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Bee Sting–Induced Ischemic Stroke with Divergent Outcomes: A Case Report and Review of 39 Cases

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Case Report

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Abstract

Background.

Bee sting–induced ischemic stroke is a rare but increasingly recognized complication of Hymenoptera envenomation, with fewer than 40 imaging-confirmed cases documented in the world literature. The pathophysiology involves concurrent mechanisms including cerebral vasospasm, immune-mediated thrombosis, disseminated intravascular coagulation, and direct neurotoxicity, and no standardized management protocol has been established.

Case presentation.

We report two Asian Indian male patients of South Asian ethnicity with honeybee sting–induced ischemic stroke and markedly divergent clinical trajectories. Case 1, a 70-year-old coal-mine worker, sustained approximately 200 stings and developed multifocal embolic infarcts across anterior and posterior circulations, complicated by acute kidney injury, recurrent tracheostomy occlusion requiring multiple bronchoscopy sessions, nosocomial carbapenem-resistant superinfection, and progressive multi-organ failure; he was discharged against medical advice in a comatose, ventilator-dependent state. Case 2, a 47-year-old previously healthy man, sustained over 100 stings and developed a delayed posterior circulation syndrome on Day 2, with diffusion-weighted imaging confirming embolic infarcts in the bilateral cerebellar hemispheres, vermis, right occipital lobe, right thalamus, and splenium of the corpus callosum; he recovered fully with anticoagulation and supportive care.

Conclusions.

A review of 39 cases demonstrates stroke onset ranging from minutes to 14 days after envenomation, involvement of all vascular territories, and outcomes spanning full neurological recovery to death. Mass envenomation burden, disseminated intravascular coagulation, and multi-organ dysfunction consistently portend the worst prognoses. Early neurological assessment, prompt MRI neuroimaging, and concurrent anti-allergic and stroke-specific management are essential in all patients sustaining multiple bee stings.

Background

Bee and wasp stings are among the most frequently encountered envenomations globally, with an estimated 100 million stings occurring annually, predominantly affecting individuals engaged in outdoor occupational and recreational activities in tropical and agricultural regions [1]. The vast majority of stings result in transient local reactions – pain, erythema, and edema – that resolve without medical intervention. However, a subset of patients develops systemic responses of varying severity, ranging from anaphylaxis and acute renal failure to rhabdomyolysis, hemolysis, and, in rare circumstances, life-threatening cerebrovascular events [2, 3].

Neurological complications of Hymenoptera envenomation, particularly ischemic stroke, represent one of the rarest yet most clinically challenging manifestations encountered in emergency and intensive care medicine [4]. Since the index fatal case reported by Day in 1962 – cerebral infarction following wasp stings – fewer than 40 cases of imaging-confirmed stroke attributable to bee or wasp venom have been published in the world literature [5, 6]. The 2022 structured literature review by Yang et al. catalogued 25 such cases across six decades, while the 2025 PRISMA-guided systematic review by Vasconez-Gonzalez et al. screened 83 publications and identified 29 cases from 28 eligible studies [1, 6]. Additional cases have since been reported through 2026, bringing the global total to approximately 35 documented cases. Despite growing recognition, this complication remains substantially underdiagnosed, particularly in low- and middle-income countries where both the burden of envenomation and the limitations of neuroimaging access are greatest [1].

The proposed pathophysiological mechanisms are multiple and often concurrent. Venom components including phospholipase A2, melittin, histamine, thromboxane A2, and leukotrienes have been implicated in platelet aggregation, endothelial injury, and cerebral vasospasm [4, 7]. Anaphylaxis-induced systemic hypotension may cause watershed cerebral hypoperfusion, while retrograde stimulation of the superior cervical ganglion by head and neck stings may produce ipsilateral carotid vasospasm [8, 9]. Immune-mediated mechanisms including mast cell degranulation, IgE-mediated hypersensitivity, and disseminated intravascular coagulation may operate concurrently, particularly in mass envenomation scenarios [1, 10].

Several isolated case reports have documented ischemic stroke involving the middle cerebral artery territory, the posterior fossa, the bilateral thalami, and the cerebral venous sinuses following Hymenoptera envenomation [6, 11–14]. However, two-case comparative reports documenting divergent clinical trajectories – particularly from the Indian subcontinent – remain scarce. In this report, we present two cases of ischemic stroke following multiple bee stings, each with markedly different presentations, degrees of systemic involvement, and outcomes. A comparative summary of both cases is provided in Table 1, and a comparison with published cases is presented in Table 2. Our aim is to contribute to the growing international literature on this rare but serious entity and to increase awareness among clinicians regarding the neurological sequelae of mass bee envenomation.

Table 1
Comparative summary of both presented cases

Parameter	Case 1	Case 2
Age / Sex	70 years / Male	47 years / Male
Ethnicity	Asian Indian (South Asian)	Asian Indian (South Asian)
Occupation	Coal mine worker	Not specified
Comorbidities	PIVD (limited mobility)	None
Insect / Species	Honeybee swarm	Honeybee swarm
Number of Stings	~ 200	> 100
Sting Locations	Face, scalp, eyes, ears, chest, back, upper limbs	Scalp, ears, neck
Presenting Symptoms	Diffuse swelling, dark urine	Scalp pain, vomiting, localized swelling
GCS at Admission	14/15	Normal
Onset of Neurological Signs	Acute – within hours of admission	Delayed – Day 2
Key Lab Findings	TLC 18,100/mm³; ALT 225; AST 54; Bilirubin 4.5 mg/dL; PT 17.8 s; INR 1.2; haemoglobinuria	No significant derangement
MRI Brain Findings	Bilateral frontal subcortical WM, right temporal, left parietal, bilateral cerebellum – embolic infarcts	Bilateral cerebellar hemispheres + vermis, right occipital, right thalamus, splenium of corpus callosum
Vascular Territory	Multifocal – anterior + posterior	Posterior circulation predominant
Cardiac Evaluation	Normal 2D echo + Holter	Normal 2D echo
Renal Involvement	AKI – haemodialysis required	None
Airway / Ventilation	Mechanical ventilation + tracheostomy	Not required
Bronchoscopy	Multiple sessions – recurrent clot	Not required
Ocular Involvement	> 15 stingers removed from eyes; live bee in ear	None
Treatment	Supportive, ICU, haemodialysis, multiple bronchoscopy, anti-allergic	Antihistamines, corticosteroids, anticoagulation
Complications	Carbapenem-resistant Enterobacteriaceae, CPR, multi-organ dysfunction	None significant
Proposed Mechanism	DIC + vasospasm + systemic inflammatory cascade	Vasospasm / thromboembolism – posterior circulation
Outcome	DAMA; GCS E1V1M1; ventilator-dependent; comatose	Discharged stable; resolving neurological deficits

