DİKLOFENAK POTASYUMUN ORAL ALIMINA BAĞLI KOUNİS SENDROMU: OLGU SUNUMU

Kounis Syndrome Due to Oral Intake of Diclofenac Potassium: A case report

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ÖZET

Allerjik reaksiyonlar, koroner spazm, akut stent trombozu veya inflamatuar sitokinlerin salınması sonucu koroner plak rüptürü yoluyla akut koroner sendroma neden olabilirler. Bu klinik antite Kounis Sendromu olarak adlandırılır. 64 yaşında erkek hasta göğüs ağrısı, nefes darlığı, bulantı ve kusma şikayetleri ile hastanemiz acil servisine başvurdu. Başvuru öncesinde baş ağrısı için oral yolla diklofenak kullandığı tespit edildi. Elektrokardiyografisinde(EKG) lateral derivasyonlarda ST-segment yükselmesi izlendi. Acil koroner anjiyografide koroner damarlar normaldi. Hastaya anafilaktik reaksiyon teşhisi kondu. Anafilaksi başarıyla tedavi edildikten sonra, ST-segment yükselmeleri çözüldü. Klinisyenler bu sendromun hızlı tanı ve uygun tedavi için farkında olmalıdır.

Anahtar Sözcükler: *Kounis sendromu; Anafilaksi; Diklofenak potasyum*

ABSTRACT

Allergic reactions may cause acute coronary syndrome by coronary spasm, acute stent thrombosis or coronary plaque rupture via releasing of inflammatory cytokines. This clinical entity is termed as Kounis Syndrome. We encountered a 64-year-old male who admitted to emergency department of our hospital with complaints of chest pain, shortness of breath, nausea and vomiting. Before the application, it was found that he used diclofenac orally for headache. His electrocardiography(ECG) showed ST-segment elevations in lateral derivations. Emergency coronary angiography revealed normal coronary vessels. He was eventually diagnosed with an anaphylactic reaction. After successful treatment of anaphylaxis, ST-segment elevations resolved. Clinicians should be aware of this syndrome for promt recognition and proper treatment.

Keywords: *Kounis syndrome; Anaphylaxis; Diclofenac potassium*

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INTRODUCTION

Kounis syndrome refers to acute coronary syndromes of varying degree induced by mast cell activation as a result of allergic and anaphylactic reactions. This syndrome was described firstly by Kounis and Zavras in 1991 (1,2). This allergic reaction leads to coronary artery vasospasm and/or rupture or erosion of coronary plaque soon after releasing of inflammatory cytokines (1). It has a wide clinical spectrum between angina pectoris and acute myocardial infarction which may coexist with allergic reactions like hypersensitivity, anaphylactoid reaction and anaphylaxis (3). A variety of conditions are responsible for Kounis syndrome such as environmental exposures and medically used drugs (4). Diclofenac sodium is a widely used nonsteroidal antiinflammatory drug in the management of pain, and it can be found easily as an over-the-counter drug. We present a case to report that anaphylactic shock and acute coronary sydrome may be encountered after the use of this drug.

CASE REPORT

A 64-year-old man was admitted to our emergency department with complaints of chest pain, shortness of breath, nausea and vomiting. His blood pressure and heart rate were 80/50 mmHg and 110 beats per minute. Body temperature was 36.7 ºC and respiratory rate 22 breaths per minute. His skin was erythematous. The patient has no prior coronary artery disease, diabetes, hypertension and smoking. He did not have any known allergic disorders such as asthma. He has been using oral prednisolon 16 mg (Prednol, Mustafa Nevzat Pharmaceutical Industry, Istanbul, Turkey) once a day for sacroiliitis. One hour before admitting to the hospital, he had used 50 mg diclofenac potassium (Dolorex; Abdi Ibrahim Pharmaceutical Industry, Istanbul, Turkey) orally for headache. Initial electrocardiogram revealed sinus tachycardia and ST-segment elevation in D1 and aVL leads with reciprocal ST depressions in D3 and aVF leads (Figure 1). Creatinin kinase myocardial band and Troponin I levels were normal [(0.25 ng/ml (normal value <0.3ng/ml), 7.3U/L(normal value <25 U/L), respectively]. Complete blood count and other biochemical tests were also normal. Bedside transthoracic echocardiography showed lateral and anterolateral wall hypokinesis with an ejection fraction of 35%. No valve abnormality was detected. Nasal oxygen, intravenous isotonic saline and hydrocortisone, subcutaneous adrenalin were administered according to anaphylaxis treatment protocol. Emergency coronary angiography was planned. Coronary angiography demonstrated normal coronary arteries without any significant lesion (Figure 2). The angina resolved afterwards. A repeat electrocardiogram after coronary angiography showed complete resolution of ST segment elevations and depressions (Figure 3). Troponin I level rised to 5,3 ng/ml. In the third day of hospitalization, ejection fraction rised to 50% without any residual wall motion abnormality and troponin I level returned to its normal level (0.23ng/ml). He was discharged uneventfully on the fourth day.

