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Ischemic Stroke Related to Bee Sting: A Rare Case Report

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ABSTRACT

Background: Stroke remains one of the leading causes of global morbidity and mortality, with most cases associated with classical vascular risk factors. Neurological complications following bee stings are rarely reported but may trigger ischemic stroke through mechanisms involving toxins, vasospasm, coagulopathy, and endothelial dysfunction.

Case Report: We report a 61-year-old female with a history of hypertension who developed left-sided weakness, facial asymmetry, and involuntary movements within eight hours following a single bee sting on her toe. A non-contrast CT scan revealed a lacunar infarct in the right lentiform nucleus. The patient was treated with dual antiplatelet therapy, levodopa, clonazepam, N-acetylcysteine (NAC), and folic acid, resulting in significant clinical improvement by the fifth hospital day.

Discussion: Bee-sting–induced stroke is an uncommon phenomenon involving complex interactions between bee venom toxins (melittin, PLA_2, and hyaluronidase) and systemic inflammatory, coagulatory, and vasospastic responses. The pathophysiology of ischemic stroke includes excitotoxicity, oxidative stress, and endothelial dysfunction. Management in this case followed standard ischemic stroke protocols, with adjunctive antioxidant and neuromodulatory therapy for symptomatic control.

Conclusion: Although rare, bee stings can precipitate ischemic stroke. This case highlights the need for clinical vigilance toward neurological complications after bee stings and underscores the importance of further research into their mechanisms. More comprehensive data are still needed regarding the diagnostic criteria and confirmation process for ischemic stroke secondary to bee sting.

Keywords: Bee sting, Involuntary movement, Ischemic stroke

INTRODUCTION

Stroke is the leading cause of global disease burden, measured in Disability-Adjusted Life Years (DALYs), and the third leading cause of death worldwide in 2021.[1] According to the report of the American Heart Association (AHA), ischemic stroke accounts for 87%, while the rest are hemorrhagic strokes and subarachnoid hemorrhage.[2] Globally, an epidemiological review based on Global Burden of Disease (GBD) 2021 data revealed that in 2021, there were approximately 7.80 million new cases of ischemic stroke (ASIR ≈ 92.4 per 100.000), with significantly declining trends in incidence, mortality, and DALYs over the last three decades.[3,4] The 2012–2014 stroke registry involving 5.411 stroke patients in Indonesia showed that the majority were

ischemic stroke (67%).[5] Rapid and accurate diagnosis is a crucial step determining the success of stroke management. Although most stroke cases are associated with cardiovascular risk factors, there are other uncommon causes such as toxins from various animals, including bee stings.

Bee stings typically cause transient local reactions such as pain, itching, erythema, burning sensations, and edema. Rarely, systemic reactions may occur, including vomiting, dyspnea, generalized edema, acute kidney injury, myocardial infarction, and cerebral infarction. [6] Stroke after a bee sting is a rare manifestation and the exact etiology is still unknown. Neurological manifestations following bee sting are rarely reported. [7] Similar to myocardial infarction following bee sting, vasoconstriction secondary to mediators released after the sting—exacerbated by exogenous adrenaline and platelet aggregation—contributes to cerebral ischemia. Bee venom contains histamine, thromboxane, leukotrienes, and other vasoactive and inflammatory mediators. Both thromboxane and leukotrienes have been shown to cause vasoconstriction leading to cerebral infarction. [8] Stroke associated with bee stings has been reported only in a few case reports, and most of these involved multiple stings. [9]

CASE REPORT

A 61-year-old woman presented to the emergency department with left-sided weakness. She could raise her left arm and leg but reported heaviness. Facial asymmetry and numbness on the left side were noted, along with brief, recurrent involuntary movements lasting less than one minute. Earlier that morning, while selling goods at school, she had been stung by a bee on her left little toe—approximately eight hours before hospital admission. She had a history of hypertension, routinely taking amlodipine 10 mg once daily.

On physical examination, the patient was fully conscious (compos mentis). Blood pressure was 154/99 mmHg, and other vital signs were within normal limits. Neurological examination revealed left central facial nerve paresis, spastic left hemiparesis (muscle strength 4/5), and left hemihypesthesia. Laboratory tests (figure 1) showed increased neutrophils (94.9%), random blood glucose (253 mg/dL), mildly elevated D-dimer (0.68 ng/mL, reference <0.5 ng/mL), decreased lymphocytes (4.7%), monocytes (0.2%), and eosinophils (0%). Electrolytes (sodium, potassium, chloride, and calcium), liver function (SGOT, SGPT), and renal function (urea, creatinine) were within normal limits.

