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# A Case Report of Kounis Syndrome in Guyana

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## ABSTRACT

**Background:** Kounis Syndrome is a fascinating and relatively rare condition that bridges the domains of Allergic Reactions and Coronary Artery Disease. It is defined as Acute Coronary Syndrome (ACS) occurring in the setting of an Allergic Reaction or Anaphylaxis and is associated with mast cell and platelet activation. The triggers for Kounis Syndrome are many but the most common includes Drugs, Foods and Environmental Exposures. There are three variants of Kounis Syndrome characterized by their pathophysiologic mechanisms and include coronary artery vasospasm, plaque erosion or rupture and stent thrombosis respectively.

**Case report:** This was a single case presentation in which a 70-year-old female patient presented to the Accident and Emergency after being stung by many bees with complaints of abdominal pain and vomiting. A diagnosis of Anaphylaxis was made and the appropriate treatment was given. Due to routine workup of critical patients, an EKG was done which showed ST elevations in the inferior leads with reciprocal changes in the anterior and lateral leads. This patient never complained about any chest pain or epigastric discomfort. A diagnosis of Kounis Syndrome was made however the patient signed out against medical advice so further investigation into what variant of Kounis she presented with could not be done.

**Conclusion:** Kounis Syndrome is considered a rare condition but it may be more common than we realize. Its diagnosis or more specifically underdiagnosis may be largely due to it being unrecognized by physicians. Based on how this case presented, an EKG should be ordered for all patients presenting with Anaphylaxis as Kounis Syndrome further complicates and worsens the outcomes of these patients.

***Keywords:*** (Kounis Syndrome, Allergic Reaction, Coronary Artery Disease, Acute Coronary Syndrome, Anaphylaxis, Allergic Angina, Guyana)

## INTRODUCTION

Kounis Syndrome also known as Allergic Angina or Allergic Myocardial Infarction was defined as ACS (Acute Coronary Syndrome) occurring in the setting of an allergic reaction in 1991 by Kounis and Zavras (1). In 2016 the definition was updated by Dr. Kounis as an ACS associated with mast cell and platelet activation in the setting of hypersensitivity and allergic insults. (2) This condition highlights the intricate connections between the immune system and the cardiovascular system, shedding light on the potential for allergic insults to precipitate severe cardiac events. The etiology of Kounis Syndrome ranges from Drugs, Predisposing Conditions, Various food sources as well as Environmental Exposures such as Hymenoptera Stings.(2) Kounis syndrome cases, although under-reported, are more

often encountered in clinical practice, and it is anticipated that many more causative factors will be implicated in the future. Any natural allergen can be a potential trigger for Kounis syndrome.(3)

The underlying pathophysiology of Kounis Syndrome is not fully understood and involves a cascade of inflammatory mediators released in response to allergens, leading to coronary vasospasm, plaque destabilization, or thrombosis.(4) Three variants of Kounis Syndrome have been described and are as follows.

| Type | Cardiac history | Pathological changes |
|---|---|---|
| I | Normal coronary arteries<br>No risk factors for IHD | Coronary artery vasospasm |
| II | Inactive preexisting atheromatous disease | Plaque erosion of rupture causes vasospasm or infarction |
| III | Previous coronary artery stenting | Stent thrombosis secondary to platelet activation |

**Table Adapted from Life in the Fast Lane**

Clinically, Kounis Syndrome presents a diagnostic challenge, often requiring a high index of suspicion, especially in patients with a history of allergies or coronary artery disease. It is associated with subclinical, clinical, acute or chronic allergic reactions along with cardiac symptomatology. Electrocardiographic changes range from ST segment elevations, depressions, varying degrees off heart blocks and cardiac arrythmias. (5) (6)

Management typically involves a multifaceted approach, including prompt identification and treatment of the underlying allergic trigger, along with appropriate cardiovascular interventions to mitigate the risk of adverse cardiac outcomes. The treatment of Kounis is challenging especially given that the drug of choice to treat anaphylaxis is epinepheline given intramuscularly and can worsen coronary vasospasm. In patients with type 1 Kounis, treatment of the underlying allergic event may abolish symptoms. In Type 2 and 3 Kounis treatment should follow and ACS event protocol with the use of nitrates and calcium channel blocks where appropriate. Beta Blockers should be avoided as they can exaggerate coronary artery vasospasm due to unopposed alpha-adrenergic activity.(4)

