Research Paper | Medical Sciences | E-ISSN No : 2454-9916 | Volume : 1 | Issue : 4 | Nov 2015

HONEY BEE STING – A MIMIC OF ACUTE MYOCARDIAL INFARCTION – CASE SERIES

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INTRODUCTION

Acute myocardial infarction (AMI) due to honeybee sting has been extensively documented in literature. Numerous authors have discussed the relationship between honey bee sting,anaphylactic shock and myocardial infarction.1-4Bee venoms can act in promoting acute coronary artery thrombosis via platelet aggregation and hypotension.The allergic reaction secondary to the stings trigger various inflammatory mediators and can induce acute coronarysyndrome. Many studies have reported single cases of honeybee bite and their manifestations1.

Herein, we report multiple cases with clinical manifestations mimickingacute myocardial infarction following honeybee sting and review the literature.

CASE REPORTS

CASE 1

A 48 years old male non hypertensive , non diabetic with no prior history of cardiac disorders , farmer by occupation presented with history of honey bee bites while working in the fields. The patient an ex smoker left the habit 2 years back .

The patient started complaining of dyspnoea on exertion and chest pain NYHA class II after 2 days of the incident .The patient was taken to a local doctor for the complaints and was then referred to this hospital for further management. The patient was evaluated for the symptoms in the form of investigations of ECG(Figure 1), Echocardiography and cardiac enzymes all of which were found to be normal.

The patient underwent Treadmill Exercise testing (figure 2) which was positive for inducible ischaemia.In view of the symptoms of presentation and TMT positive the patient was taken up for diagnostic coronary angiography which revealed triple vessel disease .

