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Myocardial injury following multiple bee stings

Balasingam Nisahan¹, Gowri Selvaratnam² and Thirunavukarasu Kumanan³

Abstract

Envenomation by the honey bee can cause multi-organ dysfunction as a result of toxic effects and may cause a severe anaphylactic reaction. Acute myocardial ischemia is an extremely rare complication of bee envenomation. We report a case of a 58-year-old woman with acute myocardial injury following massive bee envenomation.

Keywords

Multiple bee sting, myocardial infarction, vasospasm, anaphylaxis

Introduction

Envenomation by insects of order Hymenoptera (honey bee, wasps, ants) frequently occur in northern Sri Lanka. The venom contains a mixture of peptides and enzymes that cause, variously, a non-allergic or allergic local reaction, anaphylaxis, or a systemic toxic reaction such as oedema, vomiting and seizure. Unusual reactions include cardiac ischemia, cerebral infarction and encephalomyelitis.¹ We describe a patient with multiple bee stings causing myocardial damage compatible with non-ST segment elevation myocardial infarction.

Case report

A 58-year-old previously healthy woman presented with multiple bee stings while she was returning home. She was admitted to hospital in Jaffna within 20 minutes of the incident. On admission more than 300 bee sting marks were noted all over her body and 16 live bees were found under her clothing and were removed. On admission she did not complain of chest pain, shortness of breath, wheezing or palpitation, nor was there evidence of an altered level of consciousness. Her pulse rate was 96 bpm and her blood pressure was 110/70 mmHg with a respiratory rate of 16/min. Her saturation at room air was 98%. Mild swelling was noted at all the bite sites. Physical examination was otherwise unremarkable. Investigations revealed a random blood sugar of 14.8 mmol/L, leucocytes of 14,300 cell/mm² with neutrophils of 12,700 cell/mm²,

haemoglobin of 11.8 g/dl and platelets of 220,000 cell/mm². Serum electrolytes revealed sodium of 146 mmol/L and potassium of 3.9 mmol/L. Her blood urea was 12.1 mmol/L and serum creatinine 61.6 µmol/L. Her coagulation profile including bleeding time, clotting time, prothrombin time (PT/INR) and activated partial thromboplastin time (APTT) were normal.

After 1 hour, she was found to be anxious and complained of classic tightening chest pain of myocardial ischemia. A bedside 12-lead electrocardiogram (ECG) showed ST segment depression in V2–V4 (Fig. 1). The cardiac marker troponin I was positive (0.766 u/L).

She was immediately treated with intravenous hydrocortisone 100 mg and thereafter 6-hourly along with Chlorpheniramine. She was managed with dual anti-platelet drugs, atorvastatin and enoxaparin. Within 24 hours, the ECG changes reverted to normal and she did not complain chest pain thereafter. A two-dimensional (2D) ECG was normal (no regional wall motion defects and normal ejection fraction).

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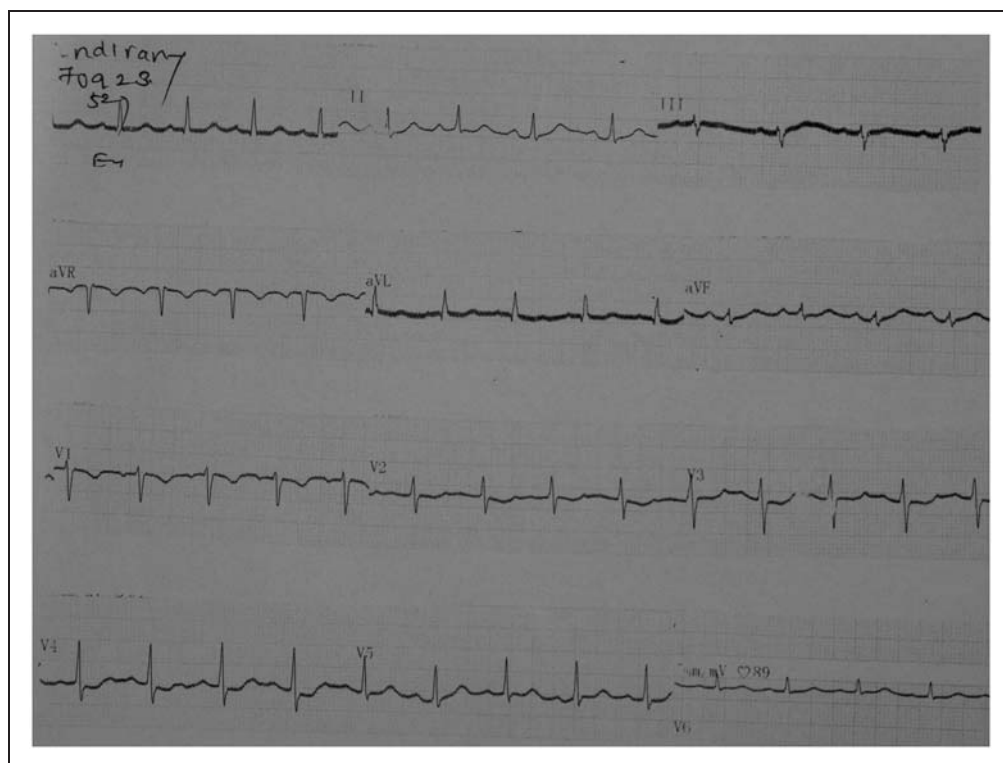


Figure 1. ECG after envenomation shows ST segment depression in V2–V4.

Polyuria which started on day 2 of admission settled after 48 hours. A coronary angiogram revealed normal epicardial coronary arteries and consequently all the anti-anginal drugs were discontinued.

Discussion

Envenomation by bees can cause acute myocardial injury due to vasospasm, platelet aggregation and thrombosis. It may also have a direct toxic effect on the heart.^{2,3} It can also occur as a result of Kounis syndrome (anaphylactic reaction).^{1,4} An acute coronary syndrome is evident where chest pain is associated with elevation of cardiac-specific marker troponin I and ECG changes related to a specific coronary artery territory. The ECG changes can be ST elevation, ST depression, pathological Qwaves or T wave changes. Rhythm abnormalities such as supra-ventricular arrhythmia, ventricular ectopics, junctional rhythm and right bundle branch block are also reported in envenomation by the bee.^{1,4} Myocarditis may also occur. In that case the ECG changes are diffuse rather than discrete. The 2D echocardiogram would show evidence of global motion defect and the cardiac marker values would be expected to be only mildly elevated.

Maintaining good hydration and urine output is pivotal in the management of bee sting envenomation. The

clinical presentation may be quite diverse with myocardial involvement after any bee sting. More often than not it is virtually completely asymptomatic.⁵ However, the treating physician should be vigilant enough and should have a high clinical suspicion to diagnose rare complications at the earliest suspicion. Serial ECGs and cardiac marker values are warranted in patients who complain chest pain regardless of the severity of the reaction to the bee sting.

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