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Ischemic stroke consequent to snake bite

Deepika Kapil[1], Uday Mahajan[2*]
[1] Departments of Obstetrics and Gynaecology, Civil Hospital Nagrota Bagwan, Himachal Pradesh, India
[2] Senior Resident, Department of Medicine, Dr Rajendra Prasad Govt. Medical College, Kangra, Tanda, Himachal Pradesh, India

Abstract
45 year post-menopausal female presented to emergency with a history of snake bite on left foot with history of tourniquet application at ankle. History of altered sensorium, sluggish responses, history of single episode of vomiting, no history of ptosis, diplopia, shortness of breath. Patient given another 100 units antisnake venom with premedication and continued to be monitored in intensive care unit the patient susequently deteriorated and had cardiorespiratory arrest.

Keywords: cord, blood, screening

Introduction

Neurological deficit following snake bite is not uncommon and is usually caused by intracerebral or subarachnoid bleed. [1] Cerebral infarction following snake bite is rare and only few cases have been reported since 1966 [1-4]. Serious neurological complications, including stroke and muscle paralysis, are related to the toxic effects of the venom, which contains a complex mixture of toxins affecting the coagulation cascade, the neuromuscular transmission, or both.

Metalloproteinases, serine proteases, and C-type lentins (common in viper and colubrid venoms) have anticoagulant or procoagulant activity and may be either agonists or antagonists of plateletaggregation; as a result, ischemic or hemorrhagic strokes may occur.

Case report

45 year post-menopausal female presented to emergency with a history of snake bite on left foot with history of tourniquet application at ankle. History of altered sensorium, sluggish responses, history of single episode of vomiting, no history of ptosis, diplopia, shortness of breath. Vitals at time of admission saturation 86 percent at room air pulse 70/ min regular, blood pressure 100/ 70 mm hg. No clubbing or cyanosis or regional lymphadenopathy was present. Abdomen was soft, non-tender, chest- vesicular breath sound bilaterally, on neurological examination cranial nerves were normal, power upper limb 5/5 bilaterally and 4-/5 in bilateral lower limbs and left plantar extensor response. Electrocardiogram of patient was normal sinus rhythm with tachycardia.

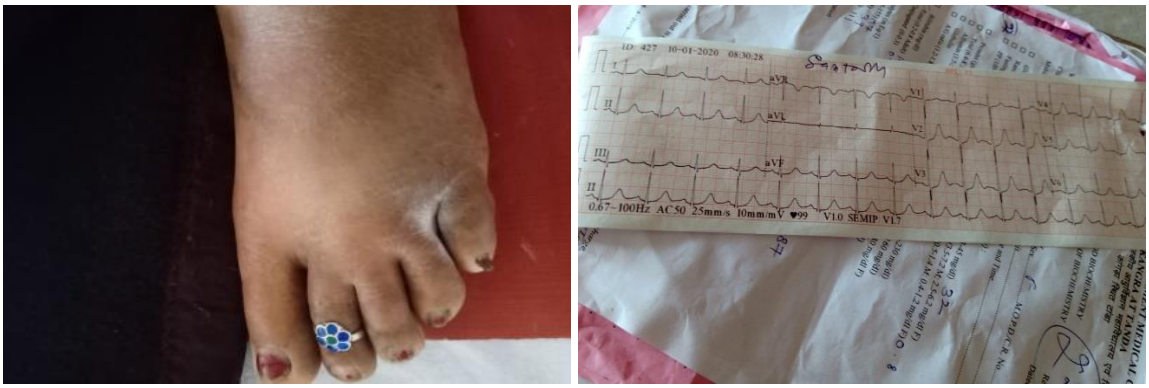

Fig 1

Image: Ecg of patient

Investigations total leukocyte count 17600/ul with neutrophilia 14.96 %, haemoglobin 10.6 gm/dl, platelet count 142000/ul, urea 32 mg/dl, serum Creatinine .8 mg/dl, total bilirubin 1 mg/dl conjugated .4 mg/dl, serum electrolytes within normal limits /Prothrombin time > 60 seconds /serum lactate dehydrogenase 1034 IU/ ml, sgot 83 IU/l, sgpt 45 IU/l, viral markers non-reactive Initial Whole blood clotting time prolonged and patient planned for imaging brain. Pateint managed with intravenous fluids, strict input output charting, oxygen inhalation, prokinetics plus antibiotics plus injection antisnake venom 100 units to be repeated according to coagulation profile Initial non contrast CT was normal without evidence of intracranial/ extracranial bleed Patient subsequentlyinvestigated- Prothrombin time reduced to 27.7 seconds with INR 2.76/ sgot/ sgpt were within normal limits Patient given another 100 units antisnake venom with premedication and continued to be monitored in intensive care unit. The patient susequently deteriorated and had cardiorespiratory arrest.

