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[: : **Special Issue**](#)
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Case Reports

Acute Myocardial Injury and Rhabdomyolysis caused by Multiple Bee Stings

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Abstract

Massive envenomation by honey bees is capable of causing multiorgan dysfunction as a result of direct toxic effect of massive envenomation and secondary to systemic anaphylactic reactions. Acute myocardial ischemia due to bee envenomation is a rare event. We report the case of a 65 year old lady who presented with acute myocardial ischemia, severe rhabdomyolysis and angioedema following massive bee envenomation.

Introduction

Insects of the order Hymenoptera, which include honey bees, wasps, ants and hornets are frequently involved in accidental

Table 1 : Serial cardiac enzymes at admission, 4 and 48 hours

	Normal	At Admission	4 hours later	At 48 hours
Creatine Phosphokinase	<190 U/L	29,022	34500	1340
Creatine Phosphokinase - MB	<25 U/L	790	844	-
Troponin I	<0.2 U/L	18.7	29.9	-

stings to human beings around the globe. Hymenoptera venom consists of a mixture of biologically active substances including enzymes (phospholipases, hyaluronidases), peptides (melitin, apamin, bombolitins) and other low molecular weight compounds (biogenic amines, acetylcholine, lipids and free amino acids).¹ Hymenoptera sting envenomation may result in a number of clinical presentations (i) Non-allergic, local reactions (pain, minor edema, redness at the sting site); (ii) Allergic, large local reactions (extensive swelling >10 cm persisting more than 24hrs); (iii) Anaphylaxis (generalized urticaria, angioedema, bronchospasm, hypotension, cardiovascular collapse and loss of consciousness); (iv) Systemic toxic reactions (edema, vomiting, diarrhea, headache, seizures and altered sensorium); (v) Unusual reactions (cardiac ischemia, encephalomyelitis and cerebral infarctions). Massive honey bee envenomation is defined as more than 50 stings at a time.¹ We report a case of massive bee sting envenomation with unusual manifestations of acute myocardial injury and rhabdomyolysis.

Case Report

A 65 year old female, a treated case of Hansen's disease, presented with a history of multiple bee stings (>100) while she

was gathering flowers in the woods. She had been admitted in a local hospital immediately, with swelling of the face and lips. She was given preliminary care at this center (treatment details are not known) and was referred to our hospital for further management. There was no history of chest pain, dyspnea, wheeze, decreased urine output, palpitations, syncope, seizures or altered sensorium. She had no past history of diabetes mellitus, hypertension, ischemic heart disease and dyspnea on exertion or angina. No history of dark coloured urine. She was brought to our Emergency Department 12 hours after the bee stings. On admission, her pulse rate was 112/min, blood pressure was 110/70 mm Hg, respiratory rate was 14/min and oxygen saturation was 96% at room air. There were multiple stings noted on the face and back. She had marked edema of the face and lips with diffuse urticaria over the body. Systemic examination was normal. Investigations revealed a hemoglobin of 12gm%, leukocyte count of 16,000 cells/cumm and platelet count of 2, 50,000 cells/cumm. Serum sodium was 137 mEq/L, potassium 3.1 mEq/L, bicarbonate 26 mEq/L and creatinine 0.7 mg/dl. Liver function tests showed total and direct Bilirubin of 2.6 mg% and 0.2 mg% respectively with Serum alanine transaminase of 105U/L and Serum aspartate transaminase of 159U/L. Her Serum Creatinine phosphokinase (CPK) as well as Creatinine phosphokinase MB and Troponin I were elevated at admission (Table 1). Urine myoglobin was not done. An electrocardiogram (ECG) done showed ST elevation in leads V1-V5, with T inversion in V4-V6 (Fig. 1). After four hours there was a rise in CPK, CK-MB and Troponin I

