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Acute myocardial infarction after a Yellow jacket sting in 22 year old man in Iran

Sajjad Ahmadi[1], Hamidreza Morteza Bagi[2], Gholamreza Farid Aalaee[1], and Seyed Hesam Rahmani[1]*

[1]Emergency Medicine Department, Faculty of Medicine, Urmia University of medical sciences, Urmia, Iran
[2]Emergency Medicine Department, Faculty of Medicine, Tabriz University of medical sciences, Tabriz, Iran
Corresponding author

KEYWORDS

Myocardial Infarction;
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A B S T R A C T

A 22-year-old previously healthy man was stung in the lip by a yellow jacket. He came to the emergency department After 15 minutes with urticaria, dyspnea and chest pain and within two hours sustained an acute myocardial infarction. Acute myocardial infarction after an insect sting is rare; only few unequivocal cases have been repotted world wide. That they were after wasp sting, but we report a case of myocardial infarction complicating a yellow jacket sting in the Iran. Possible pathogenic mechanisms include the anaphylactic reaction itself, the action of yellow jacket venom constituents, and therapeutic intervention with adrenaline

Introduction

Anaphylaxis is a medical emergency that requires immediate diagnosis and treatment. Definitions of anaphylaxis have conflicted over the years, but recent clarity has emerged based on consensus symposia. In simple terms, "anaphylaxis is a serious allergic reaction that is rapid in onset and may cause death (1-2). The incidence of anaphylaxis is 4–5 per 100,000 persons per year (1-2), with a lifetime risk of 0.5–2% (2). Currently, anaphylaxis leads to 500–1,000 deaths per year (2.4 per million) in the United States, 20 deaths per year in the United Kingdom (0.33 per million), and 15 deaths per year in Australia (0.64 per million) (2). Anaphylaxis typically presents with many different symptoms over minutes or hours(3-4) with an average onset of 5 to 30 minutes if exposure is intravenous and 2 hours for foods(5). The most common areas affected include: skin (80–90%), respiratory (70%), gastrointestinal (30–45%), heart and vasculature (10–45%), and central nervous system (10–15%)(4) with usually two or more being involved(6). Coronary artery spasm may occur with subsequent myocardial infarction, dysrhythmia, or cardiac arrest (4, 6). Those with underlying coronary disease are at greater risk of cardiac effects from

anaphylaxis (7). The coronary spasm is related to the presence of histamine-releasing cells in the heart (7). While a fast heart rate caused by low blood pressure is more common (8). Anaphylaxis can occur in response to almost any foreign substance (9). Common triggers include venom from insect bites or stings, foods, and medication (8, 10). Foods are the most common trigger in children and young adults while medications and insect bites and stings are more common in older adults(6). B-Lactam antibiotics are estimated to cause 400 to 800 deaths in the U.S. annually, with a systemic allergic reaction occurring in 1 per 10,000 exposures.' Hymenoptera stings constitute the next most common cause of anaphylaxis, with fewer than 100 deaths in the US annually.

Anaphylaxis after an insect sting usually presents as hypotension, bronchospasm, and laryngeal edema. Electrocardiographic changes (11-13), chest pain (14), and three instances of acute myocardial infarction (15) -two in North America and one in Sweden- have been described. Severe allergic reactions (anaphylaxis) to insects are relatively uncommon, and are usually due to bees, wasps or the Australian Jack Jumper ant. In the vast majority of patients, signs and symptoms begin suddenly, often within 60 minutes of exposure. In general, the faster the onset of symptoms, the more severe the reaction, as evidenced by the fact that one half of anaphylactic fatalities occur within the first hour. Anaphylaxis from stinging insect allergy results in an average of three deaths per year in Australia. Older individuals and those with severe difficulty breathing are at greatest risk and should be seen by a medical specialist. Those at greatest risk of further serious reactions are people who have suffered an episode of shock or severe difficulty breathing following a sting. Adults are at greater risk than children. Anyone with a history of a generalized reaction to an insect sting should be referred to a medical specialist (Allergist / Clinical Immunologist). The occurrence of an acute myocardial infarction following a 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. Anaphylaxis is diagnosed based on clinical criteria(6), and is a medical emergency that may require resuscitation measures such as airway management, supplemental oxygen, large volumes of intravenous fluids, and close monitoring(3). Administration of epinephrine is the treatment of choice with antihistamines and steroids often used as adjuncts (6).

Case report

A 22- year- old man was stung in the lip by a yellow jacket, while drinking the water.
He experienced lip pain, shortening of breathing and generalized urticaria rash followed by chest pain. When he arrived at an emergency department 30 minutes after being stung he was agitated, and had urticaria, with neck and face edema followed by lethargy.