Netrofil	* 94.9	50 - 70
Limfosit	* 4.7	18 - 42
Monosit	* 0.2	2 - 11
Eosinofil	* 0	1.0 - 3.0
Basofil	0.2	0 - 2
KIMIA - METABOLISME KARBOHIDRAT		
POCT Glukosa Darah (Sewaktu)	* 253	70 - 140
HEMOSTASIS		
D-dimer	★ 0.68	< 0.5

Figure 1. Laboratory results

Chest X-ray (figure 2) demonstrated cardiomegaly. A non-contrast head CT (figure 3) revealed a lacunar infarct in the right lentiform nucleus consistent with subcortical arteriosclerotic encephalopathy.

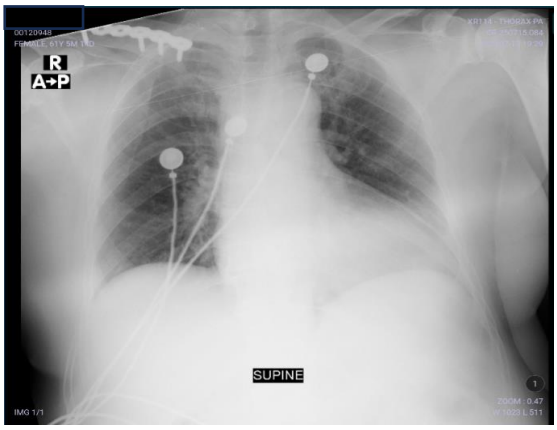

Figure 2. Chest X-Ray, AP view

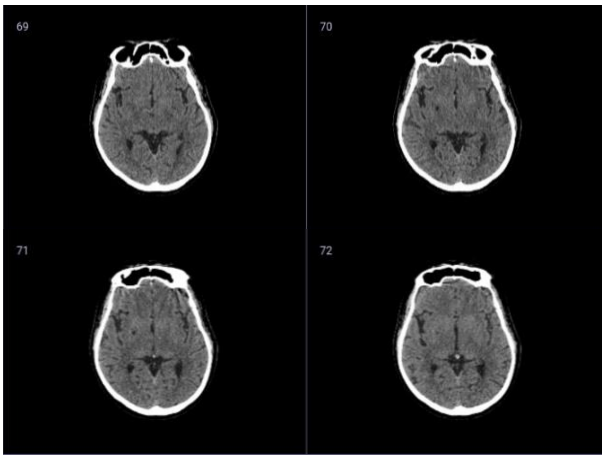

Figure 3. Non-contrast head CT-Scan, axial view

The patient was diagnosed ischemic stroke with central facial nerve paresis, left hemiparesis, and involuntary movement due to bee sting, hyperglycemia type 2 diabetes mellitus, ischemic stroke with, hypertensive heart disease, and old myocardial infarction. She was admitted to the intensive care unit and treated with dual antiplatelet therapy (aspirin 80 mg in the morning and clopidogrel 75 mg at night) levodopa three times daily, clonazepam 2 mg at night, intravenous N-acetylcysteine (NAC) 5 g single dose, folic acid 1 mg twice daily, atorvastatin 20 mg nightly, intravenous citicoline 500 mg twice daily, intravenous mecobalamin 1 ampoule per day, diphenhydramine 10 mg every 12 hours IV, dexamethasone 5 mg IV, ketorolac 30 mg IV twice daily, and intravenous fluids with 0.9% NaCl every 12 hours. From the cardiology department, additional therapy included ramipril 2.5 mg once daily, furosemide 40 mg once daily, bisoprolol 1.25 mg once daily, and amlodipine 5 mg once daily. From internal medicine, repeat random blood glucose was advised the next morning, with a plan to give 6 units of Sansulin log G at night and correctional sliding-scale rapid insulin if glucose >200 mg/dL. However, because glucose remained <200 mg/dL during hospitalization, insulin was not administered.

On the first hospital day, the patient's left-sided weakness improved; involuntary movements were still present but markedly reduced compared to admission. On the second day, no more involuntary movements were noted, though mild weakness persisted. After two days in intensive care, she was transferred to a general ward and referred to physical and rehabilitation medicine. After 5 days of hospitalization, the patient was discharged with maximal improvement.

