



EXTENSIVE ISCHEMIC STROKE FOLLOWING SCORPION STING IN A YOUNG DURING HOSPITAL STAY IN A TERTIARY CARE HOSPITAL: "A RARE CASE REPORT"

General Medicine

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ABSTRACT

Complication 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 autonomic storm, hypertension, hypotension, toxic myocarditis, disseminated intravascular coagulation, venom induced vasculitis. We present a rare case of multiple brain infarcts following scorpion sting which has rarely been reported in medical literature. In our case non-contrast CT head showed multiple infarcts involving bilateral cerebellar hemisphere, bilateral occipital lobes, medulla and Pons on right side, right temporal lobe, and right thalamus, left parietal lobe. He was investigated for other possible cause of stroke in young without positive results such as cardiac, hematological, rheumatological workup. In view of normal CT cerebral angiography and lack of other cause of stroke in young and the temporal relation and progression of events we concluded that stroke in this patient is likely as result of severe vasospasm due to autonomic storm resulting in low flow infarct. Close observation of the patient for a period of hospital stay is required. During this period an hourly check of the patient should be done as assessing the level of consciousness, pulse rate and rhythm, blood pressure, respiratory rate and new symptoms or signs such as focal neurological deficits.

KEYWORDS

autonomic storm, extensive ischemic stroke, myocarditis, scorpion envenomation

INTRODUCTION

Scorpion sting and its various manifestations are common occurrence in India especially in rural subcontinent. Stabilization of neuronal voltage gated sodium channel by scorpion venom in open state cause repetitive, prolonged depolarization of autonomic nervous system causing increase autonomic neurotransmitter including endogenous adrenal medullary catecholamine causing "Autonomic Storm" [1].

Among 80 species of scorpion in India, *Mesobutius tumulus* (Indian red scorpion) and *Palamneus gravimanus* (Indian black scorpion) are of medical importance and are common in our region. The systemic complications are commonly associated with *mesobutius tumulus* (Indian red scorpion). Scorpion envenomation in India represents a medical problem of great importance especially for rural population. The scorpion venom is a water soluble antigenic, heterogeneous mixture. This heterogeneity of the venom may reflect the different reaction. The scorpion venom is composed of neurotoxin, cardiotoxin, nephrotoxin, hemolytic toxins, histamines, serotonin phosphodiesterases, and cytokine releasers [2, 3]. By opening sodium channels at presynaptic nerve terminals and inhibiting calcium dependent potassium channels, the neurotoxin, which is the most potent of the toxin, can be continuous, prolonged repetitive firing of the somatic, sympathetic, parasympathetic neuron [4]. Severe envenomation usually appears within 1-3 hours following a sting and death most commonly occur within 24 hours results from left heart failure and pulmonary edema [5]. However patient can die suddenly within 15-30 minutes due to lethal arrhythmias. If the patient has not developed any symptoms or signs of envenomation, regular close observation of the patient during of hospital stay is required.

During this period an hourly check of the patient should be done as assessing the level of consciousness, pulse rate and rhythm, blood pressure, respiratory rate and new symptoms or signs such as focal neurological deficits.

Even false recovery followed by a complete precipitation of symptoms and signs are common. The most common systemic manifestations are vomiting, profuse sweating, salivation, priapism cold extremities, mydriasis, hypertension, tachycardia, hypotension, bradycardia [5]. However it can be present with seizure, shock, acute pulmonary edema, strokes and coma. Complication following scorpion sting are common in India mainly cardiac and can be fatal, and central nervous system complication are rare comprising only 2% of all the complication [5]. Stroke following scorpion sting is a rare

complication and can occur by various mechanisms such as autonomic storm, hypertension, hypotension, toxic myocarditis, disseminated intravascular coagulation, venom induced vasculitis. In our case non-contrast CT head showed multiple infarcts involving bilateral cerebellar hemisphere, bilateral occipital lobes, medulla and Pons on right side, right temporal lobe, and right thalamus, left parietal lobe. As per the knowledge of our authors, this is first case reported in our institute.

We present a rare case of multiple brain infarcts following scorpion sting which has rarely been reported in medical literature.

Case report

21 year old male farmer was presented in emergency department of our institute with history of scorpion sting at flexure aspect of right forearm at 10.00 am on 24.10.17 while working in field (Figure 1). It was associated with local burning pain at the site of scorpion sting, followed by vomiting and profuse sweating. Then he was taken to local town dispensary at around at 11.00am, on 24.10.17 where he was managed symptomatically scorpion anti venom was not administered because of unavailability. Then he was referred to our institute for further management with continued local burning pain and vomiting, without any focal neurological deficit here he initially was admitted in general ward at 6.25 am on 25.10.17. on evaluation at the time of admission, his general condition and vitals were stable and respiratory, cardiovascular and neurological examination were unremarkable. Patient was fully conscious with GCS 15/15 with no focal neurological deficit. On same day 12-16 hours later (7.00 pm on 25.10.17) he became dyspneic, coughing, altered sensorium, irritable then he was immediately shifted in ICU where further evaluation was made, on history we found weakness in right upper and lower limb which was sudden in onset and gradually progressed over 36-48 hours after of scorpion sting during hospital stay after scorpion sting. his blood pressure was 170/100 mmHg and equal in all four limbs, pulse rate-150/minute, regular and all peripheral pulses were normally palpable, respiratory rate -26/minute, pSo₂ -75%, on chest examination b/l fine crepitation, heart sound-normal with S3 gallop, GCS-14/15 (E "V" M"). On CNS Examination he had slurred speech with right upper and lower limb weakness with power 2/5, tone on right side was decreased. Reflexes decreased on right side. Planter-equivocal on right side while on left side power/tone/reflexes were within normal limits, he had no sensory deficit, no cranial nerve dysfunctions were noticed, no bladder and bowel disturbance was found. There were no meningeal sign.