By the time of arrival some ten minutes later, he was unconscious and his blood pressure unobtainable. He had no history of ischemic heart disease, diabetes, or allergy, and he was not a smoker. However, he recalled that he had been stung one time, in the past, by honeybees, without any sequelae, but had no previously experienced a yellow jacket sting. Blood sample was obtained. Electrocardiogram showed acute ST-segment elevation in inferior and precordial leads (Figure 1).

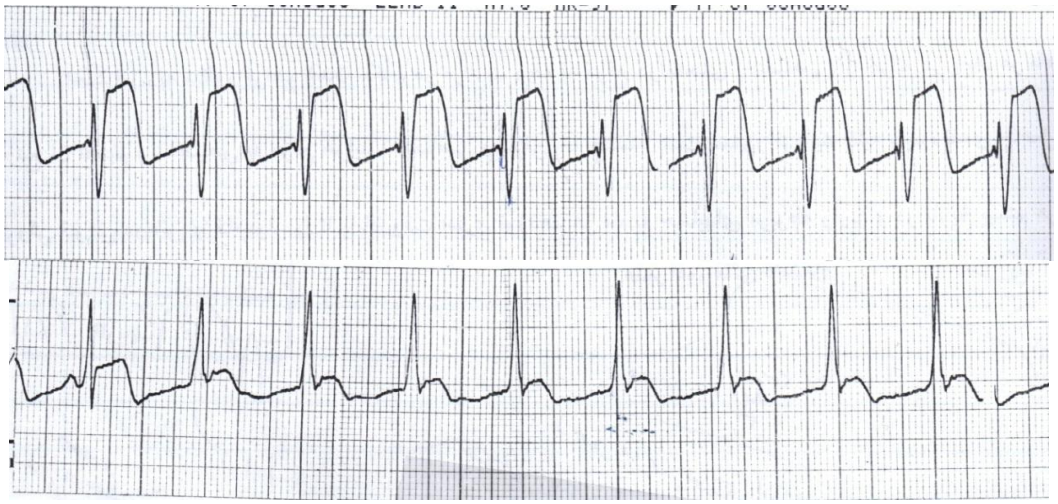


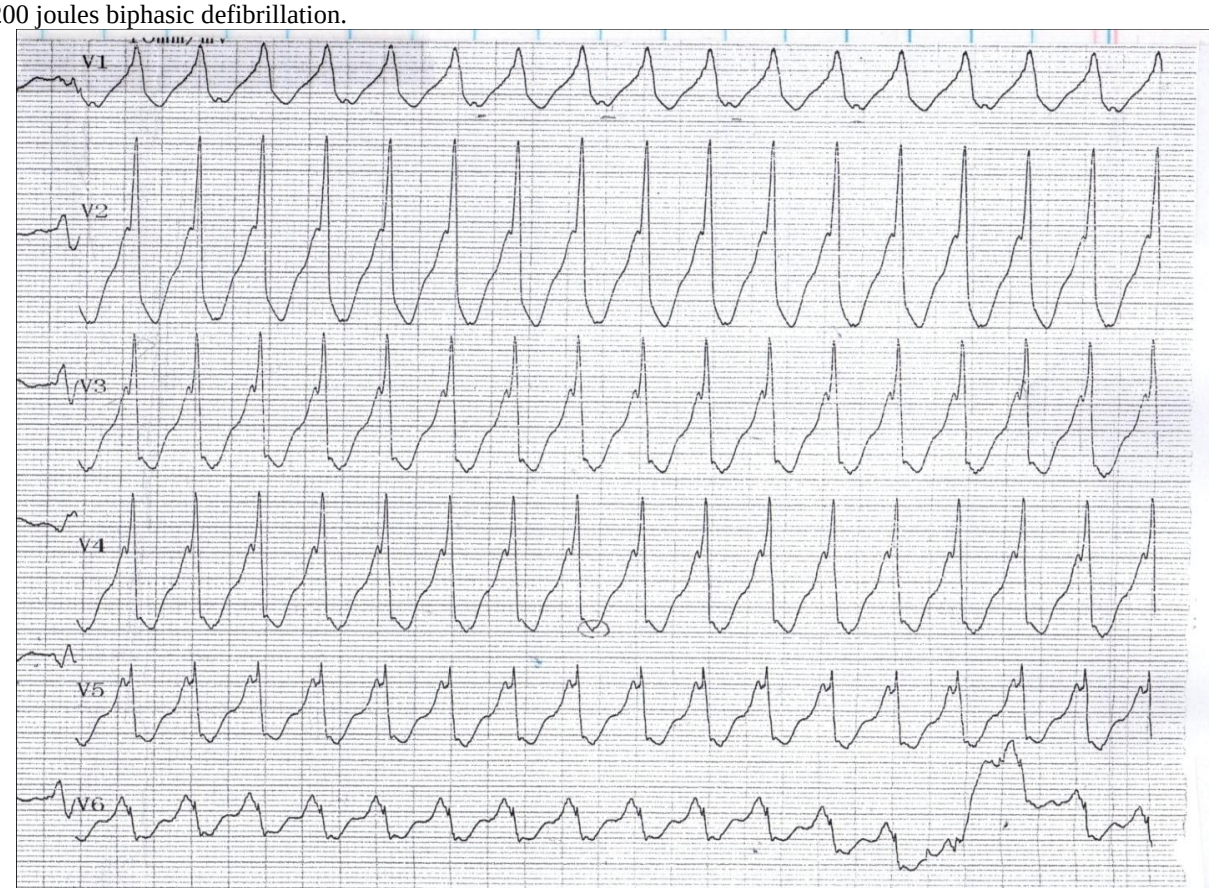
Figure.1 ST_ segment elevation (lead II & avf)

