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The Shocking Truth: A Case Study of Allergic Reaction Leading to Heart Attack

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ABSTRACT

Background: Anaphylactic shock is a symptom of a severe allergic reaction that occurs after exposure to an allergen. Manifestations of anaphylactic shock can occur in various organs such as the cardiovascular system. Kounis syndrome (KS) is an acute coronary syndrome (ACS) related to allergic anaphylactic reactions triggered by the release of inflammation cells and mediators. ***Case Description:*** A 35-year-old male presented with a problem on his left leg skin. Examination revealed a red and edematous wound on his left calf, accompanied by pain and tenderness. He was then referred to a dermatologist by the on-duty doctor. Further diagnosis revealed cellulitis of the left leg, and he received intravenous meropenem 1 gram and oral clindamycin 300 mg. Four hours after the administration of intravenous meropenem and oral clindamycin, the patient complained of chest discomfort associated with palpitations, shortness of breath, nausea, and vomiting. His physical examination was remarkable for a shock with hypoxia and features of an anaphylactic reaction. ***Conclusion:*** We report a rare case of Kounis syndrome due to a drug allergy causing anaphylactic shock reaction. The treatments depend on the cause and symptoms of the patient.

Keywords: allergic reactions; acute coronary syndrome; Kounis syndrome.

INTRODUCTION

Drug allergy is a hypersensitivity reaction caused by the induction of immunoglobulin E (IgE) that is specific to certain allergens and is caused by allergy mediators, namely mast cells. There is an allergic reaction in the form of anaphylactic shock with symptoms that arise immediately after exposure to the allergen and can be life-threatening.[1] Anaphylactic shock is relatively rare with an estimated prevalence of 0.05–2%, and according to recent epidemiological data, between 2 and 20% of cases of anaphylaxis are fatal.[2] Manifestations of anaphylaxis include hypotension and shock, cardiac arrhythmia, ventricular dysfunction, and cardiac arrest, which are emergency conditions that require appropriate and rapid treatment.[2]

Kounis syndrome is an acute coronary syndrome characterized by coronary artery vasospasm, acute myocardial infarction, and coronary stent thrombosis, all of which are linked to mast cell and platelet activation in the context of allergies or anaphylaxis.[3] This case was raised because the incidence of anaphylactic shock with manifestations in the cardiovascular system is not widely reported, so it is hoped that it can provide new insights for handling similar cases.

CASE REPORT

This is the case of a 35-years-old man who was admitted to our hospital with cellulitis. He had an erythematous macule with indistinct borders, edema, and tenderness of the left leg. The patient denied any history of diabetes, hypertension, coronary heart disease, and allergy. Vital signs were normal, while laboratory results revealed leukocytosis. The patient received intravenous meropenem 1 gram and oral clindamycin 300 mg, four hours after the administration of intravenous meropenem and oral clindamycin, the patient complained of chest discomfort associated with palpitation, shortness of breath, nausea, and vomiting.

His physical examination was remarkable for a shock with hypoxia and features of anaphylactic reaction. There was neither urticaria nor angioedema on the record. This was also the first time since his admission that he had been given meropenem. A 12-lead electrocardiogram (ECG) showed atrial fibrillation with rapid ventricle response of 114 beats per minute, Inferior ST elevation (Figure 1), and cardiac enzyme showed elevated HS-Tropinin I level (32.6 ng/L). Bedside echocardiography showed regional left ventricle wall motion abnormality (akinetic at anterior A, septal A, inferior A, and another segment are hypokinetic). Allergic acute coronary syndrome was suspected.

Thus, the patient was managed with fluid resuscitation and initiated on a loading dose of aspirin and clopidogrel, followed by coronary angiography (CAG).

Surprisingly, the CAG revealed normal coronary arteries (Figure 2). From these results, we concluded a possible allergic reaction causing side effects on the coronary blood vessels, consistent with Kounis syndrome.

