

A case report on Bradycardia: a rare manifestation of Saw scaled viper bite.

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Abstract

Saw scaled viper is one of the venomous snakes in Sri Lanka. (1) It has different species worldwide. Bleeding manifestations and mortality are varying among them. There is no reported case on saw scaled viper bite related death in Sri Lanka. (1) Coagulopathy is the commonest manifestation. Here we are reporting the case report on Bradycardia following saw scaled viper bite for the first time. It has occurred after 72 hours and recovered with isoprenaline.

Introduction

Envenoming by *Echis* species is thought to be responsible for more snakebite deaths worldwide than from any other snake species. (2) Saw scaled viper is the one of the six venomous snakes in Sri Lanka. (3) Its generic name is *Echis carinatus*. (1,2) Its clinical manifestations are local reactions that vary from tenderness to necrosis of whole area of bitten part, coagulopathy and hemorrhage. (1,3) Less commonly they can cause renal failure. (1) Very rarely they can be cardiotoxic. But there is no documented neurotoxicity. Saw scaled viper venom produces lot of hemotoxins that disturb our body coagulatory mechanism. Fortunately, we have effective antivenom to neutralize its toxins.

Case report

He is 32-year-old healthy man, father of 3 children, doing fishing, coming from Kilinochchi. He got saw scaled viper bite in the right foot while he was doing works in the garden in evening. He developed pain and abnormal sensation over bitten area. errated scales.

He was admitted to ETU and kept on monitoring. He was cannulated, and catheterized. On inquiry he didn't complain abdominal pain, nausea and

vomiting. But he felt generalized ache and malaise. He did not develop any bleeding manifestations. He had adequate urine output. There were no features of neuro toxicity such as dyspnea, swallowing difficulty, ophthalmoplegia and double vision. He did not complain of dizziness, palpitation or chest pain. Examination showed he was anxious and tenderness and erythema around the bitten area. There were no signs of neurotoxicity such as ptosis, ophthalmoplegia or cranial nerve palsy. His BP was 130/80 mmHg and PR 110bpm. His respiratory rate was 15/min. Lungs were clear with SpO₂ 99%. Abdomen examination was unremarkable.

He was reassured. His initial Whole blood clotting time (WBCT) showed more than 20 minutes twice. Then he was given with 10 vials of AVS. But his repeat WBCT 6 hours later showed more than 20 minutes. Second AVS given. Next WBCT was normal. His blood investigations from admission to general hospital showed WBC 15,000/mm³, Hb 15.4 g/dl without any significant drop, platelet dropped from 220,000/mm³ to 15,000/mm³ on second day and then raised to 160,000/mm³. Recorded high INR was 2.86 and APTT was 74s with PT 24 sec on day 2. Then following days it gradually normalized to INR 1.01 Total bilirubin was 0.91mmol/dl. Liver enzymes were AST 28 IU/l, ALT 31 IU/l and ALP 100 IU/l. Serum creatinine was 1.09mg/dl. Serum electrolytes were Na 143meq/l and K 4.1meq/l. CRP was 4.5mg/dl.

On 3rd day he developed dizziness. At that time, he had heart Rate of 38 bpm which was regular and Blood Pressure was 90/50 mmHg. He was started with isoprenaline 5ug /min infusion with fluid bolus. His HR improved around 58 bpm. He was transferred to Teaching hospital

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Jaffna for cardiologist opinion and insertion of Temporary pace maker. On arrival he had heart rate 50bpm, blood pressure 90/60 mmHg. He was asymptomatic.

We didn't give further dose of ASV because he was given twice antivenom and WBCT was normal. We contacted cardiologist and they suggested to continue isoprenaline at that moment as his heart rate was reasonable and if HR is refractory to drug then we will go for TPM. He denied any history of chest pain, palpitation during these hospital days from admission. He did not have any past history of dizziness, blackouts, presyncope or syncope. There was no family history of any heart disease or having Pacemaker implantation. He did not have any history heart disease in childhood. he was not on any long-term cardiac drugs. At this time also, he was treated with cloxacillin only. His troponin was twice negative. ECG showed sinus bradycardia. There were no features of myocardial infarction / ischemia. His follow up ECG also did not reveal any etiology. TSH and T3 was within normal range. ECHO was normal. We gradually tailed off isoprenaline infusion without any drop in HR. Once the infusion was omitted he was maintaining HR around 68 bpm. He was discharged on day 5. On reviewing after one week his heart rate was 66 bpm and BP was 100/80 mmHg.

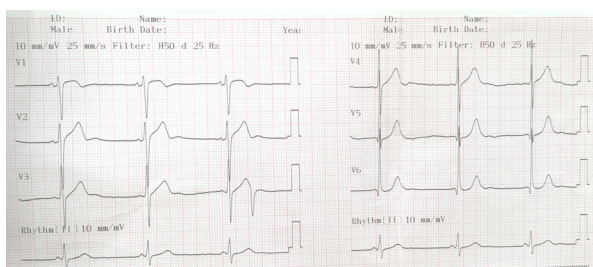


Figure : sinus bradycardia

Discussion

There are 96 species of snake, identified in Sri Lanka so far, out of which, only six are deadly venomous. (5) In which saw scaled viper is the one responsible for highest number of bites in Jaffna. (1) In Tamil it is called as 'suruddai' / 'viriyam paampoo' and in Sinhala 'vali polanga'. (1,2)

Its distribution includes the coastal areas of the Northern and Eastern Provinces extending as far south as Yala. This patient also came from one of these coastal areas.(6)

Toxins in the venom are phospholipase A2, procoagulant enzymes, metalloproteinases, prothrombin (factor II) activators; namely, ecarin and carinactivase, platelet aggregation inhibitors namely carinatin, echistatin, and echicetin, protein C activator, fibrinogenolysin, Ca²⁺ dependent carinactivase, and disintegrins (2,4) and some unknown cytotoxins including cardiotoxins(7)

Circulatory effects of *E. carinatus* venom observed in experimental animals include hypotension and bradycardia due to myocardial depression, mesenteric vasodilatation(8) or release of histamine (9) Electrocardiographic abnormalities following snake bite may be due to direct cardiotoxic action of venom, hypotension or electrolyte disturbances. (7) Some effects of cardiotoxin resemble those of digitalis.(7) ECG abnormalities which included sinus tachycardia and arrhythmia, bradycardia, tall T-waves and abnormalities suggestive of myocardial ischemia and nonspecific T-wave abnormalities. Atrioventricular blocks were also seen. (10) However, in Agarwal et al (11) bradycardia occurred due to parasympathetic stimulation and also as a result of severe fear.

This patient developed bradycardia after 72 hours and not associated with significant low blood pressure. Therefore, parasympathetic stimulation is less likely cause for Bradycardia. He is a young patient and troponin was also negative. Therefore, Ischemia is less likely. Cloxacillin also doesn't cause bradycardia. There were no precipitating factors or attributable causes. So, it is most likely due to venom effect rather than parasympathetic stimulation. As there are no case reports available and no data regarding bradycardia related to saw scaled viper bite. Further we should consider cardiac manifestations too following viper bite.

Conclusion

The manifestations of Saw scaled viper bite can vary from country to country according to their sub species and venoms production. The main

toxic effects apart from local manifestations are coagulopathy and hemorrhage. Very rarely its venom can also cause cardiotoxicity that can manifest either infarction, myocarditis, bradycardia or arrhythmias. Bradycardia can occur in first day or in 72 hours even after AVS given and it is reversible. We can manage it temporarily either isoprenaline infusion or temporary pace maker while searching for other causes.

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