Bilateral Cerebellar Infarction: A Rare Complication of Scorpion Sting

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Abstract
Introduction: Complications following scorpion sting are common in India and can be fatal. Stroke following scorpion sting is a rare complication and can occur by various mechanisms such as hypertension, hypotension, DIC, myocarditis and venom-induced vasculitis. We present a rare case of extensive cerebellar infarction following scorpion sting, which has rarely been reported in medical literature.

Objectives: ●To study the clinical profile of two patients presenting with an acute onset of cerebellar symptoms following a scorpion sting. ●To evaluate the possible causes of the stroke and to study the relation of their symptoms to the scorpion sting.

Methods: Two young women presented with a history of acute onset of dysarthria, ataxia and incoordination following scorpion sting. They did not have any known risk factors for stroke. They had cerebellar type of dysarthria and cerebellar signs on both sides along with incoordination. A CT-scan of the brain showed bilateral extensive cerebellar infarctions. They were investigated for other causes of stroke without any positive results. With treatment the patients made a gradual but complete recovery.

Conclusion: Since there was no evidence of hypertension, hypotension, myocarditis or disseminated intravascular coagulation, we can conclude that the patients had suffered a thrombotic stroke caused by the vasculotoxic action of the scorpion venom.

INTRODUCTION

Scorpion sting and its various manifestations are a common occurrence in India, and especially so in Bellary, which has one of the highest incidence of this condition. About 310 cases of scorpion sting are reported every year at our institute, with a mortality rate of 1.5%, with a peak incidence during the summer months. Among the 80 species of scorpions in India, Mesobuthus tamulus and heterometrus swammer dami (formerly Palamneus) are of medical importance and are common in our region. Systemic complications are commonly associated with Mesobuthus tamulus (Indian red scorpion). The scorpion venom is a water-soluble, antigenic, complex mixture of neurotoxin, cardiotoxin, nephrotoxin, hemolysins, phosphodiesterases, phospholipases, hyaluronidases, histamine, serotonin and other chemicals.1 By opening sodium channels at presynaptic nerve terminals and inhibiting calcium dependent potassium channels, the neurotoxin, which is the most potent of the toxins, can cause continuous, prolonged, repetitive firing of the somatic, sympathetic and parasympathetic neurons.2 Systemic complications are not uncommon but CNS complications are rare, comprising only 2% of all complications. In 1928, pulmonary edema following scorpion sting was reported for the first time from Bellary by a British physician. In this communication we present two interesting cases of cerebellar infarction, which is an exceedingly rare complication.

CASE HISTORY

A twenty eight year old lady presented with a history of acute onset of slurring of speech, unsteadiness of gait and incoordination following a scorpion sting. It was associated with burning pain at the site and was followed by chills and sweating lasting for an hour. She was asymptomatic until 4-6 hours later when she had 2-3 episodes of projectile vomiting following which she retired to bed. On waking up 6 hours later, the patient noticed slurring of speech and unsteadiness of gait along with incoordination. She could not sit or stand without support and was unable to do any tasks. She complained of swaying to either side. She did not complain of visual disturbances, sensory disturbances or weakness of her limbs. There was no history to suggest cranial nerve involvement or seizures. The patient gradually became
drowsy over a period of six hours and was admitted on the same day. She did not have any known risk factors for stroke or a family history of premature cardiac or cerebrovascular accidents. There was no history of fever, bleeding tendencies, chest pain, palpitations or dyspnea. Her previous medical history was unremarkable.

At admission, her vitals were stable and there was no evidence of autonomic disturbances. General physical examination revealed pallor. Her cardiac, respiratory and abdomen examination were normal.

Neurological examination: Patient was conscious with a GCS of 13/15 (3+6+4). Speech was slurred and of cerebellar type. Pupils were normal and equal in size, and reactive to light. Cranial nerve examination revealed bilateral papilledema. Nystagmus was absent. Hypotonia and hyporeflexia were noted in all the limbs with normal power. Plantar reflex was of extensor type on both sides. She had truncal and limb ataxia with swaying to both sides. There were no meningeal signs, sensory deficits or sphincter disturbances.

Investigations: Hemogram was normal except for mild degree of normocytic hypochromic anemia (Hb= 10.2 g/dl). Blood sugar levels, renal and liver function tests, lipid profile and serum electrolytes were normal. Coagulation profile, homocysteine levels and Protein C and S levels were normal. Urinalysis was normal. Electrocardiogram, chest x-ray and ultrasound examination of abdomen and pelvis were normal. A 2-D echocardiography and color Doppler study of her heart, both carotid and vertebral arteries did not reveal any abnormality. Cerebrospinal fluid analysis done at the time of discharge was normal. She tested negative for HIV, VDRL, ANA and antiphospholipid antibody. A contrast enhanced CT scan of the brain on the day of admission showed multiple hypodense lesions of 14-20 HU in both cerebellar hemispheres without any areas of hemorrhage (Fig. 1). The lesions did not enhance with contrast suggesting multiple cerebellar infarcts. There was perilesional edema which caused pressure effect on the fourth ventricle leading to minimal obstructive hydrocephalus (Fig. 2).

