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Case Report

Kounis Syndrome: Acute Myocardial Infarction Following Multiple Bee Stings

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ABSTRACT

Introduction: Kounis syndrome is an acute coronary syndrome triggered by allergic or hypersensitivity reactions. It is an underdiagnosed clinical entity where mast cell activation leads to coronary vasospasm, plaque erosion, or thrombosis. Hymenoptera stings, such as those from bees, are a rare but well-documented cause.

Case Presentation: A 64-year-old man presented to the emergency department with multiple bee stings, manifesting only mild cutaneous urticaria. He was discharged after observation with stable vitals. Approximately 12 hours post-sting, he experienced sudden-onset chest pain, diaphoresis, and syncope. Upon arrival, he was in a gasping state, requiring immediate intubation and mechanical ventilation. He suffered a cardiac arrest, and after 30 minutes of cardiopulmonary resuscitation, return of spontaneous circulation was achieved. Electrocardiography revealed ST-segment elevation in the inferior leads (II, III, and aVF) with reciprocal ST-depression in the anterolateral leads. Emergency coronary angiography demonstrated a complete thrombotic occlusion of the mid-right coronary artery. Successful primary percutaneous coronary intervention with a drug-eluting stent was performed, restoring TIMI grade 3 flow.

Conclusions: This case highlights the critical importance of considering Kounis syndrome in patients presenting with acute coronary symptoms following an allergic insult, even with a delayed presentation. A high index of suspicion is necessary for timely diagnosis and management, which must address both the allergic reaction and the acute coronary syndrome concurrently. ***(Iranian Heart Journal 2026; 27(2): 81-86)***

KEYWORDS: Kounis syndrome; bee sting; acute myocardial infarction; allergic angina; primary percutaneous coronary intervention

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Bee sting envenomation is a common environmental hazard, typically resulting in a self-limited local reaction characterized by pain, swelling, and erythema. Systemic allergic reactions, including anaphylaxis, occur in a small subset of individuals and are a well-known medical emergency. Beyond anaphylaxis, bee venom can rarely precipitate a spectrum of systemic complications such as serum sickness, encephalitis, and acute kidney injury.

A particularly rare and life-threatening complication is Kounis syndrome, first described by Kounis and Zavras in 1991 as "the coincidence of acute coronary syndromes with conditions associated with mast cell activation, involving interrelated and interacting inflammatory cells, and comprising of allergic or hypersensitivity and anaphylactic or anaphylactoid insults." The pathophysiology involves the release of inflammatory mediators (eg, histamine, leukotrienes, proteases, and platelet-activating factor) from mast cells in response to an allergen. These mediators can induce coronary artery vasospasm, disrupt atherosclerotic plaques, and activate platelets, leading to thrombosis and acute myocardial infarction (MI).

We report a compelling case of a 64-year-old man who developed a delayed, catastrophic presentation of Kounis syndrome (Type 2 variant) manifesting as ST-elevation myocardial infarction (STEMI) because of acute thrombotic occlusion of the right coronary artery following multiple bee stings. This case underscores the diagnostic challenges and the imperative for a dual-treatment approach in this critical condition.

Case Presentation

A 64-year-old man with no significant prior medical history, including no known history of coronary artery disease, diabetes, or hypertension, presented to the emergency department in the morning of May 2025. His chief complaint was multiple bee stings sustained earlier that day. Upon initial assessment by the emergency medicine team, the patient was found to be hemodynamically stable. His vital signs were pulse rate of 76 beats/min, blood pressure of 120/70 mm Hg, respiratory rate of 16 breaths/min, and oxygen saturation of 98% on room air. Physical examination revealed multiple bee sting marks on his torso and extremities, accompanied by mild, localized urticarial rashes. There was no wheezing, stridor, or signs of respiratory distress. Cardiovascular and abdominal examinations were unremarkable.

A diagnosis of a mild systemic allergic reaction was made. The patient was treated conservatively with intravenous antihistamines (eg, chlorpheniramine) and was kept under observation in the emergency department for 12 hours. During this period, his vital signs remained stable, and the skin rash resolved significantly. He was subsequently discharged home in a seemingly stable condition.

