Case Report

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Cerebral infarction following bee stings: Case report and literature review

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Abstract
Background – To date, only 25 cases of cerebral infarction following a bee or wasp sting have been reported. Due to its rarity, undefined pathogenesis, and unique clinical features, we report a case of a 62-year-old man with progressive cerebral infarction following bee stings, possibly related to vasospasm. Furthermore, we review relevant literature on stroke following bee or wasp stings.

Case presentation – A 62-year-old retired male presented with progressive ischemic stroke after bee stings to the ear and face. Initial magnetic resonance imaging of the brain showed small punctate infarcts in the left medulla oblongata. Head and neck computed tomography angiography showed significant stenosis in the basilar artery and occlusion in the left V4 vertebral artery. The patient received intravenous alteplase (0.9 mg/kg) without symptomatic improvement. Digital subtraction angiography later demonstrated additional near occlusion in the left posterior cerebral artery (PCA). Thrombectomy was considered initially but was aborted due to hemodynamic instability. Repeated CT brain after 24 h showed acute infarcts in the left parieto-occipital region and left thalamus. The near occluded PCA was found to be patent again on magnetic resonance angiography (MRA) 25 days later. This reversibility suggests that vasospasm may have been the underlying mechanism. Unfortunately, the patient had persistent significant neurological deficits after rehabilitation one year later.

Conclusion – Cerebral infarction following bee stings is rare. There are several proposed pathophysiological mechanisms. While the natural course of this phenomenon is not well characterized, early diagnosis and treatment are essential. Furthermore, it is important to establish standardized care procedures for this unique entity.

Keywords: bee stings, cerebral infarction, thrombectomy, case report

1 Introduction

Wasp or bee stings are common around the world. Typical allergic symptoms include mild urticaria to severe anaphylaxis. Various unusual reactions after bee stings have been reported, involving neurological, renal, cardiac, pulmonary, and ocular systems [1]. To date, only 25 cases of cerebral infarction following a bee or wasp sting have been reported since the initial index case in 1962. Here, we report a case of a 62-year-old man with progressive cerebral infarction following bee stings and review relevant literature (Table 1) [2–26]. This rare phenomenon has unclear pathogenesis and clinical features, and there is no consensus on management.
| Author             | Region/Year | Age/Sex | Wasp/Bee           | Clinical features                          | CT/MR findings                          | Onset time | Angiography               | Treatment                                      | Prognosis          |
|--------------------|-------------|---------|-------------------|--------------------------------------------|-----------------------------------------|------------|--------------------------|------------------------------------------------|-------------------|
| Day et al. [2]     | US/1962     | 36/M    | Wasp: neck, face, and arms | Headache, hemiplegia, seizure, coma         | Necropsy: left hemorrhagic cortical infarction; pontine infarction | 15 min     | NR                       | Anti-allergic phenobarbital                     | Died              |
| Romano et al. [3]  | US/1989     | 1.4/M   | Wasp: inner upper lip | Hemiparesis, facial weakness               | Left putamen and caudate infarctions    | 4 days     | Left ICA occlusion       | Anti-allergic                                      | Full recovery      |
| Riggs et al. [4]   | US/1993     | 38 y/M  | Wasp: multiple; face and neck | Hemiplegia, aphasia                        | Left MCA infarction                    | 2 days     | Left ICA occlusion       | Unrecorded                                      | NR                |
| Riggs et al. [5]   | US/1994     | 52/M    | Wasp               | Dysarthria, hemiparesis, quadriparestis     | Left parietal and insular cortical infarctions | A few hours | Right ICA occlusion      | Anti-allergic                                      | NR                |
| Crawley et al. [6] | US/1994     | 30/F    | Wasp: arm          | Visual deficits, hypotension                | Left occipital infarction              | 45 min     | NR                       | Anti-allergic                                      | Full recovery      |
| Bhat et al. [7]    | India/2002  | 30/M    | Bee: multiple; all over body | Dysarthria, vertigo, tinnitus, and bilateral cerebellar signs | Bilateral cerebellar hemorrhagic infarction | <1 day     | NR                       | Anti-allergic reduced intracranial pressure       | Died              |
| Sachdev et al. [8] | India/2002  | 40/M    | Wasp: face         | Left hemiplegia, slurred speech             | Right ventral pons and right cerebellum infarctions | 10 h       | NR                       | Reduce cerebral edema aspirin                     | Improved          |
| De-Meing Chen et al. [9] | Taiwan/2004 | 71/F | Wasp: head, face, and limbs | Facial palsy, paraplegia                   | Right MCA territory infarction          | 1 day      | Occlusion of the infrarenal aorta | Thrombectomy, anticoagulant plasmapheresis | The patient received rehabilitation programs and was discharged on the 56th day | Improved |
| Schiffman et al. [10]| US/2004   | 57/F    | Bee: neck, head, eye, face, arm | Left homonymous hemianopia               | Large right temporoparietal hemorrhagic infarction | 2 days     | Right PCA P1 occlusion  | Anti-allergic antiemetics                          | Improved          |
| Taurin et al. [11] | French/2006 | 36/M   | Wasp: location NR | Vomiting, syncope                          | Left dorsal medulla infarction          | 14 days    | NR                       | Anti-allergic                                      | Full recovery      |
| Temizoz et al. [12]| Turkey/2009| 60/M   | Bee: head, face, limbs | Hemiplegia, dysarthria                     | Bilateral frontal lobe infarcts, right temporoparietal and bilateral centrum | 2 h        | NR                       | Anti-allergic and aspirin                         | Improved slight left hemiparesis                  |
| Vidhate et al. [13]| India/2010  | 8/M     | Wasp: eyebrow nasal bridge | Hemiplegia, altered sensorium              | Infarcts in left frontoparietal region, right subcortical area, and posterior limb of the left internal capsule | 8 days     | CTA normal               | Systemic antibiotics, anticoagulants               | Improved right-sided complete ophthalmoplegia    |

