CASE REPORT
OLGU SUNUMU

Kounis syndrome in a patient who developed contrast material allergy: A case report

Kontrast madde alerjisi gelişen hastada kounis sendromu: Olgu sunumu

İsmail Tekin[1], Gözde Yılmaz[2], İbrahim Tüysüz[2], Haticenur Yıldırım[2], Necmi Baykan[2]

[1]Primary Health Care Corporation, Qatar
[2]Emergency Department, Kayseri City Hospital, Kayseri, Türkiye

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Correspondence: Necmi Baykan
Seker Mah. Muhsin Yazıcıoglu Bulvari No:77 Kocasinan/Kayseri, Turkiye
e-mail: drnecmibaykan@gmail.com

ORCID ID: İT: 0000-0003-3405-9011
GY:0009-0003-0032-4398
İT: 0009-0003-8729-5437
HY:0009-0003-9990-6928
NB:0000-0002-6845-9550

SUMMARY

Kounis Syndrome is one of the consequences of allergic reactions that should be kept in mind as a frequent cause of emergency room visits. Anginal symptoms and ECG changes accompany this phenomenon. Eliminating exposure to the causative agent is the main treatment. In addition, a medication that will provide vasodilation of the coronary arteries and suppress the allergic reaction are also included in the treatment. With this case report, we aimed to draw attention to Kounis syndrome, which may be associated with a wide variety of clinical symptoms and may be overlooked, and to increase clinical awareness.

Keywords: Allergy, kounis syndrome, vasospasm

ÖZET

Kounis Sendromu, acil servise sık başvuru sebeplerinden biri olan alerjik reaksiyonların mutlaka akılda tutulması gereken sonuçlarındandır. Hastada anjinal semptomlar ve EKG değişiklikleri bu olaya eşlik eder. Neden olan ajana maruziyeti ortadan kaldırmak esas tedavidir. Bunun dışında koroner arterlerin vazodilatasyonunu sağlayacak ve alerjik reaksiyonu baskılayacak ilaçlar da tedavide yer alır. Biz bu olgu ile çok çeşitli klinik semptomlarla birlikte görülebilen ve gözden kaçabilecek bir tanı olan Kounis sendromuna dikkat çekmeyi ve klinik farkındalığı arttırmayı amaçladık.

Anahtar Kelimeler: Alerji, kounis sendromu, vazospazm

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INTRODUCTION

Kounis Syndrome is a rare allergic reaction resulting in coronary vasospasm and can occur in patients with or without coronary artery disease. This phenomenon was first reported by Dr. Kounis in 1991. The mechanism involves the release of various cytokines and chemokines such as histamine, tryptase, chymase, platelet activating factor, leukotrienes and prostaglandins as a result of mast cell degranulation, which may lead to vasoconstriction and coronary artery spasm (1). Kounis syndrome, which develops as a result of allergy secondary to contrast media, is a rare adverse reaction. The common result of many studies revealed that Kounis syndrome caused by contrast media is type I and II, which occurs mostly in elderly male patients (2). We think that as the awareness of Kounis syndrome, which is still considered to be a rare condition, increases, the rate of case reports will also increase. Consent was obtained from the patient for this case report.

CASE REPORT

A 42-year-old woman was admitted to the emergency department with complaints of dyspnea and nausea following the administration of contrast material (gadopentetic acid dimeglumine salt) during Magnetic Resonance Imaging (MRI). She had a history of Familial Mediterranean Fever and breast cancer. At the time of admission, blood pressure was 70/40 mm-Hg, pulse rate was 113/min, SpO2 was 98%, temperature was 36.4 °C, and finger tip blood glucose was 108 mg/dL. On physical examination, Glaskow Coma Score (GCS) was 15 points and the patient was oriented, coopere, neurologic examination was normal, had redness in places on her body and uvula edema was present. There were no rales on respiratory examination, but there was wheezing and rhonchi. Electrocardiography (ECG) and chest radiography were ordered in addition to laboratory tests. When her allergy was questioned, it was learned that she had no known allergy. 1 mg of adrenaline was immediately administered intramuscularly in the vastus lateralis region. The patient's control blood pressure was 97/58 mm-Hg, pulse rate was 126/min, SpO2 was 99%. Systemic antihistamines (45.5 mg pheniramine maleate), systemic steroids (80 mg methylprednisolone) and proton pump inhibitors (40 mg ezomeprazole) were administered. 1 mg of adrenaline was administered for the second time when there was no regression in uvula edema on control examination. Laboratory tests taken before the patient was given medical treatment revealed pH:7.41, lactate:6 mmol/L, WBC:11.82 10^3/µL, EO#: 0.59 10^3/µL, Troponin I: 0.1 µg/L and other laboratory values were within normal limits. ECG was consistent with sinus tachycardia and there were no acute pathologic findings on chest radiography (Figure 1,2). Approximately 20 minutes after intravenous contrast material was administered to the patient, who developed chest pain during follow-up, a control ECG was taken and control troponin was requested. On control ECG, ST segment elevation in leads D1 and aVL (Figure3) was present and cardiology was consulted. He was hospitalized with a diagnosis of acute lateral myocardial infarction and emergency coronary angiography was planned. Coronary angiography result was normal and medical treatment was organized. No cardiac complications (such as hypotension, ventricular fibrillation) developed in the patient's management. The patient was discharged in full recovery without any cardiac sequelae.