Later that same day, at approximately 9:45 PM (approximately 12–14 hours post-initial sting), the patient experienced a sudden onset of severe, crushing substernal chest pain, associated with profuse diaphoresis. This was rapidly followed by a witnessed loss of consciousness. Emergency medical services were activated, and he was rushed to our emergency department.

On arrival, the patient was critically ill. He was in a gasping respiratory pattern and profoundly hypoxic. His pulse and blood pressure were unrecordable, indicating cardiopulmonary arrest. Cardiopulmonary resuscitation was initiated immediately according to advanced cardiac life support (ACLS) protocols. The patient was rapidly intubated, and mechanical ventilation was commenced. After 30 minutes of sustained cardiopulmonary resuscitation, return of spontaneous circulation was achieved.

Post-resuscitation, a 12-lead electrocardiogram (ECG) was urgently obtained. It revealed significant ST-segment elevation (> 2 mm) in the inferior leads (II, III, and aVF) with reciprocal ST-segment depression in the anterolateral leads (V_2-V_6, I, and aVL) (Figure 1). Bedside transthoracic echocardiography was performed, which demonstrated hypokinesia of the inferior wall of the left ventricle. Left ventricular ejection fraction was estimated at 40%. There were no

significant valvular abnormalities. An arterial blood gas analysis drawn at this time revealed a severe metabolic acidosis with a pH of 7.0, a lactate level of 15 mmol/L, and a base deficit of −20, reflecting the prolonged period of circulatory arrest.

Based on the clinical scenario and ECG findings, a primary diagnosis of acute inferior wall STEMI was made. The temporal association with the bee stings raised a strong suspicion for Kounis syndrome. After informed consent was obtained from the patient's next of kin, he was transferred directly to the cardiac catheterization laboratory for emergency coronary angiography.

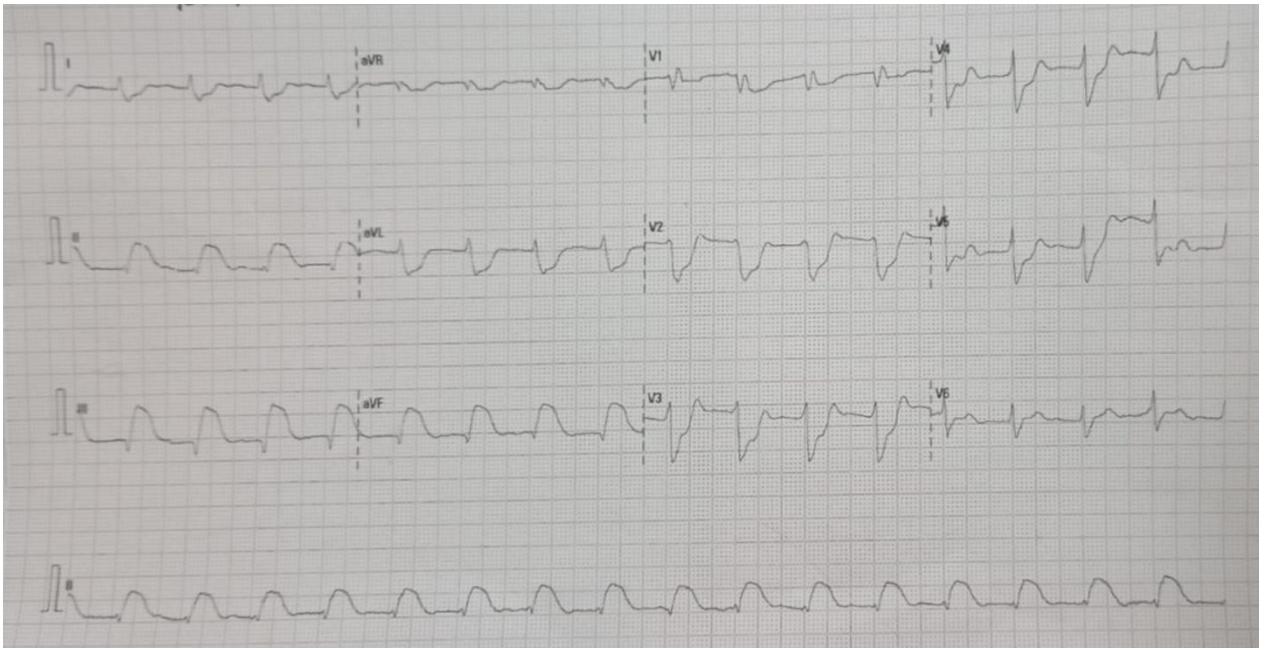

Figure 1. The 12-lead ECG shows ST elevation in leads II, III, and aVF with reciprocal depression in leads V_2-V_6, I and aVL.