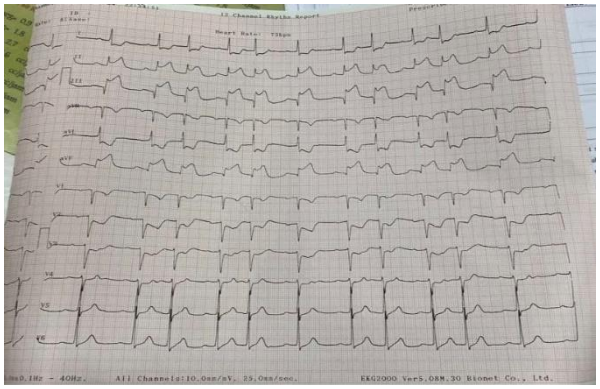

FIGURE 1: 12 lead ECG showed atrial fibrillation rhythm with a rapid ventricular response of 60-140 bpm, and inferior ST elevation.

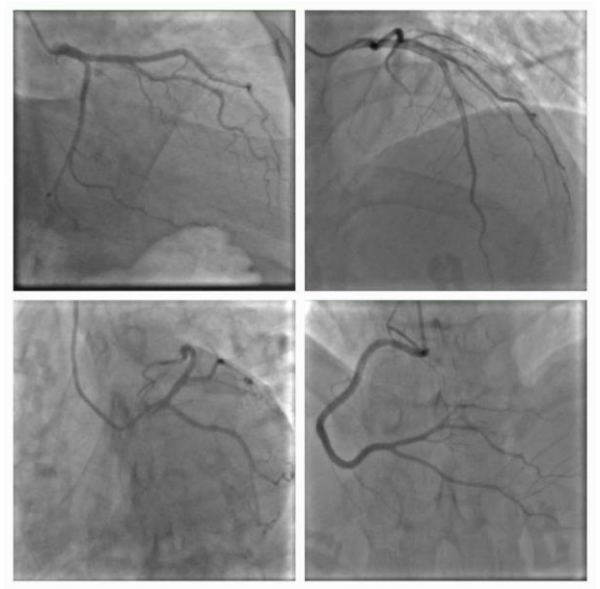

FIGURE 2: Coronary angiography showed normal coronary arteries.

DISCUSSION

Drug allergy is a type of unpredictable drug reaction that involves immunological mechanisms mediated by immunoglobulin E (IgE), immunoglobulin M (IgM), and certain complements through various mechanisms. During an allergic reaction, IgE binds to mast cells, which then release histamine, leukotrienes, and prostaglandins into the bloodstream. In the vascular system, histamine causes widespread vasodilation, leading to a drastic decrease in blood pressure and increased capillary permeability. This condition can be life-threatening if not treated immediately.[2,5]

Drug hypersensitivity reactions (DHRs) are most commonly triggered by nonsteroidal anti-inflammatory drugs (NSAIDs) and antibiotics.[4] In a study on adverse drug reactions, it was found that 15.1% of hospitalized patients and 6.7% of patients experienced severe drug reactions, including anaphylactic shock. Anaphylaxis can cause widespread dysfunction of bodily organs, with the cardiovascular system frequently involved in severe reactions.[6]

The diagnosis of anaphylaxis is based on the acute onset (within minutes to hours) involving the skin and/or mucosal tissues, and at least one of the following: respiratory symptoms, reduced blood pressure, collapse, syncope, or incontinence. Secondly, clinical manifestations are observed in two or more organs immediately after exposure. Thirdly, there is a decrease in blood pressure immediately after exposure, namely systolic blood pressure less than 90 mmHg or a decrease of more than 30% from the previous blood pressure immediately after the patient is exposed to the allergen, without any other apparent cause of shock.[7]

The term "cardiac anaphylaxis" refers to the functional and metabolic changes in the heart that result from the release of histamine and arachidonic acid cascade metabolites following a severe allergic reaction. Mast cells are the primary drivers of this inflammatory process, releasing inflammatory mediators both locally and into the systemic circulation. These mediators exert several cardiovascular effects, including coronary vasoconstriction and platelet activation, which can lead to plaque erosion and rupture. They also cause tachycardia, impaired ventricular contractility, and atrioventricular conduction block.[14]