PIVD prolapsed intervertebral disc, GCS Glasgow Coma Scale, TLC total leucocyte count, ALT alanine aminotransferase, AST aspartate aminotransferase, PT prothrombin time, INR international normalized ratio, WM white matter, AKI acute kidney injury, ICU intensive care unit, CPR cardiopulmonary resuscitation, DIC disseminated intravascular coagulation, DAMA discharged against medical advice.

Table 2
Comparison of current cases with previously published cases

No.	Author / Year	Country	Age/Sex	Insect	No. of stings / location	Onset from sting	Imaging findings	Proposed mechanism	Outco
1	Day, 1962 [5]	USA	36/M	Wasp	Multiple; neck, face, arms	15 min	Necropsy: left hemorrhagic cortical + pontine infarction	NR	Died
2	Starr & Brasher, 1977 [16]	USA	37/M	Wasp	NR	< 1 h	Left cerebral infarction	Anaphylaxis-induced	Partia hemip
3	Romano et al., 1989 [17]	USA	17 mo/M	Wasp	Single; inner lip	4 days	Left putamen + caudate infarction	ICA occlusion	Full re
4	Riggs et al., 1993 [8]	USA	38/M	Wasp	Multiple; face, neck	2 days	Left MCA territory infarction	Sympathetic innervation / vasospasm	NR
5	Riggs et al., 1994 [9]	USA	52/M	Wasp	Multiple (2nd sting)	Few hours	Left parietal + insular cortical infarctions	Immune sensitization + bilateral ICA vasospasm	NR
6	Crawley et al., 1999 [3]	UK	30/F	Wasp	Single; arm	45 min	Left occipital infarction	Anaphylaxis-induced hypoperfusion	Full re
7	Bhat et al., 2002 [18]	India	30/M	Bee	Multiple; all over body	< 1 day	Bilateral hemorrhagic cerebellar infarction	Mass envenomation DIC/vasospasm	Died
8	Sachdev et al., 2002 [19]	India	40/M	Wasp	Single; face	10 h	Right ventral pons + right cerebellar infarctions	Vasospasm	Impro
9	Chen et al., 2004 [20]	Taiwan	71/F	Wasp	Multiple; head, face, limbs	1 day	Right MCA territory infarction	Thromboembolism + DIC	Partia recov
10	Schiffman et al., 2004 [21]	USA	57/F	Bee	Multiple; neck, head, eye, face, arm	2 days	Large right temporo-occipital hemorrhagic infarction	PCA vasospasm/thrombosis	Impro
11	Taurin et al., 2006 [22]	France	36/M	Wasp	Single	14 days	Left dorsal medulla infarction	Vasospasm (delayed)	Full re
12	Temizoz et al., 2009 [23]	Turkey	60/M	Bee	Multiple; head, face, limbs	2 h	Bilateral frontal lobes, right temporoparietal, bilateral centrum semiovale	Multifocal vasospasm	Impro
13	Vidhate et al., 2011 [24]	India	8/M	Wasp	Multiple; eyebrow, nasal bridge	8 days	Left frontoparietal, right subcortical, posterior limb left internal capsule	Global hypoperfusion (anaphylaxis)	Impro
14	Dechyapirom et al., 2011 [25]	USA	64/M	Bee	Multiple; face, neck, chest, extremities	16 h	Large right MCA territory infarction	Vasospasm / concurrent Kounis syndrome	Recov
15	Viswanathan et al., 2012 [26]	India	59/M	Bee	Multiple; face, neck, scalp, chest	2.5 h	Right perisylvian, peri-insular, parietal cortices	Vasospasm / thromboembolism	Impro
16	Jain et al., 2012 [27]	India	70/M	Bee	Multiple	6 h	Left frontoparieto-occipital; bilateral gangliocapsular lacunar infarcts	DIC	Impro
17	Bilir et al., 2013 [28]	Turkey	35/M	Bee	Multiple	6 h	Left MCA infarction	Vasospasm	Resid hemip
18	Wani et al., 2014 [29]	India	40/M	Wasp	Multiple; face, head, neck	1 day	Left thalamic, left parietooccipital, bilateral cerebellar,	DIC + vasospasm	Veget state

NR not reported, MCA middle cerebral artery, ICA internal carotid artery, PCA posterior cerebral artery, AOP artery of Percheron, MRA MR angiography, DSA digita subtraction angiography, DWI diffusion-weighted imaging, SAH subarachnoid haemorrhage, DIC disseminated intravascular coagulation, DAMA discharged again medical advice, WM white matter, mRS modified Rankin Scale. Cases [17], [18], [21], [27], [29], [33], [34], [38] *were not independently indexed in the literature data and are cited through the secondary sources indicated in the reference list.*

