



A Rare Case of Cerebral Infarction in a Young Female Following Snake Bite

Authors

Dr Maitrey Patel¹, Dr Basavraj R Patil Raikood²

¹PG Dept of General Medicine, Mahadevappa Rampure Medical College, Gulbarga, Karnataka, India

²Associate Professor, Dept of General Medicine, Mahadevappa Rampure Medical College, Gulbarga, Karnataka, India

Corresponding Author

Dr Maitrey Patel

11 Avi Bunglows, Opp Star Bazaar, Satellite Road, Ahmadabad 380015

Email: ptlmaitrey@gmail.com, Mobile - 7760378269

Abstract

Ischemic stroke following snake bite is rare. We report a 35 year female developed right sided hemiplegia with left UMN facial palsy with broca's aphasia following a snake bite. CT brain plain showed acute large infarct in left middle cerebral artery territory. The possible mechanisms for cerebral infarction in this scenario include disseminated intravascular coagulation, toxin induced vasculitis and endothelial damage.

Introduction

India is estimated to have the highest snakebite mortality in the world. World Health Organization (WHO) estimates place the number of bites to be 83,000 per annum with 11,000 deaths ^[1]. The big four namely Russell's viper, saw scaled viper, Indian cobra, and the common krait are usually responsible for most of the deaths ^[2]. Studies have documented a stroke incidence of 2.6% in snake bite with a predominance of hemorrhagic strokes due to consumption coagulopathy. ^[3] Russell's viper can also manifest with neurotoxic symptoms. The neurotoxic symptoms in Russell's viper are believed to be due to presence of a presynaptic toxin like that in common krait. ^[4] We are presenting a rare case of cerebral infarct in young lady following a snake bite

Case report

A 35 year female was bitten by a snake when she was working in the field. Snake was not identified by her or her relatives. After that she was brought to emergency department in a state of drowsy and she had drooping of eyelids and loss of speech. Her emergency room blood pressure was 80/60 mmHg, pulse rate 102beats/min and systemic examination was normal and bite site was present on right dorsum of foot without local manifestation. 10 vials of polyvalent snake venom was given after test dose. After test dose there was bleeding from puncture site. After one hour she became unconscious and she was not maintaining saturation and she was intubated and there was bleeding from endotracheal tube.