One cardiovascular disorder associated with allergy is Kounis syndrome, characterized by acute coronary syndrome occurring concurrently with an allergic reaction or anaphylaxis.[8,9] Delayed cutaneous manifestations are thought to be caused by vasoconstriction, resulting from reduced cardiac output and hypotension during rapidly developing anaphylaxis.[15]

The clinical manifestations of Kounis syndrome are invariably associated with an allergic reaction, accompanied by cardiovascular symptoms. Patients may present with acute chest pain, chest discomfort, shortness of breath, headache, weakness, nausea, pruritus, skin itching, and vomiting. Physical examination may reveal hypotension, bradycardia, tachycardia, cold extremities, and even cardiac arrest. Electrocardiographic findings may include ST-segment elevation or depression, varying degrees of heart block, and arrhythmias.[10]

Cardiac troponin I levels are higher in patients with anaphylaxis compared with patients experiencing milder allergic reactions. These findings may have profound clinical, therapeutic, and pathophysiological implications related to anaphylaxis, myocardial injury, and Kounis syndrome. Guidelines recommend that physicians treating patients with anaphylaxis promptly rule out the possibility of Kounis syndrome by examining electrocardiographic findings and measuring cardiac troponin levels.[15]

Meropenem, a broad-spectrum carbapenem beta-lactam antibiotic, is used to treat a variety of gram-positive and gram-negative bacterial infections, including anaerobic bacteria. For skin infections, particularly cellulitis, meropenem is a recommended therapeutic option because of its good tolerability and low risk of allergic reactions. The reported incidence of these reactions (rash, pruritus, and urticaria) is 0.3%–3.7%. While generally safe and effective, meropenem can trigger allergic reactions in some individuals. These reactions can range from mild skin rashes to severe anaphylaxis. Rarely, these allergic reactions can lead to Kounis syndrome.[11,12,13]

The patient presented with a diagnosis of cellulitis. The patient reported no history of allergies. Four hours post-injection, the patient experienced weakness, chest pain, nausea, and vomiting. Inferior ST elevation and a rise in high-sensitivity troponin were observed. The meropenem injection is the suspected trigger for this allergic acute coronary syndrome, consistent with Kounis syndrome. The reaction was acute, with onset within 6 hours. On physical examination, the patient presented in shock, with a blood pressure of 70/60 mmHg and a heart rate of 129 beats per minute. Emergency management was immediately initiated, including resuscitation with vasopressors and fluid loading, aspirin, and clopidogrel, as indicated for inferior STEMI, followed by immediate coronary angiography (CAG). The surprising result of the CAG, which was normal, led to the conclusion of an anaphylactic reaction manifesting as acute coronary syndrome (Kounis syndrome).

Anaphylactic therapy consisted of epinephrine 0.01 mg/kg (maximum dose 0.3-0.5 mg) administered intramuscularly every 15-20 minutes as needed, up to 3-4 doses. During the initial resuscitation for hemodynamic instability, the patient received 300 ml of 0.9% NaCl crystalloid over 30 minutes, followed by 500 ml over the next 2 hours. Hydrocortisone was administered with a loading dose of 5 mg/kg body weight intravenously, followed by a maintenance dose of 2.5-5 mg/kg body weight intravenously every 4-6 hours for 48-72 hours.[7] After two days of ICU observation with stabilization and improvement, the patient was transferred to the ward and discharged with outpatient follow-up.

CONCLUSIONS

Anaphylactic allergic reactions are something we do not expect in the therapy we provide. Allergic reactions in the form of a heart attack are rare, but it is very important for us to know so that we can provide rapid and appropriate therapy for the prognosis and safety of patients.

Emergency and acute clinical therapy experienced by the patient still plays an important role, while ruling out the possibility of an acute coronary syndrome due to blockage in the blood vessels. In this case, we have provided epinephrine and steroid therapy for the anaphylactic reaction that occurred so that the patient could recover immediately.

CONFLICT OF INTEREST

There is no conflict of interest.

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