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Research Article

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Dragon Fruit-Associated Type II Kounis Syndrome Presenting After Anaphylaxis

Samuel Tan MD, PharmD[1,3*], David Song MD[1], Mahjabeen Rashid MD[4], Sabrina Daoui MD[2], Joyce Gu BS, MPH[4], Timothy Vittorio MD[3]

[1]Mount Sinai Fuster Heart Hospital, Icahn School of Medicine at Mount Sinai, New York, NY, USA.
[2]Mount Sinai Morningside Hospital, Icahn School of Medicine at Mount Sinai, New York, NY, USA.
[3]James J. Peters Department of Veterans Affairs Medical Center, Bronx, NY, USA.
[4]Lake Eerie School of Osteopathic Medicine, Greensburg, PA, USA.

[*]**Correspondence:** Samuel Tan, Mount Sinai Morningside, 419 W 114th St, New York, NY 10025, Samuel.tan@mountsinai.org

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Abstract

Background: *Kounis syndrome is an acute coronary syndrome triggered by allergic, hypersensitivity, or anaphylactic reactions. Food-associated cases are uncommon, and dragon fruit has been linked to anaphylaxis but rarely to Kounis syndrome.*

Case Presentation: *We report a 53-year-old male with a past medical history of hypertension, type 2 diabetes mellitus, peripheral arterial disease, and chronic tobacco use who developed generalized pruritus, dyspnea, nausea, vomiting, and abdominal pain one hour after first-time ingestion of dragon fruit. After treatment for anaphylaxis, he exhibited dynamic 12-lead electrocardiographic changes, eosinophilia, inferolateral hypokinesis on transthoracic echocardiography, and a progressive rise in high-sensitivity troponin T. Urgent coronary angiography revealed right-dominant two-vessel coronary artery disease with critical mid-right coronary artery stenosis; successful percutaneous coronary intervention was performed.*

Conclusion: *The temporal relationship between the allergic reaction and myocardial injury in the presence of obstructive coronary disease supported a diagnosis of type II Kounis syndrome. This case underscores the need to consider cardiac involvement in severe food-triggered anaphylaxis and to differentiate allergic myocardial injury from epinephrine-associated ischemia, highlighting an important clinical teaching point for recognition and management of this rare syndrome.*

Keywords: *Kounis syndrome; anaphylaxis; dragon fruit; acute coronary syndrome; coronary hypersensitivity; food allergy.*

1. Introduction

Kounis syndrome describes the concurrence of acute coronary syndrome with an allergic, hypersensitivity, or anaphylactic reaction.[1–4] Mast-cell activation and release of inflammatory mediators may provoke coronary vasospasm, endothelial dysfunction, plaque erosion or rupture, and, in patients with pre-existing atherosclerosis, myocardial infarction.[1–4] The syndrome is commonly categorized into type I disease in patients with angiographically normal coronary arteries, type II disease in patients with underlying coronary artery disease, and type III disease related to stent thrombosis or restenosis.[2–4] Although medications and insect stings are the most frequently described triggers, food-associated cases appear to be uncommon.[3,4,5,6] Dragon fruit has been reported as a cause of anaphylaxis, but its association with Kounis syndrome has been rarely described.[7] We report a case of probable dragon fruit-associated type II Kounis syndrome presenting as acute coronary syndrome after anaphylaxis.

2. Case Presentation

A 53-year-old male with a past medical history significant for hypertension, type 2 diabetes mellitus (A1c of 6.4%), peripheral arterial disease, and chronic tobacco use presented to the emergency department with 4 hours of generalized pruritus, dyspnea, nausea, three episodes of nonbloody, nonbilious emesis, and diffuse abdominal pain. Symptoms began approximately 1 hour after first-time ingestion of dragon fruit. He denied fever, chills, chest pain, palpitation, orthopnea, paroxysmal nocturnal dyspnea, and lower extremity edema. He reported no recent infections and no new medications, supplements, or cosmetic products. Home medications were aspirin, atorvastatin, lisinopril, and metformin.

On arrival, blood pressure was 100/58 mmHg, heart rate 134 beats/min, respiratory rate 25 breaths/min, and oxygen saturation 89% on room air. Physical examination was notable for bilateral expiratory wheezing. Chest roentgenography showed borderline cardiomegaly and minimal right basilar atelectatic change. Arterial blood gas testing demonstrated carbon dioxide retention in the setting of chronic smoking and suspected chronic obstructive airway disease. He received dexamethasone 8 mg intravenously, diphenhydramine 25 mg intravenously, and epinephrine 0.3 mg subcutaneously twice, with improvement in allergic symptoms and dyspnea.

