Stroke After Multiple Bee Sting


Abstract
Bee stings are commonly encountered worldwide. Various manifestations after bee sting have been described. Local reactions are common. Unusually, manifestations like vomiting, diarrhea, dyspnea, generalized edema, acute renal failure, hypotension and collapse may occur. Rarely vasculitis, serum sickness, neuritis and encephalitis have been described which generally develop days to weeks after a sting. We report a case of a 25-year-old male who developed left sided monoparesis and transient visual loss following multiple bee stings. Unlike the previous case reports, in our case there has been involvement of both the anterior circulation and posterior circulation territory to the brain. We report this case due to its rarity.

Introduction
Despite the common occurrence of insect stings and local and systemic allergic reactions, there are few reports of stroke following bee or wasp stings. We report on a young man who sustained a stroke after multiple bee stings.

Case
25 year old male working in electric motor workshop was bitten by multiple bees over head and neck region in afternoon. He was treated with intravenous antihistamines and antiemetics at a local district hospital in kancheepuram and was discharged as out-patient. Patient woke up the next day morning and found that he was having left upper limb weakness and with blurring of vision. He was then referred to government general hospital. Past medical and surgical history were insignificant. Physical examination revealed left upper limb weakness with a power of 2/5. Fundus examination was done which revealed a normal fundus on both sides. CT brain was immediately taken which revealed right frontal hypodensities with squashing of ipsilateral ventricles with hypodensities over both occipital lobes (Figure 1). MRI brain was obtained which revealed anterior infarct –right frontoparietal region, right occipital region, right gangliocapsular region with mild luminal narrowing of middle cerebral artery with haemorrhagic transformation (Figure 2). Carotid and vertebral Doppler was also done which was within normal limits. ECG and echocardiography were normal. Patient’s serum homocysteine, lupus anticoagulant and anticardiolipin antibodies were within normal limits. Patient was treated with antiedema measures, antiplatelet drugs and physiotherapy. His left upper limb monoparesis gradually improved and patient was discharged from the hospital. Patient’s visual acuity also improved. Patient was reevaluated 8 months after the incident. His left upper limb monoparesis had completely recovered. CT-Brain was again repeated 8 months later which revealed old infarct with gliotic changes in right high parietal and occipital region (Figure 3).

Discussion
In literature review, we had few cases of cerebral infarction occurring after bee sting. Maltzman, et al described common characteristics, such as acute to subacute onset of symptoms, moderate to severe visual loss followed by significant recovery (except in one case of a sting directly to the eye) which resulted in oedematous and haemorrhagic optic discs, and central or caecocentral scotomas. Our patient had transient subacute...
vision loss associated with left monoparesis. Seven cases of wasp and bee sting associated cerebral infarction were found in the literature.\(^3\)\(^4\) Reported neurological complications following bee sting includes seizure, hemiparesis, aphasia, apraxia, dysarthria, ataxia, and coma. None of these patients had a full eye examination, although in one patient\(^4\) a right homonymous superior quadrantanopia was demonstrated (Table 1).

The pathophysiology explaining the associated stroke is unknown. Hypotension caused by anaphylaxis may certainly induce cerebral and optic nerve ischaemia; however, this was not documented in our case. Similar to acute myocardial infarction after hymenoptera stings, it has been suggested that vasoconstriction secondary to mediators released after the sting, aggravated by exogenous adrenaline, and platelet aggregation also contribute to cerebral ischaemia.\(^4\) Bee venom itself contains histamine, thromboxane, leucotrienes, and other vasoactive and inflammatory mediators. In our patient, we postulate that the systemic immune mediated reaction to the bee sting caused vasoconstriction and a prothrombotic state with subsequent ischaemia leading to stroke. In addition, a neuropharmacological (sympathetic) mechanism of endothelial permeability involving the cerebral vasculature with a concurrent systemic thrombogenic

