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ALLERGIC CORONARY CATASTROPHE : REVISITING THE KOUNIS SYNDROME

Cardiology

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ABSTRACT

Bee stings are a frequent environmental exposure, yet myocardial infarction (MI) following a sting is exceedingly rare. We report a patient who developed electrocardiographic features consistent with a left main coronary artery (LMCA) infarction following a bee sting. Coronary angiography revealed no LMCA obstruction but demonstrated a mid–left anterior descending (LAD) myocardial bridging segment. This case exemplifies the rare coexistence of Kounis syndrome (allergic acute coronary syndrome/allergic angina) and myocardial bridging, resulting in transient ischemia mimicking left main occlusion. Awareness of this interplay is crucial for accurate diagnosis and appropriate management of allergic myocardial ischemia.

KEYWORDS

Kounis syndrome; Bee sting; Myocardial Infarction; Myocardial Bridging; Coronary Vasospasm; Left Main Syndrome.

INTRODUCTION

Bee venom contains biologically active peptides such as melittin, phospholipase A_2, and apamin, capable of inducing local or systemic allergic reactions (1,2). While most reactions are benign, cardiovascular manifestations including angina, arrhythmias, and MI have been reported under the spectrum of Kounis syndrome, first described by Kounis and Zavras in 1991 (3). Kounis syndrome refers to acute coronary events which are precipitated by allergic or hypersensitivity reactions, caused mainly by mast cell activation and the release of certain vasoactive mediators (4–6). These substances—histamine, tryptase, chymase, and leukotrienes—induce coronary vasospasm and may even trigger plaque rupture (7,8). In parallel, myocardial bridging, a congenital variant where a segment of a coronary artery tunnels within the myocardium, may provoke dynamic systolic compression and ischemia, particularly during tachycardia or increased sympathetic tone (9–11). Here, we report a case in which these two pathophysiologic processes coexisted, producing ECG features of left main ischemia following a bee sting.

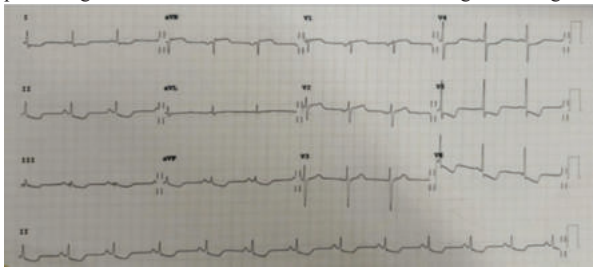

Figure 1 : ECG Showing ST Elevation in aVR and St Depressions in Inferolateral Leads (Leads II, III, avF and V5,V6)

Case Presentation

A previously healthy, middle-aged individual presented with acute retrosternal chest pain, diaphoresis and giddiness within minutes of a bee sting. He also complained to severe itching all over the body. He was immediately rushed to the emergency where his pulse rate was found to be 124 beats per minute. Blood pressure was 80 mm Hg systolic. He was given intravenous fluid. Blood pressure did not improve, hence he was initiated on low dose Nordadrenaline infusion. In view of suspected anaphylaxis, he was also given Injection Hydrocortisone 100 mg intravenous following which his itching improved, but he continued to have hypotension. Intramuscular adrenaline was not given. ECG showed ST-segment elevation in aVR with diffuse ST-segment depression in the inferior and lateral leads, as shown in Figure 1, consistent with left main or multivessel ischemia (12,13). Echocardiography showed no significant wall motion abnormality with normal ejection fraction. Troponin-T was significantly elevated. Coronary angiography was done thereafter which revealed a normal LMCA, no atherosclerotic lesions or stenosis in other epicardial vessels and a mid–LAD myocardial bridging segment showing its systolic compression and diastolic recovery(9). The patient was treated with intravenous corticosteroids, anti-histaminics. (14,15). Symptoms and ECG changes resolved within hours. Patient was also initiated on low dose beta blocker thereafter in view of significant mid LAD bridging. He was hemodynamically stable and discharged on day 3 of admission.

