



Visual Case Discussion

Shellfish-induced ST elevation myocardial infarction: A case report

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1. Introduction

Allergic acute coronary syndrome, also known as Kounis Syndrome, is potentially rare, but likely underdiagnosed. The term Kounis Syndrome was first termed “allergic angina” after Kounis et al. described the clinical occurrence of chest pain in the setting of an allergic reaction with clinical and laboratory evidence that is correlated with acute coronary syndrome.¹ However, the first case of myocardial infarction during the setting of an allergic response was first described in 1950 in a patient receiving an antibiotic infusion.² While the pathophysiology of this syndrome is not entirely understood, it is proposed that mast cell activation leads to an inflammatory-mediated vasospasms, plaque rupture, and/or thrombosis. Histamine and leukotrienes can lead to coronary vasoconstriction. Histamine has the added effect of platelet activation and subsequent aggregation which may further coronary ischemia.² There are many known allergens that trigger Kounis Syndrome with the most common causes being antibiotics and insect bites, respectively.³ We report a case of Kounis Syndrome in a patient that occurred 30 min after consuming shrimp.

2. Visual case discussion

A 57-year-old male, weighing 81 kg, with a significant past medical history of hypertension, hyperlipidemia, recurrent pulmonary embolism and chronic kidney disease, stage 3 presented with report of “throat closing after shellfish exposure” The patient reported a history of a shellfish allergy and approximately 30 min after eating the rice containing shrimp, he felt as if his tongue was swelling and hard palate was swelling. He reported having difficulty breathing with a horse voice. The

patient also reported exertional chest pain with associated diaphoresis and shortness of breath but was worried about continued oral discomfort.

Vital signs included a temperature of 98.3 F (36.8 C), pulse of 85 beats per minutes, 24 respirations per minute, and a blood pressure of 150/88. Basic metabolic panel and complete blood count were unremarkable, except for a potassium level of 2.7 mmol/L (ref 3.5–4.7 mmol/L). High sensitivity troponin was elevated at 474.6 ng/L (ref 0–79 ng/L). An electrocardiogram (ECG) revealed ST segment elevation in V1, V2 an V3 and T wave in version in V4, V5, V6 (Fig. 1). Physical exam of the skin was unremarkable for rash, petechiae or evidence of urticaria. Orally, the patient was tolerating his secretions and the uvula was midline.

The patient was given 324 mg of chewable aspirin for acute coronary syndrome. In addition, he was given 50 mg of diphenhydramine, 20 mg of famotidine, and 125 mg of methylprednisolone intravenously. The patient was also given 0.3 mg of intramuscular epinephrine. A bedside point-of-care ultrasound demonstrated mid to distal anteroseptal and inferoseptal hypokinesis. The patient went for an emergent left heart catheterization/coronary angiogram, which showed non-obstructive coronary artery disease with 30% distal left anterior descending and 20% right coronary artery occlusion. The post-procedure note, the interventional cardiologist said “Overall, presentation of coronary symptoms with EKG changes and troponin elevation suggestive of acute coronary syndrome, and in the context of non-obstructive coronary artery disease this is suspicious for coronary vasospasm as a cause for this event. Likely the vasospasm resolved by the time the angiogram was performed.”

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3. Discussion

Kounis syndrome, or allergic acute coronary syndrome, can be missed or underdiagnosed. There are three subtypes of Kounis Syndrome that have been described –vasospastic allergic angina (Type I), allergic myocardial infarction (Type II), and coronary artery stent thrombosis (Type III) secondary to an allergic reaction.⁴ The ischemia is caused by a pro-inflammatory cascade that is set off by mast cell degranulation, ultimately leading to coronary artery vasoconstriction due to histamine, leukotrienes, and even thromboxane. Treatment of Kounis Syndrome requires concomitant treatment of both the cardiac and allergic symptoms. In patients who present with Type I Kounis Syndrome, a cocktail of methylprednisolone 1–2 mg/kg and both a histamine 1 and 2 receptor antagonist is often enough to eradicate vasospasms.⁴

4. Conclusion

Anytime a patient presents with both an allergic reaction and acute coronary syndrome, Kounis Syndrome should be in the differential diagnosis. Mainstay of treatment is aimed at alleviating both the hypersensitivity and coronary symptoms simultaneously.

5. Question 1

Question Type True & False

Question Text In a patient with Kounis Syndrome, intramuscular epinephrine can induce further coronary damage, even though epinephrine is a cornerstone in the treatment of anaphylaxis.

