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# Cerebral Infarction Following Bee Sting: A Case Report and Literature Review

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**Abstract:** Bee stings typically cause local or systemic allergic reactions, but secondary cerebral infarction is an exceedingly rare complication. We report a 65-year-old man who developed motor aphasia and right-sided hemiplegia approximately 19 hours after being stung by bees on the left forehead and left upper arm. Imaging revealed a new acute infarct in the left frontotemporal lobe and basal ganglia, superimposed on severe pre-existing left internal carotid artery stenosis. Laboratory tests showed elevated fibrinogen and D-dimer, indicating a hypercoagulable state. After antiplatelet therapy, lipid-lowering statin treatment, and comprehensive rehabilitation, the patient was left with persistent right-sided motor dysfunction and aphasia. We review the 25 previously reported cases and discuss potential mechanisms, including systemic inflammation, hypercoagulability, allergy-related hypotension, vasoconstriction mediated by vasoactive substances, and sympathetic dysregulation. This case underscores that bee stings can act as a significant trigger for ischaemic stroke in patients with underlying cerebrovascular stenosis, and clinicians should maintain a high index of suspicion when neurological symptoms emerge after envenomation.

**Keywords:** bee sting; cerebral infarction; ischaemic stroke; anaphylaxis; hypercoagulability

## 1. Introduction

Bee stings are among the most common insect injuries worldwide, particularly in rural and suburban areas during warm seasons. In most instances, they cause only transient local reactions-pain, erythema, and oedema-that resolve with simple symptomatic measures. However, bee venom is a chemically complex mixture containing over 40 biologically active components, including melittin (the principal hemolytic and pro-inflammatory peptide), phospholipase A2, hyaluronidase, apamin, mast cell degranulating peptide, and biogenic amines such as histamine and serotonin [1]. These constituents can induce not only immediate hypersensitivity reactions but also multi-system toxicity, ranging from acute kidney injury and myocardial damage to hepatic dysfunction and coagulopathy [2-4]. Anaphylactic shock is the most feared acute complication, but neurological sequelae, though rarely reported, are increasingly recognised as potential serious outcomes.

Since the first description in 1962, only 25 cases of cerebral infarction following hymenopteran (bee, wasp, or hornet) stings have been documented in the English and Chinese literature [5]. The true incidence is likely underestimated because mild or transient neurological symptoms may be overlooked, and many cases occur in regions with limited neuroimaging facilities. Moreover, the pathogenesis remains incompletely understood: it is not simply a consequence of anaphylactic hypotension, as infarcts have been reported in patients without overt shock. Proposed mechanisms include venom-induced hypercoagulability, direct vasospasm, systemic inflammatory response with plaque destabilisation, and sympathetic autonomic dysregulation. Here we present a new case with detailed clinical, laboratory, and imaging follow-up, and we comprehensively

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review the literature to clarify risk factors, temporal patterns, imaging characteristics, and prognostic indicators, aiming to heighten clinical awareness and guide preventive strategies.

## **2. Case Report**

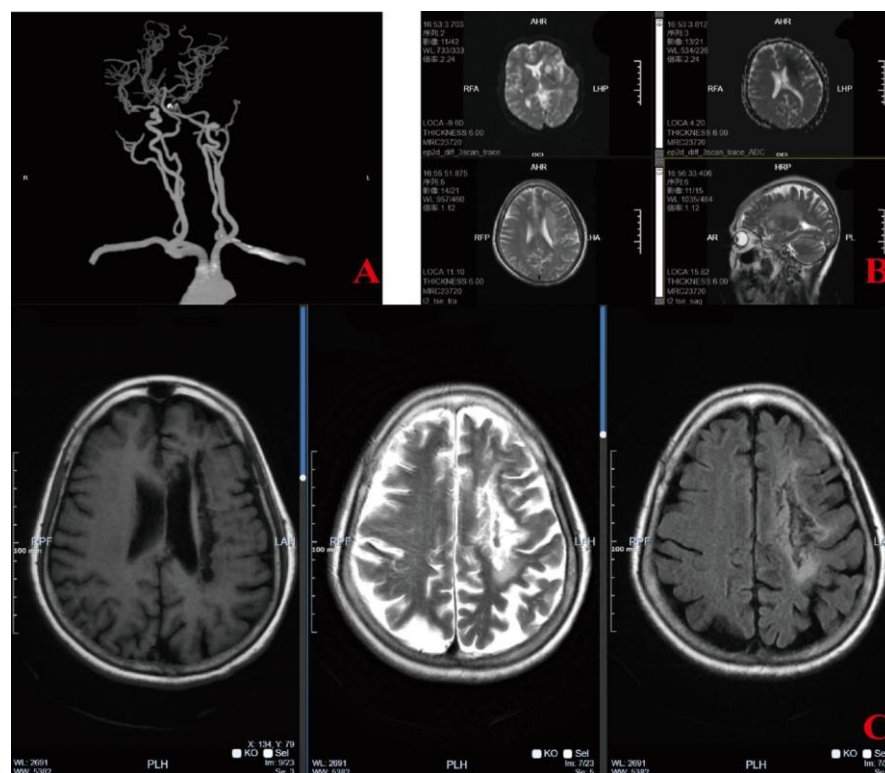
### *2.1. General Information and History*