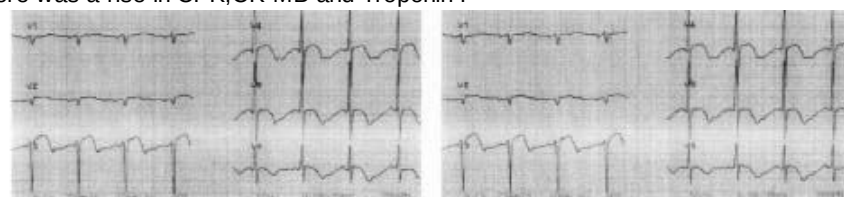


Fig. 1 : ECG at admission: ST elevation in V1-V5 with T inversion V3-V6



Fig. 2 : ECG 24 hours after admission : Normal sinus rhythm, no ST-T changes

and ECG showed no further progression of ST elevation or poor progression of R waves (Fig. 2). She was initiated on treatment with Inj. Hydrocortisone 100 mg IV q6h, Inj. Chlorpheniramine Maleate 25 mg IV q12h and Inj. Ranitidine 50 mg q8h. She was hydrated with IV fluids to maintain a urine output of 50-80ml/hr, in view of rhabdomyolysis, with careful monitoring of the cardiac status. Within 24 hours her ECG had reverted back to normal (Fig. 2) and CPK also gradually reduced. As the ECG had shown complete recovery and there was no clinical evidence of cardiac dysfunction, an echocardiogram was not done. She continued to maintain a good urine output with no deterioration of renal functions. Angioedema and urticaria also resolved completely. Inj. Hydrocortisone was gradually tapered and stopped. She was switched to oral Pheniramine Maleate and Ranitidine and discharged in five days.

Discussion

Individuals who are highly allergic to the venom may develop severe systemic anaphylaxis (type I hypersensitivity) from a single sting. Massive bee attacks can result in toxic reactions, regardless of pre-existing allergic conditions, by virtue of a high venom load potentially causing multiorgan dysfunction. Although all varieties of honey bees have the potential for these attacks, the Africanized honey bee (*Apis mellifera scutellata*) is the most commonly implicated subspecies.¹ Hymenoptera venom can cause acute myocardial injury by several mechanisms: (a) release of allergenic proteins, vasoactive, inflammatory, thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotrienes, thromboxane) which act on the coronary vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis; (b) direct cardiotoxic effect of the venom; (c) anaphylactic reaction. Anaphylactic reactions which result in acute coronary syndromes is also known as Kounis syndrome.² Mellitin, a peptide component of bee venom, hydrolyses cell membranes, changes cell permeability, causes histamine and catecholamine release and is responsible for local pain. It acts with phospholipase-A2 to trigger the release of arachidonic acid, which causes cell membrane breakdown, damage of the vascular endothelium, and activation of the inflammatory response. Peptide 401 (mast cell degranulating peptide), triggers mast cells to degranulate, releasing histamine and other vasoactive peptides. Vasoactive amines, including histamine, dopamine and noradrenaline can provoke ischemia and even myocardial infarction through profound hypotension and arrhythmia, or by increasing oxygen demands through direct inotropic and chronotropic effects in the presence of preexisting ischemic heart disease.³ The rise in serum CPK and CK-MB levels is suggestive of myocardial injury or rhabdomyolysis. Elevation of more specific markers for

myocardial injury such as cardiac Troponin I or T following the large amount of venom in the body. In patients with Acute myocardial infarction after multiple bee stings as reported in literature, the coronary arteries were normal or insignificantly stenosed. Electrocardiographic changes consistent with acute myocardial ischemia or infarction, including ST depression or elevation and even the appearance of pathologic Q-waves, are seen in these patients. Rhythm abnormalities such as supra-ventricular arrhythmias, VPC's, junctional rhythm and right bundle branch block may occur.² Mellitin and phospholipase-A2 cause rhabdomyolysis following toxic injury to the striated muscles and also act on RBC cell membrane causing hemolysis. Rhabdomyolysis and hemolysis induces acute tubular injury and consequent renal failure, especially in individuals who are hypovolemic or acidotic. These peptides are potent inhibitors of nitric oxide which leads to intra-renal vasoconstriction and ischaemia especially in those with borderline renal hypoperfusion.¹ Our patient had very high CPK levels as a result of severe rhabdomyolysis with elevated Troponin I signifying acute myocardial injury with no renal failure or electrolyte abnormalities. She was given good hydration and anti-histamines with steroids which helped her to tide over two very serious complications of massive bee envenomation. Appropriate management of non-allergic massive honey bee envenomation relies on aggressive supportive care, as no commercially available antivenin exists. Presentations consistent with massive histamine release should be managed, similar to typical anaphylactic reactions, with antihistamines, steroids, epinephrine, and airway support as needed. Maintaining adequate urine output should be instituted with aggressive hydration to reduce the likelihood of rhabdomyolysis-induced renal insufficiency. In conclusion, massive bee envenomation can cause life threatening complications like acute myocardial injury, rhabdomyolysis, hemolysis with acute renal failure and systemic anaphylaxis as a result of allergic reactions or due to direct venom toxicity.

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