Kounis syndrome – Anaphylaxis-induced acute coronary syndrome

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Keywords
case report, medicine, Kounis syndrome, Anaphylaxis, Acute coronary syndrome

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Conflict of Interest Statement
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ARTICLE

Kounis Syndrome – Anaphylaxis-induced Acute Coronary Syndrome

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Abstract

Kounis syndrome is an underdiagnosed condition in which anaphylaxis triggers vasospastic acute coronary syndrome, either with or without underlying coronary artery disease. The prevalence of this syndrome among hospitalized patients for allergic/hypersensitivity/anaphylactic reactions in the United States is 1.1%, with a 7% rate of all-cause inpatient mortality. This article presents an anaphylaxis-induced acute coronary syndrome case in a patient with underlying coronary artery disease. The pathophysiological mechanism of anaphylactic-induced acute coronary syndrome involves the inflammatory mediators of type I hypersensitivity reactions.

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1. Introduction

Kounis syndrome refers to the concurrent presentation of acute coronary syndrome with hypersensitivity and allergic or anaphylactic reactions. It is distinguished from acute coronary syndrome alone by the significantly higher degree of mast cell degranulation at plaque disruption or erosion sites.1 Mast cell activation in Kounis syndrome typically occurs days or weeks after the acute coronary event. Common risk factors associated with this syndrome include previous allergic reactions, hypertension, smoking, diabetes, and hyperlipidemia.2 The most frequent triggers of Kounis syndrome are antibiotics and insect bites, although it can be caused by various precipitants depending on an individual’s allergies.3 Despite being misconceived as a rare condition, Kounis syndrome is often underdiagnosed due to limited reports and the variability of allergens. There are three recognized variants of Kounis syndrome: type one, which occurs in individuals without coronary disease but with coronary spasm; type two, which presents in individuals with coronary disease, pre-existing atheromatous disease, and plaque erosion or rupture; and type three, which manifests in people with the pre-existing atheromatous disease with drug-eluting coronary stent thrombosis. Management of Kounis syndrome varies based on the specific variant. Type one variant requires close monitoring of vitals and anaphylactic reactions, while type two and three variants receive dual antiplatelet therapy and emergency percutaneous coronary intervention (PCI). This article presents a case of a patient with type two variant Kounis syndrome who required emergent coronary artery bypass grafting (CABG) after consuming peanuts.

2. Case presentation

A 63-year-old male with a medical history of type 2 diabetes, hypertension, and hyperlipidemia presented to the emergency room with symptoms of throat swelling, eye edema, wheezing, and a pruritic rash that had developed approximately 1–2 h before
arrival. The patient suspected these symptoms were triggered by consuming half a cup of peanuts, although he had never experienced a true anaphylactic reaction before. He did recall some previous throat itching associated with eating tree nuts. No other allergen exposures were reported. Initial vital signs revealed a blood pressure of 179/119 mmHg, pulse rate of 74 bpm, respiratory rate of 16 breaths per minute, and a temperature of 36.1 °C. Physical examination revealed a hoarse voice, lower lip edema, right eye edema, wheezing, erythema, and rash in the bilateral axillae and chest. Hematologic studies were unremarkable, but blood chemistries showed an initial elevation of high-sensitivity troponin at 78 ng/L, with a one-hour troponin level of 297 ng/L and a positive delta of 219 ng/L.