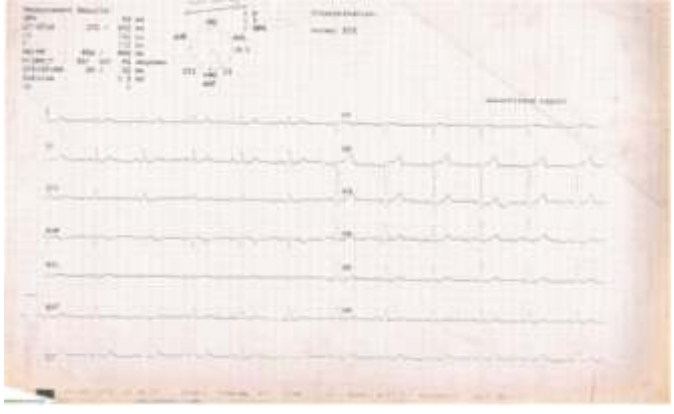


FIGURE 1

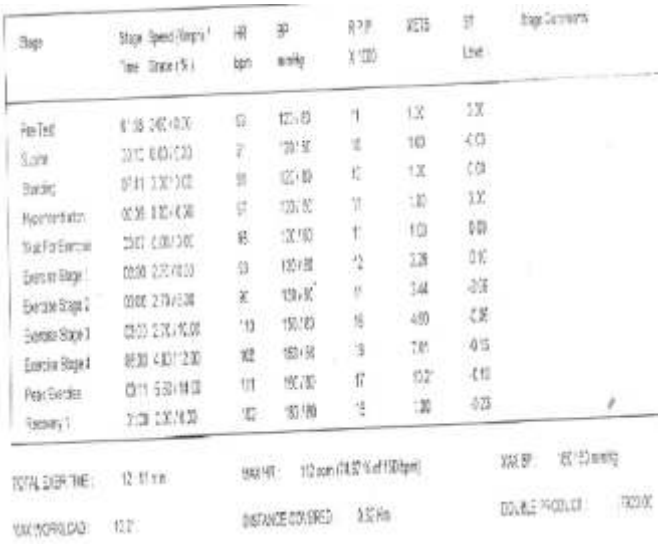


FIGURE 2

CASE 2

A 56 years old known hypertensive and non diabetic malewith history of multiple honey bees stings on his face as well as back and hand(FIG 3). He experienced immediate whole body paresthesia and a short-lasting episode of near-syncope followed by chest pain. The patient was admitted to a outside hospital two hours after the episode with complaints of chest pain and dyspnea. Blood pressure was 60 mmHg systolicwith tachycardia (104/min) and swelling of face and hands . All other systemic examination findings were normal. During hospital stay patient continued having chest pain . 12 lead ECG demonstrated acute inferior wall myocardial infarction (FIG 4A),and shortly second ECG taken demonstrated ventricular tachycardia with heart rate of 200/ min (FIG 4B),. Patient was referred for further management to our hospital . ECG on admission revealed evolved inferior wall myocardial infarction(FIG 5).The patient's initial CK-MB level was 23 U/L and TroponinT level was 0.043ng/mL (0-0.01). All hematologicaland biochemical result were normal except raised leukocyte count . Transthoracic echocardiography done showed hypokinesia of inferior wall , normal resting LV systolic function ,normal PA pressure. In view of patient being freeof chest pain and ECG revealing evolved inferior wall myocardial infarction it was decided not to thrombolyse the patient and to manage with conventional treatment . After hemodynamic stabilization ,patient was evaluated with coronary angiography (FIG 6A,6B),which revealed total occlusion of proximal right coronary artery with thrombus and TIMI 0 flow distally, distal circumflex coronary artery had 80 % type B lesion. As RCA was infarct related artery and well collateralized, patient offered stenting to only distal circumflex . Post PTCA with stenting

period was uneventful and patient was discharged with advice to follow up after 4 weeks.

