

Symmetrical thalamic and cerebellar hemorrhages following scorpion envenomation

Tekke Praveen Kumar¹, V Umamaheswara Reddy², Putcha Deekshanthi Narayan³, Amit Agrawal⁴

ABSTRACT

Clinical manifestations following scorpion envenomation are a result of autonomic outburst and direct actions of toxin on various systems. Involvement of central nervous system (CNS) following scorpion envenomation is rare. Encephalopathy, cerebral edema, subarachnoid hemorrhage, nonhemorrhagic strokes, and cortical necrosis are a few CNS complications described in the medical literature due to scorpion envenomation. We report a rare case of scorpion envenomation in which patient had symmetrical hemorrhages in the thalamus and cerebellum.

Key words: Cerebellar hemorrhage, scorpion envenomation, scorpion sting, scorpion toxin thalamic hemorrhage

Introduction

Accidental scorpion stings are common in rural India, and the most toxic of the scorpion species found in India is the *Mesobuthus tamulus* (Indian red scorpion) [1-3]. Scorpion venom is a concoction of several toxins such as the hyaluronidase, serotonin, mucopolysaccharides, phospholipase, histamine, enzyme inhibitors, and several neurotoxic, cardiotoxic, and nephrotoxic polypeptides [1,2,4-15]. Pathophysiology of scorpion envenomation is a complex phenomenon and is mainly due to hyperstimulation of the autonomic nervous system [6,7,15], resulting in a wide range of clinical manifestations ranging from mild local skin reactions to severe cardiovascular, respiratory, and neurological complications [1-7,9,15-17]. Neurologic manifestations occur as a result of blood pressure fluctuations, cardiogenic emboli, anoxia, hypoxia, disseminated intravascular coagulation (DIC), direct vasculotoxic effect of the toxin, and prolonged dehydration and vasoconstriction due to excessive catecholamine secretion. Various central nervous system (CNS) complications following scorpion sting have been described in literature, such as cerebral edema, subarachnoid hemorrhage, encephalopathy, hemorrhagic, nonhemorrhagic strokes, and cortical necrosis [2-5,8-12,16,18-21]; however, thalamic and cerebellar hemorrhagic manifestation has yet not been reported. We herein report a rare case of thalamic and cerebellar hemorrhages following a scorpion sting in a 25-year-old Indian male, which to the best knowledge of

the authors' is the first case of its kind to document such a finding in the context of a scorpion sting.

Case report

A 25-year-old male presented to our emergency room in a comatose state with Glasgow coma scale of E1V1M1, following a scorpion bite to the left hand. His vitals were unstable with a blood pressure of 90/60 mm Hg as measured over the right arm in the supine position with a heart rate of 140 beats/min. Instantaneously, ventilator support was instituted, and the patient was investigated. Complete hemogram, renal and liver function tests, serum electrolytes, coagulation profile, D-dimer were all within normal limits. Chest radiograph did not show any evidence of pulmonary edema. Electrocardiogram showed normal sinus rhythm and two-dimensional echocardiogram was normal. With no improvement in his general condition, a magnetic resonance imaging (MRI) of the brain along with an angiogram was performed following advice from neurological consult team. While MRI brain demonstrated symmetrical hyperintensities in bilateral thalami and cerebellum showing diffusion restriction and blooming on gradient echo sequences suggestive of hemorrhages [Figures 1 and 2], MR angiography was normal. A diagnosis of bilateral thalamic and cerebellar hemorrhages following scorpion envenomation was made. Patient was treated with prazosin, mannitol and other supportive measures; however, there was no clinical improvement and the patient expired.

Discussion

Our case demonstrates the fatal case of a scorpion envenomation resulting in cerebellar and thalamic hemorrhages in a young Indian residing in rural India. Cerebellar and thalamic hemorrhages resulting as a direct manifestation following a

¹Departments of Imageology, Bollineni Hospital, Departments of ²Radiology, and ⁴Neurosurgery, Narayana Medical College Hospital, Chinthareddypalem, ³Neurology, Bollineni Hospital, Nellore, Andhra Pradesh, India

Corresponding Author:

V Umamaheswara Reddy, E-mail: mahesh.rd2112@gmail.com

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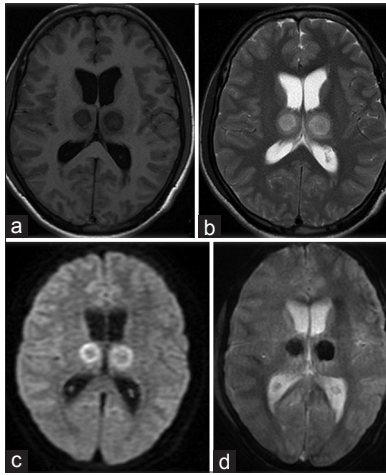