The patient initially received 0.5 mg of intramuscular epinephrine, which provided no relief. A second 0.5 mg dose of epinephrine also failed to alleviate symptoms, and the patient subsequently experienced chest pain, worsening respiratory status, diaphoresis, and bradycardia. A third dose of 0.3 mg of intramuscular epinephrine was administered, followed by the initiation of an epinephrine drip. An electrocardiogram performed during the episode of chest pain revealed ST elevation in lead aVR, as well as ST segment depression in leads II, III, aVF, V2, V3, V4, V5, and V6, along with T wave inversions in the same leads (Fig. 1). After the initiation of the epinephrine drip, the patient's symptoms improved, and bradycardia began to resolve. A repeat electrocardiogram showed improvement in ST elevation, ST depressions, and T wave inversions (Fig. 2). In addition to epinephrine, the patient received dexamethasone, diphenhydramine, and famotidine, and was subsequently admitted to the intensive care unit. High doses of epinephrine were continued for several hours before gradual titration. Bedside point-of-care ultrasound did not reveal any obvious abnormalities in left ventricular function. Cardiology consultation led to the administration of therapeutic Lovenox and aspirin for possible acute coronary syndrome. The patient remained nil per os (nothing by mouth) in preparation for a possible cardiac catheterization the following morning.

An echocardiogram performed the following morning revealed no gross abnormalities. However, considering the patient's multiple risk factors, he was transferred to a higher level of care for cardiac catheterization. The procedure revealed 100% stenosis in the left anterior descending artery, collaterals from septal to septal and right to left, 80% stenosis in the proximal vessel of the first diagonal, mild disease with less than 30% stenosis in the left circumflex and its branches (first and second marginal), and 100% stenosis in the distal vessel of the right coronary artery, with mild disease (less than 30% stenosis) in the posterior descending and posterolateral branches. Coronary artery bypass grafting was recommended to bypass the left anterior descending, first diagonal, and
posterior descending arteries. The patient subsequently underwent coronary artery bypass grafting with three grafts: left mammary to the left anterior descending artery, saphenous vein to the first diagonal, and saphenous vein to the posterior descending artery. Postoperatively, the patient recovered well and was discharged home with cardiac rehabilitation.

3. Discussion

This case report sheds light on a commonly undiagnosed condition known as anaphylaxis-induced acute coronary syndrome in the presence of underlying coronary artery disease, also referred to as Kounis syndrome. By exploring this specific case, we aim to emphasize the importance of recognizing this syndrome and its potential implications for patient management.

The patient's clinical presentation aligns with the type two variant of Kounis syndrome, which involves the activation of inflammatory mediators during an allergic or hypersensitivity reaction leading to coronary artery spasm, plaque destabilization, or thrombosis. In this case, the patient experienced an anaphylactic reaction triggered by the ingestion of peanuts, a known allergen. Although he had never previously experienced a true anaphylactic reaction, he did recall some throat itching associated with eating tree nuts. The rapid onset of symptoms, including throat swelling, eye edema, wheezing, and a widespread itchy rash, strongly suggested an allergic reaction.

Given the combination of symptoms, it was essential to consider both Kounis syndrome and demand-mediated ischemia in the differential diagnosis. While Kounis syndrome arises from the allergic reaction and subsequent inflammatory processes affecting the coronary arteries, demand-mediated ischemia occurs due to increased demand for blood flow in the presence of narrowed coronary arteries. Kounis syndrome could be viewed as a subtype of demand ischemia. In this viewpoint, the inflammatory markers associated with anaphylaxis induce vasospasm in arteries that already have pre-existing plaques. This vasospasm reduces blood flow, leading to an imbalance between oxygen supply and demand, which is characteristic of demand ischemia. Therefore, it can be argued that the mechanisms involved in Kounis syndrome, triggered by an allergic reaction, align with the pathophysiological processes of demand ischemia.

It is also important to discuss the administration of epinephrine can potentially lead to confusion with Kounis syndrome due to certain overlapping symptoms and physiological effects. Epinephrine acts by constricting blood vessels, relaxing smooth muscles, and increasing cardiac output. However, it can also cause coronary artery vasoconstriction, which may mimic the coronary artery spasm observed in Kounis syndrome.
In the context of anaphylaxis, the administration of epinephrine is necessary and potentially lifesaving. However, in individuals with pre-existing coronary artery disease or susceptible to Kounis syndrome, the administration of epinephrine can trigger or exacerbate coronary artery spasms, leading to myocardial ischemia or infarction.