Initial laboratory studies demonstrated eosinophilia of 12% (reference range ≤ 5%), NT-proBNP 564 pg/mL, and high-sensitivity troponin T 42 ng/L (reference range < 12 ng/L). Troponin rose progressively to 146 ng/L, 257 ng/L, and 347 ng/L over the next 12 hours. Compared with a prior baseline 12-lead electrocardiogram (ECG) showing sinus rhythm with first-degree atrioventricular block and right bundle branch block, the presenting ECG demonstrated persistent right bundle branch block with new ischemic T-wave abnormalities (Figure 1). Transthoracic echocardiography (TTE) showed a left ventricular ejection fraction of 50.4%, grade II diastolic dysfunction, and inferolateral wall hypokinesis.

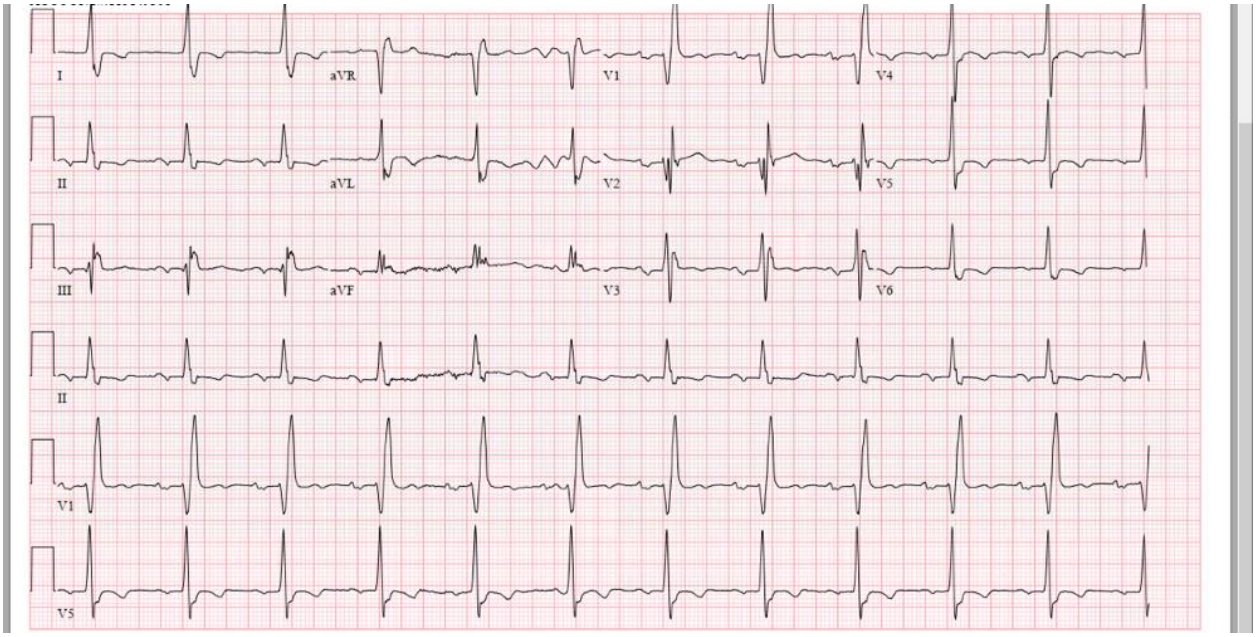

Figure 1: *Presenting 12-lead electrocardiogram demonstrating first-degree atrioventricular block, right bundle branch block, and lateral/anterior-lateral ischemic T-wave abnormalities.*

Given the evolving biomarker rise, TTE findings, and dynamic ECG changes, urgent coronary angiography was performed via right radial access. This demonstrated right-dominant two-vessel coronary artery disease with a critical mid-right coronary artery stenosis and 50% distal left anterior descending disease. Percutaneous coronary intervention of the mid-right coronary artery was successfully performed with a 3.5 × 23 mm everolimus-eluting stent. A postprocedural ECG showed resolution of the previously noted anterolateral T-wave abnormalities (Figure 2).

