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Original Research Paper

General Medicine

SCORPION STING CAUSING MULTIPLE WATERSHED INFARCTS

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ABSTRACT Scorpion sting presents with several neurotoxic local effects, cranial nerve effects (roving eye movements, blurring of vision, tongue fasiculations, dysphagia), central nervous system effects (encephalopathy, convulsions, CVA, central respiratory failure). We report a case of a 53 year old female who sustained a scorpion sting at her residence and initially presented with local site symptoms only. During the course of hospital stay, after 2 days she had a episode of hypotension followed by an episode of generalised tonic clonic seizures and right hemiplegia. MRI brain was suggestive of bilateral acute ischemic watershed infarcts, carotid Doppler was suggestive of low flow bilaterally. Patient was started on dual antiplatelets, anti edema and anti epileptic measures. The patient recovered spontaneously over a period of 2 weeks.

KEYWORDS :

INTRODUCTION-

Scorpion sting is one of the neglected diseases affecting many people. The scorpion venom is water soluble and consists of neurotoxin, nephrotoxin, hemolysins, cardiotoxins, phosphodiesterases, phospholipases and histamine. The primary target of scorpion venom is voltage dependent ion channels. It is accompanied by local manifestations such as pain, edema, burning or tingling sensation at the site of bite and several systemic manifestations like muscle weakness, arterial hypertension, pulmonary edema, cardiac arrhythmias, myocarditis and rarely cerebrovascular complications. These manifestations occur due to intracellular influx of sodium and calcium ions through sodium channels causing an autonomic storm[1]. Ischemic infarct secondary to scorpion bite is very rare and the generally caused secondary to the coagulins in venom or DIC induced intravascular thrombosis[2].

CASE REPORT-

A 53 year old female, with no known comorbidities, was stung by the Indian red scorpion (Mesobuthus tumulus) at her right index finger, at her residence. On arrival to the hospital, patient was conscious oriented, complained of pain at the site of bite. On examination blood pressure was elevated (160/100 mm Hg), other vitals and systemic examination was normal. On local examination- there was reddish discoloration and mild swelling around the area of the bite. Patient was administered inj xylocaine and a stat dose of Prazosin. All baseline blood investigations were done and showed mild derangement of renal parameters, bleeding time , clotting time and CXR PA view was normal. ECG was suggestive of old septal MI changes, 2 D ECHO showed left ventricular ejection fraction of 43%, hypokinesia of septum, no evidence.

After 2 days of in hospital admission, there was a sudden drop in blood pressure to 80/40 mm Hg, after 5 minutes patient had an episode of generalised tonic clonic seizures followed by a fall in GCS to 10/15 and right hemiplegia. MRI brain was suggestive of acute ischemic external borderzone infarcts involving bilateral MCA-ACA and MCA-PCA territory(figure 1). Carotid Doppler was done, showed significant stenosis at right bulb to ICA, proximal part of left ECA and from left bulb to proximal part of left ICA, hypoplastic vertebral artery(figure 2,3,4). CT angiogram also revealed the above findings. She was started on dual antiplatelets, statins, anti edema and anti epileptic measures. Over a period of 2 weeks the patient improved and regained right upper and lower limbs power of 4/5.

