Silent acute myocardial infarction following a wasp sting

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Key words:
Myocardial infarction; Silent ischemia.

Hymenoptera sting can lead to an acute myocardial infarction by different pathogenetic mechanisms depending on the presence of preexistent coronary atherosclerosis, the development of shock or the therapeutic use of epinephrine.

The case of a 67-year-old man with acute myocardial infarction with ST-segment elevation after a wasp sting treated with fibrinolysis and without significant coronary atherosclerosis is reported. Of particular interest in the present case report is the silent presentation and the absence of any pharmacological interference.

It follows that in any case of hymenoptera envenomation a standard ECG is advisable even when a clearly defined allergic reaction is not present.

(Ital Heart J 2003; 4 (9): 638-641)

Received March 3, 2003; revision received June 4, 2003; accepted June 12, 2003.

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Introduction

The occurrence of an acute myocardial infarction (AMI) after a bee, wasp or yellow-jacket sting has been occasionally reported in the literature[1-6]. Anaphylaxis and the therapeutic use of epinephrine can act synergically in promoting acute coronary artery thrombosis via hypotension and platelet aggregation.

The pathophysiology and clinical presentation are mainly attributable to three mechanisms: 1) an AMI following anaphylaxis and shock[1,5]; 2) a typical AMI occurring in patients with coronary atherosclerosis[1,2,6]; and 3) an AMI occurring in subjects without significant coronary artery disease in whom coronary thrombosis and vasospasm enhanced by intoxication are the postulated mechanisms[3,4].

We report the case of a patient presenting with AMI following a wasp sting. Of particular interest in the present case report is the silent presentation and the absence of any evidence of coronary atherosclerosis.

Case report

A 67-year-old man was referred to the emergency room of our hospital 60 min after he had been stung several times on his head and upper limbs by wasps. He referred a short-lasting episode of syncope followed by periorbital edema and glossopharyngeal paresthesia without respiratory discomfort. He was on treatment with irbesartan 150 mg once daily for hypertension and with theophylline 400 mg daily for chronic obstructive pulmonary disease; he was a former smoker and he had been hospitalized some months before because of an allergic reaction to cephalosporin therapy. In the past, he had already been stung by a wasp without any systemic reaction.

Clinical evaluation in the emergency room revealed only mild bronchospasm; his blood pressure was 135/75 mmHg. Maleate-clorfeniramine (10 mg i.v.) and methylprednisolone (1 g i.v.) were administered and the above-mentioned symptoms resolved.

A 12-lead ECG collected soon after presentation (75 min following the wasp sting) showed antero-lateral ST-segment elevation (Fig. 1A); the patient was asked whether he had any chest pain or dyspnea, which he denied; the serum levels of the early markers of myocardial cytonecrosis were requested and aspirin (300 mg) was given. The results of the laboratory analysis were: hemoglobin 14.9 g/dl; white cell count 24 100/mm^3; eosinophils 1.8%; fibrinogen 394 mg/dl; myoglobin 198 μg/l; troponin T 0.07 ng/ml (upper limit of normal by our hospital 50 μg/l and 0.1 ng/ml, respectively).

On subsequent admission to the cardiology unit 2 hours after the wasp sting, the patient referred no symptoms while physi-

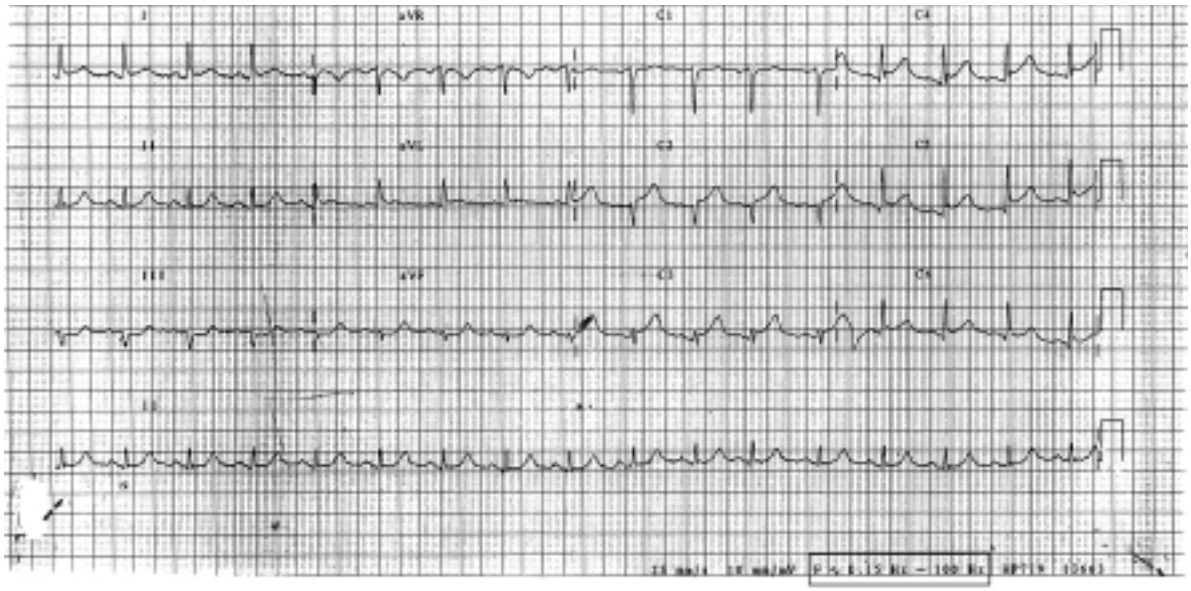
cal examination confirmed the presence of bronchospasm and of a 2/6 L mitral murmur. His cardiovascular risk factors included hypertension, a past smoking habit and LDL hypercholesterolemia (140 mg/dl).

