, AKI, cerebrovascular injury, and cardiovascular dysfunction. The renal effects may arise from direct tubular toxicity or from indirect mechanisms such as hemolysis, rhabdomyolysis, or shock.[6]

In this report, we present three patients with no prior history of allergies who developed severe systemic reactions following multiple bee stings, ultimately progressing to multiorgan dysfunction. Each bee sting is estimated to release approximately 50–140 μg of venom, a dose sufficient to cause systemic toxicity when stings are numerous. The clinical spectrum of honeybee stings ranges from localized cutaneous manifestations to severe allergic and systemic complications. In our cases, significant complications such as AKI were observed. Notably, only a limited number of such cases have been documented in the literature.

Multiple honey bee stings can lead to AKI through two major pathogenic pathways: ischemic acute tubular necrosis (ATN) and toxic ATN. Ischemic ATN results from the release of vasoactive substances, causing systemic vasodilatation and subsequent renal ischemia. Toxic ATN occurs due to the direct nephrotoxic effects of bee venom, as well as secondary complications such as rhabdomyolysis and hemolysis. These mechanisms collectively contribute to the development of acute kidney injury (Figure 4).

The condition described represents a combination of toxic and ischemic ATN. Toxic ATN is attributed to the direct nephrotoxic effects of bee venom, while ischemic ATN occurs in the setting of hypovolemia, renal vasoconstriction, hypotension, or shock. Nitric oxide, though primarily known as an endothelial relaxing factor, also contributes to renal vasoconstriction under certain pathological conditions (Figure 4).

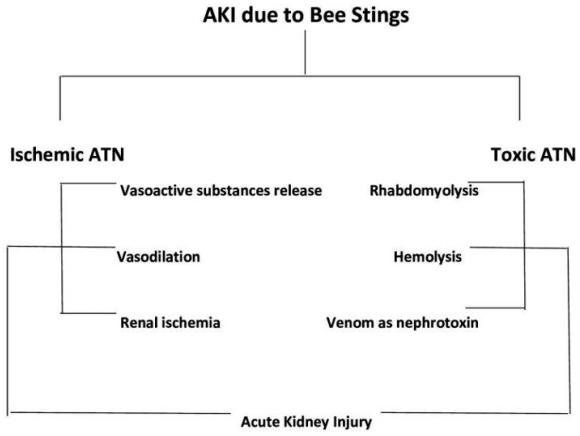

Figure 4 Mechanism of AKI due to multiple honey bee stings
ATN: Acute tubular necrosis

The occurrence of acute coronary syndrome in the context of allergic hypersensitivity or anaphylaxis is termed Kounis syndrome (KS), a medical emergency characterized by mast cell degranulation and the release of inflammatory mediators that impair coronary circulation. Three types of KS have been identified: Type I (vasospastic allergic angina), Type II (allergic myocardial infarction), and Type III (stent thrombosis) mediated by thrombus formation from eosinophils and/or mast cells. Bee venom (apitoxin) contains both vasoconstrictors, such as adrenaline and noradrenaline, and vasodilators, such as histamine. Histamine modulates inflammatory responses, activates platelets, and may also induce coronary vasoconstriction.[7]

In our cases, patients demonstrated elevated creatinine, urea, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), amylase, and other laboratory parameters. AKI following bee stings is multifactorial, arising from intravascular hemolysis, rhabdomyolysis, hypotension, and the direct toxic effects of melittin and apamin on renal tubules. The degree of liver injury appears to correlate with the total venom load, with hepatotoxicity resulting from direct cytotoxic effects.

Experimental studies support these clinical observations. Intravenous administration of bee venom in mice was associated with elevated cardiac noradrenaline levels and acute myocardial infarction.[8] Transient reductions in heart rate and mean arterial pressure were observed, alongside ECG changes resembling the Bezold–Jarisch reflex. Melittin, the principal pain-inducing component of bee venom, has been strongly linked to cardiotoxicity and hypersensitivity reactions. It promotes catecholamine

release, while venom metalloproteinases contribute to hypersensitivity responses and are implicated in coronary vasospasm and plaque destabilization. Apamin, another polypeptide, inhibits calcium-dependent potassium channels and interferes with slow calcium channel activity, thereby disrupting cardiac muscle action potentials. Studies on frog cardiac tissue demonstrated that bee venom can induce marked bradycardia, prolong the PR interval, and increase R-wave amplitude. Clinically, bradycardia was reported in fewer than 0.7% of 152 patients with honeybee stings.

Overall, bee stings typically cause transient electrocardiographic changes, mediated through vasoactive, cardiotoxic, or anaphylactic mechanisms. In some instances, bee venom may precipitate arrhythmias or destabilize atherosclerotic plaques, leading to embolic strokes. 12Stroke following a bee sting is an uncommon complication, and its exact pathophysiological mechanism remains uncertain. Several hypotheses have been proposed to explain this association:

- Anaphylaxis-related hypotension may cause ischemia, including that of the cerebral optic nerve.
- Vasoconstriction triggered by mediators released during envenomation, in combination with the effects of exogenous epinephrine and platelet aggregation, can contribute to cerebral ischemia.
- Bee venom components such as histamine, thromboxane, leukotrienes, and other vasoactive and inflammatory mediators may promote a hypercoagulable state, predisposing to cerebral ischemia. The vasoconstrictive actions of thromboxane and leukotrienes, in particular, have been implicated in the development of cerebral infarction.

Previous studies of three case reports have shown stroke following multiple bee stings. These cases highlight the potential severity of bee stings, which are often perceived as minor but can lead to serious medical complications (Table 3). They also underscore the challenges involved in managing such presentations.

3 Conclusion

This report aims to raise awareness about the risks associated with bee stings, emphasize the importance of timely medical intervention, and contribute to the limited body of literature available from data-scarce regions. When an individual sustains more than 20 stings, the cumulative venom load can be sufficient to cause multiorgan dysfunction. Such patients require appropriate counseling and should receive comprehensive intensive care management.

Declarations

Acknowledgments
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Artificial Intelligence Disclosure
Authors confirm that no artificial intelligence tools were used to prepare this article.

Authors' Contributions
Munawar Ahmed was responsible for data collection and writing the paper. Gopala Krishna supervised this research work and handled the proof-reading. Bharath Kumar conducted data analysis and assisted with proof-reading. Ravi Sankar contributed to proof-reading and was responsible for data collection and writing the paper.

Availability of Data and Materials
The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflict of Interest
The authors declared no conflict of interest.

Consent for Publication
Not applicable.

Ethical Considerations
This report describes the routine clinical management of three patients. No experimental procedures or additional diagnostic interventions were performed. According to the Institutional Ethics Committee's policy, ethical approval was not required for this case report. However, written informed consent for publication of clinical information was obtained from all patients.

Table 3 Literature studies of case reports showing stroke after multiple bee stings

Author	Age/sex	Onset of deficit	Clinical features	MRI findings	Outcome
Rajendiran C et al. [10]	25/M	> 1 day	Left upper limb monoparesis and blurring of vision	Infarct in the right frontoparietal, right occipital region	Full recovery
Bhat R et al. [11]	35/M	< 1 day	Bilateral cerebellar signs and AKI	Bilateral cerebellar infarcts	Deceased
Vidhate MR et al. [12]	8/M	< 1 day	Left hemiplegia followed by right hemiplegia and bilateral ophthalmoplegia	Non-hemorrhagic infarcts in the left frontoparietal and bilateral subcortical lesion. MRI-ICA parietal occlusion	Full recovery

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