



Clinical Communications: Adults

ST ELEVATION MYOCARDIAL INFARCTION WITH NO STRUCTURAL LESIONS AFTER A WASP STING

Sofiène Rekik, MD, Stéphane Andrieu, MD, Falah Aboukhouidir, MD, Pierre Barnay, MD,
 Gonzalo Quaino, MD, Michel Pansieri, MD, and Jean Lou Hirsch, MD

Cardiology Department, Avignon Hospital Center, Avignon, France

Reprint Address: Sofiène Rekik, MD, Cardiology Department, Avignon Hospital Center, 305 Rue Raoul Follereau, Avignon
 84902, France

Abstract—Background: Acute coronary syndromes after hymenoptera stings and other environmental exposures are referred to as the Kounis syndrome or allergic myocardial ischemia and infarction. **Case Report:** We report the case of a 58-year-old man with transient inferior ST-segment elevation consistent with myocardial ischemia after a single wasp sting. Urgent cardiac catheterization revealed normal coronary arteries with a normal left ventriculogram. The evaluation and treatment of cardiac ischemia associated with an allergic reaction is discussed. **Conclusion:** ST elevation myocardial infarction after wasp envenomation is an exceptional and interesting pathology with a partially elucidated pathogenesis. The management of cardiac ischemia in this setting is uncertain. © 2009 Elsevier Inc.

Keywords—myocardial infarction; allergy; Kounis syndrome; wasp sting; anaphylaxis; hypersensitivity reaction

INTRODUCTION

Although exposure to wasp stings is common throughout the world, severe anaphylaxis after such stings is less frequent. Rarely, acute coronary syndromes including ST-segment elevation myocardial infarction (STEMI) have been reported after hymenoptera stings (1–3). The pathophysiology of cardiac ischemia has been attributed to coronary artery vasospasm, although platelet

activation and coronary thrombosis also have been reported (2).

We report in this work the case of a 58-year-old man with no previous history of coronary artery disease, who presented soon after sustaining a hymenoptera sting. He had electrocardiographic changes consistent with inferior STEMI, and urgent cardiac catheterization revealed normal coronary arteries.

CASE REPORT

The patient was a 58-year-old man with a known history of medically controlled hypertension (amlodipine 5 mg once a day, but it was recently withdrawn due to peripheral edema). There was no history of coronary artery disease, nor allergy, cutaneous, or respiratory evidence of anaphylaxis. The patient was stung on the left elbow, as he later recounted to emergency personnel. A few minutes later, he felt severe dizziness with nausea and went to a nearby pharmacy complaining of severe chest pain with light-headedness. The pharmacist noticed extensive flushing with a large area of erythema and profuse diaphoresis, and called Emergency Medical Services (EMS).

Upon arrival of EMS, the clinical examination (by the accompanying emergency physician) revealed a confused patient with extensive erythema of the upper part

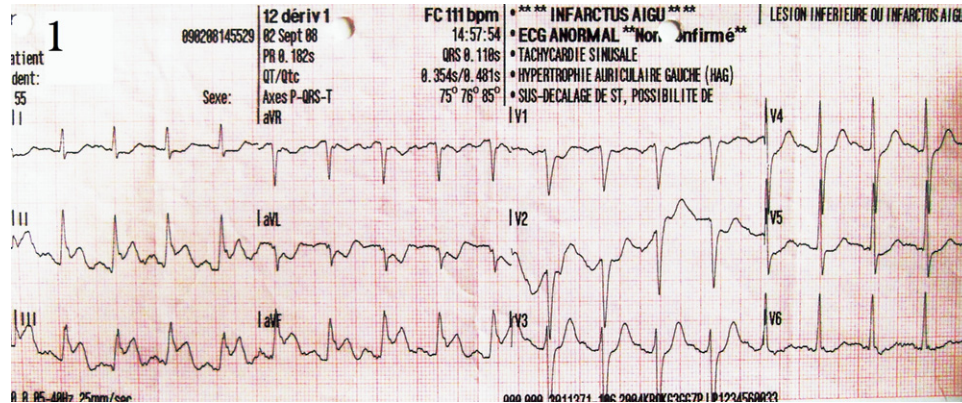


Figure 1. Initial EMS 12-lead electrocardiogram showing an ST-segment elevation in the inferior leads with an ST depression in V1, V2, and V3.