No.	Author / Year	Country	Age/Sex	Insect	No. of stings / location	Onset from sting	Imaging findings	Proposed mechanism	Outco
							pontine infarctions		
19	An et al., 2014 [30]	South Korea	50/M	Bee	Multiple	27 h	Right temporal infarction	MCA branch thrombosis	Recov
20	Alvis-Miranda et al., 2014 [31]	Colombia	74/M	Bee (African)	Multiple	NR	Left frontal hypodensity	Thromboembolism	NR
21	Guzel et al., 2016 [32]	Turkey	59/M	Bee	Multiple	Few hours	Massive right frontotemporal-parietal infarction	Vasospasm / thrombosis	Died
22	Kulhari et al., 2016 [4]	USA	44/M	Wasp	Single; leg	1 h	Multiple right MCA territory infarctions	Bilateral MCA vasospasm (DSA-confirmed)	Recov
23	Bong et al., 2017 [33]	South Korea	52/M	Wasp	Multiple	NR	Left basal ganglia, cerebral cortex	Hypoxic-ischemic	Partia impro
24	Tao et al., 2017 [34]	China	NR	Wasp	NR	NR	Posterior fossa infarction	Vasospasm	NR
25	Mirijello et al., 2017 [35]	Italy	NR	Wasp	NR	NR	MRI – ischemic stroke	Kounis syndrome	NR
26	Dalugama & Gawarammana, 2018 [11]	Sri Lanka	69/M	Wasp	3 wasps	NR	Left posterior frontal white matter infarction	Vasoactive/thrombogenic venom	Impro
27	Ramlackhansingh & Seecheran, 2020 [36]	Trinidad	70/M	Bee (Africanised)	Multiple; face, forearms, back	1 day	Left parietal + left basal ganglia infarctions	Thromboembolism	Full re
28	Elavarasi et al., 2020 [37]	India	41/M	Bee	Multiple	5 h	Massive right MCA territory infarction	DIC / thrombosis	Died
29	Gupta, 2020 [38]	India	41/F	Bee (honey)	Single; arm	3 h	Bilateral thalamic + left frontotemporal infarctions with hemorrhagic transformation	Vasospasm / thromboembolism	Died
30	Karri et al., 2021 [39]	India	40/M	Wasp	Multiple	NR	Massive anterior + middle cerebral artery infarction; right ICA thrombosis	Thrombosis – ICA	Died
31	Masaraddi et al., 2021 [10]	India	45/M	Bee	15–20; head, neck, face	6 h	Left MCA territory hyperacute infarcts	ICA stenosis (thrombotic/vasospastic)	Right hemip at 1 m
32	Abhishek et al., 2021 [40]	India	NR	Bee	NR	< 4.5 h	MRI – ischemic stroke	NR	Favor (post- throm
33	Yang et al., 2022 [6]	China	62/M	Honeybee	Multiple; face, ear	2 h	Left medulla, left parieto-occipital + thalamus; progressive	Vasospasm (PCA resolved spontaneously)	Persis defici year
34	Nittner-Marszalska et al., 2022 [41]	Poland	44/M; 24/M	Wasp	Case 1: single; Case 2: few stings	Immediate; 12 h	Case 1: bilateral caudate/lenticular nuclei; Case 2: right parietal + watershed	Case 1: global hypoperfusion; Case 2: watershed infarction	Mild d at 9 y 1); mi hemip at 2 y 2)
35	Min et al., 2022 [42]	China	NR/M	Wasp	Multiple; head + right shoulder	NR	Acute progressive large-area cerebral infarction	Vasospasm – superior cervical ganglion stimulation	Resid defici
36	Kabra et al., 2022 [12]	India	49/M	Honeybee	Multiple; hand +	5–6 h	Left corona radiata – DWI	Vasospasm / prothrombotic state	Full re at 6 m

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No.	Author / Year	Country	Age/Sex	Insect	No. of stings / location	Onset from sting	Imaging findings	Proposed mechanism	Outco
					arms		hyperintensity; MRA normal		
37	Zhang et al., 2024 [13]	China	59/M	Hornet (wasp)	Multiple; head, face, neck	13 h	Left frontal, parietal, thalamus – venous infarction + hemorrhage + SAH; superior sagittal sinus thrombosis	Venous sinus thrombosis – DIC	Perm renal impai maint haem
38	Priyadarshi et al., 2024 [14]	India	~ 40/M	Wasp	Single; left face	2 h	DWI: bilateral paramedian thalami + rostral midbrain (artery of Percheron territory)	AOP occlusion – vasogenic + thrombogenic	Full re (mRS
39	Peng et al., 2026 [15]	China	56/F	Wasp	Multiple	8 days	Right insular, external capsule, basal ganglia; MRA normal	DIC + systemic inflammatory cascade	Left l limb + at 6
40	Current Case 1	India	70/M	Honeybee	~ 200; face, scalp, eyes, ears, chest, back, upper limbs	Hours	Bilateral frontal subcortical WM, right temporal, left parietal, bilateral cerebellum	DIC + vasospasm + systemic inflammatory cascade	DAMA E1V1 ventil depe
41	Current Case 2	India	47/M	Honeybee	> 100; scalp, ears, neck	Day 2	DWI: bilateral cerebellar + vermis, right occipital, right thalamus, splenium of corpus callosum	Vasospasm / thromboembolism – posterior circulation	Disch stable resol defici

NR not reported, MCA middle cerebral artery, ICA internal carotid artery, PCA posterior cerebral artery, AOP artery of Percheron, MRA MR angiography, DSA digita subtraction angiography, DWI diffusion-weighted imaging, SAH subarachnoid haemorrhage, DIC disseminated intravascular coagulation, DAMA discharged again medical advice, WM white matter, mRS modified Rankin Scale. Cases [17], [18], [21], [27], [29], [33], [34], [38] *were not independently indexed in the literature data and are cited through the secondary sources indicated in the reference list.*

Case presentation

Case 1

A 70-year-old Asian Indian male coal-mine worker of South Asian ethnicity, with a background history of prolapsed intervertebral disc (PIVD) – his only prior medical condition – presented to the emergency department following a honeybee swarm attack during his morning walk. Unable to flee due to limited mobility secondary to PIVD, he sustained stings across the face, scalp, eyes, ears, chest, back, and both upper limbs over a period of approximately one hour. First aid was administered at a local hospital before referral to our tertiary care centre. Physical examination on arrival identified approximately 200 embedded stingers distributed across the scalp, ears, periorbital regions, and conjunctival surfaces; these were systematically removed over the initial assessment period (Fig. 1A, B). Notably, dark-coloured urine was documented on presentation, consistent with haemolysis and haemoglobinuria.