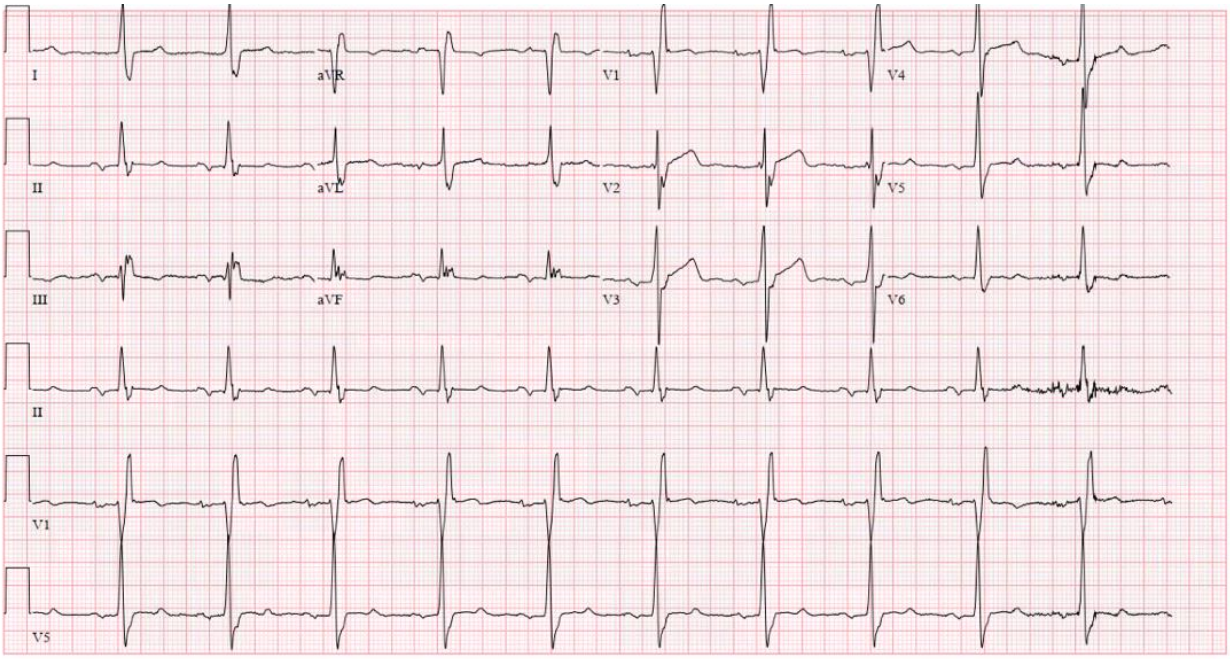

Figure 2: *Post-percutaneous coronary intervention electrocardiogram showing persistent first-degree atrioventricular block and right bundle branch block with resolution of the previously observed anterolateral T-wave abnormalities.*

The patient remained clinically stable after intervention and was discharged on dual antiplatelet therapy and guideline-directed secondary prevention. A repeat TTE performed 3 months later demonstrated preserved left ventricular systolic function with resolution of the inferolateral wall motion abnormality. At 1-year follow-up, he had no recurrent allergic events, no further cardiac hospitalizations, and no newly identified allergies.

3. Discussion

This case is most consistent with type II Kounis syndrome, defined as allergic acute coronary syndrome occurring in a patient with underlying coronary artery disease.[1–4] Several features support this interpretation: the close temporal relationship between first-time dragon fruit ingestion and systemic allergic symptoms, peripheral eosinophilia, dynamic ECG abnormalities, progressive troponin elevation, regional wall motion abnormalities on TTE,

and angiographic evidence of obstructive coronary disease requiring revascularization.

A central diagnostic challenge was distinguishing allergic myocardial injury from epinephrine-associated ischemia. Epinephrine remains first-line therapy for anaphylaxis and should not be withheld when clinically indicated[8]. However, catecholamine-mediated ischemia has been reported, particularly with intravenous administration or rapid systemic absorption[8]. In this patient, the epinephrine was administered subcutaneously, a route associated with slower and less predictable systemic absorption than intramuscular or intravenous administration. In addition, the allergic syndrome and initial blood sampling occurred essentially contemporaneously, and the subsequent pattern of biomarker rise, regional wall motion abnormalities, and culprit coronary stenosis is more compatible with allergic triggering of ischemia in an atherosclerotic substrate than with isolated pharmacologic vasospasm alone.

