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## Cardiac complication following Hemotoxic snake bite: A rare case from Eastern India

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### Abstract

Primary cardiovascular complications following viper envenomation have been sparsely observed and remain poorly characterized in literature, despite South Asia being one of the largest snake bite prevalent zones. Venom induced coagulopathy, endothelial injury and hemodynamic disturbances on the other hand have been well documented events. We report a rare case of an inferior wall ST-segment elevation myocardial infarction (STEMI) following a hemotoxic viper bite in a 55-year-old male. The patient initially presented with localized swelling at the bite site, gross hematuria, and a whole blood clotting time exceeding 20 minutes, necessitating polyvalent antivenom administration. Six hours post-envenomation, he developed chest tightness and vomiting. A 12-lead electrocardiogram supported by elevated cardiac enzymes confirmed a diagnosis of inferior wall STEMI, occurring alongside an active venom-induced coagulopathy. Because of this coexisting bleeding manifestations, standard reperfusion strategies, including thrombolysis, anticoagulation, and antiplatelet therapy were not initiated, and the patient was managed conservatively with polyvalent anti-snake venom, supportive care, and high-intensity statin therapy, achieving resolution of cardiac symptoms within a day and cessation of hematuria by the 22nd hour with improving coagulation parameters over the next 3 days. Coronary angiography performed on follow-up revealed significant proximal right coronary artery stenosis, suggesting that venom-induced mechanisms, such as hypercoagulability or vasospasm, may precipitate acute ischemia in patients with undiagnosed atherosclerotic lesions. This case underscores the rarity of viper envenomation-induced cardiotoxicity, most of which have been documented in patients without identifiable coronary risk factors. It further illustrates the added diagnostic challenges of managing myocardial infarction when conventional reperfusion protocols are precluded by severe hemotoxicity.

**Keywords:** Snake bite, STEMI, Hemotoxic coagulopathy, Viper venom, Coronary stenosis

### INTRODUCTION

Russel's viper (*Dabioa russelii*) is one of India's most toxic snakes and found in the Indian gangetic plain. It is distributed widely across South Bengal in districts such as Paschim Midnapore, Hooghly and Purba Burdwan.<sup>1</sup> Russel Viper bites account for approximately 65% of snake bite cases in West Bengal.<sup>2</sup> Its venom can produce extensive local tissue injury and systemic complications, including coagulopathy, hemolysis, bleeding, acute kidney injury, neurological manifestations, and thrombotic microangiopathy.<sup>3</sup> Although the clinical course may be predictable, severe envenoming can progress rapidly despite timely treatment. Cardiovascular complications are uncommon but potentially fatal. Reported manifestations include myocardial ischemia or infarction, arrhythmias, cardiac arrest, and, rarely, cardiac tamponade.<sup>4,5</sup> These complications may result from venom-induced endothelial injury, thrombosis, coagulation abnormalities, vasospasm, hypotension, or direct myocardial injury.<sup>6</sup> Acute myocardial infarction has been described even in patients without established cardiovascular disease or conventional cardiac risk factors.<sup>7,8</sup> Here we present a case of Inferior Wall ST segment elevated Myocardial Infarction following viper bite.



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## CASE PRESENTATION

A 55-year-old male farmer presented to the Emergency Department with complaint of Viper bite approximately 3cm above the lateral malleolus of the right leg about an hour back. The patient was a local resident and was positive regarding the identification of the snake. The patient was fully conscious, alert, and hemodynamically stable at arrival. He had a lean, moderately muscular built. His blood pressure was 130/80, pulse rate 96 beats/min, regular, respiratory rate 20/min, SpO<sub>2</sub> 96% in room air and CBG 115mg/dL. The patient was visibly anxious, and panicking about the situation. Local examination of the wound site revealed swelling, and tenderness around the alleged bite region. There were two fang marks approximately 0.5cm apart, which were oozing. The patient had tied a tourniquet approximately 5cm above the knee on advice of neighbors.

There was no history of pain abdomen, vomiting, hemoptysis, epistaxis, bleeding from other areas, ptosis, breathing difficulty or heaviness of chest. Cardiac, Neurological and Abdominal examination revealed no abnormality, and the single breath count of the patient was above 25. The patient was a chronic smoker and was a known case of COPD (GOLD 2, mMRC 1) on MDI (Group E). The patient did not have other co-morbidities or a significant medical history. The patient denied using any recreational drugs or alcohol in the preceding 1 week. There was no history of hematuria. Whole blood clotting time (WBCT) was more than 20 minutes.

