Hypoxic brain injury with unilateral hemispheric cortical involvement following multiple wasp stings

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Abstract

The most frequently reported neurological complication of a wasp sting is ischemic stroke. We treated a patient with wasp sting with unusual complications. A 52-year-old man was hospitalized for anaphylactic shock after multiple wasp stings. Although the patient recovered consciousness after 2 days, he had global aphasia and right hemiparesis. Brain magnetic resonance imaging and angiography revealed high-intensity signals in the left basal ganglia and cerebral cortex and stenosis of the left middle cerebral artery. After 2 days, the middle cerebral artery stenosis improved. After 5 days, diffusion-weighted imaging showed an enlarged lesion in the left frontal cortex. The infarct in this case was due to a predominantly unilateral vasoconstrictive hypoxic brain injury from wasp stings.

Keywords: Allergic reaction, hypoxic brain injury, neurological complication, wasp sting

INTRODUCTION

Bee stings are common causes of local allergic reactions, including edema, erythema, burn-like sensations, pruritus, and urticaria. In contrast, wasp stings can cause serious local allergies and severe systemic allergic reactions, including anaphylactic shock and even death. Although few neurological complications are associated with wasp stings, the stings can seriously damage the central and peripheral nervous systems. The reported complications of wasp stings range from simple headaches and dizziness to ischemic stroke, encephalitis, peripheral neuritis, and optic neuritis. Rare complications include myasthenia gravis, acute inflammatory demyelinating polyradiculoneuropathy, and acute demyelinating encephalomyelitis. The neurological complications of wasp stings have diverse mechanisms, which are unclear. We encountered a patient with right hemiparesis and global aphasia after multiple wasp stings. Brain magnetic resonance imaging and angiography revealed high intensity signals in the left basal ganglia and cerebral cortex, and we believe the infarct was due to a predominantly unilateral vasoconstrictive hypoxic brain injury from wasp stings.

CASE REPORT

A 52-year-old man who had experienced bee sting allergic reactions at least 4 times in the past was hospitalized for anaphylactic shock after being stung by wasps on multiple body areas, including the left neck, face, scalp, and upper extremities (Figure 1A). He visited a local hospital 15 min after being stung. The patient suddenly showed decreased consciousness, low blood pressure, and dyspnea due to airway edema. The patient had no known risk factors for stroke. The patient was given epinephrine and intravenous fluids, and his airway was established. He was then referred to our hospital. Upon arrival (2.5 h post-sting), the patient was comatose (Glasgow Coma Scale score, 6), with normal brainstem reflexes. Brain computed tomography (CT) revealed no abnormalities. Emergency department blood tests revealed a prolonged prothrombin time (16.4s) and activated partial thromboplastin time (87.7s). Both the fibrinogen degradation product (10.5 μg/mL) and D-dimer (1,323 ng/mL) levels were increased. His eosinophil count was 150 cells/mm³, and his CAP-tryptase levels were less than 200 μg/L. The patient was diagnosed with allergic reaction-mediated shock and hospitalized for treatment. The patient recovered consciousness after 2 days of hospitalization. A subsequent neurological
examination by a neurologist confirmed the global aphasia and right hemiparesis.

Magnetic resonance diffusion weighted imaging (DWI) of the brain showed high-intensity signals in parts of the left basal ganglia and cerebral cortex (Figure 1B), and angiography revealed stenosis of the superior branch of the left middle cerebral artery (MCA) (Figure 1C). However, no specific source of the embolism was found on electrocardiography, transthoracic echography, or cardiac CT and neck angiography. In the follow-up CT angiography performed 2 days later, the previously observed left MCA stenosis was improved (Figure 1D). In DWI performed 5 days later, larger lesions were observed in the left hemisphere and multiple lesions were observed in the right hemisphere (caudate, lentiform nucleus, and cortex) (Figure 1E). The patient was discharged after the global aphasia partially recovered and the right hemiparesis improved from a Motor Research Council grade 2 to grade 3.

At a follow-up outpatient examination conducted after 1 month, high-intensity signals were still observed in the previous lesion area in fluid-attenuated inversion recovery images (Figure 1F). The patient’s neurological symptoms had not changed from the time of hospital discharge.

DISCUSSION

The incidence of wasp sting-induced systemic reactions is 3–4% of the total sting cases reported in the US and Europe. Most of the neurological symptoms caused by wasp stings occur in young children. Although most cases recover after steroid treatment, neurological sequelae persist in rare cases.

The mechanisms of anaphylactic shock include extravasation of intravascular volume and reduction in venous tonicity, which subsequently causes a precipitous decrease in blood pressure. The discordant autoregulation of cerebral perfusion suggests an ischemic insult during anaphylactic shock. During anaphylaxis, factor V and VIII are markedly decreased and fibrinogen is reduced. The consumption of these coagulation factors is consistent with a concomitant thrombotic process. Several hypotheses go towards explaining a wasp sting-induced ischemic stroke. First, the wasp sting toxin contains histamine, leukotrienes,
and thromboxane, which can directly cause vasoconstriction, inflammation, and thrombosis. A wasp sting can cause vasoconstriction and platelet aggregation and result in cerebral infarction\(^5\). Second, wasp stings on the neck and head stimulate the ipsilateral superior cervical ganglion, which increases the endothelial permeability of the distal internal carotid artery (ICA), which can cause platelet activation, thrombosis, and cerebral infarction\(^6\).

In the present case, the cortical lesion was mainly in the left hemisphere and in a wide area of ICA territory. Follow-up CT angiography showed that the left MCA stenosis detected on the initial angiography improved after 2 days. We hypothesized that the wasp stings on the left side of the face and multiple points on the neck stimulated the left superior cervical ganglion, which induced thrombosis by increasing local vasoconstriction of the left distal ICA. In addition, the systemic allergic reaction that caused hypotension might have aggravated the hypoperfusion in both hemispheres, but mainly in the left hemisphere. The left distal ICA vasoconstriction made the left hemisphere vulnerable, which resulted in hypoxic damage to the left side. This pattern differed slightly from typical hypoxic encephalopathy, which commonly exhibits lesions in the cortex in both hemispheres and basal ganglia. Although extra/intracranial angiography was not performed upon hospital admittance, vasoconstriction was likely because of the improvements in the left MCA stenosis seen after 2 days. In addition, distal ICA vasoconstriction was likely because the lesions were not limited to the MCA territory but were distributed across the left hemisphere.

A few studies report stroke occurring after a wasp sting. However, there has been no report on the reversible changes seen on angiography. This patient’s hypoxic brain injury that was caused by multiple wasp stings had the peculiar pattern of being dominant one side, unlike general hypoxic brain damage. This might have been caused by a decrease in blood pressure and hypoxia that was dominant over the left hemisphere, resulting from an allergic reaction with focal vasoconstriction. Stimulation of the superior cervical ganglion from the multiple wasp stings might partly explain the characteristics of this case.

REFERENCES