Acute myocardial infarction following a hornet sting

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Abstract

Background. The occurrence of an acute myocardial infarction following a hornet sting has been very rarely reported in the previous literature. Pathogenetic mechanisms include direct action of the venom components on the coronary endothelium and allergic reaction with mediators released from mast cells. The anaphylactic reaction and venom components can produce acute coronary artery thrombosis. Case report. We reported a 45-year-old man with acute myocardial infarction after a hornet sting in the presence of anaphylaxis. We also discussed clinical implications and pathophysiological mechanisms of acute myocardial infarction caused by hymenoptera sting. Conclusion. A case report of this unusual acute myocardial infarction highlights the potential acute myocardial ischemia associated with hymenoptera sting which requests early diagnosis, thorough cardiovascular evaluation and appropriate treatment.

Key words: myocardial infarction; anaphylaxis; bites and stings.

Apstrakt


Key words: infarkt miokarda; anafilaksija; ujedi i ubodi insekata.

Background

Allergic reactions to wasp or honeybee stings are seen in up to 5% of the total population in Europe and USA. These reactions vary greatly in severity with manifestations ranging from skin reactions (rash, itching) to respiratory, cardiovascular and gastrointestinal reactions. It is well known that systemic anaphylaxis with bronchospasm, larynx edema and hypotension may ensue following hymenoptera sting. In the most severe cases, the symptoms of cardiovascular system are predominant; therefore, stenocardial troubles and accelerated and irregular heart rate may develop. The electrocardiographic (ECG) changes as well as chest pain were described. The acute myocardial infarction following an insect sting of Hymenoptera (bees, wasps, hornets) occurs rarely.

The case of acute myocardial infarction as the complication of a European hornet (Vespa cabro linnaeus) sting in Serbia was presented in this paper.

Case report

A 45-year-old previously healthy man was stung in the neck by a hornet, while he was cutting the grass. Considering that he was a beekeeper and that he saw the insect, he could have recognized the hornet. Within the following 15 minutes, the extensive itching all over the body arouse. He felt a severe asphyxiation and oppression of the lungs, and the acute pain appeared behind the sternum associated with perspiration and nausea. Two hours later he was admitted to the Intensive care unit (ICU) of the regional Health Center in the town of Kraljevo. He had no history of ischemic heart dis-
ease, hypertension, diabetes, or allergy. However, he recalled
that he had been stung several times, in the past, by honey-
bees and wasps, without any sequelae, but had no previously
experienced a hornet sting. He had a good exercise tolerance
and was a smoker. He denied myocardial infarction and
stroke in the family history.

On examination in ICU, the patient complained of se-
vere retrosternal pain accompanied by nausea, diaphoresis
and dizziness. He had generalized urticarial rash, with neck
and head edema. His heart rate was 56/min, blood pressure
100/70 mmHg, respirations 23/min, axilar temperature 36.3 ºC.
Cardiac auscultation revealed normal heart sounds without
murmurs. Respiratory and abdominal examination were
normal. The neurological examination did not reveal any
neurological damage. A 12-lead electrocardiogram (ECG) at
presentation showed sinus rhythm, and 1-mm ST-segment
elevations in leads V1-V4. The amplitude of R waves de-
creased running from V1-V3 leads (Figure 1). Later ECG re-
corded the evolution of acute myocardial infarction of an-
terolateral wall with QS wave from V2-V4 and negative T
wave in DI and aVL, V2-V6 leads (Figure 2). After admis-
sion nasal oxygen therapy was started at 2–4 l/min. After that
the patient was given aspirin 300 mg orally, morphine 5mg
intravenously and heparin infusion 1 000 unit/hour after in-
travenous 5 000 unit bolus. He was given neither adrenalin
nor hydrocortisone or antihistaminic (H1-blocker). The fol-
lowing hours the patient became asymptomatic. The results
of blood examination showed that hemoglobin, hematocrit
and platelet count were in the normal range. The leukocyte
count was 13 800/mm³ and eosinophils were raised to 11%. Erythrocyte sedimentation rate was 20 mm/h. The biochemi-
cal results revealed increased values of troponins and cardiac
enzymes confirming the diagnosis of an anterior wall myo-
cardial infarction. The pick level for troponin–I was 20.2
μg/L (normal values < 0.1 μg/L), and for creatine phospho-
kinase (CPK), and creatine phosphokinase-MB (CPK-MB)
were 1 852, and 282 Units/L, respectively. Creatine phos-
phokinase-MB decreased gradually in the next few days to
the normal range. Serum creatinine and electrolyte panel
were normal. A total cholesterol level was 7.2 mmol/L and
the triglyceride level was 4.95 mmol/L.

The patient was treated with coronary vasodilators,
antiarrhythmics, antiaggregation drugs and sedatives.
Thrombolyis was not attempted because ST-segment eleva-

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**Fig. 1 – Electrocardiogram on admission shows ST segment elevation in V1-V4 leads**

**Fig. 2 – Electrocardiogram 3 days after admission shows QS wave in V2-V4 and negative T wave in DI and aVL, V2-V6 leads**
tions in V1–V4 were borderline and without reciprocal depression. The patient had a good hospital course and was discharged from local hospital on the day 20. At the same day he was admitted to the Institute of Cardiovascular Diseases in Belgrade because of the further diagnostic evaluation. Electrocardiogram on admission confirmed Q wave anterolateral myocardial infarction. Transthoracic echocardiography showed a dyskinetic distal ventricular septum and apex with the organized thrombus of 1.6 x 1.8 cm (Figure 3).