At admission, the patient was alert with a Glasgow Coma Scale (GCS) score of 14/15 and haemodynamically stable with a blood pressure of 150/90 mmHg. Laboratory investigations revealed leucocytosis (total leucocyte count 18,100/mm³), elevated liver enzymes (alanine aminotransferase 225 U/L, aspartate aminotransferase 54 U/L), raised total bilirubin (4.5 mg/dL), and mild coagulopathy (prothrombin time 17.8 s, INR 1.2). Urinalysis confirmed haemoglobinuria. Non-contrast computed tomography (CT) of the brain demonstrated multifocal acute infarcts in the bilateral frontal subcortical white matter, right temporal lobe, left parietal lobe, and bilateral cerebellar hemispheres – a distribution spanning multiple vascular territories consistent with multifocal embolic or vasospastic ischaemia. Cardiac evaluation including transthoracic echocardiography and 24-hour Holter monitoring returned entirely normal results, effectively excluding a cardioembolic aetiology.

Despite initial clinical stability, the patient's neurological status deteriorated progressively over the subsequent hours, with GCS declining to E1V2M5, necessitating ICU transfer. Endotracheal intubation and mechanical ventilation were initiated, followed by tracheostomy in view of anticipated prolonged ventilatory dependency. Haemodialysis was commenced due to worsening acute kidney injury with oliguria. On clinical examination, massive abdominal distension with diffuse purplish-ecchymotic discolouration of the anterior abdominal wall was identified, reflecting severe systemic venom-induced microvascular injury and fluid sequestration commensurate with multi-organ dysfunction (Fig. 1C). Ophthalmological assessment identified over 15 stingers embedded in bilateral conjunctival surfaces and eyelid margins requiring specialist extraction under slit-lamp guidance (Fig. 1B). ENT assessment revealed a live bee within the external ear canal, which was removed under direct visualization.

Further complications included recurrent clot formation within the tracheostomy tube, necessitating multiple bronchoscopy sessions for airway clearance (Fig. 1D). These procedures were complicated by episodes of acute hypoxia and bradycardia during clot extraction, requiring cardiopulmonary resuscitation on one occasion. Blood and tracheal aspirate cultures subsequently grew carbapenem-resistant Enterobacteriaceae, indicating nosocomial superinfection. Despite periods of transient clinical improvement, the patient's overall course was one of progressive multi-organ failure. Given the deteriorating trajectory and at family insistence, the patient was discharged against medical advice in a ventilator-dependent state with GCS E1V1M1.

Case 2

A 47-year-old Asian Indian male of South Asian ethnicity with no prior medical history or comorbidities presented to a local facility following a honeybee swarm attack, sustaining over 100 stings to the scalp, ears, and neck region (Fig. 3A, B). He reported scalp pain, persistent vomiting, and progressive swelling of the stung areas. Stingers were removed at the local facility and he received initial management with analgesics, antihistamines, and corticosteroids before transfer to our tertiary centre (Fig. 3C, D).

Initial haemodynamic parameters and basic laboratory investigations were within acceptable limits, with no significant derangement of renal or hepatic function. He was managed conservatively. On Day 2 of admission, the patient developed new-onset persistent headache. Neurological examination revealed horizontal nystagmus on lateral gaze and impaired coordination on the left side. A positive finger-to-nose test on the left confirmed cerebellar involvement. No motor deficit or aphasia was identified.

Brain MRI with diffusion-weighted imaging (DWI) demonstrated multiple hyperintense foci with restricted diffusion consistent with acute embolic infarcts involving the bilateral cerebellar hemispheres including the vermis, right occipital lobe, right thalamus, and splenium of the corpus callosum – a posterior circulation and posterior fossa distribution (Fig. 2A, B). Transthoracic echocardiography excluded cardioembolic sources. Following neurology consultation, anticoagulation was initiated. The patient responded progressively to conservative and anticoagulant management, with resolution of nystagmus, normalization of the finger-nose test, and cessation of headache. He was discharged in stable condition with resolving neurological deficits and planned for outpatient follow-up. Table 1 summarizes the comparative features of both cases.

Discussion and conclusions

The two cases presented in this report illustrate the broad and clinically critical spectrum of cerebrovascular complications following multiple bee stings, encompassing both a devastating, ultimately fatal course with multi-organ dysfunction and a relatively benign, fully reversible posterior circulation syndrome. Together they reinforce the key themes consistently observed across the global literature: bee sting–induced stroke can occur in previously healthy individuals across a wide age range; the neurological injury may present acutely or be delayed by hours to days; multiple vascular territories may be simultaneously affected; and outcomes are determined by the interplay of sting burden, host immunological response, vascular territory involved, speed of neurological recognition, and quality of multidisciplinary intervention [1, 6].

