Manifestations Mimicking Acute Myocardial Infarction after Honeybee Sting

Ho-Pang Yang, Fu-Chung Chen, Chien-Cheng Chen, Thau-Yun Shen, Shih-Ping Wu and Yung-Zu Tseng

Acute myocardial infarction (AMI) due to arthropod envenomization has rarely been reported. Herein, we report a 65-year-old woman with clinical manifestations mimicking AMI and left ventricular wall motion abnormality after honeybee sting. The patient eventually recovered from her illness with normal left ventricular wall motion on echocardiogram and T wave inversion on electrocardiogram. We also review the literatures.

Key Words: Arthropod envenomization • Honeybee sting • Acute myocardial infarction • Echocardiogram

INTRODUCTION

Acute myocardial infarction (AMI) due to honeybee sting is rare, and only a few authors have discussed the relationship between the sting, anaphylactic shock and myocardial infarction.1-4 The allergic reaction induced by hymenoptera stings seems to have triggered inflammatory mediator release and thus induced acute coronary syndrome. Kounis syndrome is the concurrence of acute coronary syndrome with mast cell activation induced by allergic or hypersensitivity and anaphylactoid reactions.5 Takotsubo cardiomyopathy is a stress-induced cardiac syndrome mimicking acute myocardial infarction.6,7 Herein, we report a case with clinical manifestations mimicking acute myocardial infarction following honeybee sting and review the literature.

CASE REPORT

A 65-year-old woman was sent to our hospital because of honeybee sting half an hour before. Her past history was noted with only dyslipidemia. At emergency room (ER), she complained of chest pain, cold sweating, generalized flash and near fainting. Her vital signs were blood pressure: 154/98 mmHG, heart rate: 125 bpm regular and respiratory rate: 20/min. Otherwise, there were no particular findings on physical examination. AMI was suspected by the presence of chest pain, electrocardiogram (ECG) showing bigeminal ventricular premature contractions (VPC’s) and significant ST-segment elevation in leads V2-6 (Figure 1A), and high serum creatine phosphokinase (208 U/L; normal value, 12-144 U/L), creatine kinase-MB (5.21 ng/ml; normal value, < 4.89 ng/ml) but normal serum troponin-I level (< 0.2 ng/ml; normal value, < 0.2 ng/ml). This patient had some risk factors for coronary artery disease, including old age, being postmenopausal, and dyslipidemia. Her family history was unremarkable for coronary artery disease or any kind of venous and/or arterial thrombotic events. Emergency coronary angiography was performed immediately and revealed a 60% stenosis with TIMI 3 flow in the distal right coronary artery (Figures 2A, 2B). We did not perform the ergonovine or acetylcholine challenge test for coronary artery vasospasm. The patient’s echocardiogram showed apical akinesis with preserved left ventricular ejection fraction (Figure 3A). No potential sources of cardiac emboli were detected by echocardiography. The patient’s serum lipid levels were as follows:
total cholesterol, 156 mg/dL; low-density lipoprotein, 109 mg/dL; triglycerides, 35 mg/dL; and high-density lipoprotein, 84 mg/dL. The ECG of 36 hours after sting (Figure 1B) displayed evolutional changes of ST-T wave after myocardial injury. The peak levels of serum CPK (1192 U/L; normal value, 12-144 U/L), creatine kinase-MB (90.3 ng/ml; normal value, < 4.89 ng/ml) and serum troponin-I levels (24.8 ng/ml; normal value, < 0.2 ng/ml) occurred about 20 hours later. She undertook medications including antiplatelet drug and low-dose beta block. Her admission course was smooth, and she was discharged uneventfully five days later. About one month later, the patient received ECG and echocardiogram follow-up. The ECG (Figure 1C) revealed only symmetric T-wave inversion in leads II, III, aVF and V2-6. The echo (Figure 3B) showed no regional wall

**Figure 1.** Twelve-lead electrocardiograms. (A) One hour after sting, with begeminal VPCs and ST elevation in leads V2-6. (B) 36 hours after sting, with ST elevation and T-wave inversion in leads II, III, aVF and V2-6. (C) One month after sting, with only mild T-wave inversion in leads II, III and aVF, and symmetrical T-wave inversion in leads V2-6.
motion abnormality and preserved left ventricular ejection fraction.

