Case Report
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A Case Report of Kounis Syndrome Developing Anaphylaxis Secondary to Vitamin B and C Infusion

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Abstract

Kounis syndrome is a condition characterized by temporary coronary spasm, reduction in coronary blood flow and myocardial ischemia by activated mast cells as a result of an abnormal immune reaction to a drug or molecule. It is also called allergic angina and allergic myocardial infarction. It was first described by Kounis and Zarvas in 1991(1). Kounis syndrome can be observed in all age groups regardless of the history of coronary artery disease. Kounis syndrome may occur due to environmental factors such as drugs, intracoronary stent implantation, foods, insect bites, bee stings, pollen, latex exposure etc. In our case report, acute coronary syndrome occurring during anaphylaxis secondary to vitamin B and C infusion, which is frequently used in clinical practice, will be explained.

Case Report

A 26-year-old female patient with no known disease was admitted to the emergency department with complaints of widespread body pain, weakness, and fatigue. The Covid 19 PCR test performed on the patient was negative. Vitamin B complex and vitamin C infusion were started in saline solution for hydration and support. The patient described numbness in the arm, feeling unwell, flushing on the face, and difficulty in breathing within the first minute after starting the infusion. The infusion was terminated, and the patient was monitored. Progression in dyspnea was observed. As the patient was hypotensive and tachycardic, 0.5 mg adrenaline was administered intramuscularly with the preliminary diagnosis of anaphylaxis. Simultaneously, the patient developed vomiting and palpitation. In the ECG, T wave negativity was observed in d2-d3 avF and V3-6. (figure 1) The control ECG, which was taken at the 15th minute after the medical treatment, was found to be normal. (Figure 2) IV hydration was applied to the patient who was hypotensive and tachycardic in the follow-ups. Progressive increase in troponin values was observed. The 1st troponin was 62 ng/L, the 3rd hour troponin 1100 ng/L, and the 6th hour troponin 1200 (upper limit of normal 11.6 ng/L) Kounis syndrome was considered in the patient with ECG change, left arm pain and increased troponin. 300 mg acetyl salicylic acid, 600 mg clopidogrel and 6000 IU enoxoparin were administered in medical treatment. In the follow-up of the patient in the coronary intensive care unit, angina and arrhythmia did not develop, and no additional changes were detected in the control ECGs. A decrease in troponin value was observed at the 14th hour (650 ng/L) and at the 30th hour troponin 140 ng/L. The echocardiographic evaluation was totally normal. The patient, who had no known atherosclerotic history, was discharged after 72 hours of monitored follow-up in the coronary intensive care unit without coronary angiography due to the patient's disapproval. No additional pathology or symptoms were detected in the outpatient follow-up at the 1st month.

Discussion

Vitamin supplements are commonly used, often without a doctor's recommendation. Triggering anaphylaxis and subsequent progression to acute coronary syndrome in a patient without any known allergy history, risk factors and disease shows the importance of preventing unnecessary use of these molecules. Although the pathophysiology of Kounis

