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CASE REPORT

Anaphylaxis-Induced Acute Coronary Syndrome: A Critical Intersection of Allergic and Cardiac Events

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ABSTRACT

Background: A 56-year-old man arrived at the Emergency Department with shortness of breath that had persisted for 30 minutes. He developed a rash, diaphoresis, and nausea shortly after taking sodium diclofenac. While in the Emergency Department, he experienced cardiac arrest and was successfully resuscitated.
Case: he patient had a medical history of hypertension, heart failure, and coronary artery disease, and had received a stent in the left anterior descending (LAD) artery one year prior. His initial vital signs showed blood pressure of 106/64 mmHg, heart rate of 65 bpm, respiratory rate of 25 breaths/min, and SpO2 of 98% on room air. Physical examination revealed an erythematous rash over the entire body and wheezing on pulmonary auscultation. Post–return of spontaneous circulation (ROSC) electrocardiogram (ECG) showed sinus rhythm with bifascicular block, inferior old myocardial infarction (OMI), ischemic changes, and occasional ventricular extrasystole (VES). Initial troponin I level was 0.01 (normal <0.02).
Discussion: The patient received intensive care unit (ICU) management after being intubated following cardiac arrest, which led to hemodynamic stabilization. Transthoracic echocardiography revealed reduced left ventricular systolic function with an ejection fraction of 38%.
Conclusion: The patient was successfully extubated on the fourth day and discharged nine days after admission with a diagnosis of suspected Kounis syndrome, although the specific type could not be determined due to diagnostic resource limitations.

Keywords: Kounis syndrome, cardiac arrest, allergy, anaphylaxis, coronary artery disease

INTRODUCTION

Kounis Syndrome (KS) is a coronary syndrome that occurs in the context of an allergic or anaphylactic reaction. This condition is often underdiagnosed. The lack of recognition is primarily due to the combination of cardiovascular and allergic or anaphylactic symptoms and signs, as well as a lack of awareness regarding the condition in laboratory, electrocardiographic, echocardiographic, and angiographic evaluations. The purpose of this case report is to highlight the importance of recognizing Kounis syndrome in patients presenting with allergic reactions or anaphylaxis, particularly in those with a history of coronary artery disease.[1]

CASE

A 56-year-old man arrived at the Emergency Room with shortness of breath that had persisted for the last 30 minutes. He developed a diffuse erythematous rash, diaphoresis, and nausea shortly after taking sodium diclofenac for his arthralgia. The patient was already aware of his allergic history to Sodium Diclofenac, which had previously caused mild allergic reactions like skin rashes.

The patient has a medical history of hypertension, heart failure, and coronary arterial disease and is currently under treatment with Aspirin 80 mg, Ticagrelor 90mg, Candesartan 8mg, Bisoprolol 2.5mg, Furosemide 40mg, and had received a stent in his Left Anterior Descending (LAD) artery a year ago. His initial vital signs revealed blood pressure 106/64 mmHg, heart rate 65 bpm, respiration rate 25 breaths/min, and SpO2 98% on room air. Physical examination revealed an erythematous rash all over his body, wheezing during pulmonary examination, and no murmur nor gallop was detected during cardiac examination. ECG on admission showed sinus rhythm with bifascicular block due to Right Bundle Branch Block (RBBB) and Left Anterior Fascicular Block (LAFB), OMI Inferior (figure 1). Shortly after physical examination and administration of diphenhydramine and dexamethasone, the patient experienced Pulseless Electrical

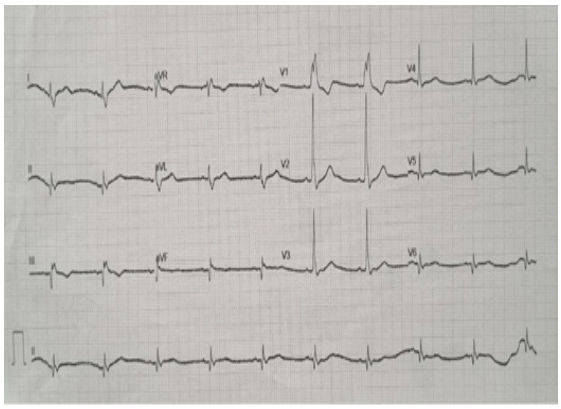

Figure 1. ECG at Admission Showed Sinus Rhythm with Bifascicular Block due to RBBB and LAFB, OMI Inferior