A 65-year-old man, a roadside plant maintenance worker with a history of hypertension for more than 10 years (controlled with oral amlodipine, but with suboptimal adherence and no regular blood pressure monitoring), and no known diabetes mellitus, coronary artery disease, or prior stroke, was stung by multiple bees on the left forehead and left upper arm while trimming shrubs on the morning of 6 July. He reported an immediate sharp pain followed by local redness and mild swelling, but he did not seek medical attention and continued working. He experienced no dyspnoea, palpitations, or dizziness at that time. Approximately 19 hours later, on the following morning, his family found him unable to speak and with complete immobility of his right side; he was promptly taken to our emergency department.

### *2.2. Admission Examination and Investigations*

On arrival, his vital signs were: blood pressure 148/90 mmHg, heart rate 82 bpm, respiratory rate 18/min, oxygen saturation 98% on room air, and axillary temperature 36.7°C. He was alert and oriented but presented with expressive (motor) aphasia—he could understand simple commands but produced only unintelligible syllables. Cranial nerve examination revealed flattening of the right nasolabial fold and deviation of the tongue to the right. Motor examination showed muscle strength grade I (out of V) in the right upper limb (shoulder abduction, elbow flexion, and wrist extension all grade I) and grade III in the right lower limb (hip flexion and knee extension weak but able to move against gravity with reduced resistance), with increased muscle tone and hyperreflexia on the right side. The right plantar response was extensor (Babinski sign positive). Sensory testing was difficult because of aphasia, but there was no clear hemi-neglect. The National Institutes of Health Stroke Scale (NIHSS) score was 15, reflecting moderate-severe stroke.

Emergency head computed tomography (CT) showed no haemorrhage but revealed old lacunar infarcts in the bilateral basal ganglia. CT angiography (CTA) demonstrated extensive calcified atherosclerotic plaques and severe luminal stenosis (estimated >80%) of the intracranial segment of the left internal carotid artery, as well as calcification of the right intracranial internal carotid artery and narrowing of the left anterior cerebral artery and the M1 segment of the left middle cerebral artery (Figure 1A). Diffusion-weighted MRI (DWI) performed within 3 hours of admission showed a large hyperintense area in the left frontotemporal lobe and left basal ganglia, with corresponding low apparent diffusion coefficient (ADC) values, consistent with acute ischaemia (Figure 1B). The infarct volume was approximately 45 mL by manual planimetry, suggesting a relatively large territory. Magnetic resonance angiography (MRA) confirmed the severe stenosis without evidence of arterial dissection or occlusion of the distal branches.



**Figure 1.** Imaging Findings in This Case. A: Admission CTA Showing Severe Stenosis of the Left Internal Carotid Artery; B: Admission Brain DWI Showing Acute Infarction in the Left Frontotemporal and Basal Ganglia; C: MRI at 3 Months Showing Softening and Gliosis of the Infarcted Area.

