Kounis syndrome due to diclofenac injection

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Abstract

Kounis syndrome is a type of acute coronary syndrome which occurs after allergic reaction or hypersensitivity. Kounis syndrome consists of coronary vasospasm with or without atheromatous plaque erosion or rupture and is also known as “allergic myocardial infarction” or “allergic angina”.
In this case report, we describe a case of a male patient who was interned with the diagnosis of ST segment elevation myocardial infarction after intramuscular diclofenac injection.

Keywords: diclofenac, Kounis Syndrome.

Introduction

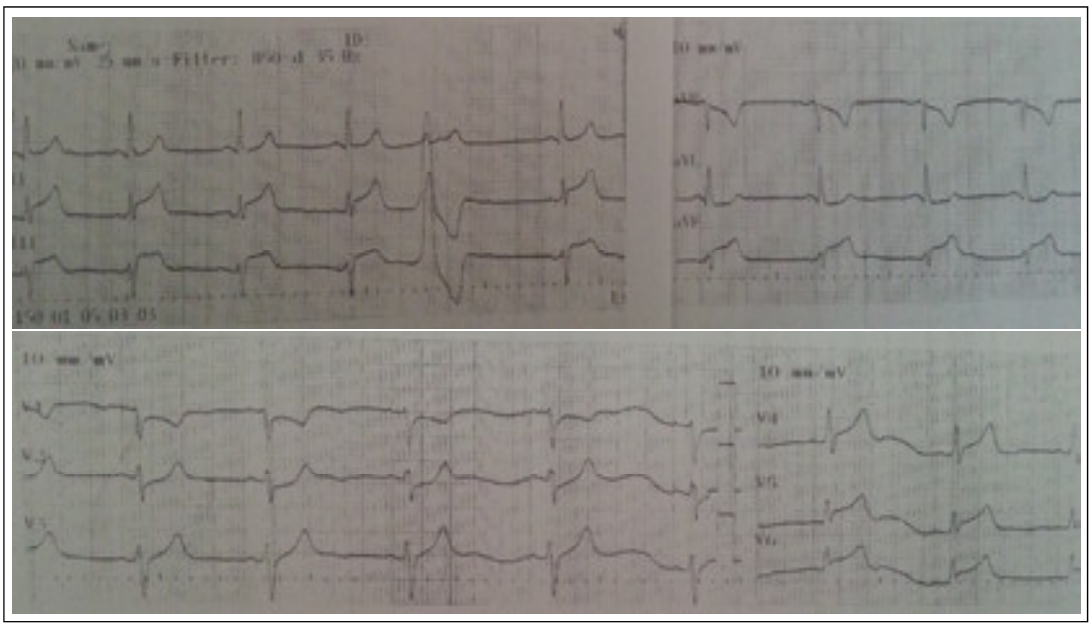
Kounis syndrome (KS) was firstly described as "allergic angina" or "allergic myocardial infarction" by Kounis and Zavras in 1991 (1). After mast cells activations, allergy, hypersensitivity, as well as anaphylactoid reactions, KS was considered as an acute coronary syndrome (ACS) (2). Drugs, foods, environmental factors and stent implantation can induce this allergic reaction. Clinical KS ranges from angina to ACS, especially inferior wall myocardial infarction (3). Hypersensitivity reactions associated with underlying coronary artery disease are not rare, despite the fact that they are not frequently documented in the medical literature especially if induced by diclofenac potassium (4-6).

In this report we describe a case of a male patient who was interned with the diagnosis of ST segment elevation myocardial infarction after intramuscular diclofenac injection.

Case report

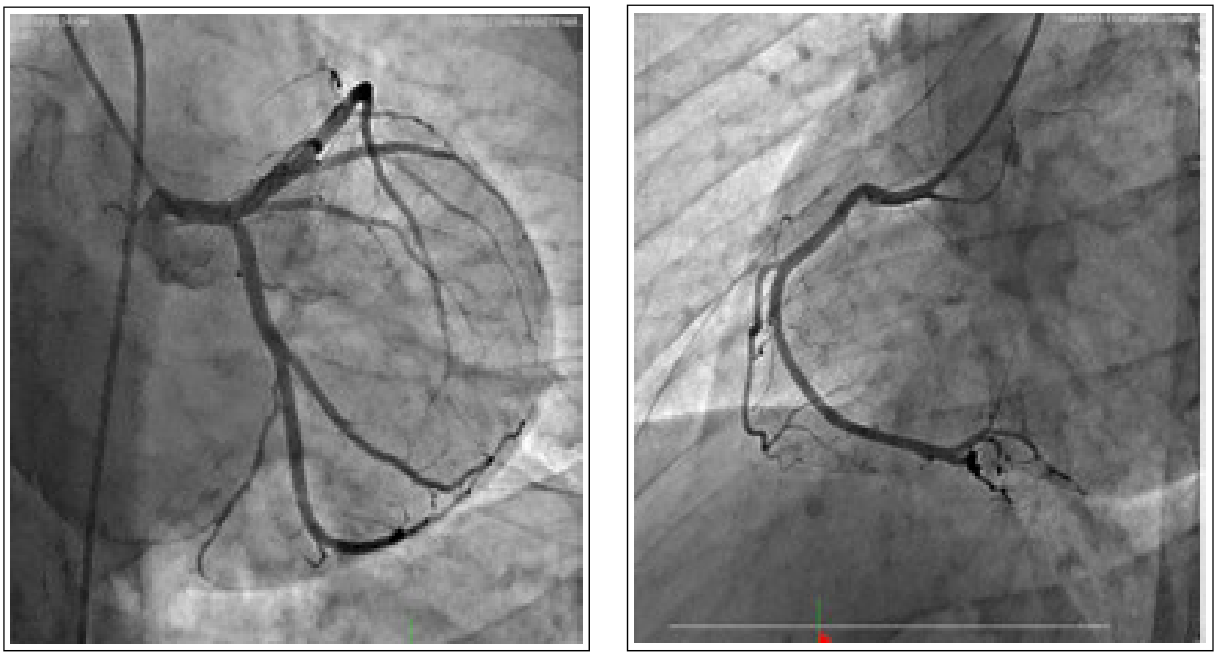
A 34-year old male patient with no history of allergy and coronary artery disease was admitted to emergency room with the complaint of chest pain which had started one hour ago. He was a smoker. Upon physical examination, small, papular, diffuse urticaria were determined. Blood pressure level was 115/70 mmHG and heart rate was 62 bpm. In the electrocardiography (ECG), there was an ST segment elevation in leads DII-DIII-aVF-V4-5-6 and reciprocal ST segment depression in leads aVL-V1-2 (Figure 1).

