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Case Report Open Access

Fatal Multi-Territory Cerebral Infarcts Following Multiple Hornet Stings in a Victim from Central Hills of Sri Lanka

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Abstract

Hornet stings are encountered frequently in clinical practice in Sri Lanka. Majority of patients sustain minor illness. However, common complication of anaphylactic shock and the rare acute kidney injury, multiple organ dysfunction and acute myocardial infarction have been reported. Most infrequently, ischemic stroke following hornet stings has been reported in scientific publications. In the foregoing case report, we discuss cerebral infarctions sustained for two days by a 42 years old female, who succumbed to the fatality.

Keywords: Lesser banded hornet; Sting; Stinger; Ischemic stroke; Vasoactivity; Thrombogenicity

Introduction

Lesser banded hornet (*Vespa affinis*) (Figure 1) is a common venomous hymenopteran that live in most parts of the tropical island of Sri Lanka [1]. Human encounter has been noted in plantation, cultivation and forest areas. They are aggressive upon disturbance and attack in swarms causing victims to sustain multiple stings. The sting is excruciatingly painful and the insect leaves the stinger (Figure 2). Attempts to remove stinger manually causes injection of further venom unless scraped out.

The majority of sufferers recover with minor illness. But fatal and non-fatal complications that arise from such stings include anaphylaxis [2], acute kidney injury [3,4], multiple organ dysfunction [5], myocardial infarction [6,7] and ischemic stroke [8-13]. Vasoactive, inflammatory, and thrombogenic peptides and amines, including histamine, leucotrienes, and thromboxane are responsible for the end organ complications [14,15]. The allergenic proteins such as phospholipases which elicit IgE responses, resulting in mast cell activation, underlie the anaphylaxis.

Case Report

A 42 years old previously healthy female was transferred to Teaching Hospital, Kandy in early December 2012, from a plantation sector hospital for further neurological management.

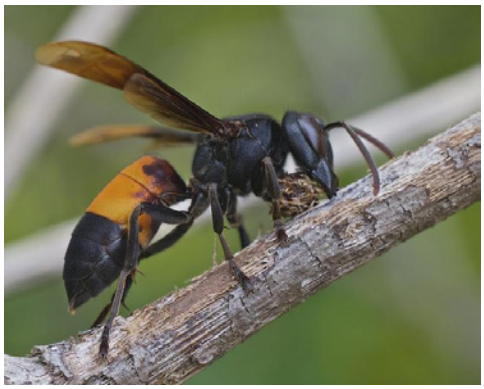

Figure 1: *Vespa affinis*, adult insect showing single yellow band in the abdomen.

Figure 2: *Vespa affinis*, showing stinger and length of adult insect.

The patient had been stung by about 50 hornets at the tea plantation where she was employed as a tea plucker. The stingers had been removed manually; the pain and itching had been treated with paracetamol (acetaminophen) and chlorpheniramine (antihistamine) at the local hospital and had been discharged without further management as she did not have significant complications.

She has suffered a single generalised tonic-clonic convulsion two days later, while at home. It has lasted two minutes, with tongue biting followed by postictal drowsiness for over thirty minutes. Upon recovery, she has had weakness of right arm and leg, but medical attention has been sought only after another three days. The teaching hospital received the patient three days after the onset of neurological disease, when the hornet stings were five days old.

The sting sites and the wound in the tongue had been healing at the time of admission but being weak on her right side, she required assistance to be mobile. Her speech, feeding and bladder and bowel

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functions were not affected. She did not have numbness of arms or legs. The patient did not complain of fever or cardiac, respiratory, alimentary and urinary symptoms.

The victim did not have a history of hypertension, diabetes, dyslipidemia, cardiac disease or neurological disease. Episodic wheezing had been treated locally. She had not been on any medication including oral contraceptive pills. Detailed inquiry did not reveal history of joint disease, skin rashes, deep vein thrombosis, pregnancy miscarriages, early morning passage of dark urine or any thromboembolic clues. She had not undergone surgical procedures or sustained trauma close to this event. She was a non-smoker and claimed no substance abuse and not a victim of passive smoking.

The family's medical history did not reveal thromboembolic disease or significant neurological and autoimmune rheumatoid disease.

The clinical examination did not reveal plethora, pallor, vasculitic skin rashes or joint disease. She had regular pulse and a blood pressure of 120/80 mmHg. There were no cardiac murmurs or vascular bruits. Respiratory, abdominal and locomotor examinations were unremarkable.