(Continued)
| Author                  | Region/Year | Age/Sex | Wasp/Bee                      | Clinical features                                      | CT/MR findings                                  | Onset time | Angiography | Treatment                        | Prognosis          |
|------------------------|-------------|---------|-------------------------------|--------------------------------------------------------|-------------------------------------------------|------------|-------------|----------------------------------|--------------------|
| Dechyapirom et al.     | US/2011     | 64/M    | Bee: face, neck, chest        | Hemiplegia, facial palsy, chest pain                   | Large right MCA territory infarction             | 16 h       | NR          | Anti-allergic rt-PA               | Recovery           |
| Rajendiran et al.      | India/2012  | 25/M    | Bee: head and neck            | Vomiting, monoplegia, transient visual loss            | Right frontoparietooccipital infarct with hemorrhagic transformation | 1 day      | NR          | Anti-allergic antiemetics         | Full recovery       |
| Viswananathan et al.   | India/2012  | 59/M    | Bee: face, neck, scalp, chest | Disorientation, dysarthria, facial palsy, hemiplegia, seizures, | Right perisylvian, perinsular, and parietal cortices infarct | 2.5 h      | NR          | Anti-allergic aspirin, atorvastatin, and heparin | Improved           |
| Jain et al.            | India/2012  | 70/M    | Bee                           | Altered sensorium, hemiplegia                          | Left frontoparietooccipital infarction, lacunar infarcts of bilateral gangliocapsular | 6 h        | MRA normal   | Anti-allergic                    | Improved           |
| Bilir et al.           | Turkey/2013 | 35/M    | Bee: multiple; NR             | Change in consciousness, dyspnea, hemiparesis         | Left MCA infarction                             | 6 h        | Neck MRA normal | Anti-allergic                   | Residual right hemiparesis |
| Wani et al.            | India/2014  | 40/M    | Wasp: multiple; face, head, and neck | Deterioration in consciousness, hemiplegia, obtundation | Left thalamic, left parietooccipital, bilateral cerebellar hemispheres, and pontine infarction | 1 day      | NR          | Anti-allergic                    | vegetative state   |
| An et al.              | Korea/2014  | 50/M    | Bee                           | Left involuntary movements                             | Right temporal infarction                        | 27 h       | Right M2 of MCA occlusion         | Anti-allergic haloperidol aspirin | Recovery           |
| Kulhari et al.         | US/2016     | 44/M    | Wasp: leg                     | Hemiparesis, facial palsy, dysarthria                  | Multiple infarctions in right MCA                | 1 h        | Vasoconstriction in the bilateral proximal MCA arteries | Anti-allergic rt-PA | Recovery           |
| Guzel et al.           | Turkey/2016 | 59/M    | Bee                           | Mild shortness of breath left hemiplegia, aphasia     | Right frontotemporoparietal infarction           | A few hours| NR          | Anti-allergic                    | Died               |
| Dalugama and Gawarammana | Sri Lanka/2018 | 69/F  | Wasp:                          | Left posterior frontal white matter infarction         | Bilateral thalami, left frontotemporoparietal infarctions, hemorrhage transformation | NR        | NR          | Aspirin and atorvastatin         | Improved           |
| Gupta et al.           | India/2019  | 41/F    | Honeybee: arm                 | Seizure, hemiparesis, dysarthria, unconscious         |                                    | 3 h        | NR          | Antiepileptics,                  | Died               |
| Author                          | Region/Year      | Age/Sex | Wasp/Bee                  | Clinical features                          | CT/MR findings                       | Onset time | Angiography | Treatment                                      | Prognosis          |
|--------------------------------|------------------|---------|---------------------------|--------------------------------------------|---------------------------------------|------------|-------------|------------------------------------------------|--------------------|
| Elavarasi et al. [25]          | India/2020       | 41/M    | Bee                       | Hemiparesis, dysarthria                    | Massive right MCA territory infarction | 5 h        | NR          | Antiepileptics, heparin, antiplatelets           | Died               |
|                                |                  |         |                           |                                            |                                       |            |             | hemicraniectomy                                 |                    |
| Ramlackhansingh and Seecheran  | Trinidad and Tobago/2020 | 70/M  | Africanised honey bee: face, forearms, shoulders, and back | Dysphasia, hemiparesis                    | Left parietal lobe and left basal ganglia infarctions | 1 day      | Normal      | Anti-allergic aspirin                            | Full recovery       |
| Current study                  | China/2021       | 62/M    | Honey bee face, neck      | Speech disorder, hemiparesis                | Left parieto-occipital lobe, basal ganglia, thalamus infarctions | 2 h        | Stenosis of bilateral VA and BA Occlusion of P1 segment of right PCA | Sequela            |

