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Case Report

Cardiovascular involvement following a hump nosed viper bite – a rare complication

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Introduction

Snake bites are commonly encountered in medical practice, especially in the dry zone of Sri Lanka [1]. The hump nosed viper is a highly venomous snake. It is a pit viper categorized under the Genus *Hypnale* of which three species are found in Sri Lanka, *Hypnale hypnale, hypnale zara* and *hypnale nepa* [2]. Hump nosed viper bites are commonly associated with local envenomation, such as haemorrhagic blisters and local necrosis. Systemic manifestation are rare and reported complications include coagulopathy and renal impairment. In general, cardio toxicity following snake bites is rare and cardiac involvement following hump nosed viper bites is extremely rare [3]. We could only find one such case in the literature. Here, we present two cases of cardiovascular complications following hump nosed viper bite.

Case report 01

A 36-year-old, previously healthy female from Anuradhapura, Sri Lanka, presented to the hospital following hump nosed viper bite. The snake was identified by the patient and, subsequently, by the hospital staff. Ten minutes after the bite she complained of chest tightness and faintishness following which she collapsed. She was in asystolic cardiac arrest on admission to the local hospital. She was successfully resuscitated with IV adrenaline and cardiac massage after 5 minutes and transferred to Teaching Hospital, Anuradhapura for intensive care unit (ICU) care. On admission, she was haemodynamically stable. She was electively intubated and managed in the ICU by a multidisciplinary team.

A 12 lead electrocardiogram (ECG) showed ST segment depression in lead II, III and aVF. Highly sensitive troponin I titre was elevated and the 2D echocardiogram (2D ECHO) showed mild inferior wall hypokinesia. An elevated white cell count with

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neutrophil predominance was noted in the full blood count. Serum electrolytes were normal. INR and APTT were both prolonged. A noncontrast CT of the brain showed gross cerebral oedema. Electroencephalogram done on day 8 of admission revealed hypoxic ischaemic encephalopathy.

She was treated with supportive management, tracheostomy and intensive physiotherapy. During the ICU stay her clotting profile and ECG changes normalized. She made a slow and steady recovery and was discharged after one month of hospital stay.

Case report 2

A 65-year-old female with a past history of hypertension and unstable angina presented with a history of hump nosed viper bite on the right hand. Offending snake was brought to the hospital and identified as a hump nosed viper. Initially, the patient was stable with mild to moderate local pain but later developed worsening ischaemic type chest pain. Her ECG showed ST segment depression in lead II, III and aVF with an elevated high sensitive troponin I titer. Her 2D echo was normal without any regional wall motion abnormality. Other basic investigations and clotting profile was normal. She was treated with antibiotics, analgesics and subcutaneous enoxaparin for three days in addition to her antihypertensive and anti ischaemic drugs. Her troponin I titer dropped with treatment with normalization of the ECG changes. Patient made a uncomplicated recovery.

Both patients underwent coronary angiogram to exclude coronary artery obstruction.

Discussion

Hump nosed viper bites commonly cause local envenomation including haemorrhagic blisters and local necrosis whereas systemic complications are rare [1,4]. Frequently reported systemic complications include coagulopathy and acute renal failure [1,4]. In the past, it was believed that hump nosed viper bite and Russell's viper bite did not cause any significant cardiac involvement [4,5]. However, there have been a few published case reports challenging this view with regard to Russell's viper [6,7]..But we could find only one case report of cardiac involvement due to a hump nosed viper bite [3].

Hump nosed viper venom is a mixture of compounds such as phospholipase A2, snake venom metalloprotease, L-amino acid, C-type lectin and snake venom serine protease (SVSP) [8]. These compounds have diverse actions. Cardiac involvement could be the combined result of several mechanisms including direct cardiac toxicity, haemorrhage and hypotensive shock following coagulopathy, vascular inflammation and thrombosis, coronary vaso-constriction, anaphylaxis, conduction abnormalities and tachycardia leading to increased oxygen demand [3,6].

Our first patient suffered an ischaemic chest tightness and cardiac arrest shortly after the bite. Most possible mechanisms could be anaphylaxis or arrhythmia due to hump nosed viper venom with possible myocardial ischaemia as evidenced by ECG changes and elevated troponin. Cardiopulmonary resuscitation can also contribute to elevated troponin. Our second patient had ECG changes and very high troponin titers which point towards direct involvement of the myocardium either due to ischaemia or direct myocardial toxicity with varying degree of contribution from other mechanisms. As coronary angiogram did not reveal any critical stenosis or occlusion, coronary artery thrombosis is less likely to be the cause behind these presentations. With the temporal association of events it was concluded that the abnormal ECG findings and elevated troponin in these patients were directly attributed to the hump nosed viper bite.

Conclusion

Hump nosed viper bite can cause significant morbidity due to cardiac involvement. Further research and studies are needed regarding this arm of envenomation as well as development of specific management protocols for a better patient outcome.

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