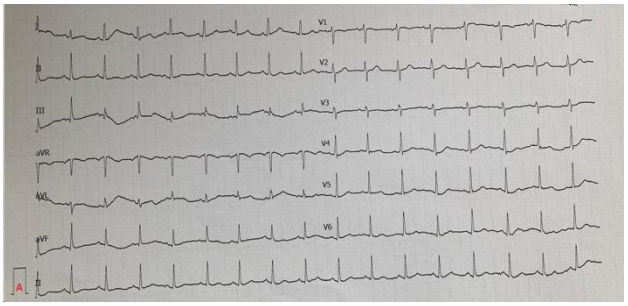

Figure 1. First ECG of the patient after admission.

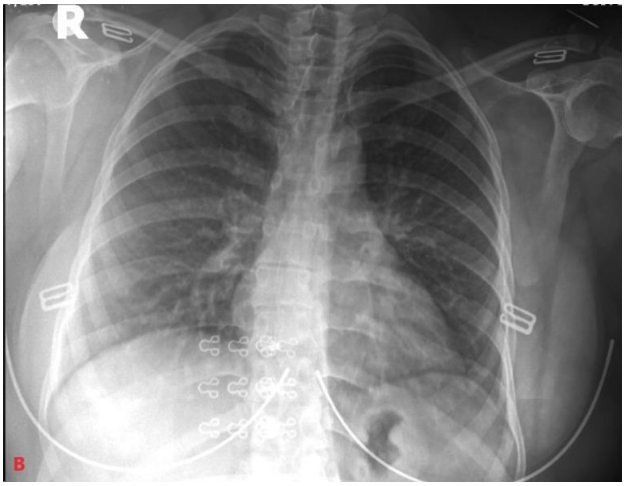

Figure 2. Chest radiograph of the patient on admission

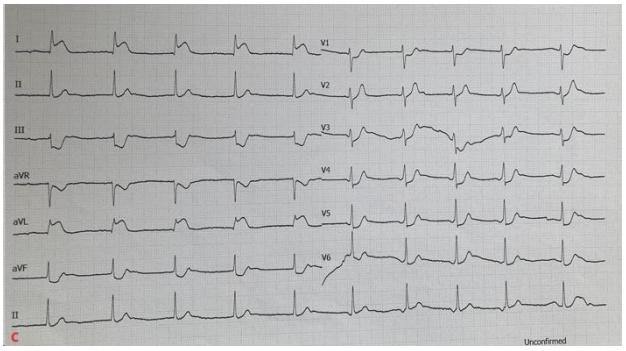

Figure 3. Control ECG of a patient with onset of chest pain

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DISCUSSION

Three types of Kounis Syndrome have been described. Type I occurs in patients with normal or near normal coronary arteries, without predisposing factors for coronary artery disease and in whom acute release of inflammatory mediators can lead to coronary artery spasm without any increase in cardiac enzymes or coronary artery spasm. It is the progression of cardiac enzymes or coronary artery spasm to acute myocardial infarction. Type II refers to patients with pre-existing atheromatous disease in whom acute release of inflammatory mediators can induce coronary artery spasm with normal cardiac enzymes or coronary artery spasm with plaque erosion orrupture that manifests as acute myocardial infarction. Type III includes patients with drug-eluting stent thrombosis with the presence of mast cells and eosinophils revealed by Giemsa and hematoxylin-eosin staining (3,4). The patient in this case had no known history of coronary artery disease or risk factors. Since coronary angiography revealed no vessel occlusion, the patient was diagnosed with type I Kounis syndrome. The right coronary artery is more susceptible to spasm after an allergic reaction; therefore, inferior myocardial wall involvement is observed more frequently in Kounis syndrome (5). Unlike the literature, lateral wall involvement was present in our case. A careful review of the clinical history of arterio sclerotic disease and allergic reactions is extremely important for appropriate treatment of different types of Kounis syndrome. In a study by Kei Shibuya et al, Kounis syndrome was associated with coronary arteriography (CAG) in five of eleven cases, and almost complete flow restoration was achieved with immediate primary stenting and/or intracoronary nitrate injection. In the remaining six patients, catastrophic events due to Kounis syndrome occurred in diagnostic radiology departments, as in our case. Three of these six patients had a history of coronary disease and were suspected of having type II Kounis syndrome. Two of these three patients underwent percutaneous coronary intervention, and the other was treated with heparin and nicorandil. The remaining three patients diagnosed with type I Kounis syndrome received anti-allergic or anaphylactic treatment, but two of these patients underwent CAG and normal findings were revealed in the coronary arteries (6). Similarly, in our case, the coronary angiography result was normal. Although almost all patients in previous case reports underwent CAG, the indication for CAG still remains a challenge in the radiology department in preventing Kounis syndrome associated with intravenous contrast injection (6).

CONCLUSIONS

Kounis syndrome, which is an acute coronary disease associated with allergic or anaphylactic reactions, can be induced by intravenous contrast material injection (6). Physicians should be careful in this regard and should definitely keep this relatively rare diagnosis in mind.

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