Bee and wasp sting–induced cerebrovascular disease was first formally documented in 1962 when Day reported a fatal case of cerebral infarction in a 36-year-old male who developed hemiplegia and seizures within 15 minutes of wasp stings; necropsy revealed left hemorrhagic cortical and pontine infarction [5]. For nearly three decades this remained an isolated anecdote. By 2022, Yang et al. had catalogued 25 imaging-confirmed cases of cerebral infarction following bee or wasp stings [6], and the 2025 PRISMA systematic review by Vasconez-Gonzalez et al. identified 29 cases from 28 eligible studies [1]. Including cases published between 2023 and 2026, the global total now stands at approximately 35 documented cases spanning six decades and more than 15 countries. Peng et al.'s 2026 case – a 56-year-old woman who developed delayed hemispheric ischemia in the context of evolving multi-organ dysfunction – remains one of only two females in all published reports, underscoring the extreme male predominance in this entity [15]. Table 2 presents the detailed comparison of all published cases alongside our two cases.

An overwhelming male predominance characterizes the published literature – 28 of 29 cases (96.6%) in the Vasconez-Gonzalez et al. series occurred in males, attributed to greater occupational and recreational outdoor exposure in farming, mining, and forest-adjacent environments [1]. Patient ages range from 17 months to 75 years, with a mean of approximately 45.8 years across that same cohort [1]. The geographic burden is concentrated in South and Southeast Asia, with major series from India, China, Sri Lanka, Turkey, and South Korea, reflecting both the ecologic abundance of Hymenoptera species and the outdoor occupational exposure of agricultural populations in these regions [1, 6].

The time from sting to neurological symptom onset has been widely variable across the literature, ranging from less than one hour to 48 hours with a reported median of 16 hours in the systematic review [1], and up to 14 days in a delayed case documented by Yang et al. [6]. The earliest onset was in the index case by Day [5]. The case reported by Vidhate et al. showed stroke emergence 8 days after the initial sting with entirely normal cerebrovascular angiography, consistent with global hypoperfusion from anaphylaxis [24], while Kabra et al. documented stroke onset 5–6 hours after bee stings, also with normal MR angiography at the time of imaging [12]. Peng et al.'s 2026 case demonstrated left hemiparesis detected only on day 8 of admission in the context of evolving multi-organ dysfunction [15]. These observations confirm that stroke can occur at any point from minutes to days after envenomation, underscoring the need for prolonged vigilance and serial neurological assessment. Our Case 2 – with Day 2 cerebellar syndrome onset – exemplifies this delayed presentation pattern and illustrates how the absence of immediate neurological signs does not exclude subsequent cerebrovascular injury.

No vascular territory has proven exempt from sting-induced ischemia. The middle cerebral artery (MCA) territory is the most commonly involved, documented in cases by Masaraddi et al. [10], Elavarasi et al. [37], Guzel et al. [32], Bilir et al. [28], Dechyapirom et al. [25], Viswanathan et al. [26], Karri et al. [39], and Kulhari et al. [4]. The posterior circulation – comprising the vertebrobasilar system, posterior cerebral arteries, and cerebellar hemispheres – has been involved in cases by Sachdev et al. [19], Taurin et al. [22], Yang et al. [6], and Min et al. [42]. Bilateral thalamic infarction in the artery of Percheron

territory was documented by Priyadarshi et al., representing a rare anatomical variant – a single common arterial trunk supplying both paramedian thalami and the rostral midbrain – estimated to exist in only 4–11.7% of the general population [14]. Bilateral infarctions involving multiple territories simultaneously have been described in at least six reported cases [6], strongly implicating a systemic rather than focal vascular mechanism. The case by Zhang et al. further expanded the spectrum by documenting cerebral venous sinus thrombosis of the superior sagittal sinus leading to venous infarction and subarachnoid hemorrhage – the first clearly documented venous rather than arterial mechanism following Hymenoptera envenomation [13]. The posterior circulation and posterior fossa distribution seen in our Case 2 – cerebellar hemispheres, vermis, right occipital, right thalamus, and splenium of the corpus callosum – resembles the distribution described by Wani et al. [29] and is consistent with stings concentrated in the cervical region stimulating the vertebrobasilar territory [6].

Ischemic stroke accounts for the overwhelming majority of reported cases at 89.7%, with hemorrhagic stroke documented in approximately 10%, including hemorrhagic transformation of ischemic infarcts and combined venous infarction with subarachnoid hemorrhage as seen in Zhang et al. [1, 13]. The mechanism underlying hemorrhagic stroke likely involves catecholamine-mediated hypertensive hemorrhage, anticoagulant activity of hyaluronidase, hemolysis-mediated coagulopathy, and in venous disease the raised intracranial venous pressure causing cortical vein rupture [1, 13].

The mechanisms by which Hymenoptera venom precipitates cerebrovascular ischemia are multiple, poorly delineated in humans, and almost certainly concurrent in most severe cases. Vasospasm and vasoactive peptide–mediated vasoconstriction is supported by the most direct clinical evidence: Kulhari et al. provided definitive angiographic proof, demonstrating bilateral proximal MCA vasoconstriction on digital subtraction angiography that normalized following rt-PA [4], while Yang et al. documented reversible posterior cerebral artery near-occlusion that resolved completely on repeat MR angiography 25 days later, providing the strongest evidence to date for transient venom-induced vasospasm as a standalone mechanism [6]. Kabra et al. and Min et al. similarly demonstrated normal MR angiography despite acute diffusion-weighted imaging–confirmed infarction, consistent with vasospasm having resolved by the time of imaging [6, 12, 42]. Venom components including histamine, thromboxane A2, leukotrienes, serotonin, and bradykinin are all established vasoconstrictors capable of inducing reversible cerebral arterial spasm [4, 7]. Retrograde stimulation of the superior cervical sympathetic ganglion was proposed by Riggs et al., who documented bilateral internal carotid artery occlusion following head and neck wasp stings and postulated that venom deposited in the facial and cervical regions stimulates sympathetic innervation to the distal internal carotid artery via the superior cervical ganglion, producing intense ipsilateral carotid vasospasm [8, 9]. This mechanism is anatomically plausible and is supported by the clustering of MCA territory strokes in cases where stings predominantly involved the head, face, and neck. In both cases presented here, stings were concentrated in the head and neck region – making retrograde ganglion stimulation a plausible contributor.

