A Case of Acute Myocardial Infarction in a Neurotoxic Snake Bite

Ronald Jaison Melit*, Siju V. Abraham, Krishna Das, Vivek Gopinathan, and Vimal Krishan S
Department of Emergency Medicine, Jubilee Mission Medical College and Research Institute, India

Abstract
We report a case of an 85 year old female, with signs of neurotoxicity following snake bite who later developed an acute myocardial infarction. This unusual case highlights the importance of diagnosis and management of myocardial infarction that simultaneously present in a victim of snake bite.

ABBREVIATIONS
ASV: Anti Snake Venom; CABG: Coronary Artery Bypass-Graft; LAD: Left Anterior Descending; D2: Diagonal 2; PDA: Posterior Descending Artery; PTCA: Percutaneous Transluminal Coronary Angioplasty; RCA: Right Coronary Artery

INTRODUCTION
Russels viper bites have been reported to cause ischemic cardiac events [1], arrhythmias [2] and cardiac tamponade [3]. Though myocardial infarction following Russell’s viper bite has been previously reported [4], neurotoxic bite following which a patient developed myocardial infarction has not been reported in any prior literature reviews.

CASE PRESENTATION
85 year old lady presented with a history of snake bite to the emergency department. Her heart rate was110 per minute and blood pressure of 150 over 90mmHg. She had a saturation of 98% in room air and respiratory rate of 22 per minute. She had developed ptosis and respiratory distress by the time she reached the emergency department which responded to a trial of Neostigmine and atropine.

On examination, bite mark was seen and the patient had local bite site edema and tenderness. 10 vial ASV was administered initially and repeated later as per institutional protocol. Clotting time at admission was 14 minutes and INR 1.2. Her whole blood clotting time was never prolonged during the course of treatment. While being treated she developed central chest discomfort with radiation to back and left upper limb. ECG taken revealed ST segment elevation in leads V [2-4] and T inversion in leads II, III, aVF, V [5,6] (Figure 1). Troponin- T was positive at 0.249 ng/ml. An ECHO done showed a mid anteroseptal and anterior wall hypokinesia, with grade 2 left ventricular diastolic dysfunction.

Cardiology opinion was sought and coronary angiogram was done which revealed a double vessel disease. She underwent a coronary artery bypass-graft (CABG) to Left Anterior Descending (LAD), Diagonal 2 (D2) and Posterior Descending Artery (PDA). Percutaneous Transluminal Coronary Angioplasty (PTCA) with stent to Right Coronary Artery (RCA) and Left Anterior Descending (LAD) artery. Post procedure period was uneventful. Patient was treated symptomatically, she improved and was discharged.

DISCUSSION
In central Kerala, Jubilee Mission Medical College acts as a referral centre for snake bite victims, where anti snake venom is supplied free of cost. Preliminary estimate of the internal audit being done over the last 2 years (2013-2015) shows that 886 adult bite victims were administered ASV in our institute. This was a unique situation where the victim with signs and symptoms suggestive of neurotoxic envenomation, developed an acute myocardial infarction.

The exact mechanism by which snake bite leads to myocardial infarction is yet to be delineated. The mechanisms which have been proposed to explain cardiac damage following snake bite includes hypovolemic shock following bleeding due to hemorrhagins, consumptive coagulopathy [5], direct cardiotoxic effect on myocardium [6,7] and coronary spasms [8,9] due to endothelins or sarafotoxins [10]. Russels viper bite may present with both neurotoxic and cardiotoxic symptoms [4]. Acute Myocardial Infarction following Russell’s viper bite and possibility of predominant coagulant in venom resulting in coronary thrombosis and anaphylactic shock leading to MI has
been described. Literature review done showed no reports of myocardial infarction following a neurotoxic bite [1-11]. The cardiotoxic component of cobra venom cobramin B has direct action on cardiac muscle causing cardiac arrhythmias, various heart block, circulatory failure and cardiac arrest. The cardiotoxic content of cobra venom has direct action on skeletal, cardiac, smooth muscles, nerves and neuromuscular junction [11]. The threat of impending death causes a surge of catecholamine [12,13]. This would lead to increased myocardial oxygen demand which would worsen the demand supply mismatch. Either the direct cardio toxicity of the venom, the catecholamine surge, or maybe even the atropine administered could have unmasked the underlying triple vessel disease [14].

CONCLUSION

Recognizing the heavy economic, physical and psychological toll snake bite has on the victim, the World Health Organization categorized snake bite as a neglected tropical disease in 2009 [15]. The magnitude of the issue is often under estimated by the developing nations probably because of the compliancy of both the victim and the physician tending to the ailment [15,16].

A case of predominantly ‘neurotoxic’ snake bite envenomation with concomitant acute myocardial infarction is rarely reported. The impact of these acute illnesses presenting along with snake bite envenomation complicates the diagnostic and management strategies. We report this case as it is an unusual presentation the ED physician has to encounter dealing with an acute Myocardial Infarction in the setting of snake envenomation with predominant neurotoxic signs and symptoms.

REFERENCES