DISCUSSION

Ischemic stroke arises not only from vascular risk factors but also from complex biomolecular cascades involving endothelial dysfunction, excitotoxicity, oxidative stress, inflammation, blood–brain barrier (BBB) disruption, and cell death. Traditional risk factors—hypertension, diabetes, dyslipidemia, and smoking—induce endothelial dysfunction through oxidative stress, reduced nitric oxide (NO) bioavailability, elevated endothelin-1, and upregulation of adhesion molecules (ICAM-1, VCAM-1), ultimately impairing vasodilation, antithrombotic function, and BBB integrity. [10, 11]

Excitotoxicity represents one of the earliest and most central events in ischemic stroke. Reduced cerebral perfusion causes neuronal energy failure due to oxygen and glucose deprivation, leading to ATP depletion and Na^+/K^+-ATPase pump failure. Consequently, widespread membrane depolarization triggers massive glutamate release into the synaptic cleft. Glutamate activates NMDA and AMPA receptors, causing excessive Ca^{2+} influx. Calcium overload activates destructive enzymes: calpain damages the cytoskeleton, endonuclease fragments DNA, and phospholipase A2 induces lipid peroxidation. Additionally, overactivation of neuronal nitric oxide synthase (nNOS) produces excessive NO, which reacts with superoxide to form peroxynitrite ($ONOO^-$), a toxic radical that worsens neuronal damage. [12, 13]

Ischemia activates innate immune responses in the brain. Microglia polarize into pro-inflammatory (M1) phenotypes, releasing cytokines such as IL-1β, TNF-α, and IL-6 that aggravate neuronal injury and upregulate endothelial adhesion molecules. Astrocytes secrete MCP-1 and VEGF, promoting leukocyte recruitment and BBB permeability. Activated endothelial cells express ICAM-1, VCAM-1, and selectins, facilitating neutrophil and monocyte migration into brain parenchyma, where they release ROS, proteolytic enzymes, and neutrophil extracellular traps (NETs)—all neurotoxic. [14, 15] Oxidative stress amplifies injury through ROS species (superoxide, hydrogen peroxide, hydroxyl radicals), damaging lipids, proteins, and DNA. Mitochondrial dysfunction leads to cytochrome c release and caspase-dependent apoptosis. [16]

An uncommon etiology of stroke is insect stings from the *Hymenoptera* order, such as honeybees and wasps. Stroke associated with *Apis mellifera* stings has been reported in only a few cases, often following multiple stings and typically within 4–10 hours of exposure. [9]

Most bee stings cause only mild local pain, erythema, edema, and pruritus that resolve within 24 hours. However, in some individuals, stings can provoke severe systemic reactions including anaphylaxis, hypotension, bronchospasm, laryngeal edema, and potentially fatal shock. [7] Some reports describe neurological manifestations such as stroke, epilepsy, polyneuropathy, cranial nerve palsy, and cavernous sinus thrombosis. [17, 18]

Bee venom (apitoxin) is a complex mixture of peptides and enzymes—including melittin, phospholipase A_2 (PLA_2), apamin, hyaluronidase, and mast-cell-degranulating peptide (MCDP)—that contribute to endothelial injury, vasospasm, and coagulopathy. [7, 8, 19]

The principal vascular complication mechanism following bee envenomation involves endothelial dysfunction, characterized by impaired regulation of vascular tone, permeability, coagulation, and inflammatory signaling, primarily mediated by hypersensitivity-related inflammatory pathways.[20] Experimental research in mice by Bistué-Rovira et al. showed that apitoxin directly causes vascular dysfunction and endothelial toxicity, reducing vascular cell viability and aortic function at clinically relevant concentrations, indicating endothelial dysfunction, NO pathway disruption, and oxidative stress.[21] Although no human studies exist, a forensic case report found

coronary atherosclerotic plaques in post-mortem myocardial tissue of a patient who died of anaphylactic shock following a bee sting.[20]

The main components of apitoxin are melittin (50–60%) and PLA_2 (10–12%).[22] Melittin exhibits potent cytolytic and hemolytic properties that disrupt cellular membranes, triggering inflammatory cascades, red cell destruction, and rhabdomyolysis, potentially resulting in multiorgan involvement. It may also interfere with complement activation and modulate bradykinin pathways.[17-19,23] The inflammatory–coagulatory interaction involves bradykinin-mediated activation of factor XII, modulating coagulation and platelet aggregation. Bradykinin also enhances endothelial NO production, inhibiting platelet adhesion via angiotensin II suppression. [24] Phospholipase A_2 (PLA_2) contributes to endothelial and membrane injury through phospholipid hydrolysis, promoting pro-inflammatory and pro-thrombotic processes that increase the risk of hypercoagulability, disseminated intravascular coagulation (DIC), and cerebral ischemic events.[17-19,23]

Other vasoactive components released after a bee sting include thromboxane and leukotrienes, both of which have been shown to cause vasoconstriction and platelet aggregation, leading to impaired cerebral blood flow and cerebral infarction.[6-8,18] Bee venom also contains vasoactive mediators such as histamine, serotonin, and catecholamines, which can provoke vasospasm, vasoconstriction, enhanced vascular permeability, and bronchial smooth muscle contraction. These mechanisms contribute to anaphylactic manifestations and circulatory instability, potentially reducing cerebral perfusion.[18,23,25] Hyaluronidase increases capillary permeability and facilitates the spread of toxins to surrounding tissues. This enzyme also has anticoagulant activity and functions as a spreading factor that enhances the effects of other bee venom components.[17-19,22,23] Apamin blocks calcium-activated potassium channels, disrupting neuronal transmission and causing seizures or focal deficits.[19,22] MCDP and mastoparan activate mast cells and trigger histamine release, producing allergic or systemic anaphylactic responses. [23]