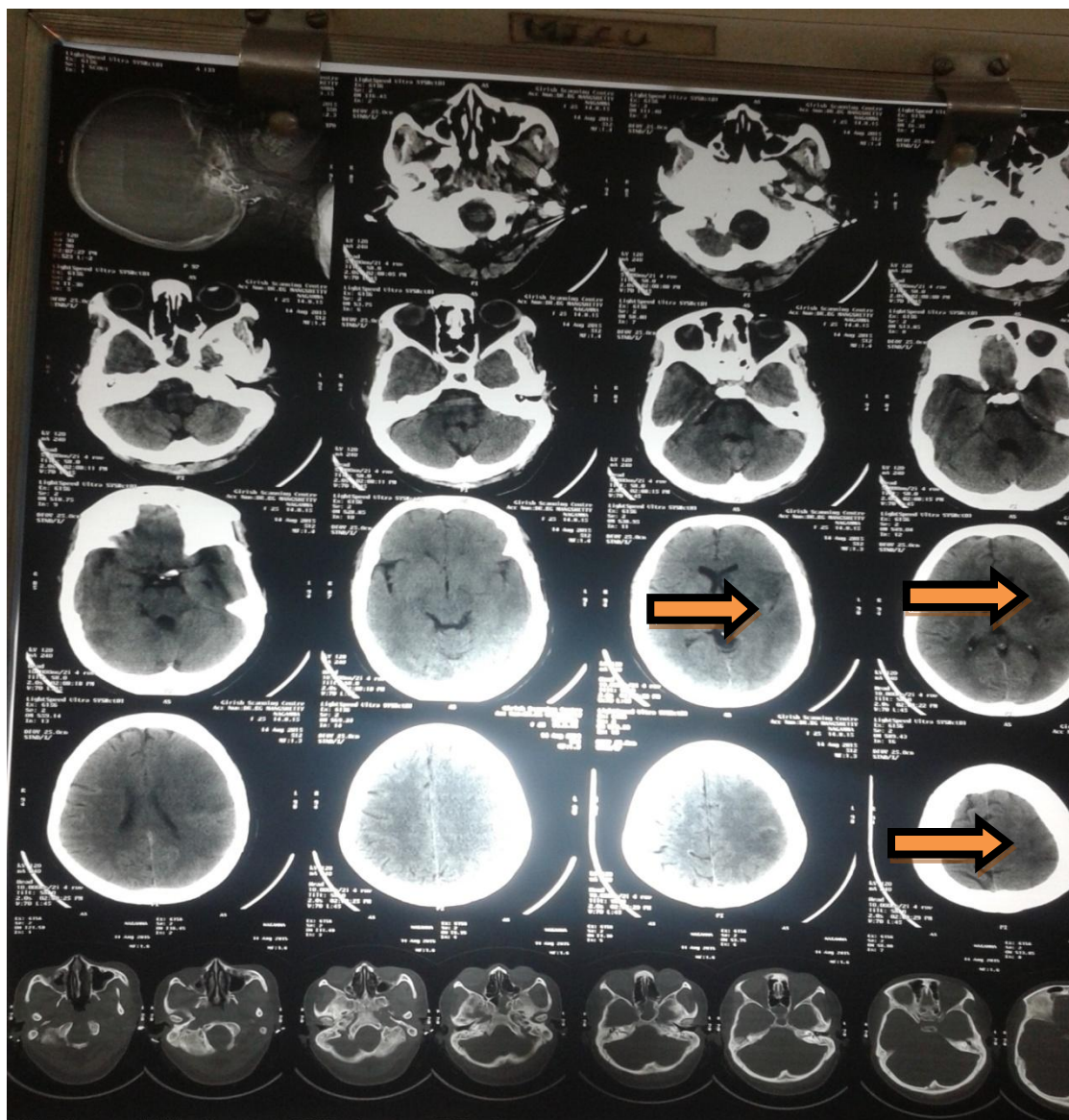
Laboratory investigation were TLC 23,000, Platelet 2.58 lac, PT 29.8, INR 3.4, D dimer-

positive, APTT 40, 20 minute whole blood clot lysis was positive. Urine examination RBCs 6-8 present

ABG, RFT, S.Electrolyte, X ray Chest, ECG were normal.

After one hour 10 more vials were repeated and atropine and neostigmine was given for 24 hours. Higher antibiotics were given and after two hour her Ptosis was reduced and bleeding from puncture site were stopped and 20 minute whole blood clot lysis was negative. On 3rd day she had left lateral gaze palsy which was false localizing sign which disappeared with antiedema measures. She was not able to raise her upper and lower limb with positive babinski on right leg and she had aspiration pneumonia. On 5th day after extubation

patient was diagnosed with broca's aphasia and left UMN facial palsy. CT Brain Plain was done it was showing large acute Left MCA territory infarct. Repeat blood investigations were normal. Further evaluation for stroke in young ECG, Lipid Profile, 2D Echo Serum Homocystine and ANA were negative. Protein C, S and Antithrombin iii levels were not done as values will be altered following a thrombotic event and ASV therapy. Patient was started antioedema measure like mannitol and anti platelet aspirin (150mg/day) and physiotherapy. Anticoagulation was not started due to potential risk of bleeding. At present patient having broca's aphasia with Left UMN facial palsy with Right hemiplegia.