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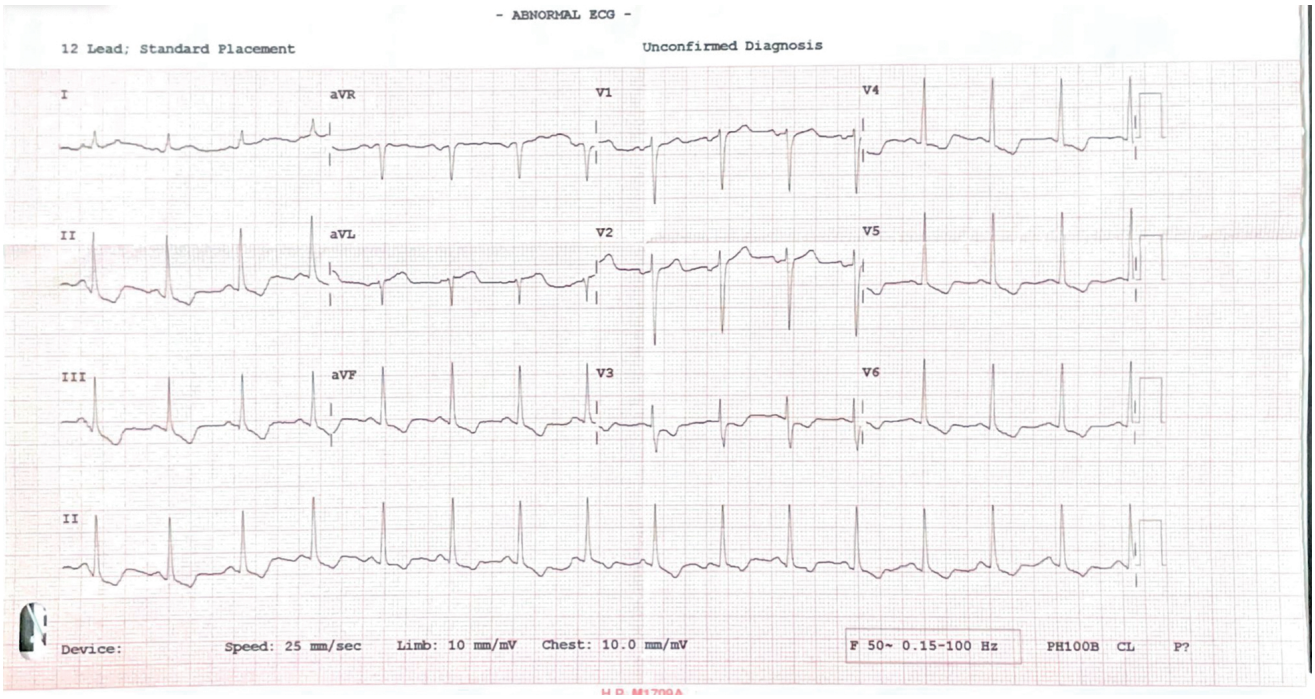