Activity (PEA) cardiac arrest. He regained spontaneous circulation (ROSC) after 3 cycles of cardiopulmonary resuscitation (ROSC) with administration of adrenaline. Post-ROSC ECG showed Sinus Rhythm with bifascicular block due to RBBB and LAFB, OMI Inferior with ischemic change, and occasional VES (figure 2). Lung infiltration was shown on chest X-ray. On laboratory test, white blood cells were high (11.97 x 103/ µ/L), hemoglobin, platelet count, kidney, and liver function all within normal limits. Initial troponin I levels were 0.01 (Normal <0.02), serial troponin was not tested due to cost limitation.

The patient received intensive care unit (ICU) management after being intubated following the cardiac arrest. He was administered intravenous Ringer Lactate, Epinephrine, Aspirin, Ticagrelor,

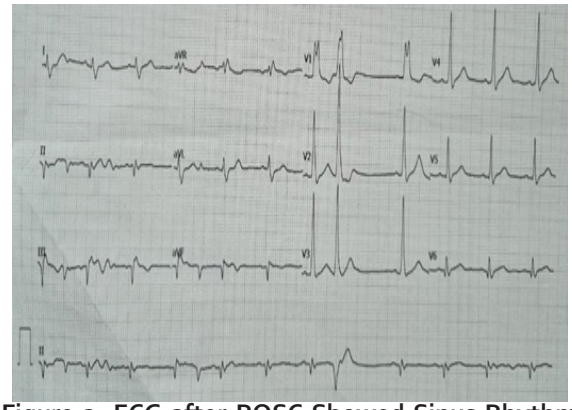

Figure 2. ECG after ROSC Showed Sinus Rhythm with Bifascicular Block due to RBBB and LAFB, OMI Inferior with Ischemic Change and Occasional VES

Ramipril, Bisoprolol, Furosemide, Atorvastatin, Fondaparinux, Ranitidine, Diphenhydramine, antibiotics for his pneumonia, Morphine, and Thiopental, which led to hemodynamic stabilization. A transthoracic echocardiography study revealed left atrial and ventricular dilatation and reduced left systolic function with an ejection fraction of 38%. Coronary angiography was not performed due to cost and resource limitation.

The patient was successfully extubated on the 4th day and discharged 9 days after admission with unspecific type of Kounis syndrome due to limitation for diagnostic.

DISCUSSION

The described patient developed an anaphylactic reaction with shortness of breath, erythema, diaphoresis, and nausea, leading to cardiac arrest 30 minutes after consuming a Non-Steroidal Anti-Inflammatory Drug (NSAID). ECG changes and a history of stent placement raised suspicion of myocardial injury. This patient had previously suffered allergic reactions to NSAIDs and was considered an atopic patient.

Kounis syndrome is formally defined as the co-occurrence of acute coronary syndromes, including coronary spasm, acute myocardial infarction, and stent thrombosis, alongside conditions characterized by mast-cell and platelet activation. These events involved interrelated and interacting inflammatory cells within the context of allergic or hypersensitivity reactions. [1,5]

Kounis syndrome has three variants. Type I KS (vasospastic angina) includes patients with normal or nearly normal coronary arteries without traditional cardiovascular risk factors, where inflammatory mediators induce coronary artery spasm or myocardial infarction.[1,5] Type II KS (acute coronary thrombosis) occurs in patients with pre-existing atheromatous disease, where inflammatory mediators may trigger plaque rupture or erosion leading to myocardial infarction.[5] Type III KS (stent thrombosis) involves coronary stent thrombosis, where thrombus specimens show eosinophils and mast cells, indicating an allergic inflammatory process.[4,5]

KS can be induced by drugs such as NSAIDs and antibiotics, as well as environmental exposure, insect stings, foods, and stents. Antibiotics and NSAIDs are the most common triggers.[2,5] NSAIDs may cause hypersensitivity

