Original Research

LEVOFLOXACIN-INDUCED ANAPHYLAXIS COMPLICATED BY KOUNIS SYNDROME: A CASE REPORT

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ABSTRACT

Background: Kounis syndrome is an underdiagnosed clinical entity characterized by the concurrence of acute coronary syndrome with an allergic or hypersensitivity reaction. The inflammatory mediators released during an allergic reaction can induce coronary vasospasm or myocardial injury, making diagnosis and management particularly challenging.

Case Presentation: We report the case of a 31-year-old female with a known history of multiple drug allergies who developed an anaphylactic reaction shortly after ingestion of levofloxacin prescribed for acute pharyngitis. Although her allergic symptoms improved following standard anaphylaxis management, she continued to experience persistent chest tightness. Electrocardiography revealed ischemic changes, and serial troponin I measurements demonstrated a marked elevation with a rising trend, confirming acute myocardial injury. In the absence of conventional cardiovascular risk factors, a diagnosis of Type I Kounis syndrome was established.

Conclusion: This case highlights the importance of considering Kounis syndrome in patients presenting with chest pain during allergic reactions. Early electrocardiographic monitoring and serial cardiac biomarker assessment are essential for timely diagnosis and appropriate management, which may be lifesaving.

Keywords: Kounis Syndrome, Anaphylaxis, Acute Coronary Syndrome, Drug Hypersensitivity, Levofloxacin.

INTRODUCTION

Kounis syndrome is defined as the occurrence of acute coronary syndrome precipitated by an allergic, hypersensitivity, or anaphylactic reaction. First described in 1991, it represents the intersection of immunological and cardiovascular mechanisms. The syndrome has also been termed allergic angina, allergic myocardial infarction, or coronary hypersensitivity disorder.

Although previously considered rare, recent literature suggests that Kounis syndrome occurs in approximately 1.1% to 3.4% of patients presenting with allergic reactions, indicating that it is more likely underdiagnosed than uncommon.

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The condition may occur in patients with normal coronary arteries as well as those with pre-existing coronary artery disease, and it can be triggered by various allergens, including drugs, foods, insect stings, and environmental exposures.

The pathophysiology involves mast cell activation with subsequent release of inflammatory mediators such as histamine, leukotrienes, prostaglandins, cytokines, and platelet-activating factor. These mediators can cause coronary artery vasospasm, platelet activation, and myocardial hypoperfusion, and in susceptible individuals, may lead to plaque erosion or rupture. Management is challenging because medications used to treat anaphylaxis or acute coronary syndrome may worsen the other condition.

We present a case of levofloxacin-induced anaphylaxis complicated by Kounis syndrome in a young female without underlying cardiac disease, emphasizing the diagnostic challenges and therapeutic considerations.

Case Presentation

Patient Information- A 31-year-old female with no known chronic medical illnesses presented to the emergency department. She had a significant history of multiple drug allergies, including azithromycin, ceftriaxone, and piperacillin–tazobactam.

Clinical History- The patient was diagnosed with acute pharyngitis and prescribed oral levofloxacin. Approximately 15–20 minutes after ingestion, she developed symptoms suggestive of an acute allergic reaction.

Clinical Findings- On examination, the patient was conscious and oriented. Her pulse rate was 106 beats per minute, blood pressure was 100/70 mmHg in the right arm in the supine position, respiratory rate was 24 breaths per minute, and she was afebrile. Respiratory system examination revealed bilateral vesicular breath sounds with bilateral rhonchi. Cardiovascular examination did not reveal any additional abnormalities.

Therapeutic Intervention- She was treated promptly with intramuscular adrenaline (0.5 mL), intravenous hydrocortisone (100 mg), intravenous antihistamine infusion, oxygen supplementation, and nebulization with bronchodilators. Baseline laboratory investigations, including complete blood count, renal function tests, liver function tests, C-reactive protein, and serum electrolytes, were within normal limits.

Diagnostic Assessment- Although the patient's anaphylactic symptoms improved, she continued to complain of persistent chest tightness. A 12-lead electrocardiogram demonstrated sinus tachycardia with diffuse ST-segment depression and T-wave abnormalities suggestive of myocardial ischemia

Cardiac biomarkers revealed a markedly elevated troponin I level of 369.7 ng/L (upper reference limit <19 ng/L). A repeat measurement performed approximately three hours later showed a further rise to 643.9 ng/L, confirming acute myocardial injury. In view of the temporal relationship between the allergic reaction and cardiac symptoms, ischemic ECG changes, dynamic troponin elevation, and absence of conventional cardiovascular risk factors, a probable diagnosis of Type I Kounis syndrome was made.

Follow-up and Outcome- The patient was managed conservatively with close cardiac monitoring and continuation of anti-allergic therapy. Her chest discomfort gradually resolved, and she remained hemodynamically stable throughout her hospital stay. Coronary angiography was not performed due to the risk of precipitating another hypersensitivity reaction to contrast agents in view of her history of multiple drug allergies and recent anaphylaxis. She was discharged in stable condition with advice regarding strict avoidance of the offending drug and follow-up with cardiology.