**Abbreviations:** BA, basilar artery; CT, computerized tomography; CTA, computerized tomography angiography; DIC, disseminated intravascular coagulation; DSA, digital subtraction angiography; ECG, electrocardiogram; F, female; ICA, internal carotid artery; M, male; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; NIHSS, National Institute of Health Stroke Scale; NR, not reported; PCA, posterior cerebral artery; rt-PA, recombinant tissue plasminogen activator; VA, vertebral artery.
2 Case presentation

A 62-year-old retired male with only mild hypertension was brought to the emergency department 1 h after bee stings to the ear and face. The initial symptoms were nausea, vomiting, and headache. On examination, he was found to have dysarthria, bulbar weakness, and right hemiparesis involving the arm and leg. The National Institutes of Health Stroke Scale score was 5. His blood pressure on presentation was 120/77 mmHg. The electrocardiogram showed sinus rhythm with a heart rate of 72 beats per minute. His initial non-contrast head CT was negative for hemorrhage or any acute processes. To treat a possible allergic reaction, he was given intravenous methylprednisolone and calcium gluconate. Given the possibility of acute stroke, intravenous alteplase (0.9 mg/kg) was also administered. Unfortunately, his symptoms did not improve.

Magnetic resonance imaging (MRI) of the brain was performed, which showed acute infarcts in the left medulla oblongata (Figure 1).

Head and neck CT angiography (CTA) was performed, which showed significant stenosis in the basilar artery, and occlusion in the V4 segment of the left vertebral artery (Figure 2). Digital subtraction angiography (DSA) showed significant stenosis in the V4 segment of the right vertebral artery, occlusion in the V4 segment of the left vertebral, and left posterior cerebral arteries (Figure 3). Thrombectomy was initially considered, but it was aborted due to hemodynamic instability. CT brain the next day showed acute infarcts in the left parieto-occipital region and the left thalamus (Figure 4). Repeat magnetic resonance angiography (MRA) head and neck was performed 25 days after presentation, which showed persistent stenosis in the V4 segments of the bilateral vertebral arteries, but patent bilateral posterior cerebral arteries (Figure 5).