The ejection fraction of the left ventricle was 50%. Coronary angiography revealed the recanalized thrombus with narrowing of 70–80% of distal left anterior descending (LAD) artery (Figure 4). There were no significant atherosclerotic lesions at the other coronary arteries. The left ventriculography showed anterolateral dyskinesis with an estimated ejection fraction of 40%. Because of the asymptomatic status and absence of any viable myocardium a decision was taken not to revascularize LAD artery in this patient.

The patient underwent the epicutaneous (prick) skin tests with insect venoms. Sensibility to bee, wasp and hornet venom was not confirmed in this case. A total serum immunoglobulin E level was in normal values. However, considering the convincing anamnestic data referring to severe anaphylactic reaction to hornet venom, the specific immunotherapy was initiated.

**Discussion**

Insects of the order *Hymenoptera* have a stinging apparatus at the tail end of their abdominal segment and are capable of delivering between 100 ng and 50 μg of venom.

There are three families of stinging insects in the order of *Hymenoptera*: *Apidae*, *Vespidae* and *Formicidae*. Honeybees and bumblebees belong to *Apidae* family (apids), wasps, yellow jackets and hornets belong to *Vespidae* family (vespids), and stinging ants belong to *Formicidae* family (formids).

Anaphylactic reactions after different insects sting may induce cardiovascular events, including acute myocardial infarction, even in patients with normal coronary arteries.

Flying hymenoptera venoms contain peptides, proteins, and vasoactive amines including norepinephrine, histamine, serotonin, bradykinin, acetylcholine, dopamine, phospholipase, leukotrienes, thromboxanes. These substances are responsible for direct venom cardiotoxicity causing vasoconstriction and platelet aggregation.

It has been estimated that about 1 500 stings would be required to deliver a lethal dose of hymenoptera venom for a nonallergic adult who weighs 70 kg. Several of the venom proteins and peptides are allergenic. These allergens, especially phospholipase A2, can cause endogenous amine release from mast cells during anaphylactic reaction. Some of these mediators released from mast cells cause vasodilatation that can lead to paradoxical coronary vasoconstriction and platelet aggregation.

These vasoactive mediators can induce myocardial ischemia either via hypotension or by increasing myocardial oxygen demand through direct inotropic or chronotropic effects. Serotonin, epinephrine and thromboxanes induce platelet aggregation accelerating thrombus formation.

Finally, epinephrine that is often administered in the setting of anaphylaxis can aggravate myocardial ischemia, especially in elderly patients with coronary heart disease.

The main mechanisms responsible for myocardial infarction might be coronary arterial spasm and/or secondary in situ thrombosis. Therefore, the management strat-
gy of choice for the final diagnosis is urgent coronary angiography. It was reported that the treatment with primary percutaneous coronary intervention (PCI) was applied in patients with acute myocardial infarction with ST-segment elevation after a bee sting.

Acute myocardial infarction after a hornet sting is very rare; only two cases have been reported in the literature. Our case is important because the patient had no signs of previous ischemic heart disease. The sequences of events suggest that anaphylaxis was the triggering factor of acute myocardial infarction. The patient’s symptoms immediately after a hornet sting are attributable to anaphylaxis. Nevertheless, epicutaneous (prick) skin tests with hymenoptera venoms were negative. Negative skin tests, such as in this case, could exist in small group of sting-sensitive persons. More sensitive intradermal skin tests with insect venoms were not performed in this patient. However, subsequent typical retrosternal pain during an anaphylaxis accompanied with ECG abnormalities, confirmed that he had an acute Q anterolateral myocardial infarction. Subsequent coronary angiography suggested that the infarction was caused by thrombosis of the LAD artery, although the formerly existing atherosclerotic plaque could not be ruled out. The appearance of recanalized thrombus in the infarct related artery and left ventricle anteroapical dyskinesis suggest that the spontaneous thrombolysis occurred too late, namely, at the time when the irreversible necrosis of myocardial tissue had already developed.

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“The allergic angina syndrome” which could progress to acute myocardial infarction (“allergic myocardial infarction”) was firstly described in 1991 by Kounis and Zavras. Allergic angina and allergic myocardial infarction are now referred as “Kounis syndrome.” This syndrome is associated with mast cell degranulation and it is caused by mediators released during degranulation. The key mediator is tryptase, short lived protease (half-live of about 90 min). There are several causes capable of inducing this syndrome. In addition to environmental exposures such as hymenoptera stings, viper venom and poison ivy, other causes inducing Kounis syndrome include several drugs and a number of conditions (angioedema, bronchial asthma, food allergy, serum sickness, after drug-eluting stent implantation et al.). Blood samples for tryptase, histamine and IgE immunoglobulins were not taken after the precipitating event so the confirm of Kounis syndrome don’t exist in this case.

Conclusion

Acute myocardial infarction after a hornet 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. In the above case, the thrombotic coronary occlusion was a possible cause of anaphylaxis related acute myocardial infarction.

REFERENCES


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