DISCUSSION

Kounis syndrome represents a unique clinical condition in which inflammatory mediators released during allergic or hypersensitivity reactions precipitate myocardial ischemia. Mast cells play a central role in the pathogenesis of this syndrome and are abundantly present in the myocardium and coronary arteries. Upon activation, mast cells release vasoactive and proinflammatory mediators capable of inducing coronary artery vasoconstriction, platelet aggregation, and myocardial hypoperfusion. Three variants of Kounis syndrome have been described. Type I occurs in patients with normal coronary arteries and is caused by coronary vasospasm, with or without elevation of cardiac biomarkers. Type II occurs in patients with pre-existing coronary artery disease, where allergic mediators may trigger plaque erosion or rupture, resulting in myocardial infarction. Type III is associated with stent thrombosis secondary to hypersensitivity reactions to stent components or drugs. In the present case, the young age of the patient, absence of cardiovascular risk factors, ischemic ECG changes, and transient myocardial injury favor a diagnosis of Type I Kounis syndrome. Drug-induced hypersensitivity reactions are among the most commonly reported triggers of Kounis syndrome. Antibiotics, including fluoroquinolones, are well-known causes of immediate hypersensitivity reactions. The rapid onset of symptoms following levofloxacin ingestion, combined with the patient's history of multiple drug allergies, supports the causal role of the drug in this case.

Management of Kounis syndrome is particularly challenging because therapeutic agents used in anaphylaxis and acute coronary syndrome may have conflicting effects. Epinephrine, although lifesaving in anaphylaxis, can aggravate coronary vasospasm and myocardial ischemia. Beta-blockers may worsen ischemia due to unopposed alpha-adrenergic activity, while nitrates can precipitate hypotension in the setting of systemic vasodilatation. Opioids such as morphine and codeine may induce mast cell degranulation and should be used with caution. Therefore, early recognition and individualized management with close monitoring are essential.

This case underscores the importance of considering Kounis syndrome in patients presenting with chest pain or ECG changes during allergic reactions and highlights the need for routine cardiac evaluation in such scenarios.

CONCLUSION

Kounis syndrome is a potentially life-threatening but often underdiagnosed condition. This case demonstrates that significant myocardial injury can occur in young patients without underlying coronary artery disease during an anaphylactic reaction. A high index of suspicion, early electrocardiographic monitoring, and serial cardiac biomarker assessment

are crucial for timely diagnosis and optimal management.

Informed Consent- Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of Interest- The authors declare no conflict of interest.

Funding- No funding was received for this study.

Figure 1. Electrocardiogram Showing Ischemic Changes during Acute Allergic Reaction

Twelve-lead electrocardiogram demonstrating sinus tachycardia with diffuse ST-segment depression and T-wave abnormalities consistent with myocardial ischemia in the setting of anaphylaxis.