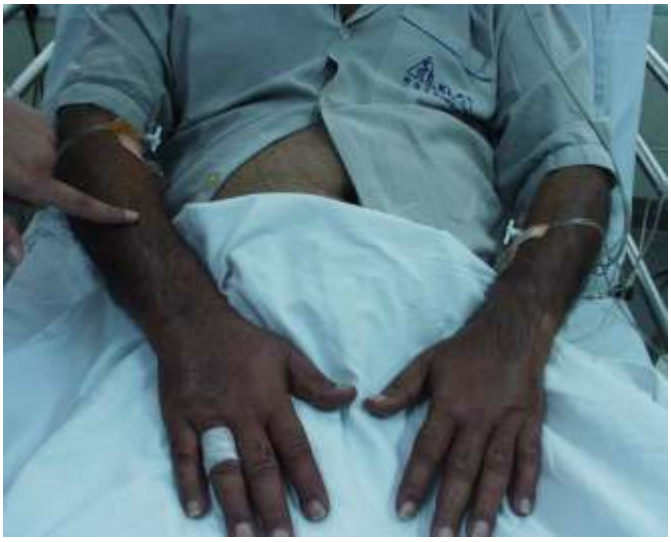


FIGURE 3

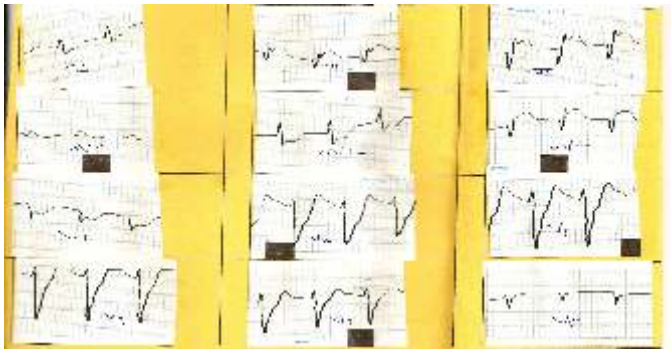


FIGURE 4A

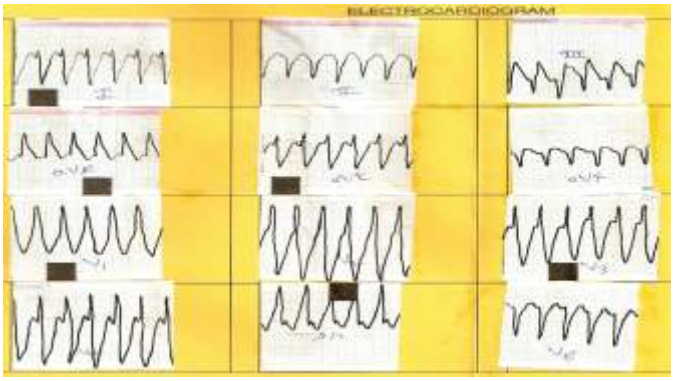
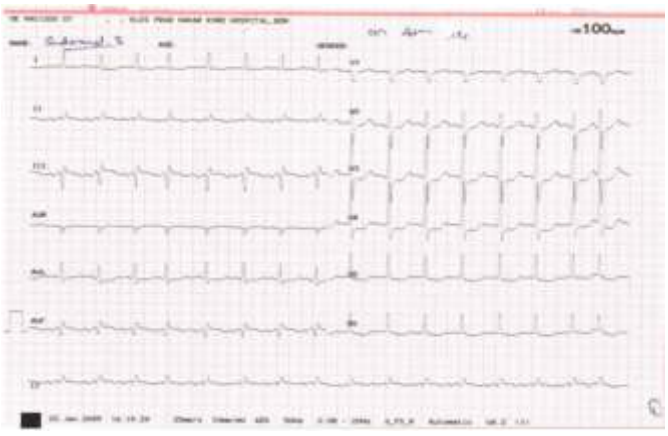


FIGURE 4B



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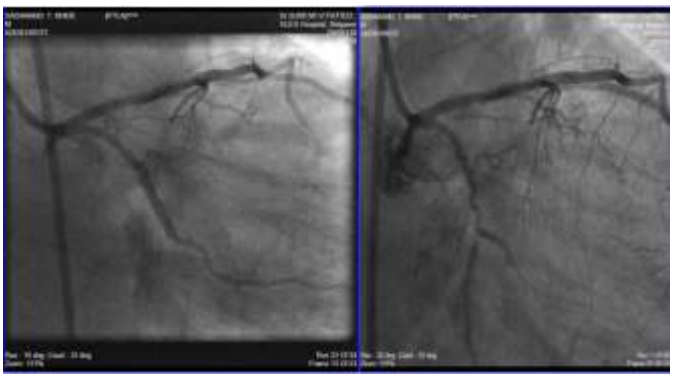

FIGURE 6A, 6B

FIGURE 7

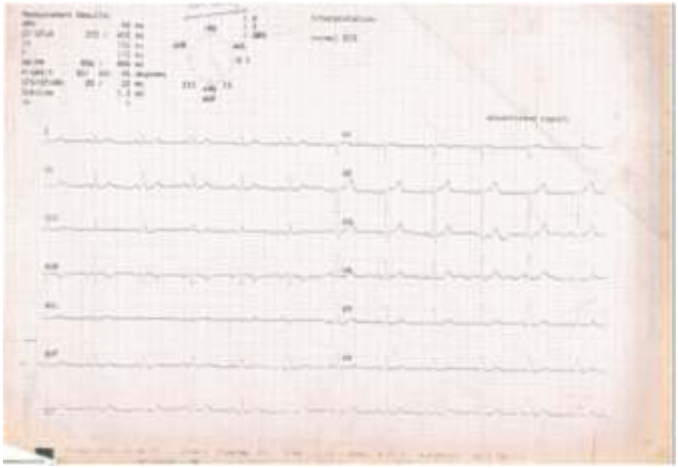

FIGURE 8

CASE 3

A 60 year old male (Fig 7)presented to hospital with complaints of chest pain squeezing type , diffuse in nature , precordial in origin radiating to the arms.The history of honey bee bite 4 hours prior to the symptoms was revealed during the history taking .The patient had no dyspnoea, no palpitation, no sweating associated with the chest pain .No history of diabetes , hypertension or any prior cardiac or medical illness was obtained .Cardiac examination was normal. ECG (Fig 8)and Echocardiography were normal although cardiac enzymes Trop I was raised on day of admission which showed a rising trend with repeat samples. The patient underwent coronary angiography in view of the raised cardiac enzymes and symptoms at presentation. CAG revealed normal coronaries.

DISCUSSION

The term "enomous animals " is usually applied to a creature capable of producing a poison in a secretorygland and delivering that toxin during biting or stingingact. Arthropods, such as spiders, scorpions, and hymenoptera(bees, wasps, yellow jacket) are found worldwide,and some of them are venomous animals.