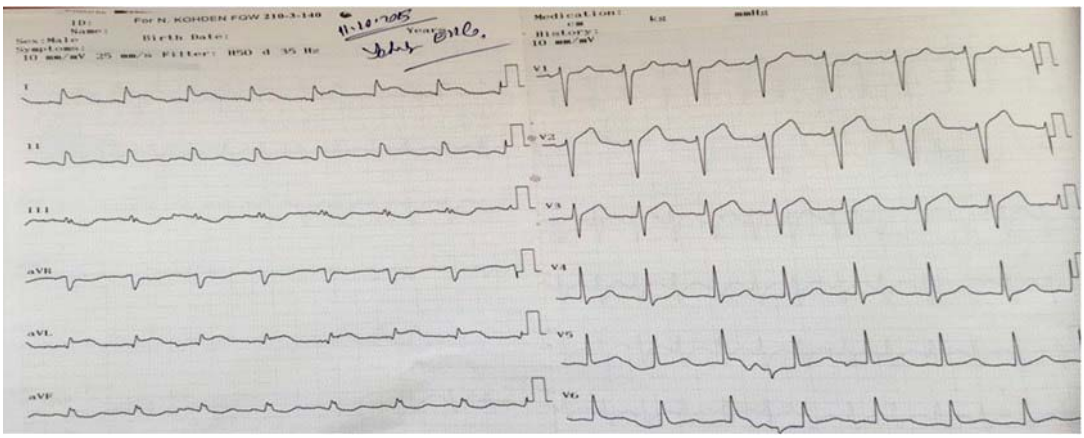

Figure 1. Emergency electrocardiogram showing ST- segment elevations in D1 and aVL with reciprocal ST-segment depressions in D3 and aVF.

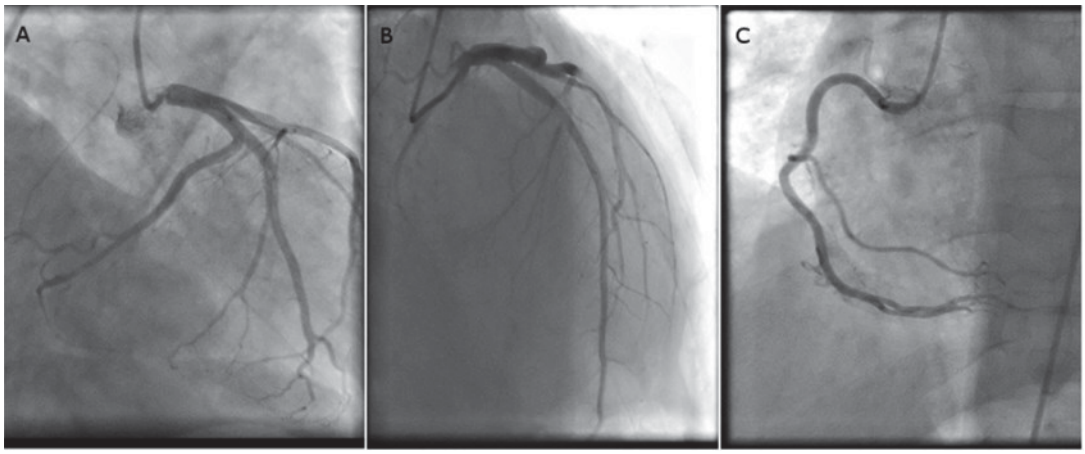

Figure 2. Emergency coronary angiography showing nearly normal coronary arteries. A, right anterior oblique caudal view of left coronary system; B, anterior oblique cranial view of left coronary system; C, left anterior oblique view of right coronary system.

