ACUTE MYOCARDIAL INJURY IN MULTIPLE BEE STINGS – A CASE REPORT

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ABSTRACT
Acute myocardial injury due to honey bee sting is rare, only a few studies have discussed the relationship between honey bee sting and myocardial injury. Massive envenomation by honey bees can cause multiorgan dysfunction due to toxic effects of massive envenomation and systemic anaphylactic reactions.

Key-Words: Bee Sting; Envenomation; Myocardial Injury

Introduction
Allergic reactions to honeybee stings are not rare. Honey bees belongs to order of Hymenoptera, other insects of this order include wasps, ants and hornets. These reactions vary greatly in severity with manifestations ranging from skin reaction to respiratory, cardiovascular and gastrointestinal reactions. It is well known that systemic anaphylaxis with bronchospasm, laryngeal edema and hypotension may occur following Hymenoptera sting. In most severe cases, the symptoms of cardiovascular system are predominate therefore chest discomfort and accelerated irregular heart rate may develop.[1]

Honey bee sting envenomation may result in number of clinical presentations[2];

a) Non-allergic local reactions (pain, minor edema, redness at the sting site)

b) Allergic large local reactions (extensive swelling >10 cm persisting >24 hours)

c) Anaphylaxis (generalized urticaria, angioedema, bronchospasm, hypotension, cardiovascular collapse and loss of consciousness).

d) Unusual reactions (cardiac ischemia, encephalomyelitis and cerebral infarctions).

Case Report
A 60 year old female with no previous history of diabetes mellitus, hypertension, and ischemic heart disease presented with multiple bee stings in her village 20 hours before admission. She was treated initially at local hospital and then referred to our hospital. At admission she did not had chest pain or breathlessness, she was conscious and oriented. There were multiple stings on face and upper limbs along with swelling and urticarial rashes all over the body.

Her vitals were- pulse rate 90 bpm, Blood pressure 100/60 mmhg, and respiratory rate 16/min. Systemic examination was normal.

At admission blood counts and biochemical parameters (Table-1) were normal except levels of Creatinine phosphokinase- MB and Troponin I (Table-2), which were elevated. After 6hours of admission, the levels of Creatinine phosphokinase- MB and Troponin I (Table-2) were further elevated. ECG at admission showed ST elevation in leads V1-V4 with T wave inversion in V3-V5 (Figure-1) which reverted back to normal after 24hrs (Figure-2).

After admission the patient was treated with IV fluids to maintain her blood pressure and then given IV. Hydrocortisone along with IV. Chlorphenaramine Maleate at regular interval. Vitals were monitored carefully along with continuous ECG monitoring. After 24 hrs ECG became normal. The medications were continued with gradual tapering of steroids till her angioedema and urticaria were resolved. The patient was discharged on day 5.
Table 1: Blood Counts and Biochemical Parameters

<table>
<thead>
<tr>
<th>Blood Investigations</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>12 gm%</td>
</tr>
<tr>
<td>Total leucocytes count</td>
<td>12,900 cells/cu mm</td>
</tr>
<tr>
<td>Platelet count</td>
<td>2,00,000 cells/cu mm</td>
</tr>
<tr>
<td>Blood urea</td>
<td>30 mg%</td>
</tr>
<tr>
<td>Serum creatinine</td>
<td>0.9 mg%</td>
</tr>
<tr>
<td>Random blood sugar</td>
<td>86 mg%</td>
</tr>
</tbody>
</table>

Table 2: Serial Cardiac Enzymes Levels at Admission and After 6 Hours

<table>
<thead>
<tr>
<th>Cardiac Enzymes</th>
<th>Normal</th>
<th>At admission</th>
<th>6 Hr Later</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine</td>
<td>&lt;25 U/L</td>
<td>226 U/L</td>
<td>468 U/L</td>
</tr>
<tr>
<td>Phosphokinase-MB</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Troponin I</td>
<td>&lt;0.2 U/L</td>
<td>10.6 U/L</td>
<td>14.8 U/L</td>
</tr>
</tbody>
</table>

Figure 1: ECG at Admission: Showing ST elevation in V1-V4 with T wave inversion in V3-V5

Figure 2: ECG after 24 Hours of Admission: Normal sinus rhythm, no ST-T changes

Discussion

The term “venomous animals” is usually applied to a creature capable of producing a poison in a secretory gland and delivering toxin during biting or stinging act. Arthropods, such as spiders, scorpions and Hymenoptera (bee, wasp) are found worldwide and some of them are venomous animals. Systemic reactions after stings are usually of immediate type. The most frequent clinical events are hypotension, dyspnoea, anaphylactic shock and angioedema. There are several reports dealing with cardiovascular complications after Hymenoptera stings. Hypotension is a common manifestation with vasodilatation and decrease of intravascular volume. Hymenoptera venom can cause acute myocardial injury by several mechanisms: (a) Release of allergic proteins, vasoactive, inflammatory, thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotriene, thromboxane) which act on coronary vasculature inducing coronary vasospasm causing platelet aggregation as well as thrombosis; (b) Direct cardio toxic effect of the venom; (c) Anaphylactic reactions. Mellitin, a peptide component of bee venom, hydrolyses cell membranes; changes cell permeability and causes histamine and catecholamine release and are responsible for local pain. It acts with phospholipase-A2 to trigger the release of arachidonic acid, which causes cell membrane breakdown and damage the vascular endothelium. Vasoactive amines including histamine, dopamine and noradrenaline can provoke ischemia and even myocardial infarction through hypotension and arrhythmia. The rise in CPK MB and more specific marker Troponin I or T levels is suggestive of myocardial injury. The “allergic angina syndrome” which could progress to acute myocardial infarction (“allergic myocardial infarction”) was first described in 1991 by Kounis and Zavras. Allergic angina and allergic myocardial infarction are now referred to as “Kounis syndrome” this syndrome is associated with mast cell degranulation. The key mediator is tryptase. In addition to environmental exposure such as Hymenoptera stings, Viper venom and poison ivy, other causes include several drugs and number of conditions (angioedema, bronchial asthma, food allergy, serum sickness). 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 pathological Q waves are seen in these patients. Rhythm abnormalities such as supraventricular arrhythmias, VPCs, junctional rhythm and right bundle branch block may occur. In our case, the patient had features suggestive of myocardial injury (ECG changes, elevated cardiac enzymes). She was given good hydration, anti-histamines with steroids which helped her to overcome massive bee envenomation. Appropriate management of non-
allergic massive honey bee envenomation relies on aggressive supportive care, as no commercially available anti-venom exists.

**Conclusion**

Acute myocardial injury after a honey bee sting is a previously very rarely reported complication. It should be considered in subjects with chest pain or hemodynamic compromise in order to diagnose it early and apply an appropriate treatment.

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**References**


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