of the body. The vital signs were: heart rate 88 beats/min, blood pressure 86/54 mm Hg, and pulse oxymetry 96%. Cardiac and pulmonary auscultation were normal. A 12-lead electrocardiogram (ECG) showed a regular sinus rhythm and an obvious 4-mm ST elevation in the inferior leads with a mirror ST depression in leads V1, V2, and V3 (Figure 1). An inferior STEMI was diagnosed. The patient was given aspirin 500 mg and clopidogrel 300 mg, and was transferred to the cardiac catheterization

laboratory. Due to an obvious severe anaphylaxis, diphenhydramine 50 mg i.v. and methylprednisone 120 mg i.v. were also administered, but no epinephrine was given.

Serial ECGs were obtained during the transport, showing a regression of ST-segment elevation and T-wave inversions (Figure 2). The patient was nevertheless taken to the catheterization laboratory according to the local protocol. Selective coronary angiography (per-

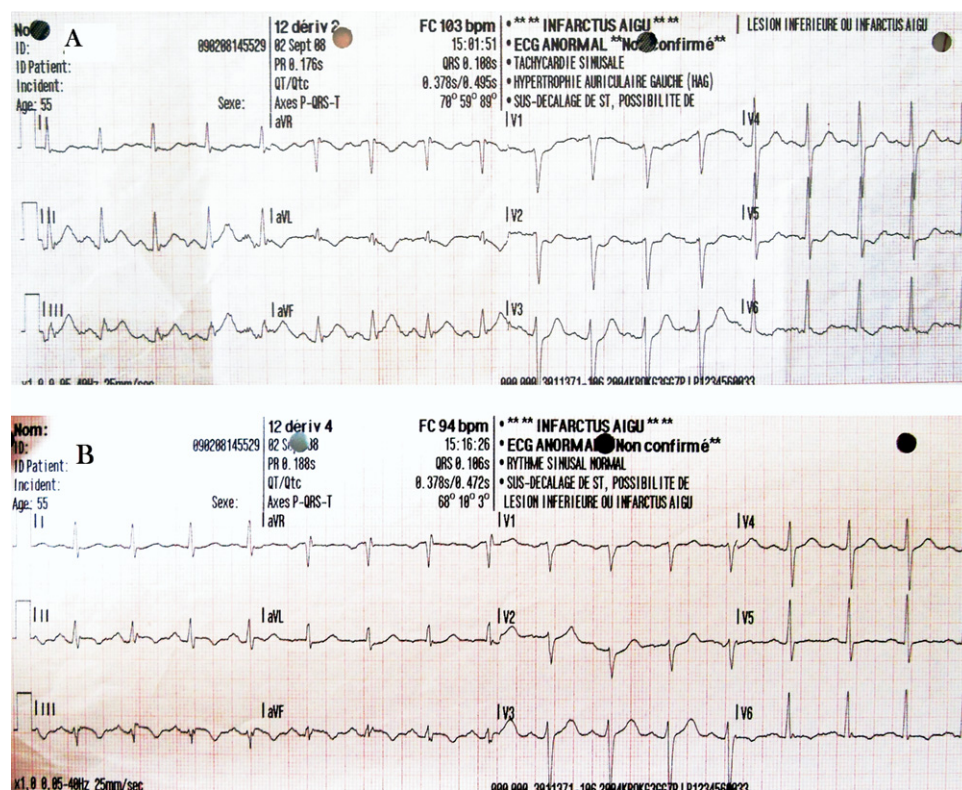


Figure 2. Serial electrocardiograms showing a regression of ST-segment elevation (A) and T-wave inversion (B).

formed about 90 min after the onset of the first symptoms) was performed via a right radial artery access route, revealing normal coronary arteries with preserved left ventricular function. Cardiac troponins T (obtained meanwhile) were positive at 0.26 $\mu\text{g/L}$ and confirmed the diagnosis of inferior STEMI with normal arteries, probably induced by the allergic reaction to the wasp sting. The patient recovered very well and was discharged home on the following day with a 7-day antihistamine treatment, and referred to allergy specialists for possible desensitization.