<table>
<thead>
<tr>
<th>Author/Reference</th>
<th>Age/Sex</th>
<th>Type of stings: location</th>
<th>Onset of neurological deficit</th>
<th>Examination findings and symptoms</th>
<th>Eye examination</th>
<th>MRI/CT findings</th>
<th>Treatment</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day(^2)</td>
<td>36/M</td>
<td>Wasp: multiple on neck, face, and arms</td>
<td>&lt;1 hour</td>
<td>Headache, seizure, right hemiplegia, coma</td>
<td>Equal and reactive pupils</td>
<td>NR; necropsy showed left haemorrhagic cortical infarct</td>
<td>Cortisone, antihistamines phenobarbital</td>
<td>Deceased</td>
</tr>
<tr>
<td>Crawley et al(^6)</td>
<td>30/F</td>
<td>Wasp: left arm</td>
<td>&lt;1 hour</td>
<td>Facial and arm swelling, widespread urticaria, acute pulmonary oedema, visual loss.</td>
<td>Right homonymous superior quadrantanopia</td>
<td>Left occipital ischaemic infarct</td>
<td>SQ adrenaline, IV gelofusine, IV hydrocortisone, IM chlorpheniramine, IV furosemide</td>
<td>Full recovery from quadrantanopia</td>
</tr>
<tr>
<td>Riggs et al(^7)</td>
<td>38/M</td>
<td>Wasp: multiple on left face and neck</td>
<td>2 days</td>
<td>Right hemiplegia, dense global aphasia</td>
<td>NR</td>
<td>Ischaemic infarction in the distribution of the left MCA; angiogram: left ICA occlusion</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Riggs et al(^7)</td>
<td>52/M</td>
<td>Wasp: single, location NR (previous history of wasp sting allergy)</td>
<td>A few hours, with worsening 24 days later</td>
<td>Anaphylactic shock with respiratory arrest, slurred speech and left hemiparesis initially, then 24 days later, acute obtundation and quadriparesis</td>
<td>NR</td>
<td>Initially, three small focal ischaemic infarcts, two in the right centrum semiovale and one in the right temporal lobe. After worsening, diffuse bilateral ischaemic white matter lesions.</td>
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<td>NR</td>
</tr>
<tr>
<td>Starr and Brasher(^7)</td>
<td>37/M</td>
<td>Wasp: 3 stings on arms</td>
<td>&lt;1 hour</td>
<td>Seizure, right hemiplegia</td>
<td>NR</td>
<td>Left cerebral infarction (CT done 14 months later)</td>
<td>Barbiturates, corticosteroids, adrenaline</td>
<td>Partial right hemiplegia, one seizure</td>
</tr>
<tr>
<td>Speach et al(^8)</td>
<td>30/M</td>
<td>Bee: single, location NR</td>
<td>&lt;1 hour</td>
<td>Decerebrate posturing, extensor plantar reflexes, left hemiparesis, hyporeflexia; after coma, patient had motor apraxia and left sensory neglect.</td>
<td>NR</td>
<td>Normal MRI and CT</td>
<td>IV adrenaline, methylprednisolone, diphenhydramine</td>
<td>Residual ideomotor apraxia</td>
</tr>
<tr>
<td>Bhat et al(^9)</td>
<td>35/M</td>
<td>Bee: multiple “all over the body”</td>
<td>&lt;1 day</td>
<td>Multiple swellings all over the body, vomiting, dysarthria, tinnitus, vertigo and swaying gait, hypertension, bilateral cerebellar signs, rhahdomyolysis with acute renal (respiratory?) failure.</td>
<td>No papilloedema</td>
<td>Bilateral cerebellar haemorrhagic infarct</td>
<td>Dexamethasone, antihistamines, mannitol, insulin, haemodialysis.</td>
<td>Deceased</td>
</tr>
<tr>
<td>Present case report</td>
<td>25/M</td>
<td>Bee sting in back of neck and body</td>
<td>1 day later</td>
<td>Left upper limb monoparesis, blurring of vision.</td>
<td>No papilloedema</td>
<td>MRI Brain-Infarct in right frontoparietal region, right occipital region. CT Brain-Right frontoparietal, right occipital hypodensities</td>
<td>Dexamethasone, antihistamines, mannitol, Aspirin,</td>
<td>Full recovery of monoparesis.</td>
</tr>
</tbody>
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NR (Not-Reported).
or immune response has also been postulated.5,6

Acknowledgement

We wish to thank the Teaching staff and Faculty of Poison control, Research and training Centre, Institute of Internal Medicine, Rajiv Gandhi Government General Hospital and Madras Medical College for their dedicated service to the patients and to Dr. Thirumalai Kolundu Subramanian, Formerly Director, Institute of Internal Medicine, for his valuable suggestions in the editing of this case report.

References


Fig. 3: Repeat CT-brain taken 8 months later reveals old infarct with gliotic changes in right high parietal and occipital region.