DOI: 10.21088/ijem.2395.311X.12226.10

Indian Journal of Emergency Medicine
Volume 12 Number 2, April–June 2026

Case Report

Antisnake Venom in Treatment of Acute Myocardial Infarction

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How to cite this article:

Srishty Vij, Sonam Kumari, Arun Kaushik, et al. Antisnake Venom in Treatment of Acute Myocardial Infarction. Ind J Emerg Med. 2026; 12(2): 108-111.

ABSTRACT

Snake envenomation is a common medical emergency in tropical regions and is typically associated with neurotoxic, hemotoxic, or myotoxic manifestations. Cardiovascular complications are rare but potentially life-threatening. Acute myocardial infarction (AMI) following snakebite is an uncommon and poorly understood phenomenon, often leading to diagnostic and therapeutic challenges in emergency settings. We report a rare case of acute myocardial infarction occurring after snake envenomation, which showed significant clinical improvement following timely administration of polyvalent snake antivenom along with standard supportive care. A middle-aged male patient presented to the emergency department with a history of snakebite, followed by chest pain, diaphoresis, and hemodynamic instability. Electrocardiography revealed ST-segment elevation consistent with acute myocardial infarction, while cardiac biomarkers were markedly elevated. The patient had no prior history of coronary artery disease or traditional cardiovascular risk factors. Early administration of snake antivenom resulted in rapid stabilization, improvement in electrocardiographic changes, and normalization of cardiac enzymes. This case highlights the importance of recognizing acute coronary events as a possible complication of snake envenomation. Early diagnosis and prompt antivenom therapy may play a crucial role in reversing myocardial injury and improving outcomes. Clinicians in snakebite-endemic regions should maintain a high index of suspicion for cardiac involvement in such patients.

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➢ **Received :** 16-01-2026 ➢ **Accepted :** 17-02-2026

KEYWORDS

• Snake Antivenom • Myocardial Infarction • Kounis syndrom

INTRODUCTION

Snakebite remains a significant public health problem in many parts of the world, particularly in South Asia, Africa, and Southeast Asia. The clinical manifestations of snake envenomation depend on the species involved, venom composition, and the amount of venom injected. Common systemic effects include neurotoxicity, coagulopathy, rhabdomyolysis, acute kidney injury, and local tissue necrosis. Cardiovascular complications are rarely reported but can be fatal if not promptly identified and managed.

Acute myocardial infarction following snakebite is an unusual presentation and is often underrecognized. Proposed mechanisms include venom-induced coronary artery spasm, direct myocardial toxicity, hypercoagulable states, endothelial injury, and hypotension leading to myocardial ischemia. In some cases, allergic or anaphylactoid reactions to venom may trigger coronary vasospasm, a phenomenon described as ***Kounis syndrome***.

The diagnosis of AMI in snakebite victims can be challenging, as chest pain may be attributed to anxiety, autonomic effects, or systemic envenomation. Additionally, standard thrombolytic therapy poses significant risks in the presence of venom-induced coagulopathy, making management decisions complex.

Snake antivenom remains the cornerstone of treatment for systemic envenomation. Its role in reversing cardiovascular complications, particularly myocardial ischemia, is not well established but is increasingly recognized in isolated Case Report. Early neutralization of circulating venom may halt ongoing myocardial injury and prevent further complications.

We present a rare case of acute myocardial infarction occurring after snakebite, which demonstrated marked clinical and electrocardiographic improvement following administration of snake antivenom. This report aims to raise awareness of this uncommon complication and emphasize the importance of early recognition and appropriate management.