Transthoracic echocardiography documented akinesis of the septo-apical segment and hyperkinesis of the basal segments of the left ventricle (left ventricular ejection fraction 45%) along with mild mitral regurgitation.

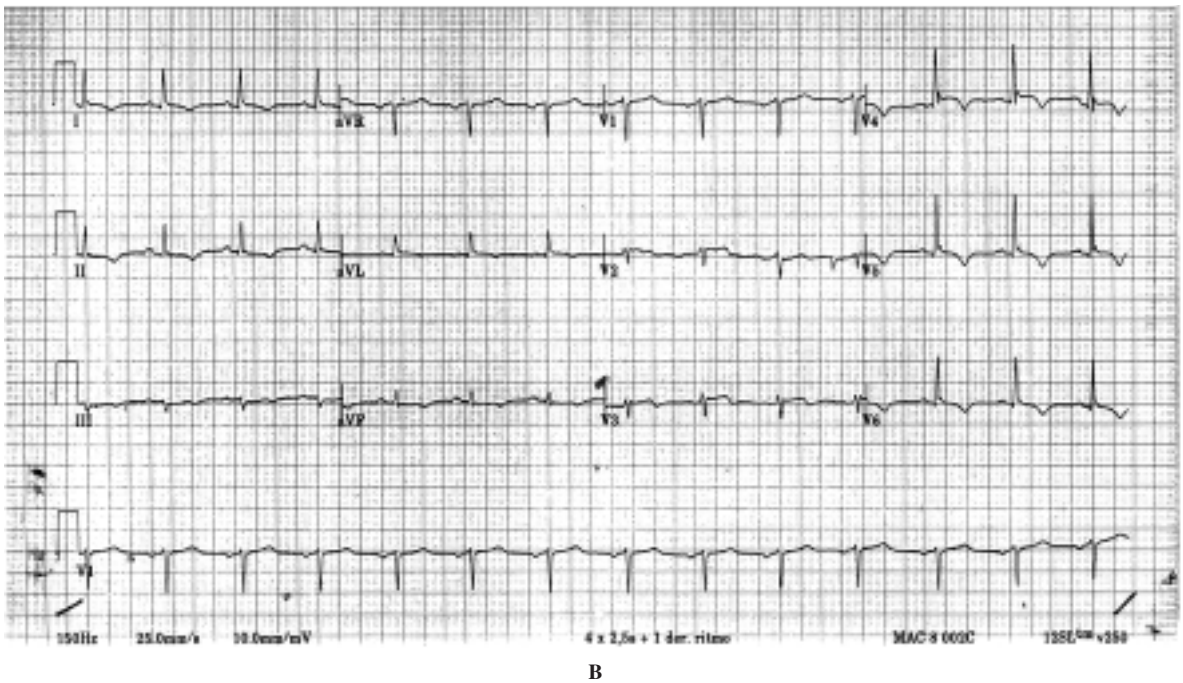
Because of the persistence of ST-segment elevation > 30 min and in view of the resistance to the i.v. infusion of nitroglycerin (40 μg/min) started in the emergency room, fibrinolysis with tenecteplase (i.v. bolus 7000 IU) was attempted within the third hour of the wasp sting.

The patient still presented with signs of a poor reperfusion, i.e. ST-segment elevation persisting for 60 min, myoglobin levels at 90 min of 178 μg/l (vs basal 198 μg/l). Subsequent monitoring showed a peak creatine kinase-MB mass value as low as 24 ng/ml (normal value 0-5 ng/ml) along with 12-hour troponin T levels of 0.48 ng/ml and a negative T wave on the ECG tracing (Fig. 1B).

The patient never complained of chest pain and a submaximal treadmill test performed on day 6 was neg-



A



B

Figure 1. A: *ECG on admission at the emergency room.* B: *ECG taken 24 hours following admission.*

ative. The hyperventilation test and Holter monitoring for spontaneous myocardial ischemia were also negative. He was discharged on aspirin, metoprolol, irbesartan and simvastatin and has been well since then. Elective angiography performed 1 month later revealed the presence of normal coronary arteries.

Discussion

Our report focuses on a case of ST-elevation AMI following a hymenoptera sting in the absence of prolonged hypotension or coronary artery disease (Table I)[1-6]. Even though coronary angiography was not performed during the acute phase[6], the pathophysiological mechanism of local coronary thrombosis activation, probably related to vasospasm, is highly suggestive[7].

