

Acute Myocardial Infarction After a European Hornet Sting

A Case Report

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The authors briefly describe an 87-year-old man who experienced an acute ST-elevation myocardial infarction in the context of an anaphylactic reaction caused by a European hornet (*Vespa cabro linnaeus*) envenomation. This unusual case highlights the potential cardiovascular complications associated with common insect stings such as those caused by hymenoptera. Thus, a thorough cardiovascular evaluation is essential in such cases since the vasoactive, inflammatory, and thrombogenic mediators of anaphylactic reactions may provoke myocardial ischemia.

Introduction

Insect stings, apart from local cutaneous manifestations or generalized anaphylaxis, can cause a variety of systemic complications. The occurrence of an acute myocardial infarction after a bee or a wasp sting has been rarely reported in the medical literature.¹⁻⁶ We report on a patient who experienced an acute ST-elevation myocardial infarction induced by a European hornet (*Vespa cabro linnaeus*) sting.

Case Report

An 87-year-old man was found unconscious by his relatives lying on his bed during the nighttime. They also noted that a large insect (European hornet) was lying by his right side, while a diffuse rash was present throughout the patient's body. The patient was immediately transferred to the Emergency Department of this hospital. On arrival, the patient was unconscious (Glasgow scale: 9) with signs of hypoperfusion. His past medical history was significant only for mild hypertension treated with losartan 50 mg/day. An urticarial rash mainly on his trunk and head was also noticed. A sign of a recent sting was evident on his right lateral chest. His vital signs were as follows: heart rate 60 beats/minute, blood pressure 80/50 mm Hg, respirations 23/minute, rectal temperature 36.2°C. The cardiac examination disclosed an S4 heart sound. The rest of the physical examination was unremarkable. A diagnosis of anaphylactic shock was initially made. A

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venous line was inserted and rapid intravenous crystalloid solution was commenced, while the nurses were preparing adrenaline and hydrocortisone solutions. Interestingly, an electrocardiogram (ECG) recorded before the administration of adrenaline and hydrocortisone showed marked ST-segment elevation in leads II, III, and aVF; ST-segment depression in leads I, aVL, and V₁-V₆; along with a first-degree atrioventricular block (Figure 1), findings consistent with an acute inferior myocardial infarction. ECG recordings obtained from the right precordial leads did not show any evidence of right ventricular infarction. The infusion of adrenaline was aborted, and the patient was admitted to the Coronary Care Unit.

On admission, his blood pressure was 110/70 mm Hg, and he had regained consciousness. An echocardiographic examination performed at the bedside revealed hypokinesia of the inferior wall with an estimated ejection fraction of 0.50, and a mild concentric hypertrophy of the left ventricle. He was given aspirin, thrombolytic treatment with tenecteplase, and placed on a regimen of aspirin, heparin, metoprolol, and loratadine.

Soon after, the patient confirmed the sting and clearly described that a few minutes after the event he felt dizziness, somnolence, and retrosternal chest discomfort. He denied any previous symptom suggestive of coronary artery disease.

The initial biochemical studies were significant only for a troponin T of 2 ng/mL, while a complete blood count disclosed a mild leukocytosis (15,629 leukocytes/mm³). Serial enzyme measurement disclosed a peak creatinine kinase (CK) of 1,437 IU/L, and CK-MB 140 IU/L. During the next few hours the electrocardiographic abnormalities gradually resolved and the patient's clinical status markedly improved.

An ECG recording obtained on the third day of hospitalization showed T-wave inversion in the inferior leads, without the presence of Q waves (Figure 2). In addition, a second echocardiographic examination performed 3 days later showed only mild concentric hypertrophy of the left ventricle with an ejection fraction of 0.60. The patient's clinical course was uneventful, and a predischARGE exercise stress test was unremarkable. He was discharged on a regimen of aspirin, metoprolol, losartan, and pravastatin. The patient, as well his relatives, refused an elective cardiac catheterization.

Discussion

To the best of our knowledge this is the second case of myocardial infarction caused by a European hornet (*Vespa cabro linnaeus*) sting re-



Figure 1.
Twelve-lead
electrocardiogram
recorded on
admission.

ported in the literature. The first case, reported by Quercia et al,⁵ described a 55-year-old man who suffered angioedema and a non-ST-elevation myocardial infarction after a *Vespa cabro* sting. This insect belongs to the family of hymenoptera vespidae and looks like a large wasp. Notably, only a handful of cases have been reported in the literature describing cardiac damage following a sting by hymenoptera (mainly bees and wasps)¹⁻⁶ In some cases, the causal relationship is not very clear since adrenaline, which is often administered in the setting of anaphylaxis,¹⁻⁵ can aggravate myocardial ischemia, especially in elderly people with coronary heart disease. Certainly, this was not the case in our patient since the ECG abnormalities revealed prematurely led us to cancel the administration of adrenaline. Even though the ECG changes on admission were indicative of marked ischemia, our patient's left ventricular systolic function as assessed by echocardiography was relatively good, indicating that the shock was probably anaphylactic and not cardiogenic. Fortunately, the fluid challenge rapidly reversed the shock and allowed us to administer the thrombolytic treatment.

Hymenoptera venom contains vasoactive, inflammatory, and thrombogenic substances such as histamine, serotonin, dopamine, leukotrienes, thromboxanes, and bradykinin.^{2,7} Moreover, these mediators are further released by mast cells dur-

ing anaphylactic reactions inducing systemic involvement.^{2,3,8} Endogenous secretion of catecholamines has been attributed to the action of histamine and serotonin. All the aforementioned substances, besides bradykinin, have been shown to cause coronary vasoconstriction, especially in individuals with coronary artery disease.^{2,3,6-8} In addition, these vasoactive mediators provoke hypotension and an increase in myocardial oxygen demand through direct inotropic or chronotropic effects, leading to myocardial ischemia. Interestingly, serotonin, adrenaline, and thromboxanes induce platelet aggregation accelerating thrombus formation.^{2,3,6-9}

Taking into account these pathophysiologic considerations it is reasonable to assume that the European hornet sting, along with the hypotension in the context of anaphylactic shock, induced the myocardial infarction in our patient. Even though our patient had not had known coronary artery disease, we cannot exclude its presence since he refused further evaluation by coronary angiography.



Figure 2. Twelve-lead electrocardiogram recorded on the third day of hospitalization.

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