Case Report

Lateral medullary syndrome after a scorpion sting

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Abstract

Scorpion bites are a common problem in Southern parts of India. The sting of *Mesobuthus tamulus* belonging to the *Buthidae* family is known for being fatal. The toxidrome of scorpion sting is known for its effect on the cardiovascular system, and there have been rare reports of cerebrovascular accidents as well. We describe a case of lateral medullary syndrome secondary to scorpion sting. As per the knowledge of the authors, this is the first case report of the same.

Keywords: Scorpion sting, stroke, toxin-induced thrombosis

Introduction

India is known to harbor 86 species of scorpion. Of these, the *Mesobuthus tamulus* and *Palamneus swammerdami* are of clinical importance. The annual sting rate world over as reported by Chippaux *et al.* was 1.23 million. These scorpions reside in dry and arid regions in India. Areas endemic for scorpion sting are Gujarat, Maharashtra, Tamil Nadu, Andhra Pradesh, and Karnataka. The case fatality due to scorpion stings has dramatically reduced due to drugs such as prazosin and antiscorpion venom. However, in rural areas of India, it continues to be a major medical problem.

Case Report

A 45-year-old manual laborer, from the Kadapa region of Andhra Pradesh, presented with a history of a scorpion sting on his left foot. Immediately following the sting, he had excruciating pain over the sting site. He was then taken to a primary health facility where he was found to have high blood pressures and was treated with prazosin and was kept under observation. He did not have any breathing difficulty, bleeding, or fever on the 1st day. On the 2nd day of admission, he was noted to have drooling of saliva with inability to swallow saliva and had an inability to get up from the bed. He was not a smoker, diabetic, hypertensive, or dyslipidemic, and he did not have any other risk factors for young hypertension or stroke.

At this point of time, he complained of ptosis of his right eye, decrease sensation over the right side of face along with instability, and swaying to the right side. In view of new onset focal neurological deficit, in spite of having a normal blood pressure, he was referred to a higher center. On examination, at the time of presentation, he was conscious and oriented, and his vitals were stable. He had a complete Horner's syndrome on the right side of his face, with decreased pain and temperature sensation over the right side of his face. He had a deviation of the uvula to the left and had dysdiadokokinesia and past-pointing involving his right upper and lower limb. His other system examinations were normal. His complete blood counts, renal function, and electrocardiogram were normal.

A clinical diagnosis of a lateral medullary syndrome was made and a magnetic resonance imaging with magnetic resonance angiogram of the brain was done. It revealed features of the right lateral medullary infarct with the right vertebral artery thrombosis [Figures 1-6]. Evaluation for prothrombotic states

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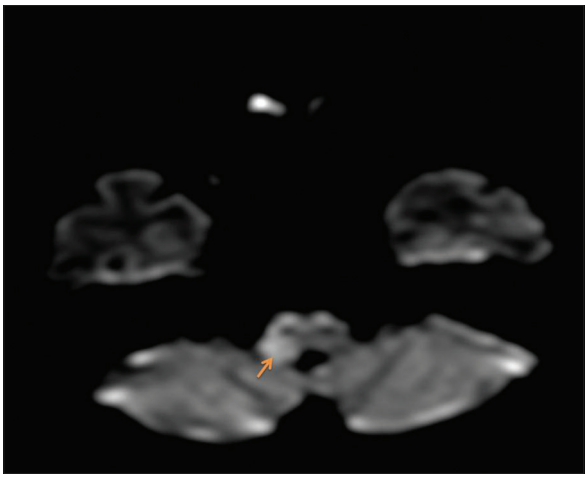

