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Kounis syndrome as a result of anaphylactic reaction to diclofenac sodium: A case report

Diklofenak sodyum kullanımı sonrası anafilaktik reaksiyon sonucu gelişen Kounis sendromu: Olgu sunumu

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Abstract

Kounis syndrome refers to acute coronary syndromes of varying degrees (myocardial ischaemia to infarction) induced by mast cell activation as a result of allergic and anaphylactic reactions. Following subclinical, acute or chronic allergic reactions, it has a clinical spectrum ranging from chest pain to ST segment elevated myocardial infarction. The trigger of the allergic reaction can be drugs, food, environmental factors (insect bite, bee sting, pollen, latex contact). We present a case of a patient who developed Kounis syndrome as a result of anaphylactic reaction to Voltaren (Diclofenac sodium) which is one of the drugs widely used in the emergency departments as intramuscular pain killer.

Keywords: Kounis syndrome, Diclofenac sodium, Hypersensitivity, Anaphylaxis

Öz

Kounis sendromu mast hücrelerinin etkinleşmesi ile seyreden alerji, hipersensitivite, anaflaksi veya anaflaktoid reaksiyonlarla ilişkili olarak akut koroner sendromun değişik derecelerini ifade eder. Subklinik olarak akut veya kronik alerjik reaksiyona eşlik eden ve göğüs ağrısından başlayıp ST elavasyonlu miyokard enfarktüsü kadar uzanan bir klinik spektruma sahiptir. İlaçlar, yiyecekler, çevresel etkenler (böcek ısırması, arı sokması, polenler, lateks teması gibi) alerjik reaksiyonu tetikleyen neden olabilir. Burada, acil servislerde yaygın olarak kullanılan Voltaren'e (Diklofenak sodyum) bağlı gelişen anafilaktik reaksiyonu olan bir Kounis sendromu vakasını sunuyoruz.

Anahtar kelimeler: Kounis sendromu, Diklofenak sodyum, Aşırı duyarlılık, Anafilaksi

Introduction

Kounis syndrome was first described by Kounis and Zarvas in 1991 [1]. The reaction may be triggered by drugs, food, environmental factors such as insect bite, bee sting, pollens, or latex contact. It is common in Europe, especially in countries like Spain, Italy, Greece, and Turkey. It is sometimes referred to "Allergic Angina syndrome" or "allergic myocardial infarction".

Kounis syndrome has been classified into two main types depending on the pathophysiology or presence of coronary artery disease (CAD). In type I the patients have no signs of arteriosclerotic CAD and symptoms develop as a result of vasospasm triggered by allergic mediators, but in type I there is no increase in cardiac enzymes and troponin. Type II patients are those with CAD in whom the allergic mediators trigger coronary vasospasm or plaque rupture, and they have associated rise in cardiac enzymes and troponin [1-3].

In our case report we present a patient who developed Kounis syndrome after anaphylactic shock as a result of intramuscular injection of diclofenac sodium (DS) which is widely used in emergency department for pain management.

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Case presentation

A 54 year old man with severe back pain was admitted to our emergency department. The patient had history of CAD and hypertension. He had no history of allergy to any drug. The findings in his physical examination on arrival were: the blood pressure was 140/90 mmHg (no difference between right and left side), pulse: 95 beat/min. temperature: 37 degrees centigrade, respiratory rate: 18/min, oxygen saturation: 98%. Apart from tenderness in intrascapular region, his other physical examinations were normal. The electrocardiography (ECG) and laboratory tests were also normal. The patient was diagnosed as myalgia and hence DS 75mg (Voltaren ® 75 mg/3 mL IM; Novartis, Stein-AG, Swiss) was administered intramuscularly for pain management. Immediately after the injection the patient collapsed. His blood pressure fell to 50/30 mmHg, pulse was 145 beats per minute, his Glasgow Coma Scale was 10 and he developed urticaria and edema of mucous membrane. He was diagnosed as having anaphylactic shock as a result of DS injection. İntramuscular adrenaline 0.3mg and pheniramine maleate 45.5mg (Avil ® 45.5 mg/2ml: Sandoz, Kurtkoy, Istanbul, Turkey) were administered. 80 mg Methylprednosolone (Prednol-l ® 40mg, Mustafa Nevzat, Istanbul, Turkey) and intravenous fluid administration was started and vasopressor support was given. In his new laboratory results after the incident the troponin level that was in normal range on arrival (0.02 ng/ml) showed a seven fold increase to 1.40 ng/ml, also an increase in CK-MB from 15 ng/ml to 68 ng/ml, and aspartate transaminase levels (from 16 U/L to 44 U/L). There were no changes in his ECG findings. Cardiology consultation was performed immediately. His echocardiography showed an ejection fraction of 25% and global hypokinesia of the left ventricle. The patient was diagnosed as Kounis Syndrome resulting from anaphylaxis to DS. Emergency cardiovascular percutaneous intervention was not found necessary and he was put on medical treatment. After 36 hours of hospitalization the patient was discharged with normal vital signs.

Discussion

"Kounis syndrome" is a condition that occurs by coincidence, accompanied by the classical angina pectoris' clinic and laboratory results that is caused by the inflammatory mediators resulting from allergic reaction [2]. Depending on the underlying pathophysiology or presence of CAD, Kounis syndrome has been classified into two main types [3,4]. In type I the patients have no signs of arteriosclerotic CAD risk and symptoms develop as a result of vasospasm triggered by allergic mediators, but in type I there is no increase in cardiac enzymes and troponin. Type II patients are those with CAD in whom the allergic mediators trigger coronary vasospasm or plaque rupture, and they have associated rise in cardiac enzymes and troponin levels as seen in our case.

The underlying pathology in Kounis syndrome is coronary artery vasospasm due to release of vasoactive mediators secondary to mast cell degranulation. The mast cells in heart tissue play a great role in anaphylaxis. They may trigger tachycardia, change ventricular contractility and cause block antihistaminic and corticosteroid treatment should start immediately and preparation for coronary intervention should be ready. In our case adrenalin, antihistaminic and corticosteroid treatment was given. His syndrome has also been referred to as "cardiac anaphylaxis" [3,4]. This has been used to explain the functional and metabolic changes occurring in the heart following an allergic reaction, caused by the release of histamine and metabolites of the arachidonic acid cascade. The most important step in the diagnosis of Kounis syndrome is to suspect it in patients with allergic symptoms accompanied by chest pain [5]. The ECG changes seen may include ST elevation or depression, heart block of varying degrees or cardiac arrhythmias. There were no ECG changes in our case, but an increase in troponin levels was detected. Ordering of ECG and cardiac enzymes in patients with hypersensitive reactions, as seen in our case, is important in the diagnosis of Kounis syndrome.

Fox DJ et al have reported a case of Myocardial infarction after aspirin treatment [6]. Non-steroidal anti-inflammatory drugs have been reported to be one of the most common classes of medications causing anaphylaxis [7-10].

In conclusion, because of extensive use of in the emergency departments, physicians should have enough knowledge about this syndrome and be aware of its complication to make a prompt diagnosis and initiate an early treatment. As seen in our case, non-steroidal anti-inflammatory drugs should be used carefully in those with type II Kounis syndrome, and if the syndrome occurs, acute coronary syndrome protocols should be activated and early anaphylaxis treatment should be given without hesitation.

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