Laboratory results on admission: white blood cell count  $8.7 \times 10^9/L$  (normal  $4.0\text{--}10.0$ ), haemoglobin  $142\text{ g/L}$ , platelet count  $208 \times 10^9/L$ . Coagulation panel: prothrombin time  $11.8\text{ s}$  (normal  $10.0\text{--}13.0$ ), activated partial thromboplastin time  $30.5\text{ s}$  (normal  $25.0\text{--}35.0$ ), fibrinogen  $4.2\text{ g/L}$  (reference  $2.0\text{--}4.0\text{ g/L}$ ), and D-dimer  $0.8\text{ mg/L}$  (reference  $<0.5\text{ mg/L}$ ), indicating a prothrombotic state. Cardiac biomarkers: high-sensitivity troponin I  $0.012\text{ ng/mL}$  (normal  $<0.040$ ), creatine kinase-MB  $2.3\text{ ng/mL}$ , N-terminal pro-B-type natriuretic peptide  $86\text{ pg/mL}$ . Renal function: creatinine  $82\text{ }\mu\text{mol/L}$ , urea  $5.1\text{ mmol/L}$ ; electrolytes, liver enzymes, and glucose were normal. Serum total cholesterol was  $5.8\text{ mmol/L}$ , low-density lipoprotein cholesterol  $3.9\text{ mmol/L}$ , and triglycerides  $1.7\text{ mmol/L}$ . No eosinophilia or elevated IgE level was tested, but there was no clinical evidence of ongoing anaphylaxis.

### 2.3. Treatment and Follow-Up

The working diagnosis was acute ischaemic stroke (large-artery atherosclerotic type, left internal carotid territory) precipitated by bee sting. Because the time from symptom onset to arrival exceeded 4.5 hours (approximately 20 hours), intravenous thrombolysis was not indicated, and mechanical thrombectomy was not performed owing to the severe stenosis rather than an acute occlusion amenable to retrieval. He was immediately started on aspirin  $100\text{ mg}$  once daily and atorvastatin  $20\text{ mg}$  at bedtime, along with intravenous fluids for volume support, antioxidants (edaravone), and neurotrophic agents (citicoline). Blood pressure was carefully managed to maintain mean arterial pressure above  $90\text{ mmHg}$  to avoid further hypoperfusion, while not exceeding  $160/100\text{ mmHg}$ . He remained haemodynamically stable throughout the hospital stay, with no episodes of hypotension, skin rash, or respiratory distress, indicating that the sting-induced allergic reaction had been mild and self-limited.

After two weeks of inpatient care, his neurological status was stable: right upper limb strength remained grade 0, right lower limb grade III, aphasia unchanged, but he could swallow and sit with support. He was then transferred to a municipal hospital of traditional Chinese medicine for integrated rehabilitation, receiving herbal decoctions

(promoting blood circulation and removing stasis), acupuncture at specific acupoints (Baihui, Quchi, Zusanli, etc.), and neuromuscular electrical stimulation on the right limbs. At discharge from that facility 1 month post-sting, right upper limb strength had improved to grade I (flicker of finger movement), and he could produce a few simple words with effort.

Over the following five months, he underwent eight additional hospitalisations in provincial and military rehabilitation centres, where he received intensive physiotherapy, occupational therapy, speech-cognitive training, and hyperbaric oxygen sessions (20 sessions total). Follow-up MRI at 3 months showed softening and gliosis of the infarcted area with no new lesions (Figure 1C). At 10 months after the sting, his right upper limb strength had reached grade II (active movement without gravity), and right lower limb grade IV (able to walk with a cane and ankle-foot orthosis); however, motor aphasia persisted with only short-phrase output, and he required assistance for most daily activities. The modified Barthel Index was 60 (moderate disability), indicating that he remained dependent in bathing, dressing, and toileting. He continued on secondary prevention with aspirin, atorvastatin, and strict blood pressure control (target <140/90 mmHg).