Kounis syndrome is well described in an article published by Memon et al where three cases were presented caused by ingestion of peanuts, bee stings and administration of ceftriaxone respectively(7). Each patient presented with ST segment elevations in the inferior leads on EKG and after treatment of their allergic reaction/anaphylaxis their ST elevations resolved. Their paper supports Kounis Syndrome as a distinct phenomenon and treatment should be focused on the allergic insult. A case report published in September/October 2023 by Lin et al; in China; describes Kounis syndrome in the setting of a bee sting and describes delayed ACS symptoms and diagnosis of Kounis Syndrome due to unfamiliarity with the condition.(8) The case that will be discussed further in this report occurred in Guyana, South America, where the presumed causative agent is a bee sting as well as unfamiliarity with the diagnosis of Kounis Syndrome.

## CASE REPORT

A 70-year-old female patient with a past medical history of hypertension presented to the Emergency Room with pain about the body, abdominal pain and vomiting. The patient referred that she was doing work in her yard when she suddenly got bitten about the body by numerous amounts of bees. She started feeling intense pain, vomited a few times and was rushed to the ER by her family. The patient was immediately triaged and placed into the critical area of the emergency room where she was immediately assessed by an Emergency Specialist while being placed on cardiac monitor, Oxygen and having her IV access established and full rainbow of labs indicated, all simultaneously. The patient's ABC's were found to be intact

and vital signs were BP: 159/94, PR: 124, RR: 26 Temp: 36.4 Sp02: 94%.

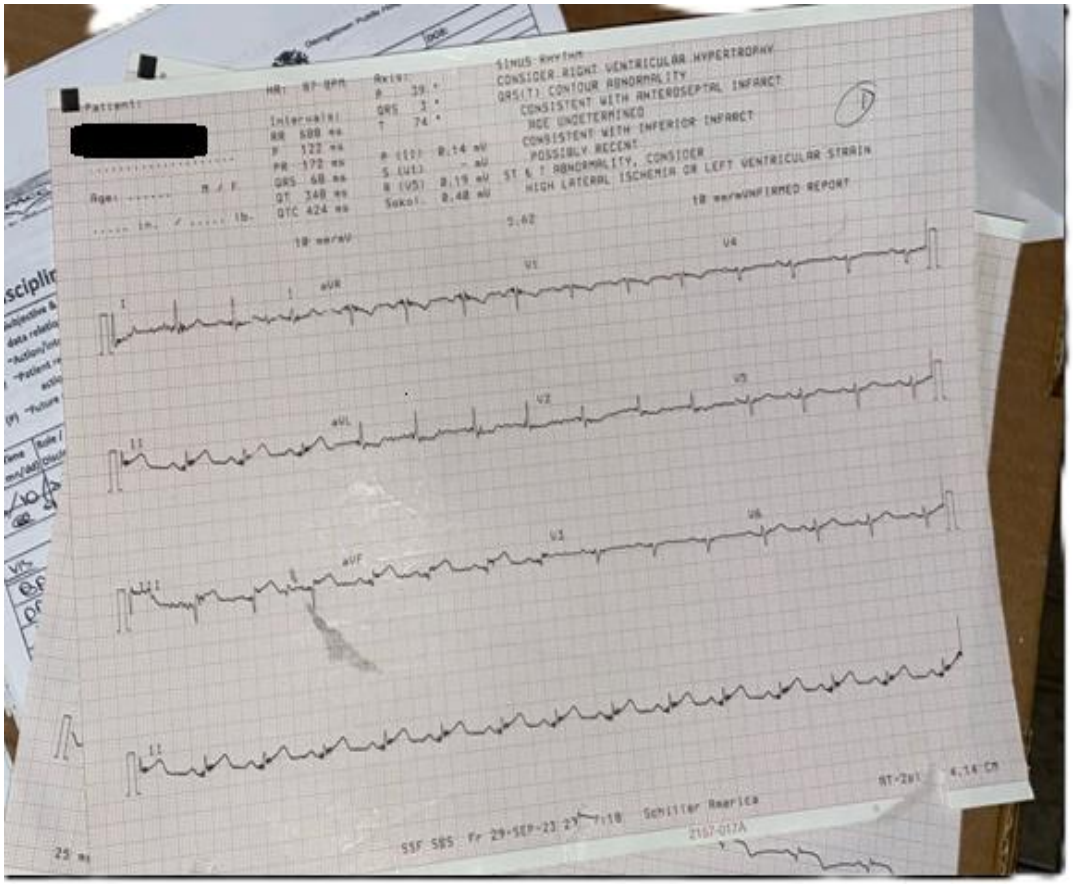
A diagnosis of Anaphylaxis was made based on the history and presentation of this patient and she immediately received 0.5mg of Epinephrine IM in the left antero-lateral thigh along with IV Fluids (2L RL), Analgesia, Hydrocortisone, Cimetidine, and Chlorphenamine (Piriton) IV. A portable Chest X ray was done which was normal.

