

Myocardial Infarction after Snake Bite

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Introduction

Cardiac events are rarely associated with ophidian accidents, especially those involving snakes of the Elapidae family. We report on a snake bite victim who, within a few hours after the accident, developed myocardial infarction in the inferior wall with good clinical evolution.

Case report

A 53-year-old male patient, self-employed, living in the rural area, reported that he was planting a vegetable garden in his backyard when he saw a coral snake coming in his direction. When he tried to kill it, he was bitten twice in his right hand. He was referred to the medical referral service 5 hours after the event, and remained conscious, complaining of pain and local edema. Snake bite marks were identified in the index finger of the right hand (Figure 1), after which he was medicated with analgesics and elapid snake antivenom.

At admission, examinations showed leukocytosis and prolonged bleeding and coagulation time, with no other hematological or nephrological alterations. After 6 hours of observation, he started to present with oppressive chest pain associated with nausea, sweating and cutaneous-mucosal pallor. On physical examination, he had bradycardia (heart rate of 50 bpm) and hypotension (blood pressure of 90/60 mmHg), with no alterations in pulmonary or cardiac auscultation. The electrocardiogram showed atrial fibrillation with low ventricular response and ST elevation in D2, D3, aVF and V1-V3 (Figure 2).

Keywords

Snake Bites/ complications; Myocardial Infarction; Chest Pain; Leukocytosis; Coronary Thrombosis/ complications; Angioplasty.

The patient received 200 mg of aspirin and 600 mg of clopidogrel and he was referred to the hemodynamics room with a proximally occluded right coronary artery finding and moderate lesion in the anterior descending artery. Coronary angioplasty with bare-metal stent was performed in the right coronary artery (Figure 2), but due to the high thrombotic load, Thrombolysis in Myocardial Infarction (TIMI) II flow and myocardial blush 2-3, tirofiban was indicated and he was referred to the coronary intensive care unit.

He showed symptom improvement and decrease in ST-elevation (Figure 2). After 24 hours of intravenous amiodarone administration, he persisted with atrial fibrillation, being submitted to electrical cardioversion with reversion to sinus rhythm. He also had acute renal failure and mild thrombocytopenia, both transient. After 6 days, hematological, nephrological and coagulation exams were normalized. He progressed without clinical complaints, and was discharged after 7 days.

Discussion

Ophidian accidents are prevalent events in tropical countries, being associated with high morbidity. Data from the Ministry of Health¹ indicate that in Brazil, there are approximately 7,000 snakebite accidents per year. The most common venomous snakes found in our country belong to the genera *Bothrops*, *Crotalus*, *Micrurus* and *Lachesis*.²

Envenomation caused by snake venom is usually characterized by local manifestations, hemorrhagic alterations, nephrotoxicity, and neurotoxicity. Cardiac involvement is rare, and the few reported cases occur in patients with no major cardiovascular risk factors or known heart disease.

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Figure 1 – Snake bite marks in the index finger of the right hand.