### 3. Discussion

#### 3.1. Clinical Characteristics and Comparison with Reported Cases

Our patient's presentation aligns with the typical profile of sting-associated stroke: an elderly male with vascular risk factors, a delay of several hours to a day between sting and neurological onset, and unilateral supratentorial infarction in a watershed or territorial distribution. In a systematic review of the 25 previously published cases (including bee, wasp, and hornet stings) [5–16], the mean age was 44.6 years (range 8–70), with a male:female ratio of 2.1:1. The sting-to-stroke interval ranged from 5 minutes to 7 days, but 76% occurred within 24 hours. Infarct topography was heterogeneous: 56% were unilateral hemispheric (mostly middle cerebral artery territory), 28% bilateral (often watershed), and 16% involved brainstem or cerebellum. Interestingly, 9 of the 25 patients had documented pre-existing vascular risk factors (hypertension, diabetes, or hyperlipidaemia), and 5 had known carotid or vertebrobasilar stenosis. This suggests that the sting rarely causes stroke in a healthy vascular bed; rather, it acts as an acute precipitant upon a vulnerable substrate. Our case reinforces this notion, as the severe left carotid stenosis clearly determined the infarct location.

Another noteworthy observation is that the number of stings does not correlate with stroke severity—some patients developed massive infarction after a single sting, while others with dozens of stings had no neurological sequelae. This points to individual host susceptibility (e.g., immune response, plaque vulnerability, and cerebral autoregulation capacity) as a critical determinant.

#### 3.2. Pathogenic Mechanisms—an Integrated View

The precise cascade from bee venom to cerebral ischaemia is multifactorial, and we propose a synergistic model comprising four interconnected pathways:

(1) Systemic inflammation and hypercoagulability -- Bee venom components, especially melittin and phospholipase A2, are potent activators of phospholipase C and nuclear factor- $\kappa$ B, leading to the release of tumour necrosis factor- $\alpha$ , interleukin-1 $\beta$ , and interleukin-6 from macrophages and mast cells [8]. These cytokines upregulate tissue factor expression on monocytes and endothelial cells, thereby activating the extrinsic coagulation pathway. Concomitantly, venom can directly activate platelets via thromboxane A2 synthesis and P-selectin exposure, promoting platelet aggregation. In our patient, the elevated fibrinogen and D-dimer levels on admission are objective evidence of ongoing coagulation activation. In patients with atherosclerotic plaques, the inflammatory milieu not only promotes thrombosis but also destabilises the fibrous cap, increasing the risk of plaque rupture and distal embolisation. Even without overt plaque

disruption, hyperviscosity and microaggregate formation can compromise microcirculatory flow, especially in regions with reduced perfusion pressure.

(2) Anaphylaxis-induced hypotension and cerebral hypoperfusion -- Histamine, leukotrienes, and prostaglandins released during immediate hypersensitivity cause widespread vasodilatation and increased capillary permeability, resulting in a drop in systemic vascular resistance. In severe cases, this leads to anaphylactic shock with mean arterial pressure below 60 mmHg, but even a modest decrease from 140 to 110 mmHg can critically reduce cerebral blood flow in the presence of a tight internal carotid stenosis, because the distal perfusion pressure is already at the lower limit of autoregulation. Watershed infarcts, which are common in paediatric cases without large-vessel disease [7], support a hypoperfusion mechanism. In our adult patient, although no hypotensive episode was recorded, it is possible that transient, self-resolving hypotension occurred during the night at home, lowering perfusion in the left middle cerebral artery territory and precipitating the infarct.

(3) Vasoactive-mediated vasoconstriction -- Bee venom contains thromboxane A<sub>2</sub>, leukotrienes C<sub>4</sub> and D<sub>4</sub>, and platelet-activating factor, all of which are potent vasoconstrictors. Experimental studies have shown that topical application of bee venom extract can induce reversible contraction of isolated cerebral arteries [11]. Some authors have proposed that stings to the face or neck may stimulate the superior cervical ganglion, causing a sympathetically mediated reflex spasm of the ipsilateral internal carotid artery [13]. However, our patient's stings were on the forehead (a region innervated by the ophthalmic branch of the trigeminal nerve, which has connections to the sympathetic plexus) and the arm, so this mechanism cannot be entirely excluded, though it is less likely to be the primary driver given the lack of angiographic vasospasm.

(4) Disseminated intravascular coagulation (DIC) and endothelial injury -- Severe envenomation can cause haemolysis, rhabdomyolysis, and direct endothelial damage, triggering widespread microvascular thrombosis. Although DIC has been reported in some fatal cases [14], our patient's platelet count, prothrombin time, and fibrinogen level did not meet the International Society on Thrombosis and Haemostasis criteria for DIC, making this mechanism unlikely here.