DISCUSSION

This case illustrates a rare interaction between allergic vasospasm and structural coronary anomaly. The Kounis syndrome Type I variant—occurring in patients with angiographically normal coronaries—likely caused transient vasospasm following bee venom exposure (3,6,16). Simultaneously, LAD myocardial bridging acted as a dynamic obstruction during tachycardia, causing flow limitation and mimicking a left main coronary syndrome in this case (9,11).Proposed mechanisms include: (1) mast cell activation with histamine, leukotrienes, and thromboxane release causing coronary vasospasm (7,17); (2) catecholamine surge leading to tachycardia and increased myocardial oxygen demand (18); and (3) systolic compression of the bridged LAD segment resulting in transient ischemia (9,11,19).The result was severe, reversible ischemia despite the absence of any anatomical occlusion. This finding aligns with previously described cases of allergic myocardial syndromes in which pre-existing structural or functional abnormalities amplify allergic coronary responses (16,20,21). The clinical presentation of Kounis Syndrome is highly variable, ranging from mild angina to life-threatening myocardial infarction. Symptoms may include chest pain, dyspnea, and various allergic manifestations such as skin rashes, urticaria, and bronchospasm. Kounis syndrome has three recognized variants (3,6): Type I, coronary vasospasm in normal vessels; Type II, allergic reaction triggering plaque rupture; and Type III, allergic stent thrombosis. Bee sting–related Kounis syndrome is rare, with fewer than 50 cases reported worldwide. Only a few have demonstrated ECG patterns mimicking LMCA occlusion. Myocardial bridging as a cofactor in allergic ischemia is exceptionally rare, though reports suggest pre-existing structural anomalies may amplify allergic coronary dysfunction (9,11). This case thus represents a unique intersection of two distinct ischemic mechanisms: allergic vasospasm and mechanical systolic compression of the left anterior descending artery.

Diagnostic Considerations

The electrocardiographic pattern of ST elevation in aVR and widespread ST depressions in inferolateral leads is strongly predictive of LMCA or triple-vessel disease (12,13). In this case, however, the angiogram was normal—highlighting the importance of recognizing functional causes of ischemia. Myocardial infarction with nonobstructive coronary artery disease is a complex condition

requiring comprehensive evaluation and management. Coronary angiography remains the gold standard and the first investigation of choice but it may also miss transient vasospasm (22). Investigations like Intravascular ultrasound (IVUS) or fractional flow reserve (FFR) can assess the dynamic compression and confirm myocardial bridging (9,11,23). Further cardiac MRI in such cases may demonstrate transient subendocardial ischemia without infarction (24). Serum tryptase levels may often support an allergic etiology if obtained early.

Therapeutic Implications

The management of allergic coronary events differs from the management of conventional acute coronary syndrome. Adrenaline may worsen vasospasm and should be used with caution in such cases. Corticosteroids and antihistamines help to further reduce the release of mast cell mediators. (15). Calcium channel blockers help relieve spasm, whereas nitrates may exacerbate systolic compression in myocardial bridging (9,11). Any unnecessary revascularization should be avoided, however in patients with significant coronary atherosclerotic lesions or occlusion, as in Type 2 or 3 Kounis Syndrome, traditional management of acute coronary syndrome should be employed(25). On the other hand, when angiographic findings are normal and an allergic trigger is clearly evident, percutaneous intervention should be deferred. (6,18).

CONCLUSION

This report highlights a rare but clinically significant phenomenon where bee sting–induced Kounis syndrome precipitated transient ischemia and left anterior descending myocardial bridging, thus producing an ECG picture of left main infarction without fixed coronary obstruction (9). Recognition of allergic coronary syndromes is vital for differentiating them from obstructive MI and avoiding inappropriate interventions, like the use of antiplatelets or percutaneous coronary intervention. Clinicians should suspect this diagnosis upfront in any acute coronary presentation following allergic exposure.

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