He had a normal hepatorenal function, interleukin-6, fibrinogen, urine analysis, erythrocyte sedimentation rate, and coagulation testing. He was discharged to an acute rehabilitation facility. Unfortunately, at a one-year clinical follow-up, he had persistent neurologic deficits. He required percutaneous endoscopic gastrostomy due to dysphasia and tracheostomy. He was also unable to ambulate independently.

Ethical approval: The research related to human use has been complied with all the relevant national regulations, institutional policies and in accordance with the tenets of the Helsinki Declaration, and has been approved by the authors’ institutional review board or equivalent committee.

Informed consent: Informed consent has been obtained from all individuals included in this study.

3 Discussion

Adverse reactions to bee or wasp stings may present with neurological, cardiovascular, renal, pulmonary, and ocular symptoms [1,2]. Stings associated with cerebral infarction are extremely rare, and the underlying pathological mechanisms remain unclear. Furthermore, there are no guidelines for the management of stroke secondary to bee stings. We list documented cases of cerebral infarctions after bee or wasp stings (Table 1). Among all the reports, the stroke symptoms presented over a wide range
of times after sting, from 15 min to 4–8 days and up to 14 days [2,3,11,13]. There were more males than females in the reviewed literature, with a ratio of 4.2:1. The ages of patients ranged from 34 months old to 71 years old [3,9]. Most individuals did not have classical stroke risk factors. Only two documented cases had a history of hypertension and smoking [10,21]. Given the rarity of this phenomenon, the TOAST classification of our case may be best described as “stroke of other determined etiology.” We believe that bee venom caused multifocal vasospasm involving different arteries. This likely resulted in the pattern of ischemic stroke confirmed on imaging. Alternatively, there could have been thrombus formation in the right V4 segment that subsequently embolized to the left

**Figure 2:** Admission CTA (225 min after symptom onset) showed significant stenosis in the basilar artery, and occlusion of the left V4 segment of the vertebral artery (arrow). Both bilateral posterior cerebral arteries were patent at this time (arrow).

**Figure 3:** DSA (8 h after symptom onset) showed (a) significant stenosis in the right V4 segment of the vertebral artery and occlusion in the left PCA (arrow); (b) occlusion in the left V4 segment of the vertebral artery (arrow).
posterior cerebral artery (PCA). Given the presentation with headache and vasoconstrictive trigger, a diagnosis of RCVS could also be considered [28]. The RCVS2 score for our case was 3. The features supportive of RCVS is a vasoconstrictive trigger, while the features inconsistent with RCVS is not thunderclap headache. Another possibility is secondary infarction related to rt-PA [29]. However, we believe this is less likely.

Most patients had favorable outcomes with immediate treatment; however, some had poor outcomes and four patients died [2,7,22,24]. The natural history of stroke following bee or wasp stings was, therefore, varied in the available literature. The youngest patient who died was 30 years old, and the mean age of death amongst cases was 41 years [7]. The locations of stings mainly involved the head, face, and neck. One case reported a near-global distribution of stings to the patient’s body; it was associated with bilateral hemorrhagic cerebellar infarctions [7]. Principal neurological manifestations following the sting included facial weakness, hemiplegia, slurred speech, seizures, involuntary movements, and coma [2,19,24]. Renal failure, acute coronary syndrome, and arrhythmias were also reported in some cases [1,14]. Two cases developed hypotension, and one had hypotensive syncope [6,18,20]. Thirteen patients underwent cerebral angiography, of which nine were found to have large artery stenosis or occlusion. Six cases were found to have bilateral infarctions [7,12,13,17,19,27]. Three cases were found to have bilateral large artery stenosis or occlusion [5,21]. Four cases had combined intracerebral or subarachnoid hemorrhage [2,7,10,15,24]. Currently, there is no evidence to suggest a particular vascular territory is more susceptible. In addition to stroke, bee stings have been associated with other neurologic signs and symptoms, such as trigeminal neuralgia [30] and Parkinsonism [31]. Future research is needed to better understand the relationship between stings and neurologic sequelae.