Local site wound care was done by slowly loosening the tourniquet as per Standard Operating Procedure, and washing the bite site with warm saline and soap. The patient was administered tetanus prophylaxis (1cc Tetanus toxoid intramuscularly) and immediately started on 10 vials of Polyvalent Antisnake Venom (ASV) as per guidelines laid down by the "Medical Officers' Training Module on Snake Bite, Animal Bite and Common poisonous bites."<sup>9</sup> Blood sample for relevant laboratory examination was sent simultaneously. Since gross hematuria was noted despite ASV, a second dose of 10 vials was given.

At the 6th hour following snake bite and 5th hour following 1st dose of ASV, the patient complained of chest tightness along with slight breathing difficulty. Initially, the patient's symptoms was attributed to the anxiety. However, when the patient reported worsening of symptoms after 30 minutes and had an episode of vomiting, an ECG was ordered.

A 12 lead ECG revealed Inferior Wall ST segment elevation (STEMI). The patient was promptly shifted to the ICU. The patient was profusely sweating and had another episode of vomiting during the ICU transfer. In the ICU, the patient had a blood pressure of 100/70, heart rate of 128 beats/min, respiratory rate of 22/min, SpO<sub>2</sub> of 90% and CBG of 123mg/dL. Rapid Trop T card test was positive. Trop I showed a value of 21ng/mL (ref <0.1ng/mL). A provisional diagnosis of Inferior Wall ST segment elevated Myocardial Infarction in a case of Hemotoxic Snake Bite was made. Another ECG taken about 20 minutes later exhibited similar findings, with slight tachycardia. (Fig 1). A complete hemogram

showed leukocytosis (13400/cu.mm) and mild anemia (10.2g/dL). Bedside ABG showed mild respiratory alkalosis. CK-MB was 8 IU/L (ref<25IU/L). Prothrombin Time (PT) was 22 seconds (control 14 sec) and international normalized ratio (INR) was 1.6. Given the patient's hematuria, bite site oozing and WBCT>20mins, thrombolysis, anticoagulants and antiplatelets were not considered. The patient was given supplemental oxygen at 4L/min through a face mask and high dose statin (Atorvastatin 80mg). Cardiac examination of the patient remained unremarkable with clear lung sounds bilaterally. Bedside X-ray showed findings consistent with COPD. Bedside Echocardiography could not be done due to logistical limitations. Urinary Catheterization was done and patient was put on fluid support along with analgesics.

The patient and his party were counselled regarding the unavailability of Primary Percutaneous Coronary Intervention (PPCI) at the facility, and the importance of such. Further exploration revealed no history of coronary or atherosclerotic disease in the patient or relatives. There was no family history of sudden or unexplained deaths.

4 hours following ICU transfer, the patient's chest pain had reduced. By this time, laboratory results were obtained which showed mild elevation in liver enzymes - SGOT 98 IU/L, SGPT 68 IU/L; and LDH 344 IU/L. D-dimer showed a value of 4.8 mg/L (ref <0.5mg/L). Urine output remained over 0.5ml/kg/hr. Based on these findings, and the persistent hematuria, a further 10 vials of ASV was given. The patient received the maximal dose of 30 vials of ASV.

Six hours after the last dose of ASV, WBCT was 13 minutes. Hematuria eventually stopped the next day. However, by this time, the local site was severely swollen and tender. Surgery opinion was taken regarding the wound site, and magnesium sulphate dressing along with antibiotics was suggested. The patient did not require oxygen support by the next morning. The urine output of the patient consistently remained above 0.5ml/kg/hr and creatinine rise was <0.3mg/dL over 72 hours - (Baseline 0.9mg/dL; 1.0mg/dL at 24 hours 0.9mg/dL at 48 hours). Dialysis was not needed for this patient. Serial PT, INR and D-dimer values showed a declining trend. The patient was discharged without further complications on day 7.

The patient followed up at the Medicine OPD a month later. He successfully underwent a Coronary Angiogram at a Cardiac facility post discharge (day 13 post bite) which showed significant stenosis at the proximal RCA region. The patient did not undergo a secondary PCI due to personal reasons. He remains on dual antiplatelet therapy along with high intensity statin, ACE inhibitor and other antianginal drugs. The patient had an unremarkable follow up in the OPD for the next 3 months.