Taken together, we postulate that the most plausible sequence in our case is: the sting evoked a systemic inflammatory response that, together with pre-existing hypercoagulability (elevated baseline fibrinogen from hypertension and age), shifted the haemostatic balance toward thrombosis. Concurrently, subtle nocturnal hypotension might have reduced flow distal to the tight left carotid stenosis. The combination of increased platelet aggregation and decreased perfusion led to a critical ischaemic event in the already compromised left hemisphere.

### 3.3. *Diagnosis and Differential Diagnosis*

For any patient presenting with acute focal neurological deficits after a sting, the diagnostic algorithm should include: (1) non-contrast CT to exclude intracranial haemorrhage (which can occur from venom-induced vasculitis or anticoagulant effects); (2) DWI/ADC MRI to confirm acute infarct; (3) vascular imaging (CTA or MRA) to assess for stenosis, dissection, or occlusion; and (4) laboratory work-up for coagulopathy, cardiac enzymes, and renal function. Differential diagnoses to consider include: (i) global ischaemic encephalopathy secondary to prolonged anaphylactic shock (usually bilateral, diffuse grey-matter injury, and not focal); (ii) direct neurotoxic encephalopathy with seizures or confusion but without a DWI correlate; (iii) cerebral venous sinus thrombosis (rarely reported after stings); and (iv) stroke from cardioembolism (atrial fibrillation, valvular disease) or arterial dissection (which CTA can rule out). In our case, electrocardiography and 24-hour Holter monitoring excluded arrhythmias, and transoesophageal echocardiography was not performed but there was no clinical evidence of embolic source.

### 3.4. Therapeutic Considerations and Prognosis

There is no disease-specific treatment for sting-associated stroke; management follows established acute ischaemic stroke guidelines. If the patient presents within the thrombolytic window ( $\leq 4.5$  hours) and without contraindications, intravenous alteplase can be administered, although the presence of concurrent anaphylaxis may pose a risk. Endovascular thrombectomy should be considered for proximal large-vessel occlusions. Beyond the window, as in our case, antiplatelet therapy (aspirin or clopidogrel), high-intensity statin, and meticulous blood pressure control are the mainstays. Importantly, if the sting is recent and there is ongoing allergic reaction, epinephrine and corticosteroids may be needed, but they should be used cautiously as they can raise blood pressure and potentially increase intracranial pressure or haemorrhagic transformation a delicate balance.

Prognosis in the published series remains guarded, as most reported cases result in residual neurological deficits, and complete recovery is uncommon [15,16]. In our patient, although right lower limb function improved substantially, upper limb motor function and speech recovery remained limited. The poor recovery of the upper limb likely reflects the extensive infarct involving the motor cortex and corticospinal tract, further aggravated by severe baseline carotid stenosis that restricts collateral perfusion.

### 3.5. Prevention and Public Health Implications

We report a case of acute ischaemic stroke occurring 19 hours after multiple bee stings in a 65-year-old man with severe left internal carotid stenosis. The combination of venom-induced hypercoagulability, mild systemic inflammation, and possible haemodynamic fluctuation precipitated infarction in a critically perfused territory. This case highlights that bee stings are not merely trivial injuries in elderly patients with vascular comorbidities; they can serve as a decisive trigger for devastating neurological events. Clinicians should remain vigilant, perform rapid imaging, and follow standard stroke protocols, while also addressing potential allergic components. Heightened awareness and preventive counselling are essential to reduce the burden of this rare but severe complication.

## 4. Conclusions

This case report demonstrates a rare cerebral infarction after bee sting, highlighting that bee stings can trigger ischaemic stroke not only through allergic reactions but also via inflammatory-hypercoagulable-hypoperfusion pathways, especially in elderly patients with pre-existing cerebrovascular stenosis. When a patient stung by a bee develops neurological deficits, clinicians should promptly perform brain imaging and initiate stroke management. Furthermore, individuals with risk factors for atherosclerosis should be closely observed after stings, and preventive measures may be considered.

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