Case Report

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Russell Viper's Cardiac Bite: A Case Report

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Abstract

Viper snake is common in South Asia. It produces a variety of clinical manifestation like hypotension, diarrhea, headache, renal failure, respiratory failure and other neurological manifestations. The toxicity depends on a combination of five different venom fractions, each of which is less toxic when tested separately. Venom toxicity and bite symptoms in humans vary within the different population and over time. Myocardial ischemia and infarction are a rare complication of Russell's viper bite. Here we report a case of anterolateral ischemia and acute kidney injury following a Russell's viper bite in a 20-year-old healthy male, without previous cardiac or renal complications. He recovered within 2 weeks following hemodialysis and anti-snake venom administration.

Key words: Acute kidney injury, Anti-snake venom, Hemodialysis, Myocardial ischemia, Snake bite, Russell viper

INTRODUCTION

In India, 50,000 people die from snake bite every year.[1] Though many types of snakes are prevalent the mortality and morbidity are more common with viper, cobra and krait.[2] Venom in many snakes affects virtually every organ system in the human body and can be a combination of many toxins including cytotoxins, hematotoxins, neurotoxins and myotoxins. Most snake bites, whether venomous or non-venomous will produce some local effects. There may be minor pain and redness in 90% of cases although this varies depending on the site of the bite. Viper bite is extremely painful and produces local cellulitis, tissue damage, neurological complications like ptosis, convulsions, loss of coordination, weakness, nephrological complications like renal failure and hematological complications like bleeding manifestations. Cardiotoxicity in a viper bite is an extremely rare complication, which is reported in <10% of the viper bites.[3] We report a case of Russell's viper bite in a 20 year male who presented with acute myocardial ischemia and acute kidney injury who recovered within 2 weeks following hemodialysis and administration of polyvalent snake venom and other supportive measures.

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CASE REPORT

A 20-year-old male presented to our emergency department 4 h after an alleged history of snake bite in his right leg above the lateral malleolus. He complained of severe pain at the site of bite, giddiness, retrosternal chest pain and also had vomiting. Patient was not a diabetic, hypertensive and did not have any cardiac, renal and coagulation disorder in the past. No history of any previous surgeries. Patient was a non-alcoholic and non-smoker. No significant family history.

Patient was conscious and oriented. His blood pressure 90/60 mmHg, pulse - 112/min regular and was comfortable with room air. On examination, fang marks was seen with surrounding erythema at the site of the bite. He developed cellulitis involving up to 5 cm below the knee. Patient had congenital ptosis, decreased urine output, his single breath count test was normal, with no other neurological deficit, no bleeding manifestations, and other system examinations were normal.

Whole blood clotting time was prolonged >20 min. Electrocardiogram (ECG) showed sinus tachycardia,

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ST flattening with T-wave inversion in lead II, III, aVF, V3-V6 suggestive of inferior and lateral wall ischemia and cardiac enzymes - Trop I was positive, creatine kinase MB - 116 U/L, echo: No regional wall motion abnormalities. Bleeding time was 3 min 35 s, clotting time was 5 min 45 s, white blood cell - 19,000 cu/mm, platelet - 123,000 cu/mm and he had normal electrolytes levels. Patients renal function test was normal initially, lactic acid dehydrogenase was 803 U/L which was suggestive of rhabdomyolysis. His repeated renal function test on the subsequent day was elevated which indicated acute kidney injury and urine for myoglobin was negative.