The conscious and well oriented patient had grade three weakness affecting right arm and leg with increased tendon reflexes and extensor plantar response in the same side. Her cranial nerve, cerebellar and sensory examinations revealed no defect as did fundi.

Both plain and contrast enhanced computerised tomography revealed infarcts in left internal capsule, bilateral deep parietal grey matter and both occipital lobes. The electrocardiograph showed sinus tachycardia only. Chest radiography and two dimensional echocardiography did not reveal cardiac disease.

The complete blood count, renal and hepatic biochemistry were without abnormality and her erythrocyte sedimentation rate was 23 mm in the first hour. C-reactive protein had been 33.4 mg/dl (ref: 0.0-5.0 mg/dl). She had a normal serum lipid profile and a homocystein level with the antinuclear antibodies remaining negative. She was managed symptomatically while being investigated for possible remediable aetiologies and to exclude causes other than hornet stinging.

On the third day at the teaching hospital- the eighth day of hornet attack- she suddenly became drowsy with a declining Glasgow coma scale and a respiratory embarrassment forcing her to be transferred to the medical intensive care unit. The clinical impression was of new infarcts in the brain stem or cerebral oedema due to infarcts elsewhere. Repeated plain computerized tomogram and biochemistry did not show new or significant finding. Despite valiant attempts to save life, the patient succumbed to her fatality.

The pathological post-mortem conducted by the Senior Consultant Judicial Medical Officer revealed multiple cerebral and brain stem infarcts (some of which were not clear in the tomograms) with minimal systemic atherosclerosis. Autopsy did not reveal any other aetiologically significant findings.

Discussion

Hornet venom contains vasoactive, inflammatory, and thrombogenic peptides and amines, including histamine, leukotrienes, and thromboxane. The venom also contains allergenic proteins such as phospholipases which elicit an IgE response, resulting in mast cell activation which is the hallmark of anaphylaxis [2,14,15]. The reaction to hornet stings can be local or systemic as well as immediate or delayed. Pain, wheal, flare, oedema and swelling, which are generally self-limiting, constitute local disease. Multiple stings can lead to vomiting, diarrhoea, generalized oedema, dyspnoea, and hypotension.

Severe systemic complications include anaphylaxis which constitutes the leading cause of death [2] acute kidney injury [3,4], multiple organ dysfunctions [5], disseminated intravascular coagulation, myocardial infarction [6,7], Rhabdomyolysis [16] and neurological disease [8-13].

Numerous neurological complications include ischemic stroke, venous sinus thrombosis [12], ocular myasthenia gravis [17] and thrombotic thrombocytopenic purpura [10].

The proposed underlying mechanism of early ischemic stroke includes hypotension of anaphylaxis and vasospasm due to treatment with adrenaline. The delayed phenomena leading to infarctions appear to be spasms caused by vasoactive substances and thrombosis induced by thrombogenic factors in venom.

The latter mechanism is the likely sequel in this patient, as neither has she had anaphylaxis nor was she treated with adrenaline to account for the multiple infarctions occurred between two to eight days after multiple hornets stinging.

In a middle aged woman as in this case, it is important to exclude other possible aetiologies that would have given rise to or precipitated a stroke in the face of stresses following multiple stings. We could investigate and exclude diabetes, hypertension, hyperlipidaemia systemic lupus erythematosus, homocystinuria and cardiac sources. Further assessment with thrombophilic and antiphospholipid antibody screening, tests for paroxysmal nocturnal haemoglobinuria, magnetic resonance imaging for multiple sclerosis and cerebral angiitis, though there were no clinical clues, were planned but untimely demise of the patient prevented such investigations.

The other neurological complications of stings which have been reported are individual case reports of ocular myasthenia gravis, optic neuritis, limb numbness, trigeminal neuralgia and encephalopathy [10]. Postulated mechanisms include both toxic effect of venom and hypersensitivity to venom.

In the case of acute myocardial infarction following hornet stings, the postulated mechanism is a combination of coronary vasoconstriction and platelet aggregation secondary to mediators released after wasp sting, aggravated by exogenous adrenaline given as part of the treatment.

This unusual fatal sequel in this case discussion signifies necessity to anticipate and treat neurological outcomes following hornet stinging. The delayed onset of vascular event poses a challenge to the clinician and individual susceptibility for such vascular phenomena which needs further scientific research.

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