

Multiple bee stings resulting in ST elevation myocardial infarction (the Kounis syndrome)

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Kounis syndrome consists of angina pectoris or myocardial infarction that is triggered by the release of inflammatory mediators in the setting of an allergic reaction. We present the case of a 61-year-old man who presented to the emergency department with anaphylaxis after being stung by >100 bees. During resuscitation, he subsequently developed ST elevation myocardial infarction.

Acute coronary syndrome is a common disease process seen in the emergency department. Many physiologic stressors can precipitate its acute presentation. Kounis syndrome, also referred to as “allergic angina,” is a rare but increasingly reported cause of acute coronary syndrome. It can be induced by any anaphylactic reaction and thus must be on a differential diagnosis for anyone with chest pain or symptoms of acute coronary syndrome who presents with the surrounding setting of an anaphylactic reaction. This syndrome requires a high degree of clinical suspicion and can be easily overlooked given the dramatic presentation of anaphylaxis.

CASE REPORT

A 61-year-old white man with no documented coronary artery disease but known congestive heart failure (cause uncertain), type II diabetes, tobacco abuse, hypertension, and hyperlipidemia presented to the emergency department via ambulance after having multiple bee stings while riding on his tractor. The patient received diphenhydramine en route for reported dyspnea and arrived in the exam room with diaphoresis, dyspnea, and innumerable bee stings located mainly on his head, face, and arms. He also presented with a large urticarial rash on his back, with >100 bee stingers remaining in his skin (*Figure 1*). The patient was given 0.3 mg of 1:1000 intramuscular epinephrine in addition to 125 mg intravenous methylprednisolone for anaphylaxis and showed initial improvement.

Two hours after initial evaluation, he began clutching his chest and reported sudden onset of new 10/10 stabbing left-sided chest pain. The initial electrocardiogram (*Figure 2a*) revealed sinus tachycardia with a right bundle branch block and frequent ventricular premature complexes and fusion beats, as well as intermittent ventricular bigeminy. Repeat electrocardiogram in the setting of chest pain (*Figure 2b*) disclosed

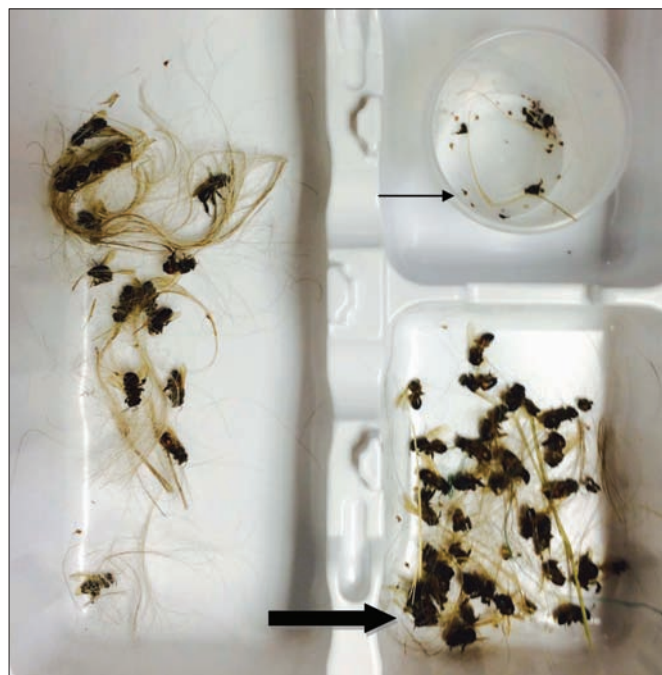


Figure 1. Bees cut from the patient's scalp and hair (thick arrow) and stingers removed from the patient's skin (thin arrow).

ST elevation in leads II, III, aVF, and V4 to V6. The cardiac catheterization revealed thrombus in the distal right coronary artery, mid left anterior descending artery, and first diagonal of the distal left anterior descending artery. Immediate percutaneous coronary intervention was performed with successful thrombectomy and placement of bare metal stents in the distal right coronary artery and mid left anterior descending artery.

The patient was transferred to the cardiac intensive care unit, where an echocardiogram revealed severe global left ventricular hypokinesis with an ejection fraction of 25%. Dual antiplatelet therapy, optimal medical therapy, and a life vest were provided.