Coronary Angiography and Intervention

Coronary angiography was performed via the right femoral artery. The left coronary system (the left main, left anterior descending, and left circumflex arteries) was found to be angiographically nonobstructive coronary artery disease. However, the right coronary artery (RCA) was noted to have a complete, abrupt occlusion in its mid-segment (Figure 2A). This finding was consistent with the inferior STEMI on ECG.

The lesion was crossed with a guidewire; then, predilation was carried out with a semicompliant balloon. A 3.5 × 28 mm drug-eluting stent was successfully deployed in the mid-RCA, covering the occluded segment. Postdilation was performed with a noncompliant balloon to ensure optimal stent apposition. The final angiographic run demonstrated excellent stent placement with Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow distally (Figure 2B).

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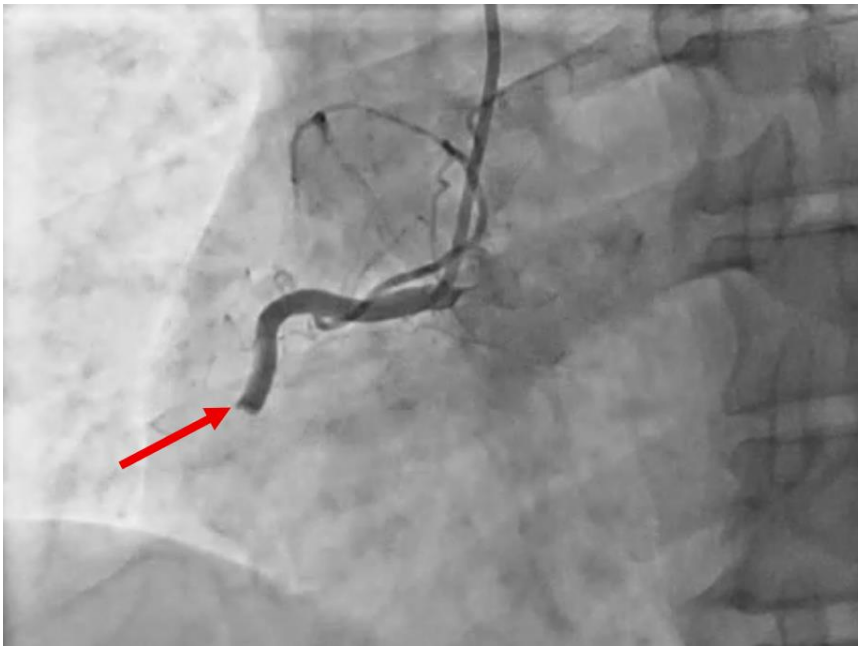

Figure 2A. The right coronary angiogram shows complete occlusion of the mid-right coronary artery (marked by the red arrow).