CASE REPORT

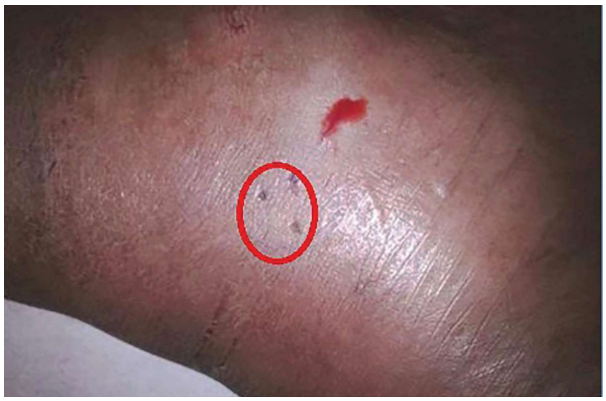

Figure 1: Snake Bite fangs mark (highlighted) (***Source:*** Department of Emergency Medicine, Max SHBG, New Delhi)

A middle-aged male with no known comorbidities presented to the emergency department approximately two hours after sustaining a snakebite to the lower limb while working outdoors. The patient initially complained of localized pain and swelling at the bite site, followed by the sudden onset of retrosternal chest pain, profuse sweating, and shortness of breath.

On arrival, the patient was anxious, tachycardic, and hypotensive. Physical examination revealed fang marks with mild local edema, but no significant bleeding or neuroparalytic signs. Cardiovascular examination showed tachycardia without murmurs. Respiratory and neurological examinations were unremarkable.

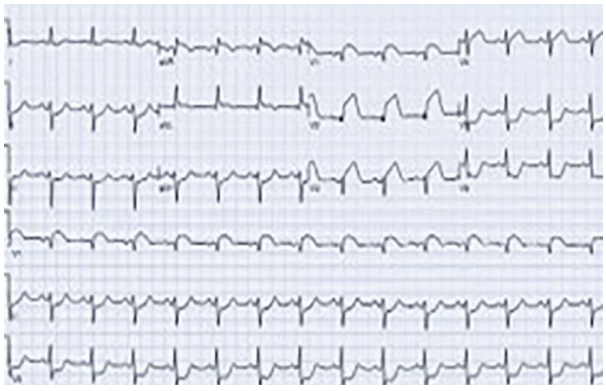

Figure 2: Classical Acute Myocardial Infarction (***Source:*** Department of ED, Max SHBG, New Delhi)

An immediate electrocardiogram revealed ST-segment elevation in the anterior leads, suggestive of acute anterior wall myocardial infarction. Laboratory investigations showed elevated cardiac biomarkers, including troponin I and creatine kinase-MB. Routine coagulation parameters were mildly deranged, consistent with early hemotoxic envenomation. There was no prior history of ischemic heart disease, diabetes, hypertension, or smoking.

Given the temporal relationship between snakebite and cardiac symptoms, a diagnosis of acute myocardial infarction secondary to snake bite was considered. Thrombolytic therapy was deferred due to the risk of bleeding associated with venom-induced coagulopathy. The patient was managed with oxygen, analgesia, antiplatelet agents under close monitoring, and prompt administration of polyvalent snake antivenom.

Following antivenom therapy, the patient showed significant clinical improvement within hours. Chest pain subsided, blood pressure stabilized, and repeat electrocardiography demonstrated partial resolution of ST-segment changes. Serial cardiac biomarkers showed a declining trend over the next 48 hours. Echocardiography revealed mild left ventricular dysfunction without regional wall motion abnormalities.

The patient was monitored in the intensive care unit and continued on supportive therapy. No bleeding complications or allergic reactions to antivenom were observed. He was discharged in stable condition after one week with advice for cardiology follow-up. At subsequent review, the patient remained asymptomatic with normal cardiac function.