Patient was managed with intravenous fluids, intravenous broad spectrum antibiotics, and anti-snake venom. He was initiated on hemodialysis and was given medical management for myocardial ischemia. Patient recovered in 2 weeks with all the abnormal parameters returning back to normal. Repeat ECG was normal with no old ischemic changes. (Table 1, Figures 1 and 2)

Table 1: Investigations

Test	Russels viper bite		
	Day 1	Day 2	Day 12
WBC count	19,500 cells/cumm	13,000 cells/cumm	8400 cells/cumm
Platelet	123,000 mm^3	126,000 mm^3	176,000 mm^3
PT and INR	C-13, T-17 INR-1.34	C-14, T-17 INR-1.21	C-13, T-16 INR-1.23
CK-MB	116 U/L	140 U/L	32 U/L
CK total	713 U/L	1336 U/L	146 U/L
Serum urea	35 mg/dl	78 mg/dl	42 mg/dl
Serum creatinine	1.0 mg/dl	3.5 mg/dl	1.3 mg/dl
Serum sodium	139 mEq/dl	133 mEq/dl	134 mEq/dl
Serum potassium	3.9 mEq/dl	4.6 mEq/dl	3.8 mEq/dl
Serum LDH	803 U/L	564 U/L	256 U/L

PT: Prothrombin time, INR: International normalized ratio, CK: Creatinine kinase, LDH: Lactate dehydrogenase

DISCUSSION

The mechanism by which myocardial ischemia or infarction occurs is not known clearly. Possible mechanisms are, disseminated intravascular coagulation[4] causing thrombus[5] formation in coronaries and direct vasculitis by snake venom causing an infarction. Some snakes have sarafotoxins,[6] which cause coronary vasoconstriction. Coronary spasm[7] due to endothelins released by snake bite is also considered to be a possible mechanism.

Other mechanisms that suggested has causative for myocardial infarction (MI) for viper bite, are: (1) Hypovolemic shock due to bleeding, (2) anaphylactic shock, (3) hypercoagulability in consumption coagulopathy, (4) hyperviscosity secondary to hypovolemia induced hemoconcentration, (5) direct cardiotoxic effect on myocardium.

Tony and Bhat[8] have reported a case of MI on day 2 following a snake bite and proposed vasospasm caused by sarafotoxins in snake venom as the possible mechanism.

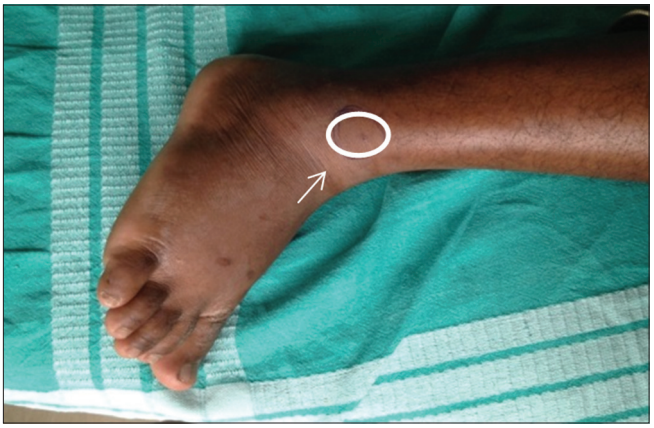

Figure 2: Bite mark: Right leg near ankle

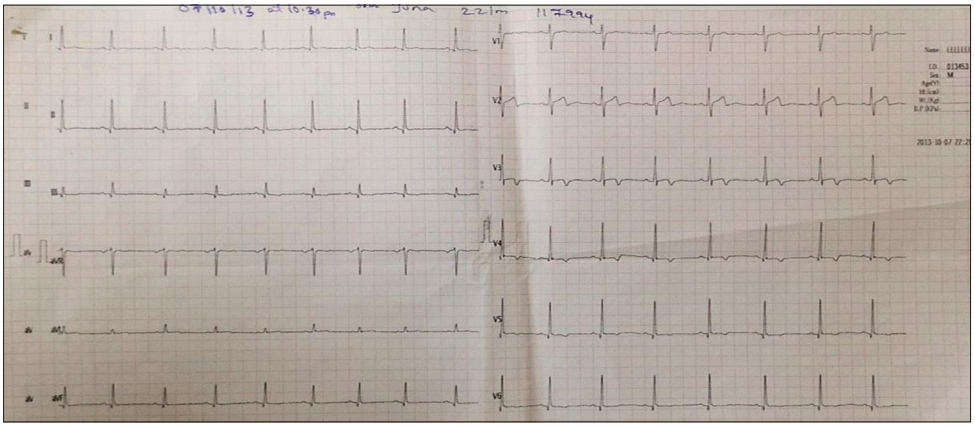

Figure 1: Electrocardiogram

Dissanayake and Sellahewa[9] have described acute MI following Russell's viper bite in a 47-year-old man and proposed a mechanism of predominant coagulant in venom resulting in coronary thrombosis – leading to MI.