Clinical diagnosis of stroke secondary to scorpion envenomation was made and his routine investigation were within normal limits and relevant investigation including non-contrast CT head was done, revealed multiple asymmetrical bilateral cerebellar hemisphere, bilateral occipital lobes, medulla and pons on right side, right temporal lobe, right thalamus, left parietal lobe (Figure 8A& 8B) and CT cerebral angiography revealed no vascular occlusion (Figure 9). He was not a known diabetic, hypertensive, dyslipidemic, drug abuser, chronic smoker. He also did not have any known risk factor for stroke in young, there was no family history of premature cardiac or cerebrovascular accident/stroke. There was no history of fever, bleeding tendency, chest pain, and palpitation. His previous medication history was unremarkable. No history of suggestive ischemic heart disease, rheumatic heart disease, transient ischemic accident or stroke. Clinical finding at the time of stroke was consistent with sign of autonomic storm and pulmonary edema. He was further investigated for other possible cause of stroke in young such as peripheral blood film, anti nuclear antibody, rheumatoid factor, serum homocysteine level, anti phospholipids antibody test, lupus anticoagulant, chronic reactive protein, erythrocyte sedimentation rate, coagulation profiles, lipid profiles, serum uric acid, serum calcium level. His RBS-120 mg/dl, CPK-1459, LDH-280, CK-MB-120 IU/L. all other investigation were in within normal limits. At the time of admission, his ECG showed sinus rhythm, without ST-T changes (Figure 2), CXR PA view showed bilateral pulmonary congestion suggestive of acute pulmonary edema (Figure 5). Then repeat ECG on next day showed sinus tachycardia with secondary ST-T changes in II, III, avF and v2-v6 leads (Figure 3) and ECG changes mimicking myocarditis. his qualitatively

Troponin-T was positive (Figure 4), his 2D ECHO showed global LV hypokinesia with severe LV systolic dysfunction (EF-30%) with mild to moderate mitral regurgitation with no intracardiac thrombosis/clots -suggestive of myocarditis (Figure 6) his B/L carotid angiography was normal (Figure 7). So in view of history positive scorpion sting, normal CT cerebral & carotid angiography and lack of other possible cause of stroke in young, he was diagnosed to be have acute ischemic stroke as result of severe vasospasm due to autonomic storm following scorpion envenomation. A neurosurgeon opinion was taken and it was decided to manage the patient conservatively with IV fluids, IV mannitol, IV diuretics, anti platelets, tab. prazosin. Scorpion antivenom was not administered as it was not available in our institute. Over the period of 7th days there was gradual improvement in neurological status but no improvement was observed in his cardiac and respiratory status and gradually he was continued to be deteriorated and put on mechanical ventilator support. But finally he died.

DISCUSSION

The outcome was equally uncommon considering the site and extent of the infarction. In our case there was evidence of autonomic storm with myocarditis with pulmonary edema and with the available information we concluded the multiple infarcts in this patient was likely to be as results of severe vasospasm due to autonomic storm following scorpion envenomation with associated myocarditis. The brain infarcts, clinically evident as 36 hours after scorpion sting are compatible with low flow infarcts [14]. which are known to develop and progress gradually over days or weeks. There was few case reported in medical literature where mortality was uncommon in ischemic stroke following scorpion sting. Severe manifestation following scorpion sting is mainly cardiac. Central nervous system complications are very rare, comprising only in 2-8% of all complication. And may be in either of two forms, both of which are associated with high mortality rates (1) encephalopathy. The venom can be directly neurotoxin resulting in seizures and encephalopathy. (2) Stroke. many mechanisms have been proposed to explain the occurrence of the stroke in patients with scorpion sting. , they are known to be fatal in 2-4% cases in India today. Mechanisms of strokes are such as

1. Rise in blood pressure due to autonomic storm may rupture unprotected or diseased vessels resulting in hemorrhagic stroke [9].
2. Toxic myocarditis may precipitate arrhythmias and embolic stroke, shock resulting in ischemic stroke [6, 12, 13].
3. Change in blood coagulation causing disseminated intravascular coagulation [4, 9].
4. The venom is vasculotoxic and has led to damage endothelial cells and cause vasculitis.
5. Catecholamine excess leads to severe vasoconstriction induced vasospasm of the cerebral vessels. causing hypoperfusion resulting into ischemic stroke [2, 6, 9].

