Abstract. Myocardial infarction is a rare complication of snakebite. The present report describes a 40-year-old Jordanian farmer who developed an acute myocardial infarction several hours after a snakebite. The diagnosis of myocardial infarction was confirmed by a typical history of retrosternal chest pain, characteristic electrocardiographic changes, and elevated serum creatinine kinase (MB-CK). The patient had no risk factors for coronary artery disease and the coronary arteries were normal on cardiac catheterization. The possible mechanisms leading to myocardial infarction following snakebite are discussed.

CASE REPORT: ACUTE MYOCARDIAL INFARCTION COMPICATING A VIPER BITE

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INTRODUCTION

Local and systemic complications of snakebite have long been appreciated. These include local tissue necrosis, hemolysis and hemorrhagic complications, nephrotoxicity, coagulation abnormalities, and neurological complications. Acute myocardial infarction caused by snakebite has rarely been reported. The author is aware of only three cases of myocardial infarction complicating a viper bite in the English literature.

CASE REPORT

A healthy 40-year-old male farmer was bitten on the left ankle by a snake while working in his field. The snake was not identified, but its description by the patient suggested Viperae palaestinae. Within two hours, the patient’s left foot and leg became swollen. He was taken first to a nearby district hospital, where he was given intravenous hydrocortisone, antibiotics, and prophylaxis against tetanus. Because snake antivenom was not available at this facility, he was referred to Princess Basma Teaching Hospital approximately one hour later. On arrival, the patient was fully conscious with no pallor or jaundice. His blood pressure was 115/75 mm Hg, and his heart rate was 92 beats per minute, his temperature 37.2°C, and respiratory rate 16 breaths per minute. Examination of the heart, lungs, abdomen, and central nervous system was unremarkable. The left leg and foot became swollen. He was taken first to a nearby district hospital, where he was given intravenous hydrocortisone, antibiotics, and prophylaxis against tetanus. Because snake antivenom was not available at this facility, he was referred to Princess Basma Teaching Hospital approximately one hour later. On arrival, the patient was fully conscious with no pallor or jaundice. His blood pressure was 115/75 mm Hg, and his heart rate was 92 beats per minute, his temperature 37.2°C, and respiratory rate 16 breaths per minute. Examination of the heart, lungs, abdomen, and central nervous system was unremarkable.

Laboratory investigations showed a hemoglobin of 13.9 g/dL, hematocrit 39%, white blood cell count 12.7 × 10⁹ per mm³ with 23.4% lymphocytes, 3.1% monocytes, and 73.5% neutrophils. The platelet count was 232 × 10⁹ per mm³ and erythrocyte sedimentation rate was 46 mm per hour. Serum electrolytes, blood urea nitrogen, creatinine, and fasting plasma glucose levels were within normal limits. Liver enzymes, total and direct bilirubin, and serum albumin were also within normal ranges. Lactic dehydrogenase was 289 U/L and creatinine kinase (CK) was 132 U/L (normal 24–195 U/L). Prothrombin time was 14 seconds (control 29 seconds) and partial thromboplastin time was 32 seconds (control 29 seconds). Serum fibrinogen level was 4.58 g/L (normal 2.0–4.0 g/L) and fibrinogen degradation products were less than 10 µg/ml. Urinalysis revealed no abnormality. Infusion of polyvalent serum anti-venom serum was started at a rate of 4 vials, each containing 10 ml of snake anti-venom serum. Creatinine kinase was 831 U/L several hours later with a positive MB-fraction. Cardiac troponin T was negative. No arrhythmias were noted and infusions of anti-snake polyvalent serum were continued every two-hours for a total of 36 vials. Chest X-ray and two-dimensional echocardiography were normal. Venography of the left leg done three days later, revealed no evidence of deep venous thrombosis. Over the subsequent two weeks, the swelling and ecchymoses gradually cleared and the patient was able to leave the hospital. One month later, the patient underwent cardiac catheterization and coronary angiography, which were entirely normal (Figure 2), except for a mild inferior wall hypokinesia. The patient was asymptomatic and the ECG showed a pattern of acute inferior wall myocardial infarction (Figure 1A). The chest pain lasted approximately 30 minutes and was not responsive to sublingual nitrates, but subsided after intravenous morphine was given. Creatinine kinase was 831 U/L several hours later with a positive MB-fraction. Cardiac troponin T was negative. No arrhythmias were noted and infusions of anti-snake polyvalent serum were continued every two-hours for a total of 36 vials. Chest X-ray and two-dimensional echocardiography were normal. Venography of the left leg done three days later, revealed no evidence of deep venous thrombosis. Over the subsequent two weeks, the swelling and ecchymoses gradually cleared and the patient was able to leave the hospital. One month later, the patient underwent cardiac catheterization and coronary angiography, which were entirely normal (Figure 2), except for a mild inferior wall hypokinesia. The patient was asymptomatic and the ECG showed a pattern of acute inferior wall myocardial infarction (Figure 1A). The chest pain lasted approximately 30 minutes and was not responsive to sublingual nitrates, but subsided after intravenous morphine was given. Creatinine kinase was 831 U/L several hours later with a positive MB-fraction. Cardiac troponin T was negative. No arrhythmias were noted and infusions of anti-snake polyvalent serum were continued every two-hours for a total of 36 vials. Chest X-ray and two-dimensional echocardiography were normal. Venography of the left leg done three days later, revealed no evidence of deep venous thrombosis. Over the subsequent two weeks, the swelling and ecchymoses gradually cleared and the patient was able to leave the hospital. One month later, the patient underwent cardiac catheterization and coronary angiography, which were entirely normal (Figure 2), except for a mild inferior wall hypokinesia. The patient was asymptomatic and the ECG showed a pattern of acute inferior wall myocardial infarction (Figure 1A).