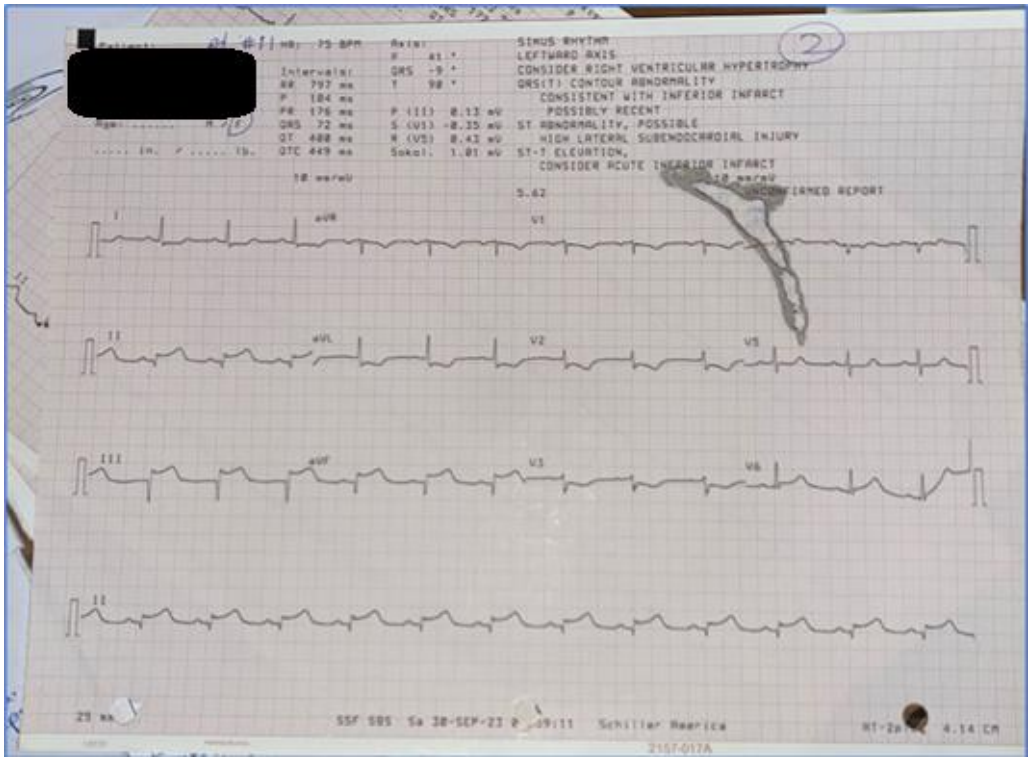
On Physical Examination the patient's Airway was clear, no wheezing heard on the lung examination and the rest of the examination was unremarkable except for the skin where numerous stingers from the bees were found and removed by the nurses.