Answer Options

- a) True
- b) False

Correct Answer = a

True. Kounis syndrome is an allergic or hypersensitivity reaction that results in subsequent acute coronary syndrome. There are many causes

that have been reported to induce Kounis syndrome. Food, environmental exposures and medications, including glucocorticoids, have been reported in medical literature. While treatment of this syndrome requires treatment of the allergic symptoms and intramuscular epinephrine is a mainstay in patients who present with anaphylaxis, the direct alpha receptor agonism can cause coronary constriction and further damage to the myocardium. While clinicians should be aware of this, epinephrine should still be used the management of an anaphylactic reaction in which there are severe symptoms such as shortness of breath, weak pulse, hives, throat tightness and/or trouble breathing/swallowing.

5.1. References

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6. Question 2

Question Type: multiple choice

Question Text Which subtype of Kounis Syndrome can often be relieved by treating the hypersensitivity reaction alone?

Answer Options

- a) Type I
- b) Type II
- c) Type III
- d) Type IV

Correct Answer = a

Subtype I of Kounis syndrome is one in which a patient has normal or near normal coronary arteries and the inflammatory cascade from the hypersensitivity reaction leads to coronary vasospasm without increased troponin levels or myocardial infarction with increased troponin. Because these patients do not have significant atherosclerotic burden,

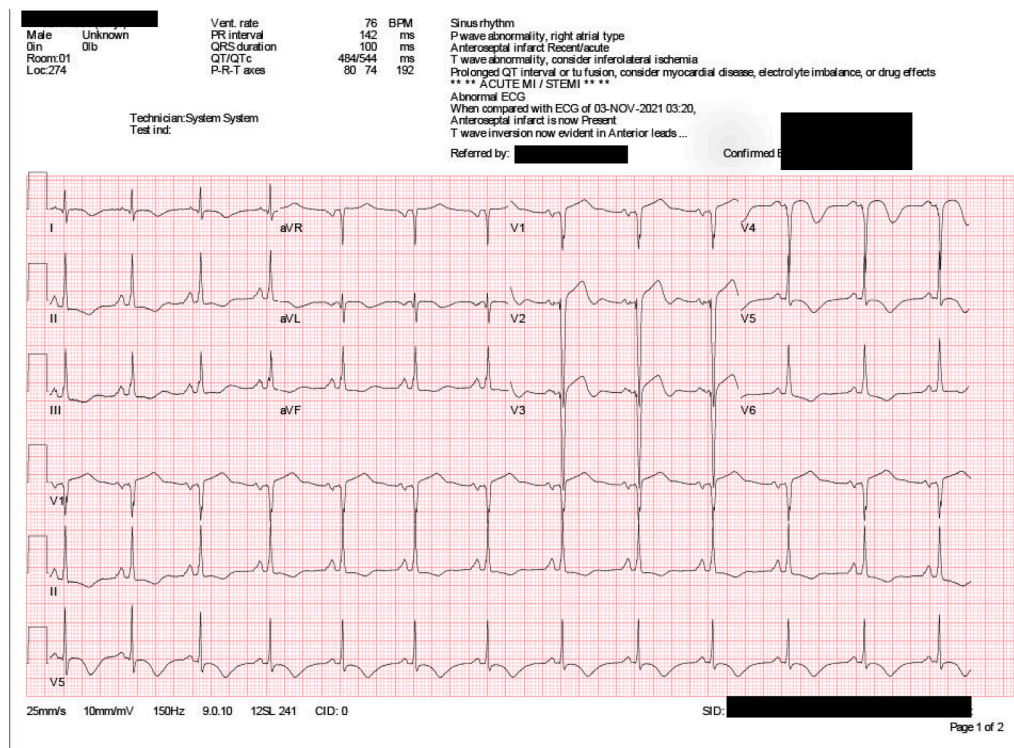


Fig. 1. ECG of a patient with ST segment elevation myocardial infarction in the setting of an allergic reaction.

relieving the hypersensitivity reaction will result in a decreased inflammatory cascade and relief of coronary vasospasm. The subtype II of Kounis syndrome occurs in a patient with significant atherosclerotic disease and have a subsequent plaque rupture leading to a myocardial infarction that is all set off by the allergic reaction. The subtype III is one which a coronary vasospasm leads a to coronary artery stent thrombosis. Type III is confirmed by the presence of eosinophils and mast cells within the thrombus. There is not a Type IV of Kounis Syndrome that has been described in the medical literature.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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