DISCUSSION

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

Acute myocardial infarction occurs very rarely after an arthropod envenomization. There are a few cases of AMI due to bee or scorpion bites reported in the literature.1-4 In these patients, coronary arteries were normal...
or non-significantly stenotic. Our case presented clinical manifestations of AMI including chest pain and changes in cardiac enzymes and ECG. The coronary angiogram in our case also showed non-significant stenosis, which is the same as in the cases in the literature review. However, the exact mechanism of AMI caused by arthropod envenomization is unclear.

Hymenoptera (bees, wasps) stings or bites are responsible for more deaths than those from all other poisonous creatures. Systemic reactions after stings are usually of immediate type. The most frequent clinical events are hypotension, dyspnea, anaphylactic shock and angioedema. There are several reports dealing with cardiovascular complications after hymenoptera stings. The important medical problem posed by hymenoptera stings is the development of anaphylaxis. Hypotension is a common manifestation, with vasodilation and decrease of intravascular volume. Patients are usually hypotensive on admission, but a few were hypertensive as in our case. Many pharmacologically active constituents of hymenoptera venoms have been isolated. All these substances can provoke ischemia and even myocardial injury via profound hypotension or by increasing oxygen demands through direct inotropic and chronotropic effects in the presence of compromised myocardial supply.

Electrocardiographic changes consistent with acute myocardial ischemia or infarction, including ST depression or elevation and even the appearance of pathologic Q-waves, have been recorded in people after stings. Rhythm abnormalities such as supraventricular arrhythmias, VPC’s, junctional rhythm and right bundle branch block were also recorded during initial stages after the sting. Animal studies of bee venom have shown that such ECG changes may be due to direct cardiotoxic effect. However, the mechanism is still not clear.

Laboratory abnormality indicating myocardial injury, such as elevated CPK and GOT, was noted in people with wasp sting. High serum creatine kinase and creatine kinase-MB levels following arthropod envenomization were reported previously. The rise in serum creatine kinase and creatine kinase-MB levels might be due to myocardial injury and/or rhabdomyolysis caused by extremely high sympathetic discharge. Elevation of more specific marker for myocardial injury such as cardiac troponin I or T following arthropod sting indicates direct or indirect myocardial injury caused by envenomization. Our case showed such change.

Transthoracic echocardiography showing regional wall motion abnormalities (hypokinesia and akinesia) and left ventricular dysfunction after an arthropod envenomization was reported. The stunned myocardium has long been known as a prolonged postischemic LV dysfunction after brief myocardial ischemia and presents a reversible LV dysfunction. Abrough et al. reported gradual normalization of wall motion abnormality in the left ventricle and septum, and also complete restoration of the systolic function in case with severe scorpion envenomation. The change in echocardiographic findings of our case is similar to that of above reported cases. Moreover, clinical, electrocardiographic, laboratory and echocardiographic findings in our case excluded the possibility of myocarditis or pericarditis.

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. The authors therefore recommend that patients presenting with AMI following an arthropod bite should be screened for any inherited thrombophilia. Another two patients who were stung by wasps and honeybee, respectively, developed Kounis syndrome as a consequence of allergic reaction. Kounis syndrome is the concurrence of acute coronary syndrome with mast cell activation induced by allergic or hypersensitivity and anaphylactic anaphylactoid reactions.

Takotsubo cardiomyopathy (Takotsubo CM) is a novel cardiac syndrome characterized by transient and severe LV apical ballooning and basal hyperkinesias in acute stage. Although Takotsubo cardiomyopathy shows striking initial manifestations mimicking AMI, the minimal change of cardiac enzymes are not consistent with the extent of LV change in acute stage, and unusual LV morphology was restored to normal, usually within several weeks, in most cases. In patients with Takotsubo CM, the ECG in acute stage shows concave ST-segment elevation, usually in leads V3-6, there is less dynamic change for days, followed by T-wave inversion and resolved in approximately 2-3 weeks associated with QTc prolongation. Abnormal Q-wave and reciprocal changes are rarely seen. Our case has similar initial manifestation as seen in Takotsubo cardiomyopathy, but the changes in cardiac enzyme and dynamic change of ECG were quite different from these of typical case.
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 vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis; direct cardiotoxic effect of the venom; and anaphylactic reaction. This sequence of events, compounded by thrombosis at the site of spasm, can precipitate AMI. In our case, the findings mimicking acute myocardial infarction (including clinical symptoms, dynamic changes of ECG, cardiac enzyme and regional wall motion) were noted after a honeybee sting. But the exact mechanism of AMI after hymenoptera sting is still unclear. It may be an atypical type of Takotsubo cardiomyopathy.

REFERENCES