Figure 1 Axial magnetic resonance imaging showing well-defined T2 hyperintense (a) and T1 hypointense areas (b) involving thalami, lesions are showing restriction on diffusion weighted images (c) and blooming on gradient echo sequence (d)

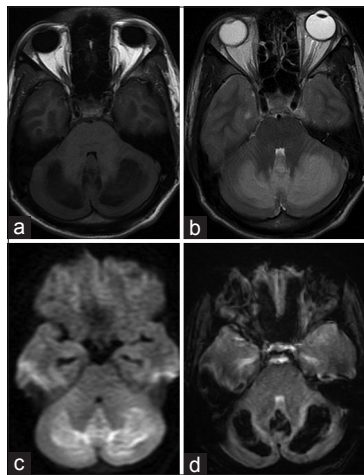


Figure 2 Axial magnetic resonance imaging T2-weighted, T1-weighted, and diffusion weighted images and gradient echo sequences (a-d) showing symmetrical hyperintensities in inferior cerebellum and vermis showing diffusion restriction, blooming

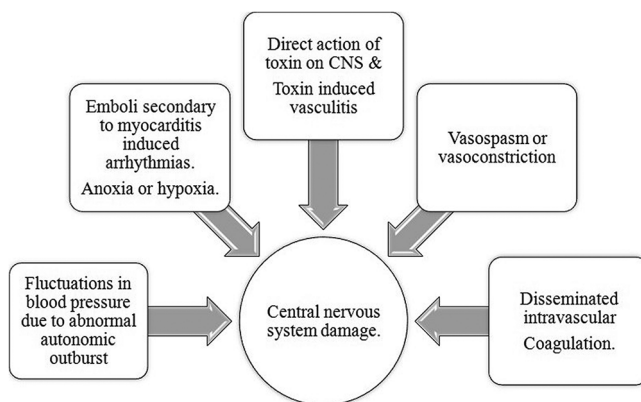


Figure 3 Graphic illustration showing mechanisms by central nervous system damage can occur following scorpion envenomation

scorpion envenomation has not been reported in the medical literature and to the best knowledge of the authors', is the first case report documenting such clinical manifestation. Mechanism of action of toxins in scorpion venom has been implicated to the presynaptic nerve endings targeting the sodium and potassium channels [7,22,23]. These toxins possibly create an imbalance in sympathetic and parasympathetic systems, leading to an autonomic outburst and massive secretion of catecholamines [9]. Clinical manifestations are a likely result of an autonomic outburst and direct actions of toxin on various systems; role of cytokines in pathophysiology of scorpion envenomation is currently debated [19]. Clinical picture varies highly depending upon species of scorpion, dose of venom, site of injection and individuals inflammatory response to the toxins [15]. CNS injury following scorpion sting occurs in many ways as depicted in Figure 3: (1) Direct action on CNS by neurotoxin results in encephalopathy [24,25]. (2) Blood pressure undulations due to counteraction of sympathetic and parasympathetic systems can result in strokes [9,25]; (3) direct damage to endothelium by toxins causes vasculitis and can lead to thrombosis of vessels and infarction [9,25]; (4) DIC may result in infarction or hemorrhagic stroke [9,25]; (5) prolonged hypoxia, anoxia, dehydration are other mechanisms by which CNS damage can occur following scorpion sting [9,25]; isolated symmetrical involvement of bilateral thalami or cerebellum is not uncommon. However, symmetrical involvement of the cerebellum and thalamus together is very rare, in our case, possibly acute rise in blood pressure and loss of cerebral autoregulation could have eventually led to these hemorrhages [21,26]. Despite adequate treatment and supportive measures, involvement of CNS in cases of scorpion envenomation carries a very bad prognosis [19]. Early diagnosis and prompt treatment can reduce mortality and morbidity caused by neurological manifestations of scorpion sting.

Conclusion

Hemorrhagic strokes following scorpion envenomation are infrequent with a few case reports mentioning lobar and intra-ventricular hemorrhages. As for our knowledge, this is the first case report where we encountered intracranial hemorrhages symmetrically involving the thalamus and the cerebellum following a scorpion sting. CNS involvement in these cases generally carries bad prognosis. Variable imaging appearances may mislead radiologist when blinded to the clinical scenario. Especially, this can happen frequently when patient becomes unconscious or comatose following scorpion sting and sting site is not obviously visible. Radiologists and clinicians should be aware of various neuroimaging appearances following scorpion sting to ensure correct diagnosis and prompt treatment.

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Authors' Contribution

All authors contributed equally to the manuscript. All authors read and approved the final manuscript.

Consent

The authors certify that a written informed consent was obtained from the parents of the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal.

Competing Interests

The authors declare that they have no competing interests.

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