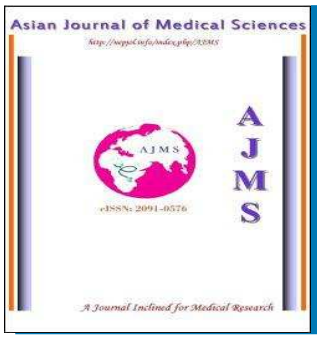

AN ACUTE ISCHEMIC STROKE AFTER MULTIPLE BEE STINGS IN AN ELDERLY PATIENT

CASE REPORT, Vol-4 No.2

Asian Journal of Medical Science, Volume-4(2013) http://nepjol.info/index.php/AJMS

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ABSTRACT

Although bee stings can cause local and systemic allergic reactions, neurological complications are rare. There are few reports of stroke following bee or wasp stings. We report a 70-year-old healthy man who developed a focal neurologic deficit 5 hours after multiple bee stings, which was confirmed to be an acute ischemic stroke on computed tomography (CT) scan.

Key Words: **Bee Sting, Ischemic Stroke, Computed Tomography**

"It is noteworthy to perform neurological examination to all patients with bee stings especially in elderly individuals"

INTRODUCTION

Bee sting envenomation is quite common worldwide. Local and systemic allergic reactions at the site of stings such as edema, erythema, burn-like sensation, pruritus, urticaria, and angio-edema are the most common manifestations of bee venom poisoning.[1] Symptoms like vomiting, diarrhea, dyspnea, oliguria, hypotension and collapse also have been described. Rarely vasculitis, serum sickness, neuritis and encephalitis have been reported which generally develop days to weeks after a sting.[2] Unusual and more severe complications may also be seen such as anaphylactic shock, myocardial infarction, acute renal failure, pulmonary hemorrhage, acute hemorrhagic pancreatitis, and atrial fibrillation.[3– 7] Neurologic complications are rare in bee venom poisoning. We report a case of an elderly man who developed focal neurologic deficits following multiple bee stings.