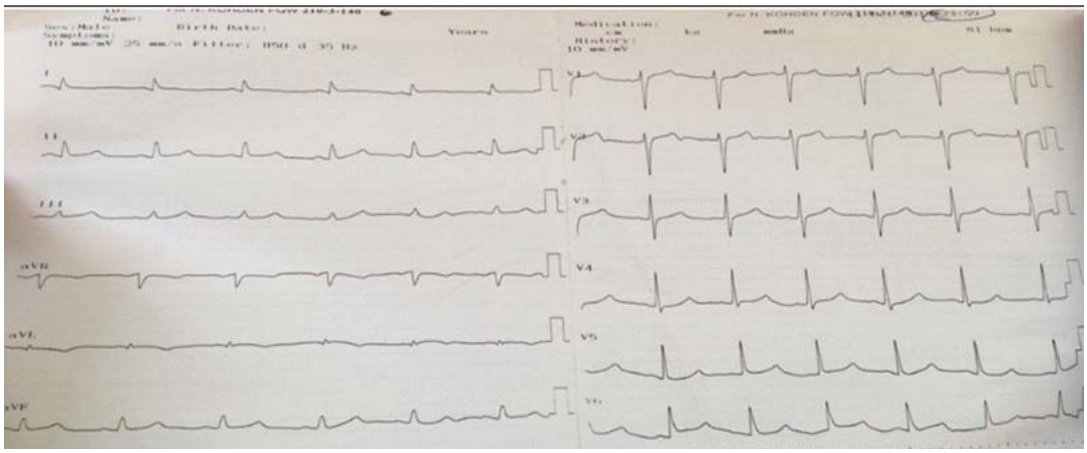

Figure 3. A repeated electrocardiogram showing complete resolution of ST- segment elevations and depressions.

DISCUSSION

Here, we presented a case with an anaphylactic shock and acute coronary sydrome due to ingestion of diclofenac potassium. Although the patient was a male and relatively elderly, absence of other well-known atherosclerotic risk factors such as smoking, hypertension, diabetes, hyperlipidemia and family history of coronary artery disease initially led us to think that acute coronary syndrome was due to ingestion of diclofenac potassium. Moreover, the time frame between ingestion of diclofenac potassium and onset of acute coronary syndrome as well as resolution of ST-segment elevations on ECG soon after the treatment of anaphylactic shock were also in line with our initial diagnosis. The patient in our case report did not have any allergic disorders like asthma but he has been using oral prednisolon 16 mg once a day for sacroilitis. It may be proposed that oral prednisolone treatment may have led to an acute coronary syndrome such as de novo coronary thrombosis or acceleration of existing coronary plaques. However, this is unlikely when taking into consideration normal coronary arteries at coronary angiography. The patient has diclofenac potassium allergy. Formerly, he suffered allergic reaction by using diclofenac potassium. The patient suffered Kounis syndrome while he was on prednisolon treatment which is rather interesting. Three types of Kounis

syndrome have been reported. Type 1 was defined as normal or nearly normal coronary arteries without predisposing coronary artery disease risk factors (5). Type 2 describes patients with nonsignificant coronary plaques in whom allergic mediators lead to coronary spasm or infarction. Type 3 was described as stent trombosis due to allergic mediators (6). According to this classification, our case was likely type 1 Kounis syndrome.

Underlying pathogenesis responsible for Kounis syndrome is the releasing inflammatory mediators such as histamine, platelet activating factor, tryptase, cytokines and prostaglandins which eventully lead to coronary artery spasm and ischemia (1). Kounis syndrome requires careful selection of various commonly used drugs. Morphine should be avoided because it may aggrevate histamine releasing. Beta-blockers may also cause coronary spasm if alpha adrenergic action is unopposed (6).

There are several reports citing diclofenac potassium-induced anaphylactic shock and acute coronary sydrome (7-10). De groot et al reported a case with both anaphylaxis and ST-segment elevational myocardial infarction induced by oral diclofenac intake in whom non-critical coronary plaques were detected at angiography (7). Çakar et al reported an elderly case who suffered an acute coronary syndrome after oral diclofenac intake and subsequently underwent coronary stenting (8). Tiwari et al reported a case who suffered acute coronary syndrome without anaphylactic shock induced by intarmuscular injection of diclofenac potassium in whom normal coronary arteries were found at angiography (9). Lastly, Colak et al reported a case with only anaphylactic reaction without acute coronary event due to intramuscular usage of diclofenac potassium (10). Unique to our case is that there were both anaphylaxis and ST-segment elevations induced by oral diclofenac potassium intake with normal coronary arteries without any further coronary intervention. Of above mentioned reports, two are from Turkey, none of which had any kind of allergic or immonological disorder. This may be a multifactorial issue including pollen exposure, undiagnosed autoimmun or allergic disordes or just a casual relationship.

CONCLUSION

Early recognition and treatment of this situation is of paramount importance and therefore clinicians should keep this sydrome in mind when managing an anaphylactic reaction with accompanying chest pain. After successful treatment of anaphylaxis, serial electrocardiograms should be planned. As mentioned before, in type 2 form of this syndrome, allergic reaction may trigger rupture of stable coronary plaque, at last myocard infarction may occur due to coronary flow cessation. Therefore physician should perform emergency coronary angiography when encounter this type of patient on emergency room. Clinicians should focus on removal of responsible allergens for secondary preventation.

The case report has written in an anonymous characteristic, thus secret and detailed data about the patient has removed. Editor and reviewers can know and see these detailed data. These data are backed up by editor and by reviewers.

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