Bee sting venom generally consists of complex mix of proteins, peptides and enzyme.The venom is mixed with water, so the actual

composition of the substance it injects into you is around 88% water and 12% venom.The main toxic component of bee venom, also referred to as apitoxin, is melittin. Melittin is a peptide that comprises around 50-55% of dry venom, and is a compound that can break up cell membranes, resulting in the destruction of cells. However, it's not considered the most harmful component of bee venom; that prize goes to an enzyme that makes up around 10-12%, phospholipase A. This enzyme destroys phospholipids, and also breaks down the membranes of blood cells, resulting in cell destruction; additionally, unlike the majority of larger molecules in the venom, it causes the release of pain-inducing agents. Yet another enzyme, hyaluronidase, aids the action of the venom by catalysing the breakdown of protein-polysaccharide complexes in tissue, allowing the venom to penetrate further into the flesh.

Other, smaller molecules can also contribute towards painful effects. A small amount of histamine is found in bee venom; histamine is one of the compounds released by the body during the allergic response, and can cause itchiness and inflammation. The proteins in the sting can cause an allergic reaction, leading to the release of even more histamine, and possible anaphylaxis. MCD peptide, another minor component of the venom, can also cause mast cells in the body to release more histamine, worsening inflammation.

Hymenoptera (bees, wasps) stings or bites are responsible for far more deaths than those due to all other poisonous creatures. The most frequent clinical events are due to immediate type of hypersensitivity reactions leading to hypotension, dyspnea, anaphylactic shock and angioedema.Acute myocardial infarction occurs very rarely afteran arthropod envenomation. There are a few cases ofAMI due to bee or scorpion bites reported in the literature.[1-4]In these patients, coronary arteries were normalor non-significantly stenotic.

However, the exact mechanism of AMI caused by arthropodenv-enomation is unclear.Several reports deal with the cardiovascular complications after hymenoptera stings.[2,5,6,7] Theimportant medical problem posed by the stings is the development of anaphylactic shock. Anaphylaxis leads to hypotension causing vasodilation and decrease of intravascular volume.[2,5,6,7].

Many pharmacologically active constituents ofvenoms have been isolated.[8]These substances can provoke ischemia and even myocardial injury due to profound hypotension or by increasing oxygen demands through direct inotropic and chronotropic effectsin the presence of compromised myocardial supply.

Electrocardiographic changes consistent with acute myocardial ischemia or infarction, including ST depressionor elevation and even the appearance of pathologic Q-waves, have been recorded in people after stings.[2,9,10]

Rhythm abnormalities such as supraventricular arrhythmias, VPC's, junctional rhythm and right bundle branchblock have been recorded during initial stages after the sting.[2,6,9]Animal studies of bee venom have shown thatsuch ECG changes may be due to direct cardiotoxic effect. However, the mechanism is still not clear.

Laboratory parameter, such as elevated CPK ,SGOT, Trop I and T have been noted in people with stings which indicates myocardial injury.[2,6]The rise in serum creatine kinase and creatine kinase-MB levels may be attributed to myocardial injury and /or rhabdomyolysis caused by extremely high sympathetic discharge. Elevation of the more specific marker for myocardial injury like cardiac troponin I or T following sting indicatesdirect or indirect myocardial injury.

Transthoracic echocardiography showing regional wall motion abnormalities (hypokinesia and akinesia) and left ventricular dysfunction after a bite has been reported.[4] The stunned myocardium is known as a prolonged postischemic LV dysfunctionafter brief myocardial ischemia and represents a reversible LV dysfunction.[8]Abrough et al. reportedgradual normalization of wall motion abnormality in the left ventricle and septum, and also complete restorationof the systolic function in cases with severe bites.[10]

The change in echocardiographic findingsof our cases are similar to that of above reported cases.[4,10]Moreover, clinical, electrocardiographic, laboratory andechocardiographic findings in our cases excluded the possibility of myocarditis or pericarditis.

Various differential diagnosis that could be possible with such a clinical and laboratory presentation are Hereditary Thrombophilia, Kounissyndrome ,Takotsubo cardiomyopathy.