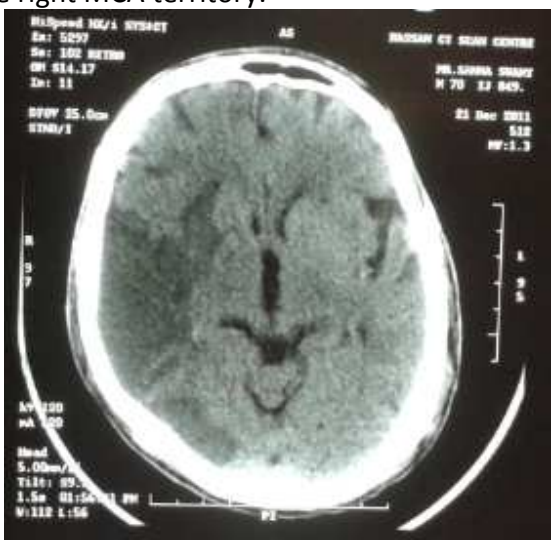
CASE REPORT

A 70 year old healthy gentleman reported to emergency department with history of sudden onset of weakness of left upper and lower limb after five hours of multiple bee stings. When he was working at the field he was attacked by 100–200 bees, and stung all over the body, Initially he developed itching, swelling and painful sensations at the venom stung sites, then he was treated with intravenous antihistamines and corticosteroids at a local hospital, later he experienced a severe headache with nausea and vomiting and developed left-sided upper and lower limb weakness with right facial deviation and got admitted in our hospital. There was urinary incontinence suggestive of UMN (Upper Motor Neuron) Type of bladder dysfunction. There was no history of seizures, altered sensorium, or any sensory symptoms. He was a non-smoker and not been suffered from diabetes or hypertension. There was no past history of any wasp or insect bites. Examination revealed his blood pressure of 136/70 mm Hg, He was conscious and had mild spastic dysarthria. There was left UMN 7th cranial nerve palsy along with motor power of grade II on left side and had left sided hyper-reflexic deep tendon jerks with extensor plantar response. An ocular examination revealed left homonymous hemianopia, with normal ocular fundoscopy. There was no carotid bruit or cardiac murmur. Rest of the systemic examination was normal. Past medical and surgical history was insignificant. Investigations revealed hemoglobin of 11.8 g/dl and total leukocyte count of 9,800/cubic-mm. Differential count revealed polymorphs-79% and lymphocytes-21%. Platelets were 2.8 x 10^5 cubic-mm. Prothrombin time and activated partial thromboplastin time were 14 seconds and 38 seconds (normal) respectively. Fasting blood sugar was 87 mg/dl. Lipid profile was normal (Serum cholesterol-180mg/dl, LDL (Low Density Lipoprotein)-151mg/dl, (Very Low Density Lipoprotein)-32mg/dl, (High Density Lipoprotein)-55mg/dl, triglycerides-190mg/dl). Renal and liver function tests were normal. Antinuclear antibody and VDRL were negative. ECG and chest X-ray were normal. Echocardiography and carotid doppler revealed no abnormality. Head computed tomography (CT) showed an ischemic infarct involving a large right MCA (Middle Territory Artery) territory (Fig.1) and revealed an acute infarct in the right parietal and temporal region, He was managed conservatively with anti-cerebral edema measures along with aspirin. Over the next five days, his motor power in the left limbs improved to grade III. However there was persistence of facial weakness. On follow up after two months, he showed remarkable motor power improvement to grade IV.

DISCUSSION

Bee sting envenomation is quite common. Various manifestations after multiple bee sting have been

Fig. 1
CT Scan showing an ischemic infarct involving a large right MCA territory.



described. Local reactions are very common. Systemic involvement such as vomiting, diarrhea, dyspnea, generalized edema, acute renal failure, myocardial infarction, and cerebral infarction are rare manifestations.[1–8] Seven cases of wasp and bee sting associated with cerebral ischemia have been reported in the english language literature. Reported neurologic complications include seizure, hemiparesis, aphasia, apraxia, dysarthria, ataxia, and coma.[9-11] The exact pathophysiology explaining the associated cerebral infarction is unknown. However the five major causes of cerebral ischemia are-vascular thrombosis, cerebral embolism, hypotension, hypertensive hemorrhage, and anoxia/hypoxia. Similar to acute myocardial infarction in bee stings, the vasoconstriction secondary to mediators released from the stings and aggravated by exogenous adrenaline and platelet aggregation, contributes to cerebral ischemia.[8] Bee venom itself contains histamine, thromboxane, leukotrienes, and other vasoactive inflammatory mediators. Both the thromboxane and leukotrienes have been shown to cause vasoconstriction resulting in cerebral infarction.[10] In addition, a neuro-pharmacological (sympathetic) mechanism of increased endothelial permeability involving the cerebral vasculature with a concurrent systemic thrombogenic or immune response has also been postulated.[12-16] Speach et al[14] described a case of an young male presented with left hemiparesis with predominant motor apraxia. Bhat et al[16] reported a 35 year old man presented with predominant cerebellar feartures with rhabdomyolysis causing renal failure and Maltzman et al[17] described the common characteristics such as acute to subacute onset of symptoms, moderate to severe visual loss followed by significant recovery which resulted in oedematous and haemorrhagic optic discs, and central or caecocentral scotomas. In our case the elderly patient was presented with features of ischemic stroke. The systemic immune mediated reactions to the bee stings causing vasoconstriction and a pro-thrombotic state was probably the cause to the present episode of stroke.

CONCLUSION

Therefore when an elderly patient presents to emergency department with history of multiple bee stings, we need to look carefully for any new onset of neurological symptoms and signs, thereby we can diagnose and treat the neurological deficits early. This case report strongly adds the high risk of stroke in the context of multiple bee stings in an elderly individual in whom age itself is a risk for cerebrovascular accidents.

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