Figure 1: Diffusion-weighted imaging image

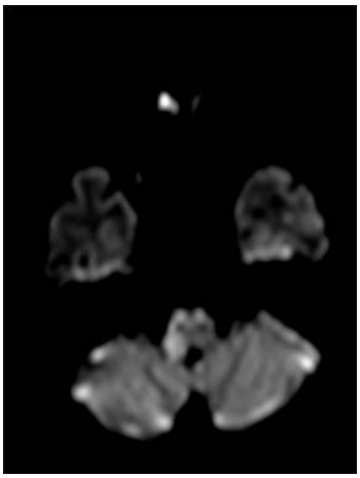

Figure 2: Diffusion-weighted imaging image of infarct region

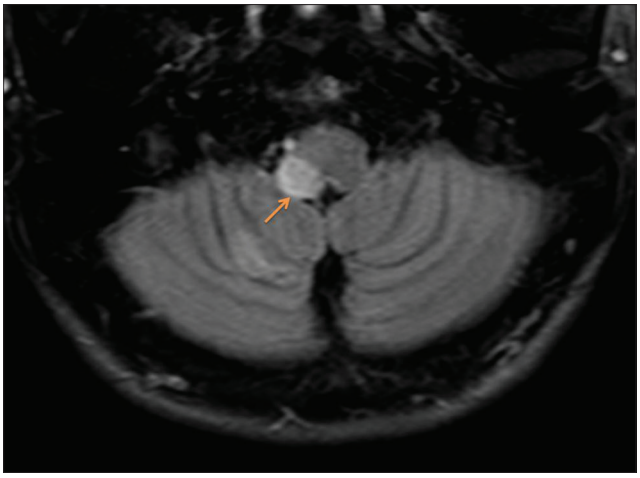

Figure 3: Fluid attenuation inversion recovery images of infarcted region

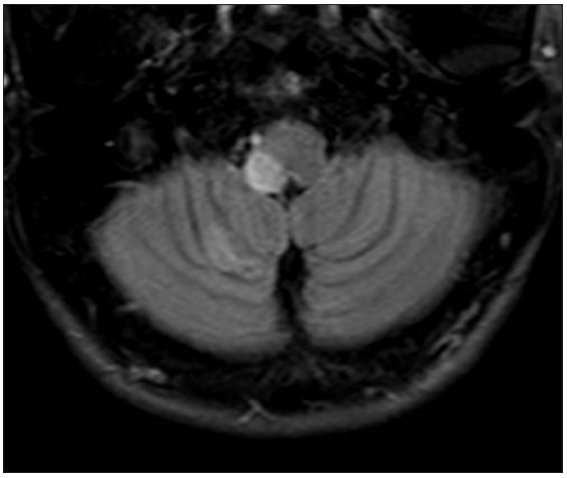

Figure 4: Fluid attenuation inversion recovery image without arrow

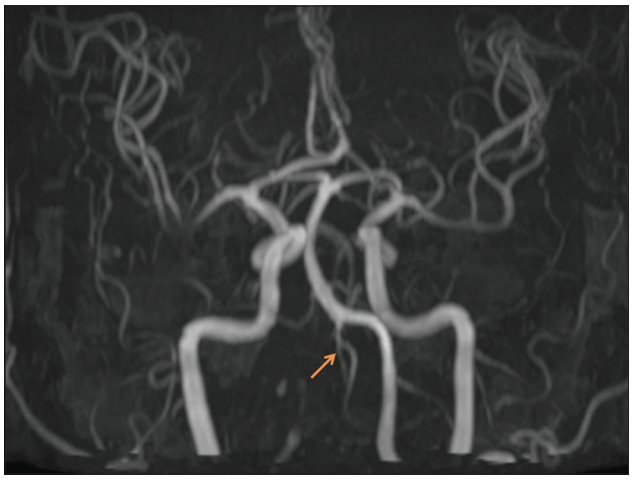

Figure 5: Original magnetic resonance angiogram image

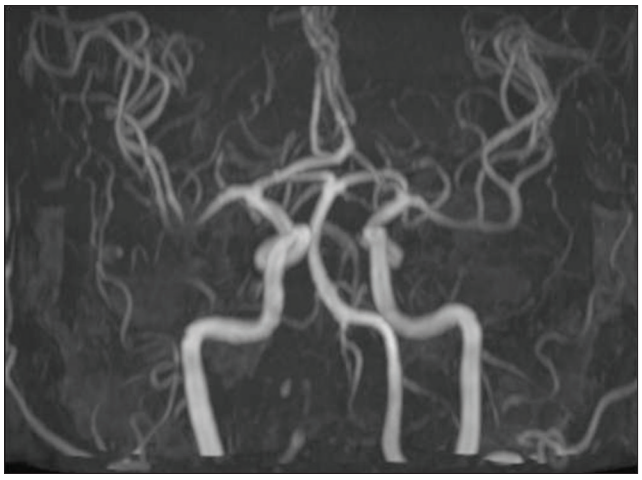

Figure 6: Magnetic resonance angiogram image of patent posterior inferior cerebellar artery

including antinuclear antibody, antiphospholipid antibodies, anticardiolipin antibody, and homocysteine levels was normal. His bleeding parameters and peripheral smears ruled out disseminated intravascular coagulation. His echocardiogram was normal as well. In view of the lack of other causes for a thrombotic event and the temporal relation and progression of events, he was diagnosed to have a scorpion sting-induced thrombosis of the posterior inferior cerebellar artery causing a lateral medullary

syndrome. He was started on antiplatelet medications and showed a gradual improvement with physiotherapy.

Discussion

Scorpion sting envenomation has been deemed to be more poisonous than certain snake bites.[1] However, just like snake bites, certain scorpion stings can be dry. They are known to be fatal in 2%–4% cases in India today. This small proportion of fatalities is due to the discovery of drugs such as prazosin, antiscorpion venom, and better intensive care facilities.[2]

Scorpion stings have been known to cause excitatory responses in the autonomic nervous system, leading to the sudden alterations in blood pressure and heart rate. Scorpion venom consists of sodium and potassium channel-directed toxins that lead to prolonged depolarization and hence excitation of autonomic nerves and the release of neurotransmitters. Hence, the toxidrome is aptly called an "autonomic storm." The other systemic manifestations are predominantly mediated through this autonomic excitation and directly by the toxin. Patients can present with bradycardia and excessive salivation secondary to parasympathetic stimulation. This can be followed by more prolonged sympathetic stimulation which can cause tachycardia, hypertension, and pulmonary edema.