The pathophysiology of Kounis syndrome is thought to involve mast-cell activation and release of histamine, tryptase, leukotrienes, platelet-activating factor, and other inflammatory mediators.[1–4] These mediators may induce coronary vasoconstriction, enhance platelet activation, and promote plaque destabilization.[1–4] In type II disease, the allergic cascade acts on pre-existing coronary atherosclerosis, leading to vasospasm, plaque disruption, or both.[1–4] The prompt improvement in allergic symptoms with standard treatment does not exclude evolving coronary involvement, and clinicians should maintain vigilance when ECG abnormalities, biomarker elevation, hemodynamic instability, or cardiovascular risk factors are present.

Food-triggered Kounis syndrome remains uncommon in the published literature.[5,6] Dragon fruit has been implicated in anaphylaxis, which strengthens the biologic plausibility of the trigger in this case.[7] The case also reinforces a practical point: absence of chest pain does not exclude cardiac involvement during severe allergic reactions. Patients with marked dyspnea, gastrointestinal symptoms, or wheezing may have myocardial injury that becomes apparent only through serial ECG and cardiac biomarker testing.

This report has limitations. The diagnosis remains a clinicopathologic inference rather than definitive mechanistic proof. No confirmatory allergy testing was performed, serum catecholamine levels were not measured, and intravascular imaging was not obtained to define plaque morphology or identify features of plaque erosion or rupture. Nevertheless, the temporal sequence and the objective evidence of myocardial injury make type II Kounis syndrome the most plausible unifying diagnosis.

4. Conclusion

Dragon fruit-associated anaphylaxis may precipitate type II Kounis syndrome in a patient with underlying coronary artery disease. This case emphasizes the importance of considering allergic acute coronary syndrome when anaphylaxis is followed by ECG changes or troponin elevation, even in the absence of chest pain. Early recognition is essential because management requires simultaneous treatment of both the allergic reaction and the acute coronary syndrome.

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References

1. Kounis NG. Coronary hypersensitivity disorder: the Kounis syndrome. Clin Ther. 2013;35(5):563-571. doi: 10.1016/j.clinthera.2013.02.022.
2. Kounis NG. Kounis syndrome: an update on epidemiology, pathogenesis, diagnosis and therapeutic management. Clin Chem Lab Med. 2016;54(10):1545-1559. doi:10.1515/cclm-2016-0010.
3. Sciatti E, Vizzardi E, Cani DS, et al. Kounis syndrome, a disease to know: case report and review of the literature. Monaldi Arch Chest Dis. 2018;88(1):898. doi:10.4081/monaldi.2018.898.
4. Fassio F, Losappio L, Antolin-Amerigo D, et al. Kounis syndrome: a concise review with focus on management. Eur J Intern Med. 2016; 30:7-10. doi: 10.1016/j.ejim.2015.12.004.
5. Mejia-Renteria HD, Viana-Tejedor A, Sanchez-Enrique C, et al. Kounis syndrome after ingestion of undercooked fish: new role of intracoronary imaging techniques. Int J Cardiol. 2014;177(2):e58-e60. doi: 10.1016/j.ijcard.2014.09.161.
6. De Gennaro L, Brunetti ND, Locuratolo N, et al. Kounis syndrome following canned tuna fish ingestion. Acta Clin Belg. 2017;72(2):142-145. doi:10.1080/17843286.2016.1265756.
7. Kleinheinz A, Lepp U, Hausen BM, Petersen A, Becker WM. Anaphylactic reaction to mixed fruit juice containing dragon fruit. J Allergy Clin Immunol. 2009;124(4):841-842. doi: 10.1016/j.jaci.2009.05.025.
8. Campbell RL, Bellolio MF, Knutson BD, et al. Epinephrine in anaphylaxis: higher risk of cardiovascular complications and overdose after administration of intravenous bolus epinephrine compared with intramuscular epinephrine. J Allergy Clin Immunol Pract. 2015;3(1):76-80. doi: 10.1016/j.jaip.2014.06.007.
9. Urushidani S, Kuriyama A. A potential association between myocardial ischemia and epinephrine for anaphylaxis. Am J Emerg Med. 2020;38(6): 1297.e1-1297.e3. doi: 10.1016/j.ajem.2020.01.033.
10. Jayamali WD, Herath HMMTB, Kulathunga A. Myocardial infarction during anaphylaxis in a young healthy male with normal coronary arteries: is epinephrine the culprit? BMC Cardiovasc Disord. 2017;17(1):237. doi:10.1186/s12872-017-0670-7.