Immune-mediated hypercoagulability and Kounis syndrome – allergic vasospasm extending the concept of anaphylaxis-induced coronary spasm to the intracranial circulation – represents an additional major pathway, where mast cell degranulation triggered by bee venom releases platelet-activating factor, tissue factor, and pro-inflammatory cytokines that initiate a thrombotic cascade [7, 10]. Phospholipase A2 directly injures vascular endothelium and activates the arachidonic acid pathway, generating thromboxane A2 and leukotrienes that further promote platelet aggregation [1, 10], and this mechanism is supported by the findings of Priyadarshi et al., who documented artery of Percheron occlusion in the context of anaphylaxis with tachycardia and normal downstream large vessel patency on MR angiography [14]. Global cerebral hypoperfusion from anaphylaxis-induced systemic hypotension was clearly documented in the two cases by Nittner-Marszalska et al., where the first patient suffered bilateral caudate and lenticular infarctions consistent with global hypoperfusion during cardiorespiratory arrest, and the second patient developed right hemispheric watershed infarcts following brief hypotension with CT angiography demonstrating only minimal right posterior cerebral artery narrowing [41]. The child described by Vidhate et al. – bilateral watershed infarctions with entirely normal cerebrovascular angiography – is the purest illustration of this mechanism among the documented cases [24]. Dalugama and Gawarammana noted that in their Sri Lankan patient, the single-territory posterior frontal infarction made anaphylaxis-induced global hypoperfusion an unlikely primary mechanism, pointing instead to vasoactive properties of Vespa tropica venom as causal [11]. Hemolysis-induced disseminated intravascular coagulation and systemic thrombosis dominates in mass envenomation: melittin and other membranolytic venom components lyse erythrocytes, releasing free hemoglobin, tissue thromboplastins, and erythrocyte membrane phospholipids that activate the extrinsic coagulation pathway and deplete fibrinogen [13]. Zhang et al. demonstrated antithrombin III depletion to 50% activity alongside extreme creatine kinase elevation, providing direct biochemical evidence that venom-induced proteases and phospholipases activate the coagulation cascade and precipitate both arterial and venous thrombotic events [13], while Peng et al. showed that plasma exchange combined with continuous renal replacement therapy may attenuate multi-organ dysfunction but does not prevent delayed cerebral infarction when the systemic inflammatory cascade is already established [15].

Across all full-text readable published cases, no evidence-based treatment protocol exists for sting-induced stroke, and management has been empirical and individualized. Prompt stinger removal is critical – bee stingers continue to inject venom after detachment for up to 60 seconds, and every stinger left in place perpetuates the toxin load [6]. Anti-allergic treatment comprising intravenous methylprednisolone, antihistamines, and calcium gluconate was initiated in all reported cases, with epinephrine administered for frank anaphylaxis as in the case documented by Priyadarshi et al. [14]. In mass envenomation with multi-organ failure, hemoperfusion, plasma exchange, and continuous renal replacement therapy have been employed by Zhang et al. and Peng et al. with partial success in organ recovery [13, 15]. Intravenous rt-PA has been administered in at least four reported cases: Dechyapirom et al. and Kulhari et al. both documented clinical recovery following rt-PA for MCA territory stroke [4, 6, 25], while Yang et al. administered rt-PA without clinical benefit, subsequently documenting progressive vertebrobasilar territory infarction – suggesting that multifocal vasospastic disease may not respond to thrombolysis in the same way as embolic stroke [6]. Abhishek et al. reported the first clearly documented favorable outcome with intravenous rt-PA administered within the 4.5-hour window for bee sting–induced stroke, establishing a precedent for applying standard stroke protocols in this atypical etiology [40]. Masaraddi et al. could not offer rt-PA as their patient presented at 14 hours post-onset, beyond the thrombolytic window – a recurring limitation given the delayed symptom presentation in many sting-induced stroke cases [10]. Anticoagulation was initiated in our Case 2 and in Nittner-Marszalska et al.'s cases, and was similarly employed in the cerebral venous sinus thrombosis case of Zhang et al. [13, 41]. Venom immunotherapy was subsequently prescribed to both patients in the Nittner-Marszalska et al. series – confirming IgE-mediated venom allergy through elevated specific IgE to wasp venom – providing 100% protection from systemic allergic reactions in subsequent stings and representing the only preventive intervention available for IgE-dependent recurrence [41].

Outcomes in the published literature span a wide spectrum. Four to six deaths have been documented [5, 6], including the index case by Day [5], bilateral hemorrhagic cerebellar infarction (Bhat et al. [18]), massive MCA infarction (Guzel et al. [32]), bilateral thalamic infarction with hemorrhagic transformation (Gupta [38]), post-hemicraniectomy death (Elavarasi et al. [37]), and fatal internal carotid artery thrombosis (Karri et al. [39]). Permanent disability has been documented in Yang et al.'s patient – requiring tracheostomy and gastrostomy at one year [6] – and in Nittner-Marszalska et al.'s first patient, who had persistent right hemiparesis and cognitive impairment 9 years after his single-sting stroke [41]. Conversely, full neurological recovery has been documented in multiple cases – particularly in younger patients with posterior circulation, single-territory, or watershed infarctions treated promptly [4, 11, 12, 14]. Kabra et al. documented full neurological recovery with return to normal activities at 6 months [12]; Priyadarshi et al. documented an mRS of 0 following artery of Percheron infarction despite being out of the thrombolytic window [14]; and Peng et al. reported left lower limb recovery to MRC grade 4 + at 6-month follow-up following delayed insular and basal ganglia infarction in the setting of multi-organ dysfunction [15]. Our Case 2 aligns with the favorable recovery group, while Case 1 represents one of the most severe reported outcomes – a clinical trajectory paralleled most closely by Wani et al. [29] and the Zhang et al. cerebral venous sinus thrombosis case that resulted in permanent dialysis dependence [13].