DISCUSSION

Viper bites are common in Jordan, as in other parts of the world. There are six types of poisonous snakes in Jordan. Four belong to the family Viperidae and include: Echis coloratus, Cerastes cerastes, Cerastes gasperetti, Pseudocerastes persicus fieldi, and Vipera eurystoma. Vipera eurystoma is the most prevalent and most poisonous snake in North Jordan, from where this case is reported.

The exact mechanism by which snake envenomation leads to myocardial infarction is unclear. Hoffman and others have reported myocarditis with extensive myocardial necrosis at postmortem in two horses after injection of Viperae palaestinae venom for commercial production of antibodies. Rowlands and others reported myocardial damage in a fatal case after snakebite by a species of the Australian elapid family in which small (foci) of myocardial damage and massive skeletal rhabdomyolysis were seen. Focal myocardial dysfunction was demonstrated.
hemorrhage and microvascular fibrin deposition was also reported by Than and others\(^\text{17}\) in a fatal case after envenomation by Russell’s viper in Burma. In experimental envenomation of dogs, Tibballs and others\(^\text{18}\) observed microthrombi in the pulmonary and coronary arteries. These pathologic changes were associated with hypotension, ST elevation, and T-wave inversion.

Although the patient reported here had hemorrhage of the envenomated limb, there were no manifestations of systemic bleeding and the coagulation profile was normal. Bleeding into the skin was therefore not due to a coagulation abnormality. Toxic vasculitis or extravasation of the venom into the peri-lymphatic tissue may be the cause of local hemorrhage.\(^\text{19,20}\)

In a series reported by Laloo and others, ECG abnormalities were observed. There was either T-wave inversion, sinus bradycardia, or, rarely, atrioventricular block.\(^\text{21}\) Creatinine kinase was elevated in 25 of 51 patients while only 2 of 24 had elevated plasma cardiac troponin T.

Arterial thrombi following viper bites may occur near to or distant from the location of the bite.\(^\text{10,22,23}\) These thrombotic complications may or may not be associated with coagulation abnormalities.\(^\text{10,23}\) It has been shown that hemorrhagins, typical components of Crotalidae and Viperidae venoms, cause vasoconstriction followed by vasodilation of the arterioles.\(^\text{24}\) Some venoms also contain endothelins or sarafotoxins that have coronary vasoconstrictor effects.\(^\text{25}\) Sathyanathan and Mathew reported Raynaud’s phenomenon and gangrene occurring in the opposite limb following envenomation with snakebite.\(^\text{26}\) Considering the above, it is likely that the cause of myocardial infarction following viper bite may involve more than one mechanism. Possibilities include a direct toxic effect on the myocardium and coagulation abnormalities or vasospasm induced by hemorrhagins or endothelins contained in the venom of some snakes.

Coronary angiography was normal and there was no coagulation abnormality in the case reported here. The absence of arterial occlusion one month after the infarction does not rule out the possibility of thrombosis that may have resolved. Toxic myocarditis as the cause of the ECG changes with elevated cardiac enzymes is unlikely because of the lack of associated arrhythmias, conduction defects, or cardiac dilatation by chest X-ray and echocardiography.

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**FIGURE 1.** Electrocardiogram taken: A. on admission and B. two months later.

**FIGURE 2.** Coronary angiography: A. right coronary artery and B. left coronary artery.
REFERENCES
