

Non-ST-elevation myocardial infarction after a wasp sting

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Abstract

We report a case of a 56-year-old woman with loss of consciousness transferred to the cardiology unit where non-ST-elevation myocardial infarction was diagnosed. The patient reported a wasp sting a few minutes before the accident. The cardiac troponin level was high and confirmed damage of the heart muscle. Finally, echocardiography and myocardial perfusion scintigraphy revealed an ejection fraction of 50% with hypokinesia of posterior walls. Cardiac consequences of the insect venom sting are discussed in this article.

Key words: anaphylaxis, wasp, non-ST-elevation myocardial infarction myocardial infarction.

Case report

Insect venom induced allergic reaction is a life-threatening disorder. Insect stings can cause severe effects, especially in atopic subjects, which may lead to cardiovascular collapse and death. The mortality rate is estimated to be approximately 1–5% in different countries [1].

Besides the typical, immediate type reactions, in very few cases of insect stings, unusual reactions are observed.

A 56-year-old woman who became unconscious in the mountain forest (picking wild mushrooms), after a sting of the wasp was presented for evaluation in the emergency department of the cardiology unit. She reported that 5 min after she was stung by the wasp, generalized urticaria and itching occurred. She complained of a chest pain. After another 5 min she developed a reaction with angioedema and 0.5 h later, unconsciousness and prolonged arterial hypotension. She was found fortunately by an incidental tourist. She was transferred to the emergency department. There, at the time of investigation, she regained consciousness and complained of stenocardial pain. On examination the patient appeared very weak. Urticarial changes were present on the skin of the face, neck and thorax. The mucous membranes of the throat and tonsils were normal, without inflamma-

tory changes. Lungs were clear to auscultation. Cardiovascular examination revealed a holosystolic murmur at the base of the heart. Abdominal examination was unremarkable. Neurologic examination was without deficits.

Her past medical history was unremarkable. She was in excellent health, had no history of immune-mediated diseases and denied any history of allergic, respiratory and cardiological illnesses. We did not prove preceding ischemic heart disease.

The first electrocardiography examination performed after one hour was normal. Because of prolonged stenocardial complaints of the patient, another electrocardiography examination was performed in the 3rd h after a sting and was also normal.

The alanine aminotransferase activity reached 38 U/l (3–26 U/l) and the aspartate aminotransferase 117 U/l (6–18 U/l). The creatine kinase-MB/total Ck ratio was 3.8, and the troponin level was 74.2 ng/ml (cTnI). The serum creatinine level was 1.52 mg/dl (< 1.5 mg/dl). The c-reactive protein level was 24 mg/l (normal value < 5 mg/l). In spite of absence of typical electrocardiographic changes we suspected myocardial infarction and decided to make further investigations. The diagnosis was confirmed by echocardiography which revealed an ejection fraction of 50% with hypokinesia of posterior walls, as well as the septum, showing a mild reduction of the general contrac-

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tility and a sign of valvular vegetation. Following cardiac investigation, myocardial perfusion scintigraphy showed a local area of myocardial infarction of the posterior wall. The consulting cardiologist recognized non-ST-elevation myocardial infarction (NSTEMI).

Chest radiographs were normal, ultrasonographic examination of the abdomen was normal.

Immunologic studies showed an elevated serum total IgE level of 288 IU/l (ImmunoCAP, Phadia) and elevated allergen-specific IgE levels against wasp venom allergen of 14.9 IU/ml. The total tryptase serum concentration in blood collected 5 h after the incident was 124 µg/l (normal < 11.4 µg/l). An intracutaneous test with wasp venom performed 4 weeks later was positive at a concentration of 10^{-5} g/l (ALK-Abello, Spain).

The patient was treated with nitrates, thrombolytics, antihistamines, and corticosteroids with a marked improvement. She was released in good condition and further cardiological and allergy treatment, and venom immunotherapy were advised.