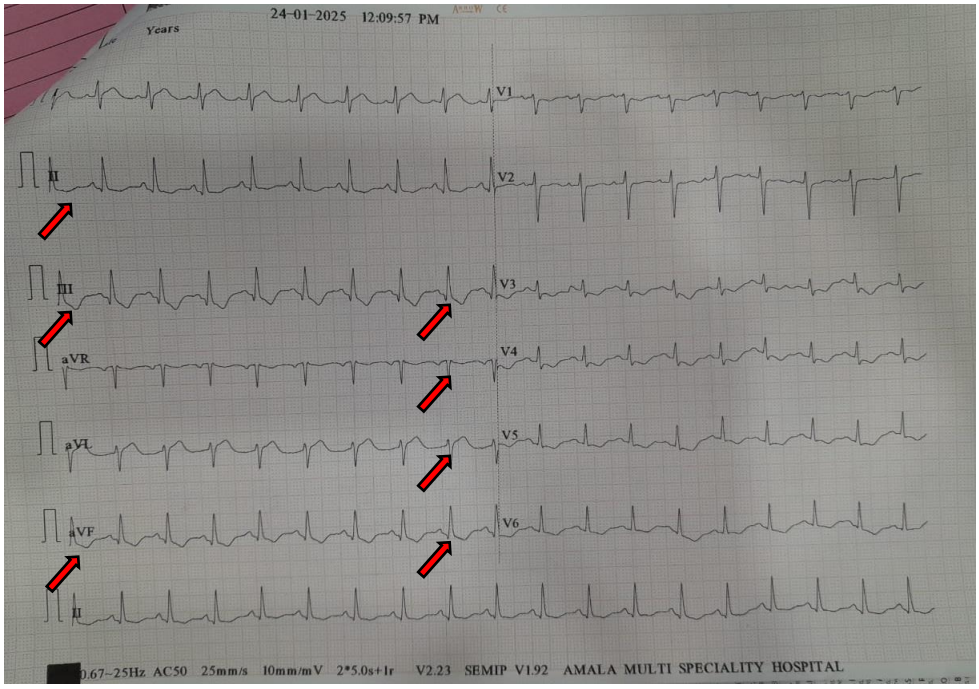
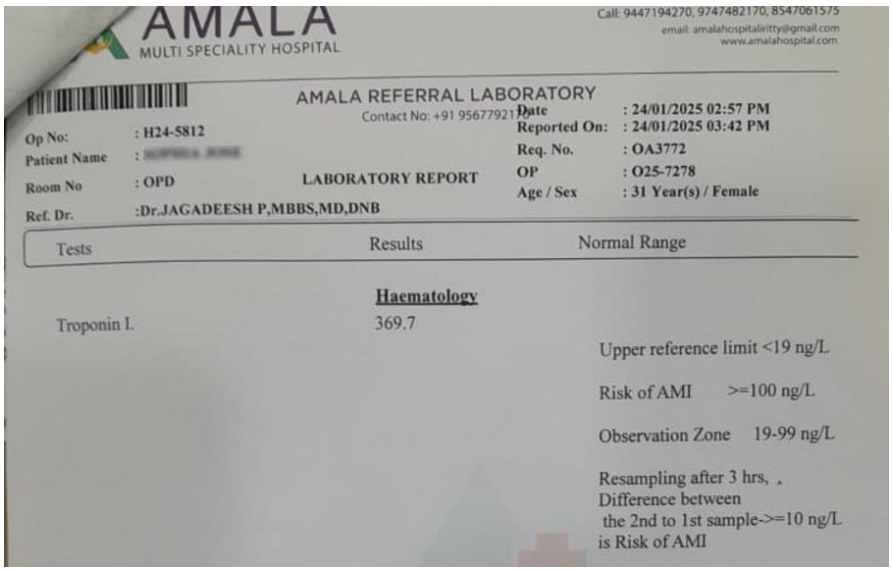


Figure 2. Serial Troponin I Elevation in Levofloxacin-Induced Kounis Syndrome

Serial cardiac biomarker measurements demonstrating marked elevation and a dynamic rise in troponin I levels during the acute allergic reaction.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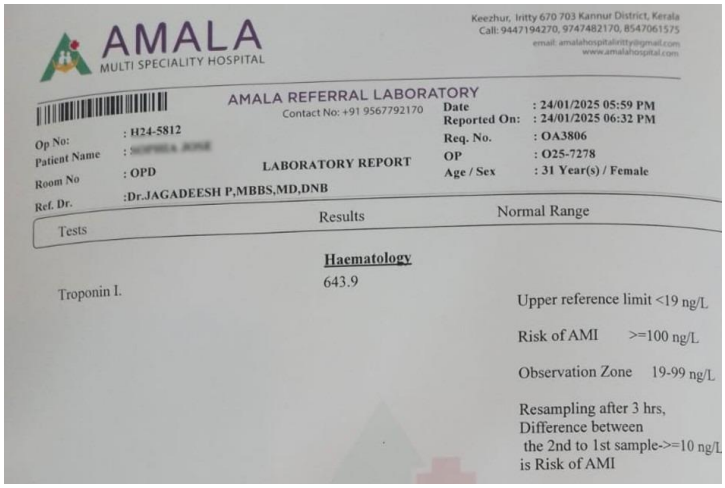
Op No: : H24-5812
Patient Name : [illegible]
Room No : OPD
Ref. Dr. :Dr.JAGADEESH P,MBBS,MD,DNB

LABORATORY REPORT

Date : 24/01/2025 02:57 PM
Reported On: : 24/01/2025 03:42 PM
Req. No. : OA3772
OP : O25-7278
Age / Sex : 31 Year(s) / Female

Tests	Results	Normal Range
Haematology		
Troponin I.	369.7	Upper reference limit <19 ng/L Risk of AMI >=100 ng/L Observation Zone 19-99 ng/L Resampling after 3 hrs, Difference between the 2nd to 1st sample->=10 ng/L is Risk of AMI

Figure 2A: Initial Troponin I Level of 369.7 Ng/L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Op No: : H24-5812
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LABORATORY REPORT

Date : 24/01/2025 05:59 PM
Reported On: : 24/01/2025 06:32 PM
Req. No. : OA3806
OP : O25-7278
Age / Sex : 31 Year(s) / Female

Tests	Results	Normal Range
	Haematology	
Troponin I.	643.9	Upper reference limit <19 ng/L Risk of AMI >=100 ng/L Observation Zone 19-99 ng/L Resampling after 3 hrs, Difference between the 2nd to 1st sample->=10 ng/L is Risk of AMI

Figure 2B: Repeat Troponin I Level of 643.9 Ng/L Obtained Three Hours Later

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