Treatment: A neurosurgical opinion was taken and it was decided to manage the patient conservatively. She was managed with mannitol, aspirin and supportive therapy. Antivenom to scorpion sting could not be administered as it is not available in India. Over a period of ten days there was a gradual improvement in her deficits. At the time of discharge, patient still had mild dysarthria and unsteadiness of gait, but was able to walk without support and do her routine activities without any help. A follow-up examination fifteen days later showed that the patient had recovered fully and had resumed her household activities.

Three months later, a 30 years lady presented to us with a history of scorpion sting followed 6-8 hours later by slurring of speech which was gradual in onset and progressed over 2-3 hours. The scorpion sting was not associated with features of autonomic storm or cardiac symptoms. About 12 hours later patient noticed unsteadiness while walking, which progressed to a degree where the patient was unable to sit or stand without support. There was associated limb ataxia but no weakness or alteration in the sensorium. She did not have any significant past history and there were no risk factors for stroke.

Examination revealed a normal heart and carotids and bilateral cerebellar signs. There was no evidence of weakness or raised intracranial tension. A contrast enhanced CT scan of the brain showed extensive areas of infarction involving both cerebellar hemispheres (Fig.
Table 1: Systemic complications following scorpion sting

<table>
<thead>
<tr>
<th>Complication</th>
<th>Description</th>
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<tbody>
<tr>
<td>Autonomic storm</td>
<td>Disseminated intravascular coagulation</td>
</tr>
<tr>
<td>Allergic reactions including anaphylaxis</td>
<td>Dyselectrolytemia</td>
</tr>
<tr>
<td>Acute pancreatitis</td>
<td>Encephalopathy^2</td>
</tr>
<tr>
<td>Acute hepatic injury</td>
<td>Myocarditis with pulmonary edema</td>
</tr>
<tr>
<td>Acute renal failure^1</td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Cerebrovascular accidents</td>
<td>SIRS^3</td>
</tr>
</tbody>
</table>

1 = Acute renal failure may occur due to hypotension or acute tubular necrosis due to toxin or rhabdomyolysis.
2 = Encephalopathy can also result from the autonomic storm because of metabolic disturbances and hypertension.
3 = The venom can initiate a systemic inflammatory response mediated by various cytokines like IFN-α and IL-6.

3). All other investigations were within normal limits and the stroke work-up was not helpful. With antiedema measures and antiplatelet agents the patient made a gradual and complete recovery over a period of 8 days. A repeat CT did not show any areas of infarction and the patient was discharged without any deficits.

**DISCUSSION**

Though local symptoms are the commonest manifestation following a scorpion sting, various systemic complications can ensue (Table 1). Central nervous system complications are very rare and may present in either of two forms, both of which are associated with high mortality rates.

1. Encephalopathy: the venom can be directly neurotoxic resulting in seizures and encephalopathy. This is more common in children.
2. Cerebrovascular accidents: numerous mechanisms have been proposed to explain the occurrence of strokes in patients with scorpion sting.
   - An acute rise in the blood pressure during the autonomic storm may rupture unprotected or diseased vessels, especially the perforating arteries resulting in hemorrhagic stroke.^3

- Toxic myocarditis may precipitate arrhythmias that may give rise to embolic stroke. Changes in the blood coagulation profile may play a contributory role.
- Stroke can occur due to disseminated intravascular coagulation. This has been confirmed by the demonstration of fibrin deposits in the affected vessels in autopsy studies of victims of scorpion sting. The venom is also known to increase platelet aggregation.
- The venom is vasculotoxic with the ability to damage endothelial cells and cause vasculitis. This can initiate thrombosis.^4
- Watershed infarcts can result from the hypotension that may occur due to myocarditis, parasympathetic overactivity and dehydration.^5
- Catecholamine excess, with firing of alpha receptors, enhances endothelin secretion leading to severe vasoconstriction of the cerebral vessels. This can result in low flow infarcts.

The patients under discussion presented with extensive infarction of both cerebellar hemispheres which is extremely rare. Not only is this a rare complication, the outcome was equally uncommon considering the site and extent of the infarction. There was history suggestive of a brief autonomic storm, but there was no evidence of hypertension, hypotension, myocarditis or disseminated intravascular coagulation. With the available information we can conclude that the patients had suffered a thrombotic stroke caused by endothelial damage resulting from the vasculotoxic action of the venom.

**REFERENCES**