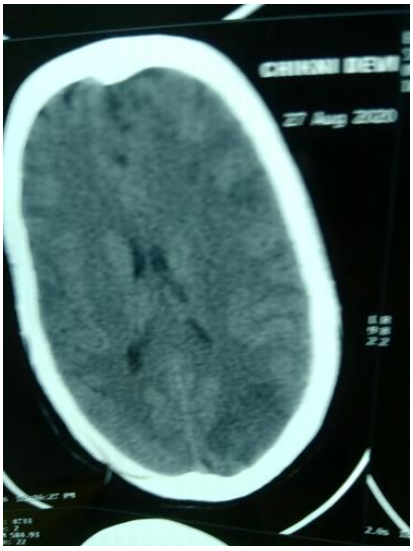

Fig 2: Diffusion weighted MRI suggestive of multiple areas of diffusion restriction suggestive of acute embolic infarct bilateral PICA nad bilateral MCA

Discussion

Snake venoms can cause cellular injury through enzymes, polypeptide toxins, cytokines, and mediators. Important venom enzymes consist of proteases, hydrolases, hyaluroxidase, oxidases, phospholipases, and esterases. Among these enzymes, phospholipases A2 and proteases (especially metalloproteases) contribute significantly to tissue injury. Cytokines and vasoactive mediators are responsible for inflammatory changes and hemodynamic alterations that can ultimately lead to cellular injury [5]. The occurrence of vascular thrombosis in vessels adjacent to the site of envenomation is common. It is extremely rare for thrombosis to occur in distant vessels. Cerebral infarction following snake bite is rare and only few cases have been reported. There are several mechanisms by which cerebral infarction occur in snake envenomation [1].

- Hypotension due to hypovolemia from sweating, vomiting, decreased fluid intake, and bleeding tendencies. This leads to low flow state and watershed infarct [1, 2].
- Hypercoagulability can be due to procoagulants in the venom such as hydrolase, consumption coagulopathy phase of DIC [1, 6].
- Endothelial injury due to toxic vasculitis by the components of venom can lead to thrombosis [1, 7].

The infarct in our patient was not in classical watershed territory and hence hypotension may not be the cause. Clotting time and bleeding time were normal, which ruled out DIC; there are no other clinical manifestations of DIC (only 20WBCT was positive). The possible cause of infarct in this case was toxic vasculitis which causes injury to the endothelial cells by snake venom toxin.

The 20-min blood clot test was adopted as the standard test of coagulopathy. It is simple to carry out and give reliable indication of consumption coagulopathy. Evidence of coagulopathy determines that the biting species is viperine. Neither of the Elapids, i.e., cobra or krait are known to give anti-hemostatic symptoms [8].

Conflict of interest

All authors declare they have no conflict of interest.

Financial disclosure

Nil

References

1. Mugundhan K, Thruvarutchelvan K, Sivakumar S. Posterior circulation stroke in young male following snake bite. J Assoc Physicians India,2008:56:713-4.
2. Rifant B, John J. Cerebral Infarction in a young female following snake bite. Stroke,1985:16:328-30.
3. Upadhyaya AC, Murthy GL, Sahay RK, Srinivasan VR, Shantaram V. Snake bite presenting as acute myocardial infarction, ischaemic cerebrovascular accident, acute renal failure and disseminated intravascular coagulopathy. J Assoc Physicians India, 2000:48:1109-10.
4. Narang SK, Paleti S, Azeez Asad MA, Samina T. Acute ischemic infarct in the middle cerebral artery territory following a Russell's viper bite. Neurol India,2009:57:479-80.
5. Sitprija V. Snakebite nephropathy (Review Article). Nephrology,2006:11:442-8.
6. Christ A, Loannidis PJ, Ktenasjohn. Acute M1 and CVA in a young girl after a viper bite. Br Heart J,1982:47:500-3.
7. Panicker JN, Madhusudhanan S. Cerebral infarction in a Young Male following Viperenvenomation. J Assoc Physicians India,2000:48:744-5.
8. Ghosh S. Management of Snake Bite-An Update. Med Update,2008:18:691-6.