DISCUSSION

Hypersensitivity reactions can occur in response to a variety of stimuli, including drugs, environmental exposures, and autoimmune disease. Such reactions, including those to wasps and more generally to hymenoptera stings, can range from localized inflammation and urticaria to severe anaphylaxis (4). Acute coronary syndromes occurring in such a setting constitute a rare finding, especially for STEMI, with, to date, only several cases reported in the literature (1–3). The reported cases are attributable to severe hypersensitivity, and are referred to as the Kounis syndrome, with two identified subdivisions: type I, occurring in patients with angiographically normal coronary vessels (as was the case in our patient); and type II, occurring in patients in whom concomitant atheromatous lesions are found (5,6).

Several factors may contribute to the genesis of an STEMI after a hymenoptera envenomation. Their venom usually contains not only vasoactive, inflammatory, and thrombogenic peptide and amine constituents, including histamine, serotonin, kinin, bradykinin, epinephrine, dopamine leukotriene, and thromboxane, but also allergenic proteins such as melittin, hyaluronidases, phospholipases, and acid phosphatases that may cause endogenous inflammation mediator and amine release, and further promote the process. Almost all of the cited substances have an established capacity either to directly induce coronary vasoconstriction or to favor platelet aggregation and thrombosis (3). In our patient, the vasoconstrictive action of these substances may have been enhanced by the lack of the protective counterbalancing vasodilator effect of amlodipine, a calcium channel blocker with known coronary vasodilatation action that was withdrawn several weeks before the incident (7). This is of particular interest considering the fact that the patient reported having been previously stung by wasps in the same geographic region (assuming it was the same species) but without developing any particular reaction.

Besides the undoubted contributive role of the above-mentioned substances, an individual susceptibility probably also exists, and the presence of an underlying endothelial dysfunction could constitute a favoring substratum for the development of STEMI in some particular individuals (5,8).

It is noteworthy that it was decided not to administer epinephrine to this patient during the initial management. In this particular case of anaphylaxis, the major manifestation was a vasoconstriction-induced STEMI, or vasoconstriction had at least a major role. Therefore, administering a powerful vasoconstrictive agent could only prove to be deleterious, as it may further aggravate the ongoing ischemia and potentially induce severe dysrhythmias. This particular form of severe anaphylaxis constitutes an exception to the established paradigm for the use of epinephrine in serious allergic reactions, provided there is an absence of severe hypotension or anaphylactic shock.

CONCLUSIONS

ST elevation myocardial infarction after wasp envenomation is an exceptional and interesting pathology with a partially elucidated pathogenesis. The management of cardiac ischemia in this setting is uncertain. In those patients with reversal of the ECG changes, that is, the ST elevations and reciprocal depressions, as was the case with the patient presented here, it may be reasonable to defer emergent coronary angiography.

REFERENCES

1. Valkanas MA, Dailey MW, Bowman S. Electrocardiographic myocardial infarction without structural lesion in the setting of acute hymenoptera envenomation. *Am J Emerg Med* 2007;25:1082.e5–8.
2. Taggar JS, Watson T, Musarrat K, Millane T. Kounis syndrome presenting as ST-segment elevation myocardial infarction following a hymenoptera (bee) sting. *Int J Cardiol* 2008 Jul 21 [Epub ahead of print].
3. Wagdi P. Acute myocardial infarction after wasp stings in a patient with normal coronary arteries. *Am Heart J* 1994;128:820–3.
4. Freeman TM. Clinical practice. Hypersensitivity to hymenoptera stings. *N Engl J Med* 2004;351:1978–84.
5. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): a natural paradigm? *Int J Cardiol* 2006;110:7–14.
6. Kogias JS, Sideris SK, Anifadis SK. Kounis syndrome associated with hypersensitivity to hymenoptera stings. *Int J Cardiol* 2007;114:252–5.
7. Singh S, Doherty J, Udhoji V, et al. Amlodipine versus nadolol in patients with stable angina pectoris. *Am Heart J* 1989;118:1137–8.
8. Camici PG, Crea F. Coronary microvascular dysfunction. *N Engl J Med* 2007;356:830–40.