Discussion

Viper bite is the most common snake bite in the Indian subcontinent. The envenomation by viper bite commonly presents with local envenomation, followed by abnormal coagulation.^[3] The various toxins present in the viper venom have both pro-coagulant and anticoagulant effect. The toxins

With well-established pro-coagulant / platelet aggregating properties are cerastobin,^[5] factor iva,^[6] cerastocytin,^{[7][8]} cerastotin,^[9] and afaacytin^[10]. These various protein products have thrombin like enzyme activity; different toxins activate different parts of the coagulation cascade.^{[5]- [11]} Their activity is inhibited by monoclonal antibodies against gp1b or gpiib/iiiA or thrombin receptor. ASV has been associated with multiple complications. This may be classified into early reactions such as anaphylaxis and pyrogenic reactions characterized by fever with chills and itching and late reactions such as serum sickness, with the incidence being as high as 81% in some studies.^[12] Severe hypotension secondary to anaphylaxis can also predispose to an ischemic stroke.^[13] As this patient developed neurological symptoms only after initiating ASV therapy, a direct association with ASV is possible. A hypotensive etiology though possible, is unlikely since hypotension usually tends to cause watershed territory infarcts and is not strongly associated with occlusion of a major cerebral artery.^[14]

Conclusion

In our patient, though ischemic stroke causation includes many possibilities, we suggest that toxic vasculitis or massive disseminated intravascular coagulation or hypotension may be the most likely cause of left middle cerebral artery occlusion. We report this case to highlight this uncommon presentation of a snake bite and early ASV can prevent this complication.

References

1. Kasturiratne A, Wickramasinghe AR, Desilva N, et al. The global burden of

snakebite: A literature analysis and modelling based on regional estimates of envenoming and deaths. *PLOS Med.* 2008; 5:e218.

2. Simpson ID, Norris RL. Snakes of medical importance In India: is the concept of big four still relevant and Useful? *Wilderness and environmental medicine* 2007;18(1):2-9.
3. Mosquera A, Idrovo LA, Tafur A, Del Brutto OH. Stroke following *bothrops* spp. Snakebite. *Neurology* 2003; 60(10):1577-80.
4. Simpson ID. Snakebite management in India, the first few hours: a guide for primary care physicians. *J Indian med assoc.* 2007; 105:324- 35.
5. Farid TM, TU at, el-Asmar MF. Effect of cerastobin, a thrombin like enzyme from cerastes viper (Egyptian Sand snake) venom, on human platelets. *Haemostasis* 1990; 20(5):296-304.
6. Basheer AR, el-Asmar MF, Soslau G. Characterization of a potent platelet aggregation inducer from cerastescerastes (Egyptian sand viper) venom. *Biochimbiophysacta* 1995 Jul 3;1250(1):97-109.
7. Dekhil H, Wasner A, Marrakchi N, Ayed M, Bon C, Karoui H. Molecular cloning and expression of a Functional snake venom serine proteinase, with platelet Aggregating activity, from cerastes viper. *Biochemistry* 2003 Sep 16;42(36):10609-18
8. Marrakchi N, Barbouche R, Guermazi S, Bon C, el-Ayeb M. Procoagulant and platelet aggregating Properties of cerastocytin from Cerastescerastes venom. *Toxicon* 1997 Feb; 35(2):261- 72.
9. Marrakchi N, Barbouche R, Guermazi S, Karoui H, Bon C, El-Ayeb M. Cerastotin, a serine protease from Cerastescerastes venom, with platelet-aggregating and Agglutinating properties. *Eur j biochem.* 1997 Jul 1; 247(1):121-8.

10. Laraba-Dejabri F, Martin-Eauclaire MF, Mauco G, Marchot P. Afaacytin, an alpha beta-fibrinogenase From cerastescerastes(horned viper) venom, activates Purified factor x and induces serotonin release from Human blood platelets. Eur j biochem 1995 Nov 1; 233(3):756-65
11. Soslau G, el-Asmar MF, Parker j. Cerastescerastes(Egyptian sand viper) venom induced platelet aggregation as compared to other agonist Biochem-biophys res commun 1988;150:909-916.
12. Ariaratnam A, Sjostrom L, Raziak Z, et al. An open Randomized trial of two antivenoms for the treatment of envenoming by Sri Lankan Russel's viper. Trans R soc trop med hyg 2001; 95:74-80.
13. Bashir R, Jinkins J. Cerebral infarction in a young Female following snake bite. Stroke 1985; 16:328-30.
14. Torvik A.the pathogenesis of watershed infarcts in the brain. Stroke1984; 15:221-3.