Two cases are described with the possible association between hereditary thrombophilia and arthropod bite giving rise to AMI without any evidence of atherosclerotic heart diseases.[4] The authors therefore recommendthat patients presenting with AMI following an arthropod bite should be screened for any inherited thrombophilia.[4] Another two patients who were stung by wasps and honeybee, respectively, developed Kounis syndromeas a consequence of allergic reaction[11].Kounis syndrome is the concurrence of acute coronary syndrome with mastcell activation induced by allergic or hypersensitivity and anaphylactic of anaphylactoid reactions.

Takotsubo cardiomyopathy (Takotsubo CM) is a novel cardiac syndrome characterized by transient and severe LV apical ballooning and basal hyperkinesia in acute stage.[12,13] Although Takotsubo cardiomyopathyshows striking initial manifestations mimicking AMI, the minimal change of cardiac enzymes are not consistentwith the extent of LV change in acute stage, and unusual LV morphology was restored to normal, usuallywithin several weeks, in most cases.[12,13] In patients with Takotsubo CM, the ECG in acute stage shows concaveST-segment elevation, usually in leads V3-6, there is less dynamic change for days, followed by T-wave inversionand resolved in approximately 2-3 weeks[6,16] associated with QTc prolongation.[16] Abnormal Q-wave and reciprocalchanges are rarely seen.[14]

It is wellknown that systemic anaphylaxis with bronchospasm, larynxedema and hypotension may ensue following hymenopte-rasting. In the most severe cases, the symptoms of cardiovascular system are predominant; therefore, stenocardial troubles and accelerated and irregular heart rate may develop [15].

The electrocardiographic (ECG) changes[16,17,18] as well as chestpain [19] were described. The acute myocardial infarction followingan insect sting of Hymenoptera (bees, wasps, hornets)occurs rarely.[20-25]Anaphylactic reactions after different insects sting mayinduce cardiovascular events, including acute myocardial infarction,These substances are responsiblefor direct venom cardiotoxicity causing vasoconstrictionand platelet aggregation.[20,26]

Several of the venom proteins and peptidesare allergenic. These allergens, especially phospholipase A2, can cause endogenous amine release from mast cells duringanaphylactic reaction.

The main mechanisms responsible for myocardial infarctionmight be coronary arterial spasm and/or secondaryin situ thrombosis.[22,18,28-30].

Our report focuses on cases of acute myocardial Infarction after a bee sting. Bee venom contains epinephrine, dopamine, leukotrienes and thromboxanes, which cause severe platelet aggregation and direct vasoconstriction, therefore paradoxical vasoconstriction is a possible explanation as an underlying mechanism. Severe coronary arterial spasm or secondary in situ thrombosis may also play role in such cases.[31] Clinical and pathophysiological background ofAMI after bee sting are generally related with three different mechanisms; AMI due to anaphylaxis and shock, a typical AMI occurring in patients with coronary atherosclerosis and an AMI occurring in subjects without significant coronary artery disease In whom coronary thrombosis and vasospasm enhanced by intoxication.[5]Several cases of AMI were reported after envenomation with different animals such as snakes, wasps and several different insects. Authors have postulated that venom constituents can cause endogenous amine release and vasodilatation leading to endothelial dysfunction[32], also postulated that it was possible that adverse effects of therapeutic doses of epinephrine could be responsible for the reaction. Primary coronary artery vasospasm (usually associated with chest pain and an ischemic patternon the ECG) was postulated to be the alternative pathophysiological hypothesis. Matucci et al. also recommend a consequence of an immunoglobulin E-related allergic reaction as another potential mechanism.[33] Clinical presentation may be quite different in AMI patients after bee stings. It may be completely silent[32]

or ECG changes with overt ST wave elevation may take place several hours after admission of the patient, as in our cases. Therefore higher grade clinical suspicion is absolutely necessary in order to come up with the correct diagnosis. Also, serial ECG recordings with assessment of laboratory parameters are recommended in every patient who had encountered chest pain regardless of the severity of a patient's reaction to a bee sting.

In conclusion, hymenoptera (bee) venom can cause acute coronary syndrome by several pathogenetic mechanisms:release of allergenic proteins, vasoactive, inflammatory, and thrombogenic peptides and amine constituents(histamine, serotonin, bradykinin, leukotrienes, thromboxane), which act on the coronary vasculatureand induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis; direct cardiotoxiceffect of the venom; and anaphylactic reactions.

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