Acute Myocardial Infarction Complicating Snake Bite

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Abstract
A case of acute myocardial infarction following viper snake bite is reported. Possible mechanisms are discussed.

INTRODUCTION
Clinical picture following viper bite is usually characterized by local tissue reaction, haemorrhagic manifestations and nephrotoxicity. Cardiac involvement is rare. There are few case reports of acute myocardial infarction (MI) following snake bite.1-3 We describe herein a case of inferior myocardial infarction following viper snake bite.

CASE REPORT
A healthy 47 years farmer was bitten by a viper snake. Shortly after the bite, he experienced severe pain at the site of bite, with retrosternal chest pain, nausea, vertigo, episode of haemetemesis and collapsed. He was brought in the emergency department of hospital in a state of shock. On examination he was cyanosed, tachypnoeic, with respiratory rate 48/mt, systolic blood pressure 50 mmHg, pulse - 114/minute and a gallop sound audible on auscultation. Two fang marks were present on outer aspect of right big toe and whole right limb was swollen, painful and cyanosed.

Coagulation profile revealed prothrombin time prolonged to 28 seconds (control 12 sec). Platelet count was reduced to 82,000/cumm. Urine examination revealed 10-12 erythrocytes/high power field. Blood biochemistry was within normal limits. Electrocardiogram at the time of admission showed ST segment elevation in leads II, III, aVF (Fig. 1). On 3rd day subsequent ECG showed appearance of Q wave with T wave inversion in same leads (Fig. 2). Patient had no other risk factor for coronary artery disease nor did he have any past history of effort angina. Creatinine phosphokinase (MB subfraction) was elevated (68.8ng/ml) and there was hypokinesia of inferior wall of left ventricle on echocardiography. Diagnosis of concomitant acute inferior MI was considered. Patient was treated with snake antivenom, intravenous fluids, antibiotics and analgesics. After stabilization of blood pressure to more than 100/70 mmHg, nitroglycerine was started. Thrombolysis, anticoagulants and antiplatelets were not considered in view of history of haemetemesis and haemotoxicity of viper venom. Patient’s condition stabilized gradually. He did not agree for coronary angiography and was discharged 12 days after admission.

DISCUSSION
Viperine bites accounts for nearly half of snake bites in India. Viper venom is predominantly haemotoxic. Tony JC et al1 reported acute MI following snake bite in a 45 year old

Fig. 1 : ECG on 1st day showing ST segment elevation in inferior leads

Fig. 2 : ECG on 3rd day showing sequential changes of inferior infarction

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man who complained of breathlessness 29 hours after being bitten by viper. ECG showed evidence of recent MI. It was presumed to be due to action of sarafotoxin in snake venom, having vascular toxic effects leading to vasospasm in coronary vessels. David S et al² have also described acute MI following viper bite in a 28 year old man who presented in emergency department in state of shock, cyanosed and tachyoeic. ECG showed elevated ST segments in multiple leads. It was presumed that myocarditis with extensive myocardial necrosis secondary to toxic viper venom resulted in MI. Christ Aravanis et al³ have reported acute MI in a 17 year old girl who immediately after viper bite experienced severe substernal chest pain, nausea and then collapsed. Immediate direct toxic effect of venom on myocardial tissue was considered to be responsible. Coronary artery obstruction by ensuing thrombosis induced by hypercoaguability and hyperviscosity secondary to hypovolemia induced haemoconcentration were considered alternate explanations for myocardial damage.

Various mechanism that have been proposed as causative of MI following snake bite are as follows: -

1. Hypovolemic shock: - Snake venom has haemorrhagins, which increase vascular permeability as part of capillary leakage syndrome and cause bleeding tendency. Acute MI can occur by profound hypotension and hypovolemic shock secondary to this haemorrhagic tendency of snake venom. However, it is less likely in our case because retrosternal chest pain, and cardiovascular collapse occurred soon after snakebite, while systemic haematological effects of viper bite are likely to take few hours and chest pain preceded the shock.

2. Hypercoaguability: Snake venom has procoagulants such as arginine esterase hydrolase.⁴ Haemoconcentration secondary to haemorrhage-induced hypovolemia also leads to hyperviscosity and hypercoaguability. All these factors lead to disseminated intravascular coagulation (DIC). This possibility is also less likely in our case because large doses of venom are required to be injected to produce intravascular clotting and coronary artery thrombosis with ischaemic myocardial damage, which is usually not possible in human beings. Our patient did not have other features of DIC.

3. Myocarditis: Direct cardiotoxic effect of snake venom can result in myocarditis and extensive myocardial necrosis. It has been reported in two horses injected with Viper-palaestinae venom for commercial production of antibodies.⁵ This possibility is also less likely in our case because myocarditis should damage myocardium diffusely while in our case, there was localized hypokinesia of inferior myocardial wall with ST segment elevations limited to inferior leads only.

4. Coronary spasm:⁶ The time sequence of retrosternal chest pain and cardiovascular collapse immediately after bite is strongly suggestive of coronary spasm as an important possible etiological factor in causing myocardial infarction in our case. This coronary spasm may be either due to direct intravascular injection of cardiotoxic viper snake venom or it may be secondary to panic associated with snakebite.

REFERENCES