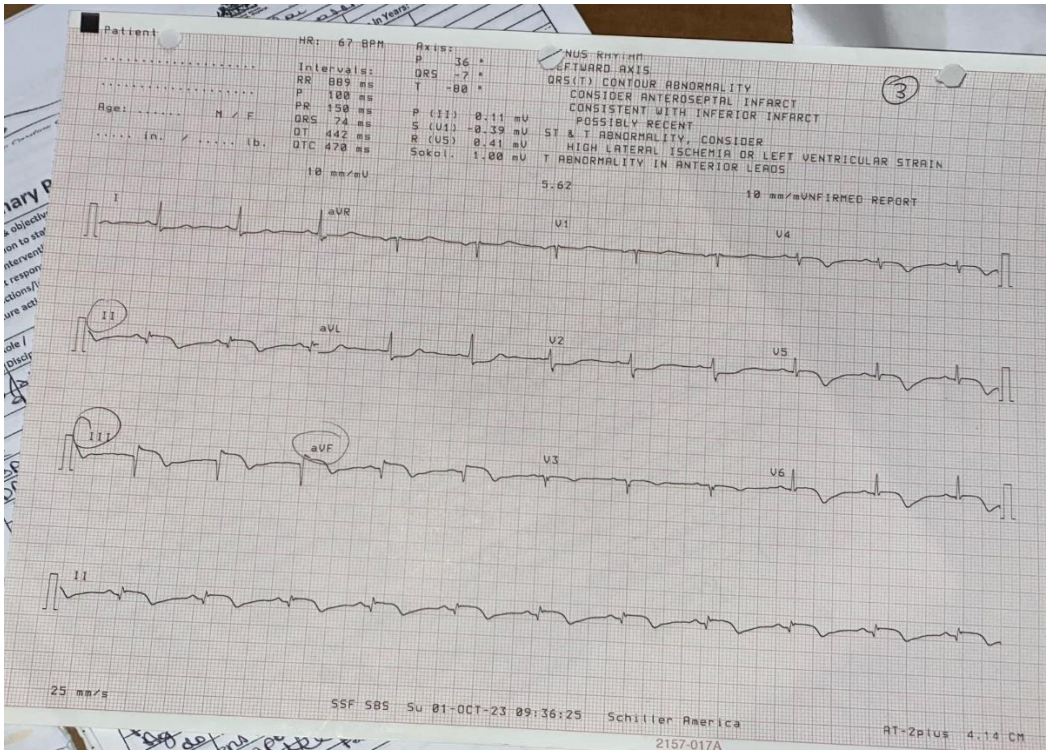
An EKG was ordered as part of the initial workup and it showed ST elevation in the inferior leads (II, III, avF) with reciprocal changes in the septal leads (V1, V2). A cardiology consult was called for a diagnosis of Kounis Syndrome and a troponin was ordered. Of note this patient never complained about any chest pain, chest discomfort or epigastric pain. The result for the troponin test came back at 100ng/ml which was severely elevated. The patient was given Aspirin 300mg to chew and was admitted to the Cardiac ICU by the cardiology team. It should be noted that all other labs were within normal parameters and given the EKG being abnormal, the patient had serial ekgs done.

The expected outcome of this treatment plan and case was resolution of anaphylaxis and admission by the cardiology team for angiogram, trending of troponin results and to determine the variant of Kounis (type I, II OR III) that this patient presented with. However, only part of this outcome was achieved. The anaphylaxis resolved and the patient was admitted, but she did not want to stay in the hospital the following morning and willingly signed out against medical advice and went home. As such no angiogram was done and the case was forced to conclude.

**PATIENT'S EKGs IN EVOLUTION**






## DISCUSSION

Bee stings can and much more frequently cause Anaphylaxis than it does Kounis Syndrome. Anaphylaxis is a severe and life-threatening allergic reaction that occurs minutes to hours (in delayed cases) after being exposed to an allergen (bee venom-apitoxin, in this case) resulting in the immune system releasing chemical mediators (histamine, leukotrienes and prostaglandins) that cause widespread inflammation, generalized vasodilation and eventually shock.(9) Anaphylaxis affects multiple organ systems and is a true medical emergency.

Immediately upon arrival, patient was identified as very sick and given her history of being stung by multiple bees an hour before, along with clinical presentation (Hypertension, Tachycardia, vomiting, increased respiratory rate etc.), anaphylaxis was identified as primary diagnosis given the involvement of multiple organ systems. As such, epinephrine was immediately given as is the medication of choice for the first aid treatment of anaphylaxis since it causes widespread vasoconstriction and prevents upper airway mucosal edema, hypotension and shock.