The first patient's diffuse infarcts across multiple vascular territories – combined with renal dysfunction, hepatic enzyme elevation, coagulopathy, hemoglobinuria, and diffuse systemic ecchymotic discolouration – point toward a systemic inflammatory cascade and toxin-mediated microvascular injury, as has been described in cases of large-vessel stroke and multi-organ involvement following mass envenomation [6, 13, 15]. The striking abdominal distension with ecchymotic discolouration (Fig. 1C) and the airway compromise necessitating repeated bronchoscopic intervention for recurrent tracheostomy tube clot (Fig. 1D) further underscore the severity of systemic envenomation in this patient. The emergence of carbapenem-resistant Enterobacteriaceae substantially reduced the treatment options available and ultimately contributed to his fatal trajectory [13]. In contrast, Case 2 experienced a more focal, cerebellar-predominant embolic pattern with complete recovery under anticoagulation, aligning with reports in which vasospasm was transient and reversible or in which posterior circulation thromboembolism responded to anticoagulant therapy [4, 11, 41]. The diffusion-weighted MRI (Fig. 2A, B) clearly demonstrated restricted diffusion in the posterior fossa, corroborating the clinical cerebellar signs. The distribution of infarcts across the cerebellar hemispheres including vermis, right occipital lobe, right thalamus, and splenium of the corpus callosum implicates the posterior circulation and is consistent with prior reports of posterior fossa predominance following cervical-region stings [6, 8, 9]. Differential diagnoses including cardioembolic stroke, infective endocarditis, thrombophilic states, and cerebral vasculitis were systematically excluded in both patients through normal echocardiography, normal Holter monitoring, and coagulation profiles that, while mildly deranged in Case 1, did not indicate primary thrombophilia – a pattern of exclusion consistently replicated across the full-text published literature [4, 6, 10–14, 41].

Several unique features distinguish our cases from the existing literature. The combination of over 200 stings, live bee removal from the external ear canal, more than 15 intraocular stingers requiring specialist ophthalmological extraction, documented ecchymotic abdominal wall discolouration, recurrent tracheostomy clot requiring repeated bronchoscopy, and nosocomial carbapenem-resistant superinfection in Case 1 represents a degree of total-body envenomation burden and secondary morbidity that has not been previously described with such procedural and photographic documentation. Case 2's involvement of the splenium of the corpus callosum – in addition to bilateral cerebellar, right occipital, and right thalamic infarcts – adds a unique vascular territory combination to the posterior circulation subset of sting-induced stroke cases. The limitations of this report include the absence of inflammatory marker profiling including serum phospholipase A2 activity, tryptase levels, and cytokine panels; the lack of formal cerebrovascular angiography in either patient; the absence of venom-specific IgE quantification; and the limited long-term neurological follow-up data due to discharge against medical advice in Case 1 and the short outpatient observation period in Case 2.

In conclusion, bee sting–induced ischemic stroke is a rare but clinically serious complication that can manifest across a wide spectrum – from isolated, self-resolving posterior circulation syndromes to devastating multisystem failure with fatal neurological outcomes – even in previously healthy individuals. Both cases reported here add to the growing international literature of imaging-confirmed cerebrovascular events following Hymenoptera envenomation and contribute unique features including the posterior fossa multi-territory infarction pattern of Case 2, and the full spectrum of mass envenomation complexity including intraocular stingers, intra-aural bee removal, ecchymotic abdominal wall discolouration, recurrent airway clot, and nosocomial superinfection in Case 1. Early neurological assessment and prompt MRI neuroimaging are essential following mass envenomation, particularly when patients develop new headache, focal neurological deficits, or altered consciousness even hours to days after the initial sting. Clinicians should simultaneously consider vasospasm, immune-mediated thrombosis, and disseminated intravascular coagulation as concurrent mechanisms, and should initiate anti-allergic therapy in parallel with stroke-specific evaluation. Multidisciplinary management involving emergency medicine, neurology, nephrology, pulmonology, ophthalmology, and ENT is essential in severe cases. Larger prospective registries and multicentre collaborations are needed to establish evidence-based protocols for this rare but potentially fatal envenomation-related cerebrovascular emergency.

Abbreviations

AOP
Artery of Percheron
CT
Computed tomography
DAMA
Discharged against medical advice
DIC
Disseminated intravascular coagulation
DSA
Digital subtraction angiography

DWI
Diffusion-weighted imaging
ENT
Ear, nose and throat
GCS
Glasgow Coma Scale
ICA
Internal carotid artery
ICU
Intensive care unit
INR
International normalized ratio
MCA
Middle cerebral artery
MRA
Magnetic resonance angiography
MRI
Magnetic resonance imaging
mRS
Modified Rankin Scale
NR
Not reported
PCA
Posterior cerebral artery
PIVD
Prolapsed intervertebral disc
rt-PA
Recombinant tissue plasminogen activator
SAH
Subarachnoid haemorrhage
WM
White matter

Declarations

Ethics approval and consent to participate

Not applicable. Both patients (or their legal representative in Case 1) provided written informed consent to participate.

Consent for publication

Written informed consent was obtained from the patients (and the next-of-kin/legal representative in Case 1) for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal. Patient identifiers and ocular regions have been masked in all clinical photographs to preserve anonymity.

Availability of data and materials

The dataset(s) supporting the conclusions of this article is(are) included within the article. Additional anonymised clinical and imaging data are available from the corresponding author on reasonable request, subject to institutional data-sharing policies and patient confidentiality.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

PP performed the literature review, drafted the manuscript, and was involved in the clinical management of both patients. MKR supervised the clinical management, provided critical review of the manuscript, and approved the final version. Both authors read and approved the final manuscript.

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Reporting guideline statement

This case report was prepared in accordance with the CARE (CAse REport) reporting guidelines. A populated CARE checklist is provided as a supplementary file accompanying this submission.