Histamine, serotonin, kinins such as SRS-A and bradykinin, acetylcholine, apamine, phospholipase A and B and ialuronidase have all been sought and identified as constituents of the hymenoptera venom. Almost all of these substances are able to cause endogenous amine release and vasodilation which lead to paradoxical vasoconstriction in the presence of endothelial damage or dysfunction. Moreover, just as snake venom, the hymenoptera venom also contains epinephrine, dopamine, leukotrienes and thromboxanes, which cause severe platelet aggregation and direct vasoconstriction[1].

As an AMI is a rare consequence of the otherwise widespread exposure to the hymenoptera venom, other factors are likely to be necessary if vasoconstriction and/or platelet aggregation leading to occlusive intracoronary thrombosis are to occur. As a matter of fact, many of the previously published case reports refer the concurrent use of therapeutic dosages of epinephrine, a drug known to promote both vasoconstriction and platelet aggregation, which has not been administered to our patient (Table I).

The fact that the coronary arteries appeared normal at angiography does not exclude the possible presence of endothelial dysfunction, in view of the presence of three established cardiovascular risk factors. Paradoxical vasoconstriction due to the exogenous mediators contained in wasp venom is therefore also possible.

Primary coronary artery vasospasm (usually associated with chest pain and an ischemic pattern on the ECG) could be the alternative pathophysiological hypothesis which has been suggested by Brasher and Sanchez[8] in the setting of a hymenoptera sting and by Matucci et al.[9] as a consequence of an immunoglobulin E-related allergic reaction.

The persistence of ST-segment elevation (transmural ischemia) after the infusion of high-dose nitroglycerin is against the hypothesis of the mechanism of vasospasm. Neither hypereosinophilia nor hyperfibrinogenemia were present, both of which have been recently suggested as markers of vasospastic angina[10]. The traditional tests for vasospasm such as hyperventilation and Holter monitoring (transient spontaneous ST-segment elevation) have turned out to be negative[11]. The intracoronary infusion of acetylcholine, which has been reported to be diagnostic for endothelial dysfunction, has not been performed because of possible serious adverse reactions[12].

A distinctive aspect of the present case report is the absence of ischemic chest pain which has always been included in previous clinical descriptions, irrespective of the lag between the time of intoxication and the onset of ECG ischemic modifications.

A similar case of silent myocardial ischemia but without evidence of AMI associated with wasp sting anaphylaxis has been reported by Brasher and Sanchez[8].

The prevalence of a silent AMI has been estimated to range from 10 to 30% in the Framingham study and the prognostic significance of silent myocardial ischemia, whether spontaneous or provoked by the stress test and revealed by imaging techniques, has been assessed during the last 20 years[13]. Yet, we still do not know why myocardial ischemia is not associated with chest pain in some individuals.

Table I. Cases of an acute myocardial infarction following a hymenoptera sting reported in the literature.

Author	Sex/age	ECG pattern of presentation	Hypotension or shock	Epinephrine therapy	Coronary arteriography*/ reperfusion therapy
Levine[1]	M/39	STE anterior	+	+ (s.c.)	- / -
	M/66	STE infero-lateral	+	+ (i.v.)	- / -
Jones and Joy[2]	M/56	STE anterior	+	+ (s.c.)	+ / -
Wagdi et al.[3]	F/50	STE anterior	-	+ (s.c.)	+ / + (SK)
Ceyhan et al.[4]	F/22	NSTE infero-lateral	-	?	+ / -
Quercia et al.[5]	M/44	STE lateral	?	+	- / -
	M/55	STE inferior	?	-	- / -
Calveri et al.[6]	M/77	STE postero-lateral	+	+ (i.v.)	+ / + (PCI)
Lombardi et al.	M/67	STE anterior	-	-	+ / + (TNK)

NSTE = non-ST elevation; PCI = percutaneous coronary intervention; SK = streptokinase; STE = ST elevation; TNK = tenecteplase. + yes; - no; ? not reported. * case 3: three venous graft thromboses and occlusion of the left anterior descending coronary artery; cases 4, 5 , and 9: normal coronary arteries; case 8: proximal subocclusive stenosis of the left circumflex and second obtuse marginal arteries.

The increased production of cytokines and a specific pattern of the activation of the inflammatory response have recently been shown to form the basis of the absence of pain in humans during acute myocardial ischemia[14]. This could have been the case of our patient (whose medical history did not include any disease, such as diabetes mellitus, that may modify one's perception of pain), exposed to the simultaneous activity of the wasp venom and endogenous inflammatory promoters.

In conclusion, two interesting aspects of the present clinical case are to be mentioned as being unique if compared to previous descriptions in the literature: 1) an ST-elevation AMI related to the left anterior descending coronary artery occurred without concomitant epinephrine therapy that could independently lead to acute coronary thrombosis; and 2) the silent presentation of AMI following the wasp sting[15,16].

The diagnosis of AMI was possible by the use of simple instrumental exams which are not usually performed in patients presenting with no or only a mild allergic-like reaction. We therefore recommend that regardless of the severity of a patient's reaction to a hymenoptera sting, an ECG be performed in order to rule out myocardial ischemia.

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