There are no specific guidelines or expert consensus on treating cerebral infarction associated with bee or wasp stings due to limited reported cases and variations in presentations. According to reported cases, it is reasonable to consider treatment with epinephrine, methylprednisolone, antihistamine, and other suitable anti-allergic drugs at an early stage. Bees generally leave their stinging apparatuses

![Figure 4: Head CT (18 h after symptom onset) showed ischemic infarcts in the left parieto-occipital lobe and thalamus (arrow).](image)

![Figure 5: Repeat head and neck MRA (25 days after symptom onset) showed persistent stenosis in the bilateral V4 segments of the vertebral arteries and patency in bilateral posterior cerebral arteries (arrow).](image)
in patients’ lesions after envenomation; the Vespidae attached to the sting site often persistently inject venom. Prompt removal of such stinging apparatuses is likely beneficial [17]. Two patients obtained favorable outcomes after intravenous rt-PA. Prompt administration of intravenous rt-PA should be considered if there are no contraindications [14,21]. It is also essential to promptly correct hypotension or insufficient perfusion [6,18]. Attempts of mechanical thrombectomy have not yet been reported; our case abandoned this procedure due to hemodynamic instability. However, mechanical thrombectomy may be considered if affected individuals have large vessel occlusions and no contraindications. The possible clinical benefits require further validation alongside attention to detail concerning related comorbidities and complications. For certain patients in whom the mechanism is consistent with vasospasm, intra-arterial vasodilators are another consideration.

To date, the pathophysiological mechanisms of bee or wasp sting-associated cerebral infarction are not fully elucidated. Wasps are members of the order Hymenoptera, suborder apocrita. Sensitization to wasp venom requires only a few stings. Also, symptoms may occur after a single sting [27], including a variety of reactions related to neurological, renal, pulmonary, ocular, muscular, and cardiovascular systems [1].

Postulated mechanisms include the following:

1) Immune system hyper-functionality: Riggs et al. [5] reported a patient who experienced a wasp sting 14 years prior to presentation. Upon being stung a second time, severe allergic reactions and bilateral occlusion of the internal carotid arteries were reported. Riggs et al. considered a possible mechanism of immune system hyper-functionality.

2) Global cerebral hypoperfusion: Hypotension post-sting may be attributed to histamine and prostaglandin-2 induction [19]. Vidhate et al. [13] reported an 8-year-old boy with no abnormalities of the cerebral vasculature. However, he suffered symmetrical watershed infarction bilaterally. Additionally, two patients [6,18] were also reported to have anaphylaxis-induced cerebral infarction related to hypotension and likely cerebral hypoperfusion.

3) Retrograde stimulation of the superior cervical ganglion: [4,5] Riggs argued that venomous insect stings may lead to systemic immune responses and increased endothelial permeability of the distal ICA. Multiple ipsilateral facial or neck wasp stings can stimulate sympathetic innervation to the distal ICA via the superior cervical ganglion.

4) Disseminated intravascular coagulation (DIC): Jain [17] proposed that hemolysis and endothelial damage via toxins in honeybee venom contributed to DIC development resulting in the occlusion of blood vessels by widespread fibrin thrombi in the microcirculation.

5) Vascostriction: Bee venom contains vasoactive peptides such as thromboxane, leukotrienes, and other vasoactive mediators, causing vasoconstriction that can lead to ischemic stroke [10,21]. The patient we reported had previous MRA three years prior that was negative for intracranial vessel stenosis or occlusion. Furthermore, the PCA stenosis resolved on repeat MRA 25-days after the initial presentation. This suggests vasoconstriction may be a possible mechanism underlying cerebral infarction in our patient.

4 Conclusion

In summary, cerebral infarction following bee stings is rare. Various mechanisms have been proposed but there is no consensus on the most common pathophysiology or management approach. Further experiments, including in animal models, would be of great interest to elucidate the cellular responses to the venom that lead to vascular compromise and cerebral ischemia.

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Conflict of interest: Authors state no conflict of interest.

Data availability statement: The data sets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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