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Figures



Figure 1

Mass envenomation burden and multi-organ manifestations in Case 1. (A) Lateral view of Case 1 (70-year-old male) in the ICU receiving supplemental oxygen via nasal cannula, with residual sting marks visible across the face, consistent with the documented neurological decline. (B) Close-up of the periorbital region of Case 1 demonstrating severe ecchymosis and oedema, with the eyelid manually retracted to facilitate stinger removal from the conjunctival surface – over 15 stingers were ultimately removed from the eyes. (C) Massive abdominal distension with diffuse purplish-ecchymotic discolouration in Case 1, with foley's catheter visible, reflecting severe systemic envenomation, venom-induced microvascular injury, and fluid sequestration associated with multi-organ dysfunction. (D) Bronchoscopy monitor displaying an endoscopic view of the tracheal lumen in Case 1, showing dark mucus plugs and organized clot obstructing the airway, corresponding to the need for multiple bronchoscopy sessions to clear recurrent tracheostomy tube occlusions.

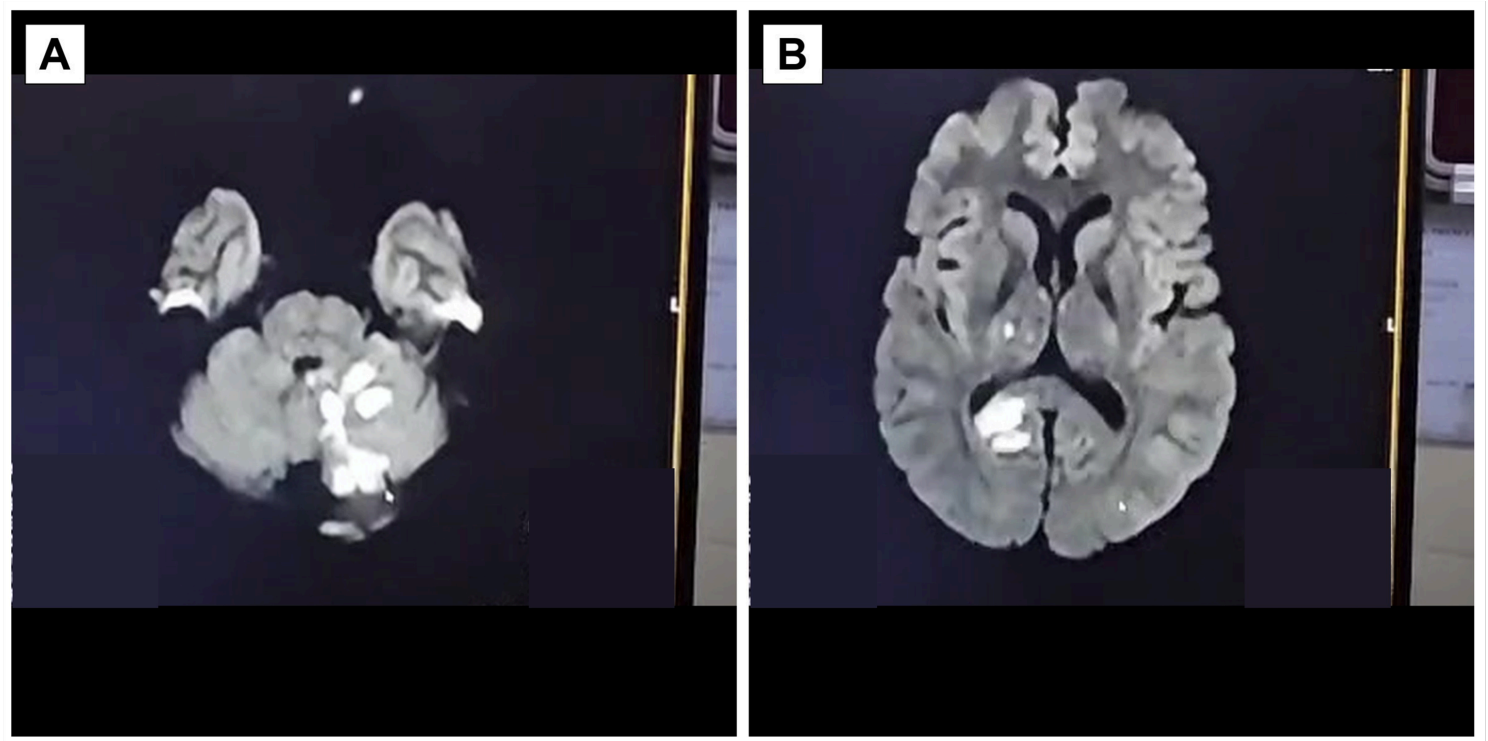


Figure 2

Diffusion-weighted magnetic resonance imaging (DWI) of the brain in Case 2 (47-year-old male), performed on Day 2 of admission. (A) Axial DWI at the level of the posterior fossa demonstrating multiple foci of restricted diffusion in keeping with acute embolic infarction. (B) Axial DWI at a higher slice level demonstrating an additional focus of restricted diffusion.



Figure 3

Bee sting burden and mechanical stinger extraction in Case 2. (A) Frontal view of Case 2 (47-year-old male) demonstrating dense embedding of bee stingers across the forehead, bilateral cheeks, periorbital regions, nose, and chin, with associated facial oedema and erythema. (B) Lateral view of Case 2 showing extensive stinger embedding with marked periorbital swelling and soft-tissue oedema involving the ear, cheek, and neck region. (C) Gloved medical personnel using instruments and a collection bowl to extract embedded bee stingers from the forearm of Case 2, illustrating the active decontamination procedure required at the tertiary care centre. (D) Dorsum of the hand and wrist of Case 2 showing numerous embedded stingers being removed with forceps, with surrounding erythema and ecchymotic spots marking each sting site.