Because bee stings can activate systemic coagulation, laboratory evaluation for coagulation biomarkers—complete blood count, PT, INR, aPTT, fibrinogen, and D-dimer—is needed to screen for coagulopathy or DIC.[9] Jung et al. reported a Korean case of DIC after bee-sting acupuncture, showing initial hemoglobin 14.5 g/dL, platelets 70,000/mm³, PT 10.5 s (normal 12–14 s), APTT 34.4 s (normal 22–40 s). Six hours later, hemoglobin dropped to 3.3 g/dL, platelets 68,000/mm³, PT >60 s, APTT >110 s; the patient died within 24 hours from hypovolemic shock and GI bleeding due to DIC. [26] In this case, appropriate hematology and D-dimer tests were performed, though other coagulation biomarkers were unavailable due to insurance limitations.

Non-contrast head CT remains the first-line imaging for stroke diagnosis in emergency settings. [27] Basal ganglia stroke can cause both hyperkinetic and hypokinetic involuntary movements such as ballism, chorea, or asterixis, usually acutely. Lesions in the basal ganglia damage inhibitory pathways involving GABA and dopamine, leading to thalamic disinhibition and excessive motor output. [28]

In this patient, choreiform involuntary movements and left hemiparesis were treated with dual antiplatelet therapy (aspirin 80 mg and clopidogrel 75 mg daily), along with levodopa, clonazepam, intravenous NAC (5 g single dose), and folic acid (2×1 mg). By the second day, involuntary movements resolved; left-sided weakness improved progressively, with full recovery by the fifth day.

Current ischemic stroke management guidelines recommend DAPT (aspirin + clopidogrel or ticagrelor) for secondary stroke prevention. [27] A comprehensive meta-analysis of 16,547 patients with non-

cardioembolic minor ischemic stroke or TIA found that DAPT significantly reduced recurrent stroke risk (ischemic or hemorrhagic) and cardiovascular events, albeit with an increased moderate-to-severe bleeding risk—higher with aspirin + ticagrelor than with aspirin + clopidogrel. Short-term DAPT (within 72 hours) also significantly reduced 90-day recurrence risk. [29]

In basal ganglia stroke with involuntary movements, dopaminergic precursors and GABA agonists are indicated. Levodopa, a dopamine precursor, crosses the BBB and binds D1/D2 receptors in the basal ganglia, normalizing motor output through the thalamo-cortical circuit. D1 activation enhances cortical excitation, while D2 activation suppresses excessive inhibition, balancing motor control.[30,31] Clonazepam, a benzodiazepine, augments GABAergic activity, reducing neuronal hyperexcitability and hyperkinetic movements. [32]

N-acetylcysteine (NAC), a cysteine and glutathione precursor, increases intracellular glutathione reserves and decreases reactive oxygen species (ROS). Its thiol group modulates disulfide bonds in proteins associated with hemostasis and endothelial function. Randomized clinical trials have shown NAC reduces oxidative and inflammatory biomarkers and improves NIHSS-assessed neurological outcomes without significant adverse effects.[33,34]

Folic acid (vitamin B9) plays a key role in methylation metabolism and homocysteine regulation. Beyond lowering homocysteine, it exhibits antioxidant and endothelial-protective effects, maintaining vascular wall integrity. Its supplementation prevents recurrent stroke through vascular protection and enhanced antioxidant capacity. [35]

The therapeutic regimen—aspirin, clopidogrel, NAC, folic acid, levodopa, and clonazepam—combines antithrombotic, antioxidant, and neuromodulatory mechanisms. In this case of right lentiform nucleus infarction following a bee sting, it served as a strategy to prevent microischemia, restore endothelial integrity, and modulate basal ganglia circuitry, promoting both acute symptom control and functional recovery.

CONCLUSION

Stroke caused by bee sting is rare, with most reported cases involving multiple stings. There have been no prior reports of stroke caused by a single sting. The treatment of bee-sting-induced stroke does not differ from that of conventional ischemic stroke. The therapeutic regimen combines antithrombotic, antioxidant, and neuromodulatory mechanisms. More comprehensive data are still needed regarding the diagnostic criteria and confirmation process for ischemic stroke secondary to bee sting, as well as further detailed case studies to strengthen the current understanding of this rare phenomenon.

Declaration by Authors

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