The resemblance of symptoms between Kounis syndrome and the administration of epinephrine can lead to confusion. Both conditions can present with similar manifestations, including chest pain, shortness of breath, palpitations, and electrocardiographic changes indicative of myocardial ischemia. Furthermore, the temporal association between the administration of epinephrine and the onset of these symptoms can further complicate the diagnostic process.

In this specific case, our patient experienced chest pain while undergoing treatment for anaphylaxis with epinephrine. However, it is noteworthy that subsequent doses of epinephrine, along with the administration of an epinephrine drip, resulted in the resolution of the symptoms and normalization of the electrocardiogram changes associated with acute coronary syndrome (ACS). This response to the continued use of epinephrine suggests that the patient's symptoms were more likely related to an anaphylactic reaction, Kounis syndrome, rather than an effect from epinephrine administration.

The unique aspect of this case is the coexistence of anaphylaxis and acute coronary syndrome, which posed a diagnostic challenge. The initial presentation resembled anaphylaxis, with features such as throat swelling and wheezing, but the subsequent development of chest pain and electrocardiographic changes indicative of myocardial ischemia necessitated a comprehensive evaluation for acute coronary syndrome. This highlights the need for heightened awareness among healthcare professionals to consider the possibility of Kounis syndrome in patients presenting with symptoms of both anaphylaxis and acute coronary syndrome.

It is crucial to highlight the limited availability of literature on Kounis syndrome, as well as the underdiagnosis of this condition. Many cases likely go unrecognized or misdiagnosed, leading to potential complications and suboptimal patient outcomes. By presenting this case, we aim to contribute to the existing medical knowledge and raise awareness about the existence of Kounis syndrome, particularly among clinicians involved in the management of allergic reactions and cardiovascular disorders.

Furthermore, the prevalence of Kounis syndrome among hospitalized patients with allergic/hypersensitivity/anaphylactic reactions in the United States is estimated to be around 1.1% according to the International Journal of Cardiology. The associated all-cause inpatient mortality rate is approximately 7%, underscoring the importance of early recognition, accurate diagnosis, and prompt intervention in optimizing patient outcomes. Recognizing the interplay between allergies and cardiovascular disease is essential for providing comprehensive care to patients, as both conditions may coexist and influence each other's management.

This case report also highlights the need for a multidisciplinary approach in managing patients with Kounis syndrome. In this case, a collaboration between emergency medicine, cardiology, and hospital medicine specialties played a pivotal role in the patient's care. The administration of epinephrine, a cornerstone of anaphylaxis management, proved insufficient in relieving the patient's symptoms and even led to chest pain and worsened respiratory status. The subsequent initiation of an epinephrine drip and adjunctive medications such as corticosteroids, antihistamines, and acid suppressants eventually improved the patient's symptoms and allowed for stabilization.

The diagnostic workup, including electrocardiography, echocardiography, and coronary angiography, played crucial roles in confirming the presence of underlying coronary artery disease and guiding therapeutic interventions. The identification of significant stenosis in multiple coronary arteries necessitated coronary artery bypass grafting to restore adequate blood supply to the affected myocardial regions.

This case report offers valuable insights into the identification and treatment of Kounis syndrome, anaphylaxis-induced acute coronary syndrome. Through the presentation of a real-life clinical scenario and a discussion of the encountered challenges, this report enhances our understanding of managing this condition.

4. Conclusion

In conclusion, we have presented a case report highlighting a frequently undiagnosed instance of anaphylaxis-induced acute coronary syndrome, also known as Kounis Syndrome, in the presence of underlying coronary artery disease. Our patient's clinical presentation aligns with the type two variant of Kounis syndrome, necessitating immediate coronary artery bypass grafting (CABG). The prevalence of this syndrome among hospitalized patients experiencing allergic/hypersensitivity/anaphylactic reactions in the United States is estimated to be 1.1%, with a 7% inpatient mortality rate for all causes. Given the scarcity of literature on Kounis
syndrome and the significant number of undiagnosed cases, it is crucial to share this report with the medical community.

Conflicts of interest

None to disclose.

References


