Case Report

Multiple cerebral infarctions with severe multi-organ dysfunction following multiple wasp stings

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Abstract

Wasp and bee sting are commonly encountered worldwide. Local reactions are more common, generally are self-limiting and settle within a few hours. Multiple stings can lead to various clinical manifestations like vomiting, diarrhea, dyspnea, generalized edema, hypotension, syncope, acute renal failure, and even death. Rarely, they can cause vasculitis, serum sickness, neuritis, and encephalitis. We are reporting a case of 40-year-old male who presented with stroke, right hemiparesis with severe multi-organ dysfunction due to multiple wasp stings.

Key Words

Disseminated Intravascular Coagulation, stroke, wasp sting

Introduction

Wasps (vespidae) belong to the order of hymenoptera along with the bees (apidae), hornets (vespidae), and the ants (formicidae). Nearly 100 million cases of hymenoptera stings are reported every year throughout the world.[1] 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.[2] Stroke after a massive attack of bees is a rare complication. Other reported neurological complication of venomous honey bee included seizure, aphasia, dysarthria, apraxia, ataxia, and coma. There are only handful cases reported in the world literature.[3] We reported a case of multiple wasp stings presented with stroke, Disseminated Intravascular Coagulation, and Acute Renal Failure.

Case Report

A 40-year-old male, normotensive, non-diabetic, reported was stung by 50-60 wasps all over the body including face, head, and neck region. He was complaining of redness, swelling, and pain all over the body. He was treated at peripheral hospital with chlorpheneramine and Hydrocortisone injection. He remained stable for next 16-18 h, after which he developed rapid progressive deterioration in consciousness and became unresponsive within a period of next 4-6 h with associated history of vomiting and incontinence of urine. There was no history of headache, fever, breathlessness, trauma, abnormal body movement, drug intake, bleeding from any site, and jaundice. He was then brought to our emergency, where his initial assessment showed Glasgow Coma Scale of 6/15 with Blood Pressure of 170/100 mmHg. He was febrile to touch, multiple wasp sting marks [Figure 1] with swelling of right upper limb (a total of 73 sting marks), moving left side of body on deep painful stimuli, gaze preference towards left side and with bilateral extensor response.

A provisional diagnosis of stroke after multiple wasp sting was made, baseline laboratory investigation [Table 1] (complete blood count, renal and liver function tests, arterial blood gas analysis, electrocardiograph, x-ray chest, blood glucose, electrolytes, urine examination, Creatine Phosphokinase/ Lactate Dehydrogenase, and coagulogram) were sent along with Non contrast Computed Tomography of head and was started on i.v hydrocortisone, i.v avil, and i.v antibiotics along with supportive measures.
Non contrast Computed Tomography of head was suggestive of hypodense areas in left thalamic region and occipital area. Later, further investigation like Fibrin degradation product, D-Dimer, and C-reactive protein was done which were also positive and next day magnetic resonance imaging of brain with diffusion weighted images was performed which revealed multiple hyperintense lesions in cortical and subcortical areas with loss of grey white contrast in both cerebral hemispheres, pons bilateral thalami, and left parieto-occipital region with evidence of marked diffusion restriction [Figure 2].

His ultrasound abdomen, X-ray chest, electrocardiograph, electrolytes, arterial blood gases, liver function tests, urine examination was normal and antinuclear antibody, anti-double stranded DNA was negative.

After 4 days, his NCCT head was repeated which showed hypodensities in left thalamic, left parieto-occipital, bilateral cerebellar hemispheres, and in pontine region.

During the hospital stay of 23 days, he remained hemodynamically stable but his clinical condition did not improve much and was finally discharged in a vegetative state with GCS of 5/15 on oral steroids and antibiotics.

**Discussion**

Worldwide annual incidence of immunological reaction to hymenoptera bee stings ranges from 0.3% to 3%. Yellow jacket, hornets, and some wasps [Figure 3] attack even without provocation in contrast to the honey and bumble bees. The bees generally lose their stinging apparatuses during stinging and subsequently die. Sting of the vespidae family can remain attached to the stung site, continuing to inject venom unless manually removed.[1]

Phisalix mentioned that the amount of venom injected by a single bee sting is to be 0.33 mg. It contains a variety of amines, peptides, and enzymes that cause various clinical manifestations. Direct toxic effect of bee venom are mediated by polypeptide toxins (mellitin), which damages cell membranes and hyaluronidase, phospholipase enzymes, and other compounds such as histamine, serotonin, thromboxane, and leucotrienes.[3]
Bee and wasp venoms are different, each containing distinct major allergens, which are well defined. Phospholipase A2 and mellitin occur only in bee venom, and antigen 5 only in wasp venom, but both venoms contain hyaluronidases. Patients allergic to wasp venom are rarely allergic to bee venom.[4]

Uncomplicated stings cause pain, erythema, pallor, urticaria, numbness, tingling, sweating, and weakness, which subside within few hours whereas systemic symptoms are angina, hypotension, syncope, cough, respiratory failure, diarrhea, vomiting, dysphagia, convulsions unconsciousness, and even death.[3]

Several cases of acute or delayed stroke having occurred in the setting of anaphylactic reaction due to hymenoptera sting have been reported to date[5] and usually the onset of clinical feature of stroke is within 30 s to 96 h of the sting.[1]

Different pathogenic mechanisms were proposed. First, hypotension is a constant part of anaphylactic reaction due to a sting of hymenopteras. The hypotension is induced by histamine and Prostaglandin 2 and can proceed up to vascular collapse.

The second pathogenic determinant is vasoconstriction induced by histamine. The vasoconstriction after exposure to histamine, due to the H1 receptor activation or thromboxane was described in the human coronary arteries. The effect of vasoactive substances producing vasoconstriction in cerebral vessel is known from reversible cerebral vasoconstriction syndrome.

Finally, the third pathogenic factor is the thrombogenic effect of thromboxane, leukotrienes, and other substances contained in the venom of hymenopteras.[5]

The other neurological complications of stings which have been reported are individual cases of ocular myasthenia gravis, optic neuritis, limb numbness, and trigeminal neuralgia and three cases of encephalopathy, one of which was fatal.[5]

Acute renal failure is an unusual complication of wasp stings. Although acute renal failure after wasp stings is typically caused by acute tubular necrosis in the setting of hemolysis or rhabdomyolysis, in some patients, acute renal failure may result from a direct nephrotoxic effect or acute interstitial nephritis from a hypersensitivity reaction.[7]

The patient reported here had acute life-threatening multi-systemic involvement that included stroke, DIC, and renal failure which were evident with brain images, positive FDP, D-Dimer, C-reactive protein increased CPK/LDH, and deranged renal parameters. We postulated that tissue damage caused by wasp venom was the possible reason for development of multi-systemic injury.

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