In our case bilateral multiple brain infarcts are compatible with severe vasospasm induced by autonomic storm following scorpion envenomation. Myocarditis induced by autonomic storm following scorpion sting are also known to synergistic effect on already compromised cerebral ischemia. These explanation are likely in this patient as significantly raised cardiac specific enzyme, qualitatively positive troponin-T, electrocardiographic and echocardiographic evidence of myocarditis are present. In a case report by Thacker *et al.* [6] also concluded vasospasm due to autonomic storm as a cause of multiple brain infarctions in a case of scorpion sting. But in previous studies of multiple brain infarctions due to scorpion sting, the favorable prognosis is usually reported but in our case the outcome was unfavorable because of combined complication of both central nervous system and cardiovascular system due to autonomic storm following scorpion sting. Compared to previous reports and studies it is exceedingly rare complication following scorpion sting and also as per knowledge of our authors this is first case in our institute. Multiple brain infarcts are reported in few case reports such as Thacker *et al.* [6], Nagaraja *et al.* [7], Gadwalkar SR *et al.* [8] and further review of literature also made but there was no case report of such extensive brain infarct with associated myocarditis and pulmonary edema found.

CONCLUSION

Early presentation and timely diagnosis and early administration of appropriate therapy such as alpha receptor blocker and scorpion anti venom can save the life of patients and can offer a better prognosis to the patient. Appropriate protective clothing and shoes for villagers during working in field is highly recommended to prevent the sting and reduce the incidence and training for doctors at primary health centre have to be done and physicians at higher centre must be aware about these rare fatal central nervous system complication of scorpion sting while evaluating the patients and regular close monitoring in intensive care unit for period of hospital stay is required.



Figure 1: Scorpion sting mark on right forearm flexor surface, blackening because of burn as a result of local ritual.

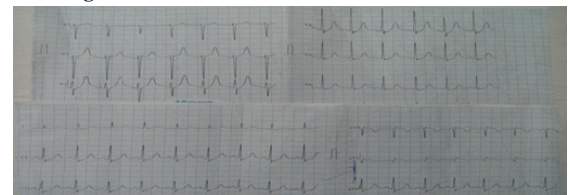


Figure 2: ECG on day of admission showing sinus rhythm with no ST-T changes

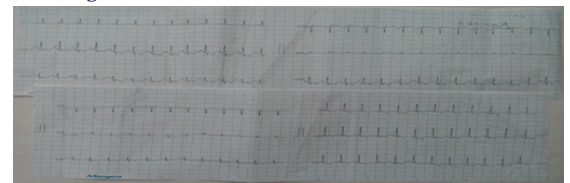


Figure 3: ECG on next day showing sinus tachycardia with ST depression and T wave inversion in leads II, III, avF and v2-v6.

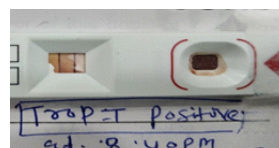


Figure 4: qualitative positive Troponin-T.

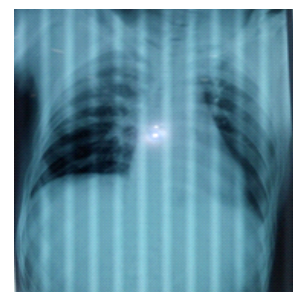


Figure 5: CXR PA view showing pulmonary edema

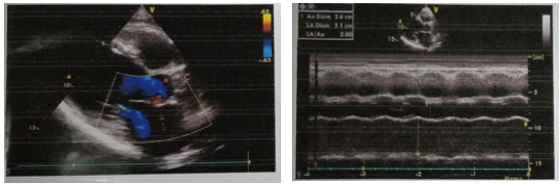


Figure 6: 2D ECHO showing global LV hypokinesia with severe LV dysfunction with mild to moderate mitral regurgitation

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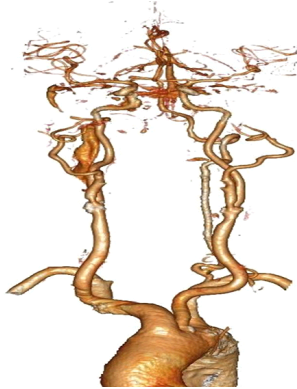
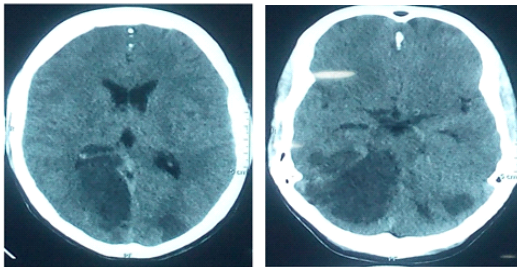


Figure 6: B/L carotid angiography showing normal carotids.



[A]

[B]

Figure 8: (A) non-contrast CT head scan showing bilateral occipital lobes, right temporal lobe, right thalamus, left parietal lobe. (B) Bilateral cerebellar hemisphere, medulla and pons on right side.



Figure 9: CT cerebral angiography showing no vascular occlusion.

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