## DISCUSSION

Snake bit is a common hazard in rural India. Viper bites are especially common because of water logging and the infestation of rodents in villages.<sup>10</sup> Viper envenomation is characterized primar-



ily by local tissue reaction, hemorrhagic manifestations and nephrotoxicity.<sup>11</sup> Cardiac manifestations, however are uncommon and have only sporadically been referenced for snake bites, particularly viper bites.<sup>12</sup> Even more sporadic are coagulopathies such as DIC, or stroke syndromes.<sup>13,14,15</sup>

Hemotoxicity is a classical feature of viper snakebite. Envenomation can cause hypotension through cardiovascular and hemostatic effects. Snake venom metalloproteinases damage capillary basement membrane, increasing vascular leakage.<sup>16</sup> In the present scenario, however, shock was not encountered. Snake venoms commonly cause local and systemic hemorrhage, with signs such as bleeding from the gums, wounds, bite site, gastrointestinal or genitourinary tracts, hematemesis, and hemoptysis.<sup>17</sup> The present patient had hematuria, which resolved following ASV administration. In regions where snakebite is common, bedside tests such as the 20-minute whole blood clotting test are widely used to detect incoagulable blood. However, patients with venom-induced consumption coagulopathy (VICC) also show markedly prolonged prothrombin time, international normalized ratio, and activated partial thromboplastin time, often beyond the assay's measurable range.<sup>18</sup> Although systemic bleeding can worsen shock-related deaths, hemorrhage itself can be fatal, especially intracranial bleeding.<sup>19</sup>

The precise mechanism of Acute Myocardial Infarction (AMI) following viper bite has not been well understood. Although mechanisms such as hypovolemic shock due to hemorrhage, direct toxic effect on heart muscle, coronary spasm induced by sudden release of tourniquet or endothelins have been proposed, the exact mechanism remains inconclusive.<sup>6,20,21</sup> Direct cardiotoxicity have only been demonstrated in mammalian models.<sup>22</sup> Myocarditis still remains the most common among the cardiac complications encountered following viper bites.<sup>23</sup>

In our patient, the initial presentation was typical of a viper bite, with cardiac symptoms appearing 6 hours following envenomation. Attribution of chest tightness to anxiety is a major teaching point. Anxiety like presentation, although atypical, may be a presenting or early feature of Myocardial Infarction.<sup>24</sup> Tachycardia which, was experienced by the patient is a rare, but known finding with Inferior Wall AMI.<sup>25</sup> Smoking was a major risk factor in our case and a previous case report has considered atherosclerosis, venom-induced hypercoagulability and vasospasm as interplaying factors for AMI in a pre-existing atherosclerotic lesion.<sup>26</sup>

Managing AMI in such patients is difficult because standard reperfusion therapies are contraindicated when coagulopathy is present or suspected. Although the patient party refused transfer to a PCI center in our case, Primary angioplasty in snake bite raises its own issues.<sup>26</sup> Improvement with ASV alone has been demonstrated in snake bite induced AMIs.<sup>27</sup>

## CONCLUSION

This case highlights that chest symptoms in snake bite/animal bites should not be dismissed as anxiety, and early ECG and cardiac markers allow rapid escalation of treatments even in resource poor settings. Furthermore, the importance of ASV in any snake bite

case demonstrating envenomation has been underlined.

## CONFLICT OF INTEREST

None Declared

## FUNDING

None Declared

## AI USE DECLARATION

AI model (Claude, Opus 5.5) was used for background research and grammatical correction of the manuscript.

## DECLARATION OF PATIENT CONSENT

Appropriate patient consent was obtained by the authors for taking and sharing pictures.

## FIGURES

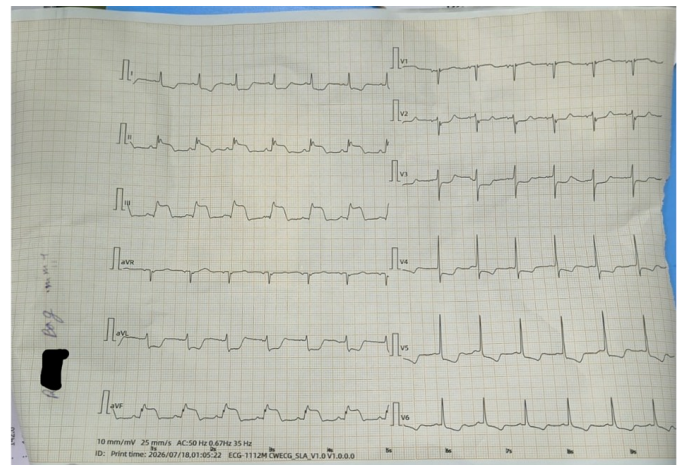


**Figure 1:** Showing Inferior Wall STEMI (Repeat ECG)



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