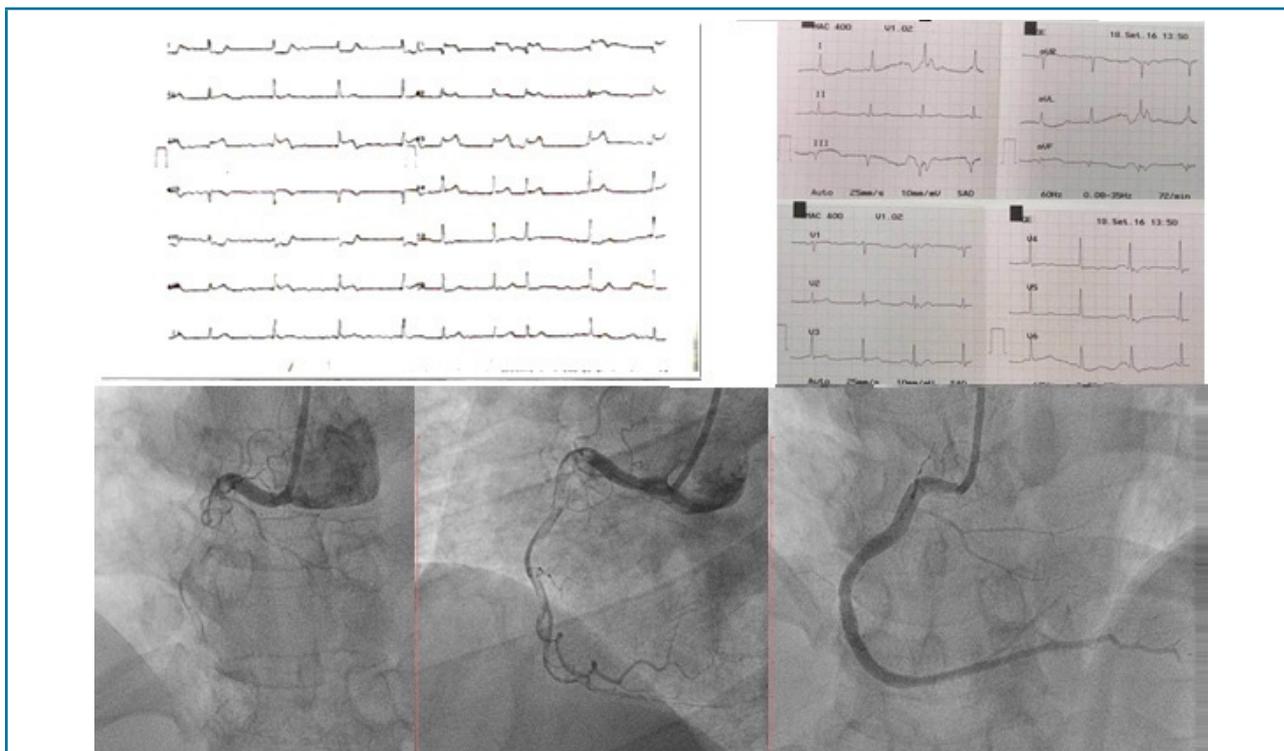


Figure 2 – Electrocardiogram and cardiac catheterization at admission and after coronary angioplasty.

A Nigerian study³ found only 2% of myocardial infarction after the analysis of 108 patients, victims of ophidian accidents. A study carried out in Papua New Guinea⁴ identified 8.3% of electrocardiographic alterations associated with a large increase in troponin-T levels, after 69 patients were analyzed. A Korean study⁵ identified 13.8% of myocardial injury, defined as troponin I elevation in 48 hours or electrocardiographic alteration, after analyzing 65 cases of consecutive ophidian accidents.

The biological plausibility for this association is due to the thrombotic and vasoconstricting properties of snake venom.⁶ In addition to the effect of sarafotoxins and the stimulus for endothelin release, the anemia secondary to coagulation alterations and anaphylactic shock mediated by cardiotoxic effects also collaborate in this process.⁷

The treatment of these patients is a major challenge. The snake venom causes thrombocytopenia and coagulopathy, predisposing to bleeding events. These phenomena may limit the use of thrombolytic agents and coronary stent implantation. However, patients who have already received snake antivenom and have normal basal tests seem to be at low risk for bleeding events.

The use of glycoprotein IIb / IIIa inhibitors is an option in very thrombotic patients and has been used in another case report,⁸ also in a patient who had already received antivenom and whose coagulation tests were within normality.

In our case, the temporality of the ophidian accident, with the development of myocardial infarction and the finding of a high thrombotic load on cardiac

catheterization reinforce the idea of the association of these two conditions.

Conclusion

Although rare, we should always be alert to the association of an ophidian accident as a coronary event precipitant. Early administration of snake antivenom and monitoring for coagulation disorders are essential for the proper management of this population.

Author contributions

Conception and design of the research: Gomes RAF. Acquisition of data: Gomes RAF, Cantarelli FL, Vieira FA, Macedo Jr ARA. Analysis and interpretation of the data: Gomes RAF, Cantarelli FL, Vieira FA, Macedo Jr. ARA. Writing of the manuscript: Gomes RAF, Macedo Jr. ARA. Critical revision of the manuscript for intellectual content: Gouveia MMA, Feitosa ADM.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

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