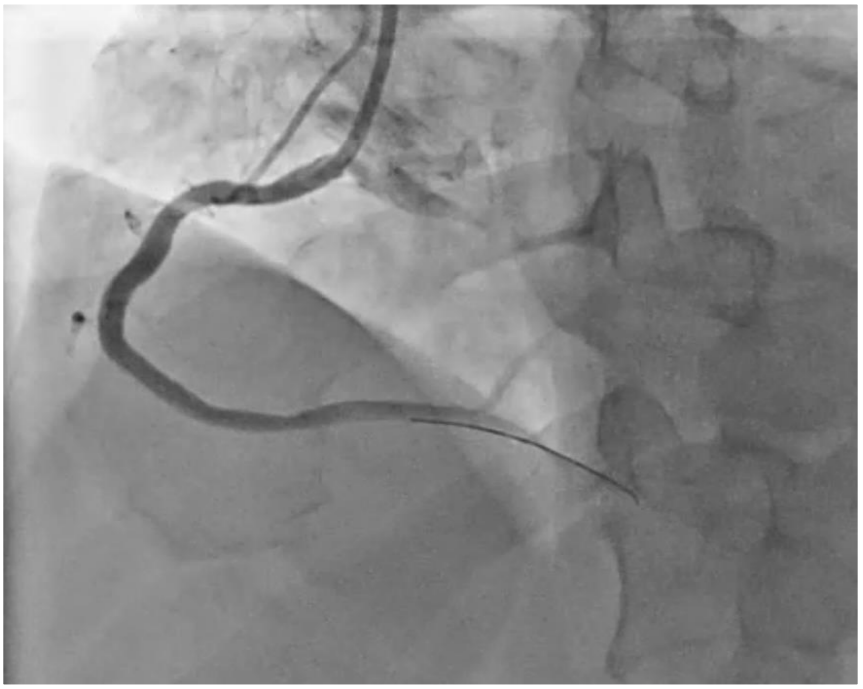

Figure 2B. The image illustrates primary percutaneous coronary intervention performed on the right coronary artery, showing Thrombolysis in Myocardial Infarction grade 3 flow.

Hospital Course and Outcome

After the procedure, the patient was transferred to the cardiac care unit for continued management. He remained intubated and on vasopressor support for hemodynamic stability. His management included dual antiplatelet therapy (aspirin and ticagrelor), high-dose statins, and

supportive care for postcardiac arrest syndrome, including targeted temperature management. His severe metabolic acidosis was managed with controlled ventilation and sodium bicarbonate infusion.

Over the next 48 hours, his hemodynamic parameters gradually improved, and vasopressors were weaned off. He was successfully extubated on the third day of hospitalization. Serial ECG monitoring showed resolution of the ST-segment elevations, with the development of Q-waves in the inferior leads, confirming a completed inferior MI. Follow-up echocardiography before discharge showed persistent inferior wall hypokinesia with an improvement in left ventricular ejection fraction to 45%. The patient was discharged on day 10 on guideline-directed medical therapy for STEMI. He was counseled on bee sting avoidance and prescribed an epinephrine auto-injector for future emergencies.

DISCUSSION

Kounis syndrome is classified into 3 types.[1, 2] Type 1 occurs in patients with normal coronary arteries, where allergic mediator release causes coronary vasospasm. Type 2 occurs in patients with pre-existing atherosclerotic disease, where the allergic insult leads to plaque erosion or rupture and acute thrombosis. Type 3 is associated with coronary stent thrombosis secondary to an allergic reaction to the stent itself.[3] Our patient most likely represents a type 2 variant, as angiography revealed a localized thrombotic occlusion without evidence of diffuse coronary disease, suggesting a focal allergic-inflammatory reaction over a potentially vulnerable, albeit previously nonobstructive, plaque.

The unique aspect of this case is the delayed presentation. Most reported cases of Kounis syndrome occur within minutes to a few hours of the allergic trigger.[2] Our patient's symptoms manifested after a 12-hour latency period. This delayed reaction could be attributed to a late-phase inflammatory response mediated by eosinophils and other cells recruited after the initial mast cell degranulation.[4] This highlights that a significant temporal gap between the allergic event and the coronary ischemia does not rule out Kounis syndrome.

The management of Kounis syndrome is complex and requires a balanced approach. It involves simultaneously treating the acute coronary syndrome and the anaphylactic reaction. Corticosteroids and antihistamines are cornerstone treatments for the allergic component.[1, 5] However, the use of epinephrine, a mainstay in anaphylaxis, is controversial in Kounis syndrome as it can potentially exacerbate coronary vasospasm or provoke arrhythmias in the setting of ischemia.[2, 5] In our patient, the primary focus was the management of cardiac arrest and STEMI, with percutaneous coronary intervention being the definitive life-saving intervention.

CONCLUSIONS

This case serves as a critical reminder for clinicians, especially emergency physicians and cardiologists, to maintain a high index of suspicion for Kounis syndrome in any patient presenting with acute coronary symptoms following an allergic reaction, even with a delayed onset. A thorough history taking, including recent environmental exposures, is paramount. Early recognition and a multidisciplinary approach addressing both the cardiac and allergic pathologies are essential to improve outcomes in this potentially fatal condition.

Conflict of Interest: There are no conflicts of interest regarding the publication of this case report.

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