Figure 1

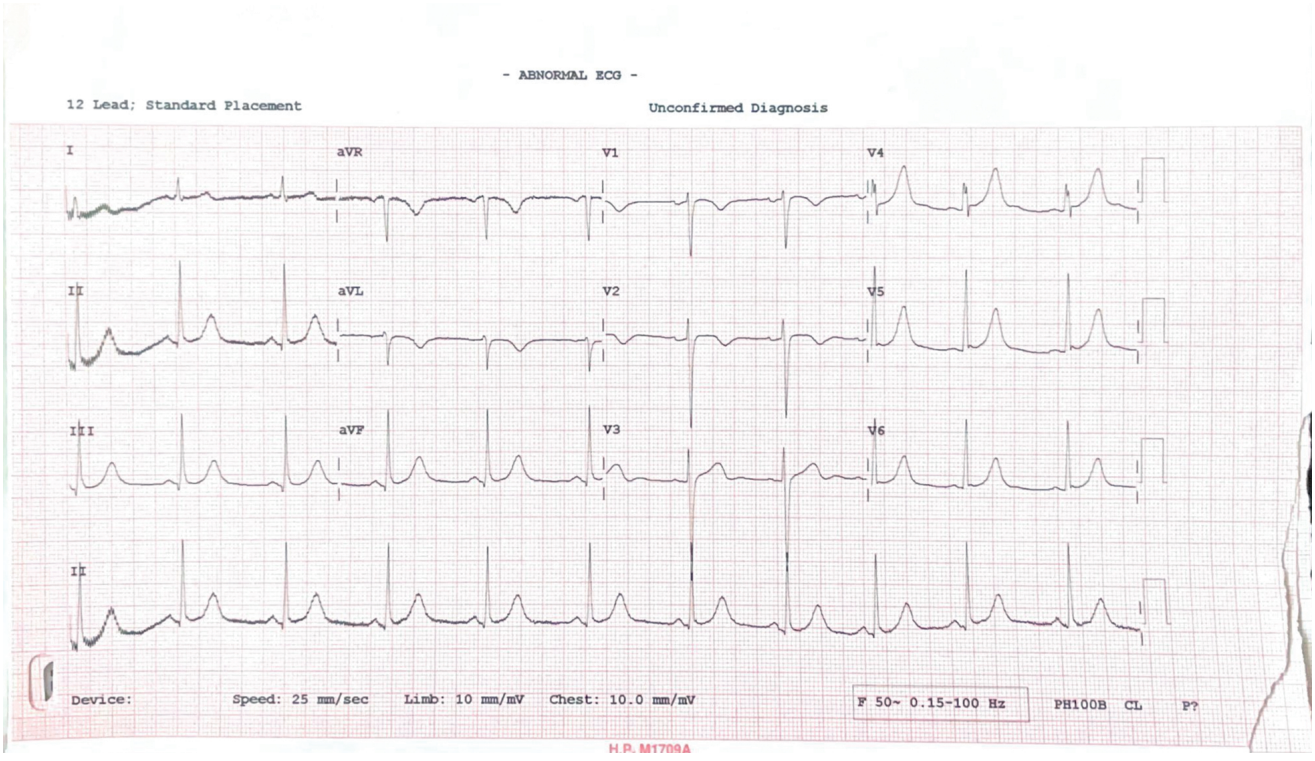

Figure 2

syndrome is not clearly defined, the most likely cause is activation of mast cells secondary to allergic stimuli and triggering of coronary spasm, plaque rupture and plaque erosion by vasoactive mediators such as histamine, tryptase, chymase, paf, cytokines, prostaglandins and leukotrienes released from these cells is considered (2). In addition, hypotension, which is one of the main clinical consequences of anaphylaxis, also plays a triggering role in the decrease in coronary blood flow and triggers myocardial ischemia. Angina, ECG changes, myocardial wall movement disorders, and cardiac enzyme elevations, especially troponin, can be detected in patients with acute coronary syndrome.

Kounis syndrome is basically classified into 3 types: Type 1 kounis syndrome is also called allergic vasospastic angina. Patients have normal coronary arteries or noncritical coronary artery disease. Depending on the severity of the clinic, an increase in troponin can be detected. In the treatment, nitroglycerin and calcium channel blockers can be used in patients who do not have hypotension in addition to intramuscular adrenaline (3,4). Type 2 kounis syndrome is an acute coronary syndrome triggered by coronary spasm or plaque erosion in individuals with asymptomatic coronary artery disease. In type 3 Kounis syndrome, stent thrombosis is observed. Activation of mast cells, which are densely located in atherosclerotic areas, triggers thrombosis (5). If the thrombus is stained with Giemsa and Hematoxylin eosin, the presence of eosinophils and mast cells can be demonstrated. Thrombus aspiration or stent implantation is the appropriate treatment option in these patients (6). Intramuscular adrenaline is recommended for the initial treatment, especially in patients presenting with anaphylaxis. However, the use of intravenous adrenaline is also recommended in patients with persistent symptoms despite intramuscular adrenaline therapy. It should be kept in mind that the effect of adrenaline is limited, especially in patients using beta-blockers, and the use of glucagon should be considered. In our case, we describe a 26-year-old young patient who developed anaphylaxis and concurrent acute coronary syndrome symptoms in the first minute of the infusion. In our patient, intramuscular adrenaline was administered for anaphylaxis due to the development of hypotension, tachycardia and airway obstruction. After symptomatic treatment, the patient's complaints regressed. Simultaneously, an improvement was detected in the ECG. Due to limited access to mast cell activation markers such as histamine, tryptase, and chymase some tests could not be performed. Angiography could not be performed because the patient's complaints regressed and did not accept coronary angiography. Although it is currently thought that the patient has Type 1 Kounis syndrome, the possibility of alpha receptor-mediated coronary vasoconstriction secondary to the effect of adrenaline cannot be excluded. In the case report of a 15-year-old who developed angioedema after exposure to cats and dust but developed ST elevations on the ECG after iv administration of epinephrine, it could not be distinguished whether the cardiac pathology was secondary to an allergic reaction or due to a side effect of epinephrine, similar to our case. However, while cardiac symptoms developed after epinephrine administration in this case, the fact that the onset of allergic reaction and the onset of cardiac symptoms were simultaneous in our case shows that our case is primarily compatible with Kounis Syndrome (7). Adrenaline, one of the cornerstones of anaphylaxis treatment, acts on both alpha- and beta-adrenergic receptors, causing vasoconstriction, positive chronotropic and inotropic effects, and bronchodilation. It also suppresses histamine and histamine-related mediators released from mast cells. However, it may cause transient myocardial ischemia with its coronary vasoconstriction effect. At this stage, a great dilemma is observed in making the treatment decision of the patients. The use of nitroglycerin and calcium channel blockers is recommended due to their effects on coronary vasospasm, especially in Kounis syndrome cases without hypotension (8).

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