The first value of cardiac troponin–I and creatine phosphokinase - MB (CPK-MB) was elevated [CTNI =120 ng/dl (normal values < 0.1 ng/dl)].

Treatment for anaphylactic shock was started with hydrocortisone, chlorpheniramine, ranitidine, salbutamol spray, supplemental oxygen and subcutaneous adrenaline.

Serial CTNI and CPK-MB were elevated, and patients brain CT scan was normal.

During his treatment in cardiopulmonary room, he showed ventricular tachycardia figure 2 which was treated with 200 joules biphasic defibrillation.



At the second day echocardiography was done and showed decrease the ejection fraction (35%).at the third day the patient was conscious. And at the seventh day echocardiography showed localized partial akinesia in left ventricle and 35-40% ejection fraction.

Discussion

More specifically, coronary arteries are known to have two kinds of histamine receptor, H_1 and H_2 (16). The role of the H_2 receptors is not considered to be particularly important. However, stimulation of the H1 receptors with small doses of histamine in patients with healthy coronary arteries and with no history of ischemic heart disease causes vasodilation in both the epicardial coronary arteries and the smaller resistance vessels, via the release of NO (endothelium-dependent vasodilation)(17). It has also been observed that stimulation of those receptors in some patients who have a history of angina causes spasm of the epicardial coronary arteries (18). This is a particularly interesting finding that could be due to an increased concentration of mast cells in the adventitia of the patients' coronary arteries (19-20) and hence to the release of histamine in relatively high concentrations, and/or to the coexistence of endothelial damage in those coronary arteries, something that would disturb their tone and lead to spasm rather than vasodilation after the local release of histamine. The fact that experiments have shown large concentrations of histamine to cause vasoconstriction rather than vasodilation supports the former view (21).

The reaction to a wasp sting can be prolonged or severe in allergic individuals and in some cases anaphylaxis may ensue, with urticaria, circulatory collapse, and bronchospasm. This is the result of a sequence of events including the release of serotonin and histamine and the formation of leukotrienes(22). Some of which are potent coronary vasoconstrictors in various animal species(23-24). Many pharmacologically active constituents of wasp venom have been isolated including histamine, serotonin, dopamine, noradrenaline , and a bradykinin-like substance which may itself induce histamine release(25). Endogenous secretion of adrenaline and noradrenaline is stimulated by histamine and serotonin. All these substances can provoke myocardial ischemia either via profound hypotension or by increasing myocardial oxygen demand through direct inotropic or chronotropic effects in the presence of a compromised myocardial blood supply.

Platelet aggregation is induced by serotonin(26) and adrenaline(27-28). Adrenaline also accelerates thrombus formation in animals and in man, possibly by increased factor V activity(29-30), and has been shown in animals to release a thromboplastin-like substance from the walls of blood vessels(31). It causes –both coronary vasodilatation and increased myocardial oxygen demand by direct inotropic and chronotropic effect. It has been used historically as a provocation test for angina pectoris and is often used in the treatment of anaphylactic shock (32-34).

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 (35).

Anaphylactic reactions after different insects sting may induce cardiovascular events, including acute myocardial infarction, even in patients with normal coronary arteries(36-38). Active venom substances are responsible for direct venom cardiotoxicity causing vasoconstriction and platelet aggregation(32,36).

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(33-34, 39-43). 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 (34,37, 44-46). Therefore, the management strategy 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(46). "The allergic angina syndrome" which could progress to acute myocardial infarction ("allergic myocardial infarction") was firstly described in 1991 by Kounis and Zavras(47). Allergic angina and allergic myocardial infarction are now referred as "Kounis syndrome"(48-50). This syndrome is associated with mast cell degranulation and it is caused by mediators released during degranulation.

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.

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