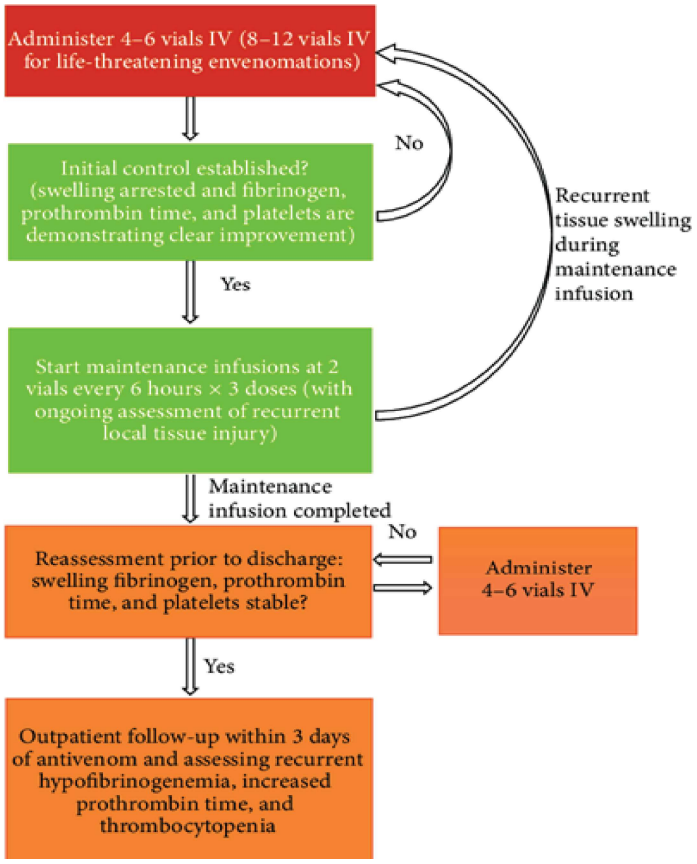

Figure 3: Snake-Bite Management-Envenomation
(***Source:*** CDC, WHO guidelines)

DISCUSSION

Acute myocardial infarction following snakebite is a rare but serious complication that requires high clinical suspicion. The pathophysiology is multifactorial and not fully understood. Proposed mechanisms include direct cardiotoxic effects of venom, coronary artery vasospasm, platelet aggregation, venom-induced hypercoagulability, and systemic hypotension leading to myocardial ischemia.

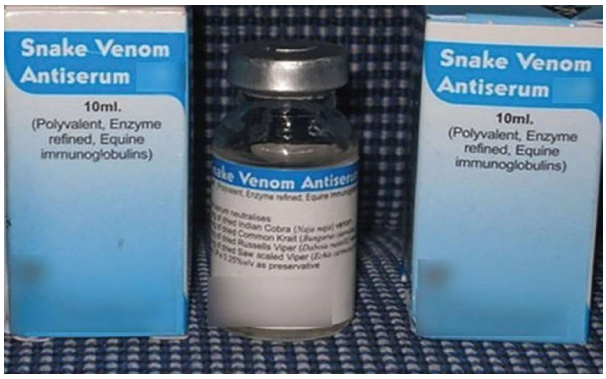

Figure 4: Anti-Snake Venom (***Source:*** Google Search Engine, CDE, Guidelines)

In the present case, the absence of traditional cardiovascular risk factors and the close temporal association between snakebite and symptom onset suggest a venom-mediated mechanism. The rapid improvement following antivenom administration supports the role of circulating venom in ongoing myocardial injury. Early neutralization of venom may prevent progression of ischemia and reduce myocardial damage.

Management of AMI in snakebite patients poses unique challenges. Standard reperfusion strategies, such as thrombolysis, carry a significant risk due to venom-related coagulopathy. Therefore, treatment must be individualized, balancing the benefits of antithrombotic therapy against bleeding risks. Antivenom remains the most critical intervention, as it addresses the underlying cause.

Previous Case Report have described variable outcomes, ranging from spontaneous recovery to fatal arrhythmias. Early diagnosis, continuous cardiac monitoring, and multidisciplinary management are essential for favourable outcomes.

This case underscores the need for awareness of cardiac complications in snake envenomation, especially in endemic regions. Prompt ECG evaluation and cardiac enzyme assessment should be considered in snakebite patients presenting with chest pain or hemodynamic instability.

CONCLUSION

Acute myocardial infarction is a rare but potentially life-threatening complication of snake envenomation. Early recognition and prompt administration of snake antivenom may play a pivotal role in reversing myocardial ischemia and improving patient outcomes. Clinicians should maintain a high index of suspicion for cardiac involvement in snakebite victims presenting with chest pain or cardiovascular instability. Careful monitoring and individualized management are essential to minimize complications and ensure recovery.

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