Having recognized anaphylaxis as a severe form of allergic reaction and initiating its primary treatment (epinephrine), the allergic reaction was subsequently treated with IV Hydrocortisone, IV Chlorphenamine (piriton), IV Cimetidine and IV Fluids all having anti-inflammatory properties targeting the inhibition of synthesis of the chemical mediators (prostaglandins, leukotrienes, histamine) responsible for the widespread inflammation leading to the allergic reaction with subsequent anaphylaxis.

After the gambit was opened and patient was diagnosed and treated for anaphylaxis, as part of routine workup for any critically ill patient, an EKG was obtained, and to the surprise of many, revealed 1mm ST segment elevations in the inferior leads (ii, iii, avf) accompanied with T wave inversions in septal leads (v1, v2) which correspond to reciprocal changes. A troponin I was therefore indicated and returned positive (100ng/ml).

Given EKG findings consistent with an Inferior Stemi (ST Segment Elevation Myocardial Infarct), an elevated troponin along with clinical history, Kounis Syndrome was diagnosed. Patient was subsequently treated with dual antiplatelet therapy (DAPT) and Cardiology was contacted. No nitrates were given since patient never had any chest pains.

Kounis Syndrome as defined by LIFE IN THE FASTLANE is a hypersensitivity coronary disorder characterized by acute coronary syndrome due to an allergic or anaphylactic reaction. It is also commonly known as allergic angina or allergic myocardial infarction. The most commonly recognized triggers are antibiotics (28%) followed closely by insect bites (23%). (10)

The exact pathophysiology responsible for Kounis Syndrome is poorly understood. However, it is thought to be as a result of one of two mechanisms:

1) Myocardial Ischemia due to widespread inflammation due to cytokine release and inappropriate platelet activation through massive mast cell activation from allergen due to hypersensitivity reaction (allergic reaction) which leads to coronary artery vasospasm (Type 1 Kounis) and or atheromatous plaque erosion or rupture (Type II Kounis).
2) Myocardial Ischemia due to global myocardial hypoperfusion due to systemic vasodilation from the anaphylaxis causing decreased venous returns (Type II MI).(10)

Regardless of the mechanism of action, the outcome is the same, which is the infarction of the myocardium. Nevertheless, the treatment is dependent on the variant of Kounis Syndrome that presents, with Type 1 responding to treating the cause (anaphylaxis) while Types II and III requiring angiograms for definitive management.

In the case presented, the patient suffered an Inferior Wall Stemi (ACS) that was treated with DAPT before being handed over to Cardiology and Internal Medicine for definitive care. Heparin was withheld by A&E given likelihood of her going immediately to the CATH Lab. Nevertheless, it was started by Cardiology due to the time the patient presented to the hospital (Evening, CATHS are done primarily early in the day).

It should be noted that patient had serial EKGs done while in the Emergency Room with the second showing worsening ST elevation of 2mm in the inferior leads and even 3mm in the third. Nevertheless, the

patient remained chest pain free throughout the visit.

Patient was admitted by both Cardiology and Internal Medicine and was scheduled for angiogram the following day at 7:00am. However, while receiving treatment in the ER, patient's vital signs normalized and she began to feel much better (no vomits or skin pains), so much so that she signed her own self discharge and left against medical advice. For the purpose of this publication, patient was contacted via telephone almost one year later and referred to be alive and well.

Given no prior history of coronary artery disease along with clinical improvement with only medical management, one can infer without having done an angiogram that patient suffered from Type 1 Kounis Syndrome which is due to coronary vasospasm and thus had a favorable outcome despite leaving the hospital prematurely.

## CONCLUSION:

In conclusion, each patient with anaphylaxis should get an EKG to screen for Kounis Syndrome as it could be a matter of life or death. Anaphylaxis is already a true medical emergency and Kounis Syndrome further complicates it and worsens prognosis.

### *Declaration by Authors*

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**Conflict of Interest:** The authors declare no conflict of interest.

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