Central nervous system involvement secondary to scorpion sting has been described in 2%–8% of patients with complications. Anoxia due to cardiovascular complications can cause central nervous system manifestations such as coma, seizures, and hypoxic ischemic encephalopathy.[3-10]

Case series from India have shown that only 7%–8% of patients with scorpion stings present with cerebrovascular accidents. Of these, only 2%–4% present with thrombotic strokes.[4,5] The various mechanisms proposed have been:

1. Alterations in coagulation of blood leading to disseminated intravascular coagulation[4]
2. High level of catecholamines induced, vasospasm causing hypoperfusion, and ischemia in previously compromised areas of the brain[3,6,7]
3. High blood pressure during autonomic storm resulting in rupture of vessels, causing hemorrhagic stroke[8-10]
4. The presence of myocarditis, thromboembolic phenomenon, or shock leading to cerebral infarction.[6,9,10]

The direct effect of toxin either leading to endothelial damage and vasculitis or resulting in prothrombotic state is yet to be established.

The mean duration to develop such complications, especially thrombotic events, is considered 2–3 days after the scorpion sting. These infarcts are seen as low-flow infarcts in imaging as early as 36 h.[6] The most common territory reported to be involved in patients with infarcts is middle cerebral artery and basal ganglia in case of hemorrhagic strokes.[10] Prompt diagnosis and early administration of alpha receptor blockers may prevent such events. However, in those with thrombotic complications, little is known about management and prevention of such complications. Thus, the physician must be aware regarding these rare central nervous system manifestations of scorpion sting while evaluating patients.

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Conflicts of interest

There are no conflicts of interest.

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