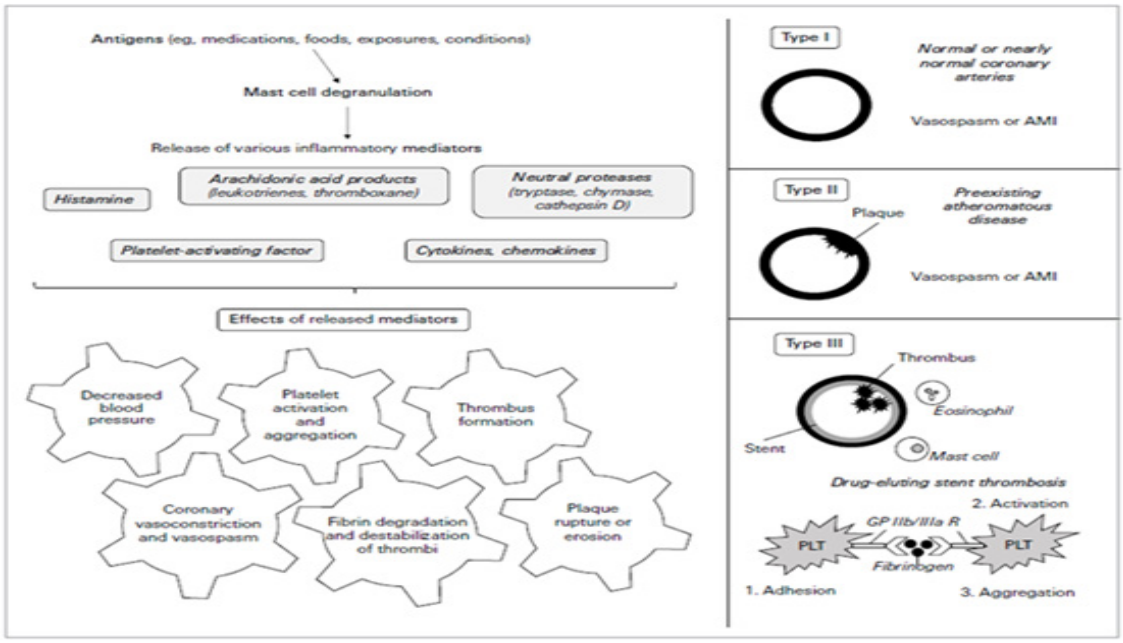

Figure 3. An Overview of Kounis Syndrome Pathophysiology

reactions via IgE-mediated mechanisms or through cyclooxygenase inhibition, leading to increased leukotriene production and release of histamine and tryptase from mast cells and eosinophils.[5] In patients with coronary artery disease, the number of cardiac mast cells is increased, making anaphylaxis more severe or fatal (6). In a series of 36 fatal cases of anaphylaxis due to drug allergy, atherosclerotic coronary disease was reported in 15 patients.[7,10]

The reported time from diclofenac use to symptom onset ranges from immediately up to 5 hours, with common symptoms including chest pain, hypotension, and multisystem allergic manifestations.[3] In this case, the patient exhibited dermatologic and respiratory symptoms without chest pain but progressed rapidly to cardiac arrest.

The diagnosis of KS relies on clinical history, symptoms, and evidence of multisystem involvement, supported by laboratory findings, ECG changes, echocardiography, and angiography.[5] Approximately 25% of patients have a history of allergy.[5] Laboratory tests such as serum tryptase, histamine, and cardiac troponins may aid diagnosis, with troponin elevated in about 60% of KS cases.[5] ECG findings, particularly ST-segment elevation, are common in diclofenac-induced KS.[3]

Currently, there are no specific guidelines for KS management. Treatment depends on the variant: allergic management in Type I, combined cardiac and allergic treatment in Type II, and standard myocardial infarction protocols with thrombus aspiration in Type III.[5]

The use of cardiovascular medications in anaphylaxis remains controversial. ACE inhibitors may worsen hypotension by interfering with bradykinin metabolism and the renin-angiotensin system.[6] However, a multicenter observational study found that beta blockers and ACE inhibitors did not increase the severity of anaphylactic reactions.[8]

CONCLUSION

Kounis Syndrome (KS) has a broad spectrum of clinical manifestations. It is important for general practitioners in the emergency room to be aware of the possibility of this condition in patients presenting with allergic reactions, especially if the patient has a history of coronary artery disease.

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