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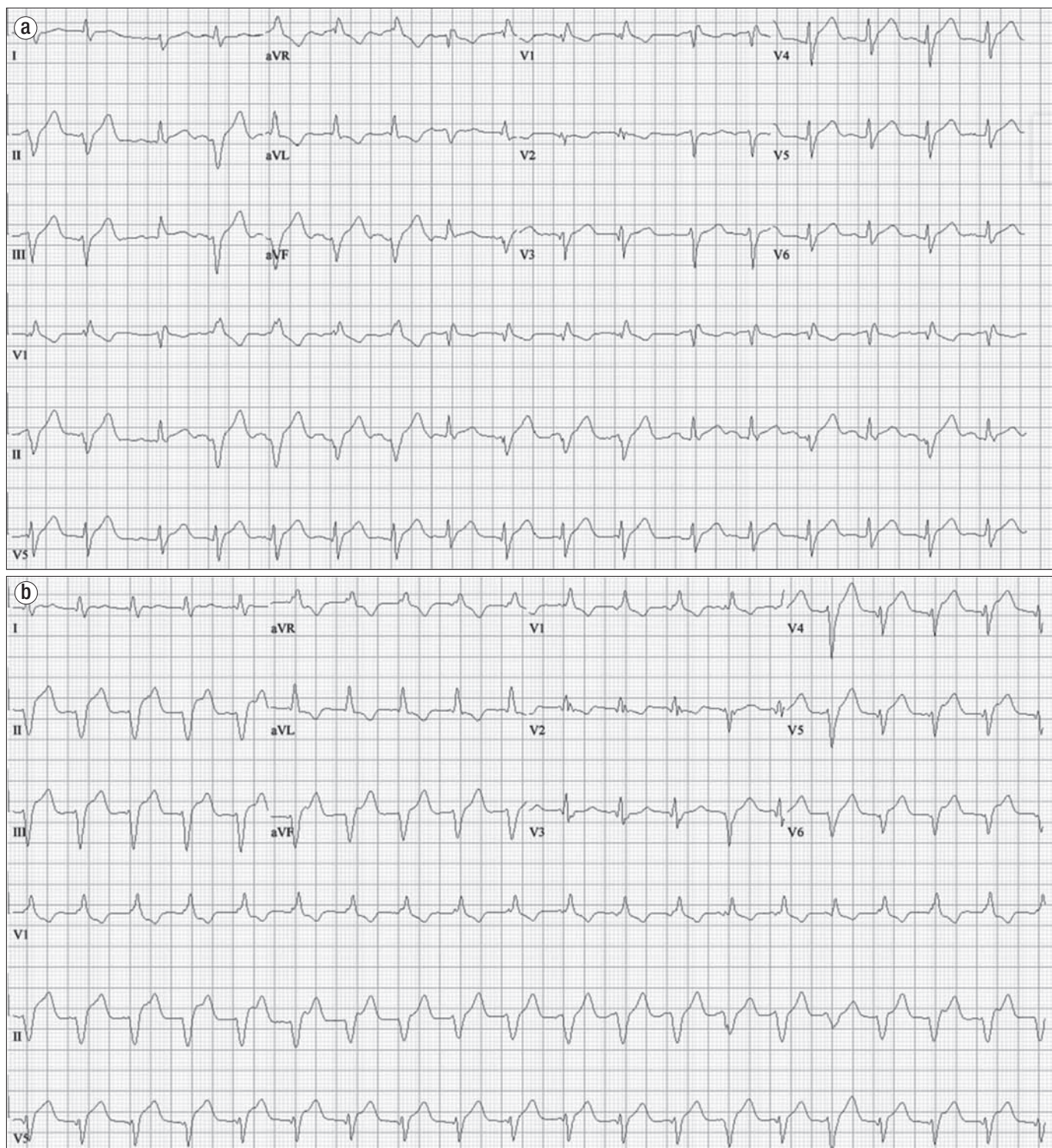


Figure 2. Electrocardiogram (a) on arrival, showing sinus tachycardia, and (b) 2 hours later, showing ST elevation myocardial infarction in leads II, III, aVF, and V4 to V6.

The patient was later discharged, only to return within a week with severe chest pain after medication noncompliance. He had an ST elevation myocardial infarction, developed ventricular tachycardia, and went into cardiac arrest. The patient was resuscitated and taken back to the catheterization lab, where his left anterior descending artery stents were found to be thrombosed, likely due to noncompliance with antiplatelet therapy. The thrombus was aspirated from the stents and balloon angioplasty was

performed. He required inotropic support. A repeat echocardiogram disclosed that his ejection fraction had dropped to 15% and a left ventricular thrombus was present. His heart failure regimen was optimized and he was started on anticoagulation.

Over the next 5 months, the patient continued to have multiple admissions for decompensated heart failure and a growing list of comorbidities, including health care–acquired infections. His cardiac function continued to decline, with an ejection

fraction decreasing to 10%. His symptoms ultimately became refractory to treatment, and he was transitioned to hospice care.

DISCUSSION

The order *Hymenoptera* consists of families of stinging insects, specifically honeybees and bumblebees in the *Apidae* family and wasps and yellow jackets in the *Vespidae* family. In the United States, *Hymenoptera* envenomations are estimated to cause >40 deaths per year (1). The venom of insects within these families consists of histamine and phospholipase A2, which can induce hypersensitive reactions leading to mast cell activation and degranulation. The combined physiologic effects of these envenomations can subsequently result in coronary vasospasm or potentially acute myocardial infarction (2).

The mechanism is a complicated and multifactorial cascade of endogenous allergic mediators. In the anaphylactic degranulation of mast cells, several collagen-degrading and vasoconstricting substances, such as histamines, neutral proteases, arachidonic acid products, platelet-activating factors, cytokines, and chemokines, are locally released. The physiologic result of the above milieu results in the constellation of coronary vasoconstriction, hypercoagulability, and even atheromatous plaque erosion or rupture (3).

There are three commonly described variants of “allergic angina” based on the patient’s current coronary artery status.

The type I variant consists of patients without coronary lesions in whom the allergic insult leads to coronary vasospasm with either normal or subsequently elevated cardiac enzymes. The type II variant describes patients with existing atheromatous disease. In these patients, an anaphylactic insult leads not only to vasospasm, but to atheromatous plaque rupture and subsequent myocardial infarction. Type III involves coronary artery stent thrombosis, which stains positive for eosinophils and mast cells (4, 5). In the case described above, we suspect an incidence of the type II Kounis syndrome variant, given the patient’s clinical presentation and comorbidities of congestive heart failure, severely uncontrolled diabetes, tobacco abuse, hypertension, and hyperlipidemia.

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