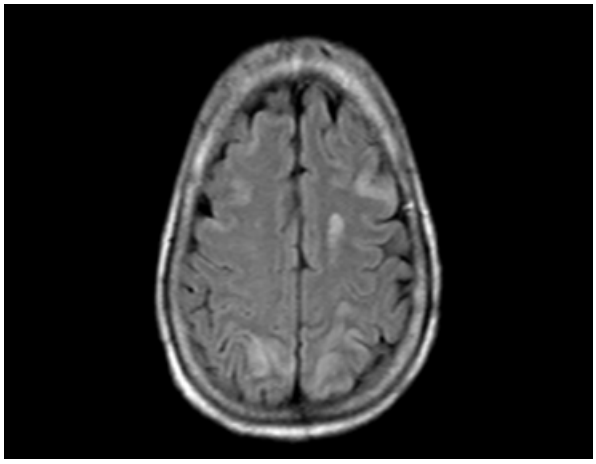

Figure 1- MRI Brain(T2 FLAIR) Showing Water Shed Infarcts

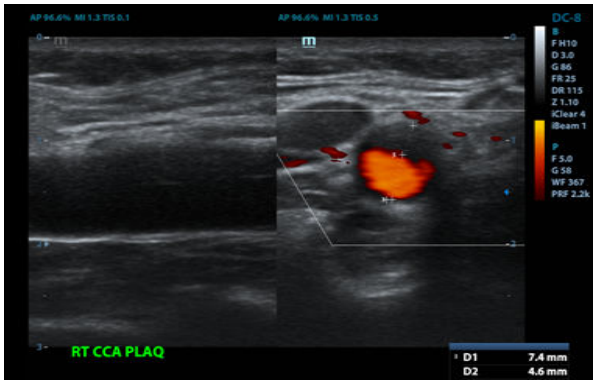

Figure 2- Carotid Doppler Showing Right Common Carotid Artery Plaque

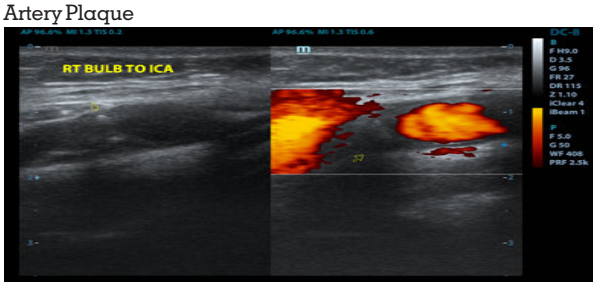

Figure 3- Carotid Doppler Showing Stenosis In Right Bulb To ICA

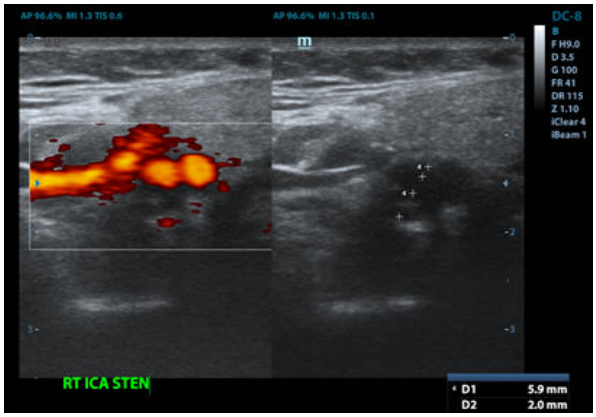

Figure 4- Carotid Doppler Showing Right ICA Stenosis

DISCUSSION-

Cerebral infarcts occurring after 36 hours of envenomation are usually low flow infarcts, develop and progress gradually over days to a week[3].

In this case, patient's initial rise in blood pressure can be attributed to catecholamine excess, activation of alpha receptors leading to vasoconstriction[4]. Post which the patient had multiple bilateral asymmetric watershed cerebral infarcts are likely to due to reduced blood flow in carotid arterial system. Several causes like, autonomic storm, myocarditis induced cardioembolic stroke, venom induced DIC causing platelet aggregation, vasculitis caused by endothelial damage, hypotension may induce watershed infarcts[5,6].

In our case, the cause of watershed infarcts can be attributed to myocarditis or vasospasm induced secondary to catecholamine excess. Since our patient recovered spontaneously, all haematological parameters were normal, we may attribute the cause as vasospastic.

Scorpion sting may be associated with initial short lasting hypotension secondary to acetyl choline excess. Hypotension in this case worsened the CVA, in already compromised carotid circulation due to vasospasm.

CONCLUSION-

Watershed infarcts are a rare manifestation in scorpion sting, but should be considered as one of the neurological complications.

Ethical Considerations

Informed consent was obtained from the patient for publication of this report.

Declarations Of Interest

None

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