Figure 1. ECG on admission



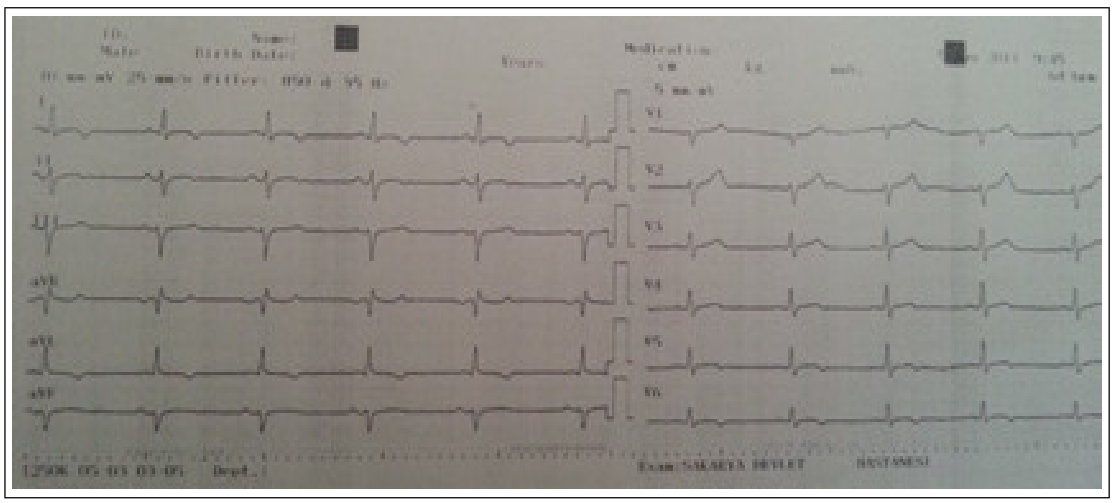
The patient was diagnosed with acute inferolateral wall myocardial infarction and, therefore, 300 mg acetylsalicylic acid and 600 mg loading dose of clopidogrel were given to him peroral. Next, the patient was taken to the catheterization laboratory. There were no pathological changes such as plaque rupture, thrombus or dissection in the coronary angiography (Figure 2).

Figure 2. Left Anterior Descending (LAD), Circumflex (CX) Artery and Right Coronary Artery (RCA) in the coronary angiography



The conclusion was that the ischemic ECG changes developed due to the allergic reaction secondary to drug use. In the laboratory indices, troponin I levels peak was 16.7 ng/ml (reference range: 0-0.04), eosinophil count was normal and total immunoglobulin E levels was increased to 106.2 IU/ml (reference range: 20-100 IU/ml). Ejection fraction was 60% and left ventricle wall motion was normal in the transthoracic echocardiography. Neither complication nor recurrent ischemic events grow up. After 3 days of hospitalization he was discharged by medical therapy (5 mg/daily desloratadine, isosorbid-5-mononitrat 50 mg/daily) and referring allergical disease polyclinics (Figure 3).

Figure 3. ECG at discharge



Discussion

Even though ACS usually involves atheromatous plaque rupture or erosion following thrombus formation, sometimes it may appear in patients with no prior coronary artery disease (CAD) (7). KS, firstly described in 1991, is also called "allergic angina" or "allergic myocardial infarction" (1). KS is a hypersensitivity or allergic event that occurs after drug intake (analgesics, antibiotics, antineoplastics, contrast agents, steroids), stent implantation (nickel, chromium, titanium), food and environmental factors (1-8). Pathogenesis of this condition involves direct allergen or allergen-antibody complex activates mast cells, and inflammatory mediators (histamine, leukotrienes, platelet activating factor) are released with mast cells degranulation. These mediators lead to vasoconstriction and platelet activation, after which the ACS occurs (3). KS is divided into two groups: Type 1, in patients who do not have prior CAD and risk for atherosclerosis, where coronary vasospasm starts after allergen factor exposure; Type 2, where KS occurs in patients with established CAD (9). There is also a newly defined Type 3, where mast cells and eosinophils are detected in the pathological examination with giemsa staining of the thrombus in patients with stent thrombosis (10). The main treatment of KS is management of the ACS and the allergic reaction. Nitrates and calcium channel blockers should be given because of the coronary vasospasm. Beta blockers are unfavourable because they may lead to vasospasm. Sometimes only antihistamines or corticosteroids may be sufficient (8,9).

In conclusion, even in individuals with normal coronary arteries, ACS may develop as a result of vasospasm secondary to allergic reaction, especially after drug use. Physicians dealing with cardiovascular disease should be familiar with the new information about the diagnosis and treatment of KS. Furthermore, follow-up by allergic disease specialists is recommended.

Conflicts of interest: None declared.

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