Hoffman *et al.*[10] have reported myocarditis with extensive myocardial necrosis at post mortem in two horses after the injection of *Viperae palaestinae* venom for the commercial production of antibodies.

Rowlands *et al.*[11] reported myocardial damage in a fatal case after snake bite by a species of the Australian elapid family in which small foci of myocardial damage and massive skeletal rhabdomyolysis were seen.

Sathyanathan and Mathew[12] reported Raynauds phenomenon and gangrene occurring in opposite limb following envenomation with a snake bite.

In our case, the patient was a non-smoker, non-diabetic and normotensive individual with no family history of dyslipidemias or adverse cardiac events and hence pre-existing coronary stenosis was considered to be unlikely. So the mechanism could be direct cardiotoxicity or coronary thrombosis due to hypercoagulopathy. Myocardial ischemia with acute kidney injury in snake bite is very rare. The physician should have a high index of suspicion to look for cardiac complication too, hence reported.

CONCLUSION

Snake bite is common in India. Young people are affected more commonly.[13] Even though myocardial involvement in a Russells viper bite is rare, recording an electrocardiogram in all snake bite patients may detect myocardial involvement earlier and devastating sequel of MI can be prevented in these cases if diagnosed earlier and managed with anti-snake venom. The postulated mechanisms and its various complications should be kept in mind while treating a patient of Russel viper's bite.

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REFERENCES

1. Chippaux JP. Snake-bites: Appraisal of the global situation. Bull World Health Organ 1998;76:515-24.
2. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, Premaratna R, *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.
3. Nayak KC, Jain AK, Sharda DP, Mishra SN. Profile of cardiac complications of snake bite. Indian Heart J 1990;42:185-8.
4. Copley AL, Banerjee S, Devi A. Studies of snake venoms on blood coagulation. Thromb Res 1973;2:487-508.
5. Blondheim DS, Plich M, Berman M, Khair G, Tzvig L, Ezri J, *et al*. Acute myocardial infarction complicating viper bite. Am J Cardiol 1996;78:492-3.
6. Wollberg Z, Bdolah A, Kochva E. Cardiovascular effects of mammalian endothelins and snake venomsarafotoxins. In: Abrahams S, Amitai G, editors. Calcium Channel Modulators in Heart and Smooth Muscle. Deerfield Beach, FL: VCH; 1990. p. 283-99.
7. Maheshwari M, Mittal SR. Acute myocardial infarction complicating snake bite. J Assoc Physicians India 2004;52:63-4.
8. Tony JC, Bhat R. Acute myocardial infarction following snake bite. Trop Doct 1995;3:137.
9. Dissanayake P, Sellahewa KH. Acute myocardial infarction in a patient with Russell's viper bite. Ceylon Med J 1996;41:67-8.
10. Hoffman A, Levi O, Orgad U, Nyska A. Myocarditis following envenoming with viperae palaestinae in two horses. Toxicon 1993;31:1623-8.
11. Rowlands JB, Mastaglia FL, Kakalus BA, Hainsworth D. Clinical and pathological aspects of a fatal case of mulga (*Pseudechis australis*) snakebite. Med J Aust 1969;1:226-30.
12. Sathyanathan VP, Mathew TM. Raynaud's phenomenon and gangrene following snake envenomation. J Assoc Phys India 1993;41:122-3.
13. Suchithra N, Pappachan JM, Sujathan P. Snakebite envenoming in Kerala, South India: Clinical profile and factors involved in adverse outcomes. Emerg Med J 2008;25:200-4.

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