Discussion

Allergic reactions to the venom of some stinging insects, such as honey bees, yellow jackets, hornets, wasps or fire ants can be life threatening. However, only a small number of people with insect bite or sting allergies suffer fatal reactions. Anaphylaxis is the most severe and prevalent insect sting reaction. There are also rare cases of unusual systemic reactions to insect stings, e.g. serum sickness, cerebral infarction, diffuse alveolar hemorrhage, rhabdomyolysis, glomerulonephritis, acute renal failure, thrombocytopenic purpura, vasculitis and disseminated intravascular coagulation (DIC) [1, 2]. Myocardial infarctions after insect stings are very rarely reported in the literature [3–5]. Most of acute coronary syndromes occur with ST segment elevation [3, 5, 6]. Rapid development of symptoms are assigned either to myocardial ischemia or anaphylaxis especially without cutaneous symptoms. Hemodynamic events in the course of anaphylaxis may reduce coronary blood flow, but it should be also remembered that histamine, serotonin, noradrenaline, dopamine released during the allergic reaction is a potent coronary vasoconstrictor and has direct inotropic and chronotropic effects increasing myocardial oxygen demand. Deep hypotension during anaphylaxis might be responsible for myocardial ischemia. Mediators released by activated mast cells as a cause of coronary symptoms were described by Kounis as allergic angina or allergic myocardial infarction in 1999 [7]. Adrenalin used in treatment of anaphylaxis is able to induce vasoconstriction and itself has thrombogenic effects. Our patient was not treated with adrenaline, so this pathomechanism of myocardial injury could be excluded. Wagdi *et al.* reported myocardial infarction after a wasp sting preceded by an allergic reaction without any angiographic changes in

coronary arteries [4]. There were even described cases of acute myocardial infarction after a wasp sting without any developed allergic reaction, indicating venom components as a cause of ischemia [5, 8]. Phospholipase A₁ present in the Hymenoptera venoms can induce thrombogenic reactions which could lead to acute arterial thrombosis. The case of acute thrombosis in the stent of the right coronary artery of a bee keeper stung by a wasp was described [8]. The patient has been stung by bees many times without any symptoms presumably because bee venom contains less phospholipase A₁ than wasp venom [9]. We should also be aware of other reasons for cardiovascular complications in the course of anaphylaxis and its treatment. Arslan *et al.* reported a case of a young, nonatopic man with a history of anaphylaxis after a wasp sting, complicated by acute ST-elevation myocardial infarction after intravenous methyl prednisolone administration [10]. To date, over 100 reports of immediate hypersensitivity reactions occurring after oral and parenteral administration of corticosteroids were published.

Conclusions

Indication of anaphylaxis as the cause of myocardial infarction without ST elevation in an unconscious patient is sometimes astonishing because these conditions occur infrequently and demand additional diagnostic procedures and adequate treatment.

Conflict of interest

The authors declare that they have no interests to disclosure that are relevant to this publication.

References

1. Bilo B, Rueff F, Mosbech H, et al.; the EAACI Interest Group on Insect Venom Hypersensitivity. Diagnosis of Hymenoptera venom allergy. *Allergy* 2005; 60: 1339-49.
2. Gawlik R, Rymarczyk B, Rogala B. A rare case of intravascular coagulation after honey bee sting. *J Invest Allergol Clin Immunol* 2004; 14: 250-2.
3. Ioannidis T, Mazarakis A, Notaras S, et al. Hymenoptera sting-induced Kounis syndrome: effects of aspirin and beta-blocker administration. *Int J Cardiol* 2007; 121: 105-8.
4. Wagdi P, Mehan VK, Burgi H, Salzmann C. Acute myocardial infarction after wasp stings in a patient with normal coronary arteries. *Am Heart J* 1994; 128: 820-3.
5. Bongo AS, Fornaro R, Sansa M, et al. Acute myocardial infarction after wasp sting without anaphylactic reaction. *Ital Heart J Suppl* 2005; 6: 178-8.
6. Valla M, Moulin F, Angioi M, et al. Myocardial infarction in a 45-year-old man following an anaphylactic reaction to a wasp sting. *Int J Cardiol* 2011; 148: 63-5.
7. Kounis NG, Grapsas ND, Goudevenos JA. Unstable angina, allergic angina, and allergic myocardial infarction. *Circulation* 1999; 100: 156.

8. Greif M, Pohl T, Oversohl N, et al. Acute stent thrombosis in a sirolimus eluting stent after wasp sting causing acute myocardial infarction: a case report. *Cases J* 2009; 2: 7800.
9. Binder M, Fierlbeck G, King T, et al. Individual hymenoptera venom compounds induce upregulation of the basophil activation marker ectonucleotide pyrophosphatase/phosphodiesterase 3 (CD203c) in sensitized patients. *Int Arch Allergy Immunol* 2002; 129: 160-8.
10. Arslan Z, Iyisoy A, Tavlasoglu M. Acute myocardial infarction after prednisolone administration for the treatment of anaphylaxis caused by a wasp sting. *Cardiovasc J Afr* 2013; 24: 4-6.