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Etofenamat enjeksiyonu sonrası gelişen akut inferiyor miyokart enfarktüsü

Acute inferior myocardial infarction developed after injection of etofenamate

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Özet– Alerjik semptomların miyokart iskemisi semptomlarına eşlik etmesi Kounis sendromu olarak tanımlanır. Etofenamat sık olarak kullanılan güvenli ve etkin bir steroid olmayan antienflamatuvar ilaçtır. Bu yazıda Kounis sendromlu 71 yaşında erkek hasta sunuldu. İntramüsküler 1 gram etofenamat enjeksiyonu sonrası eritamatöz döküntüler, kaşıntı, bulantı ve kusma, baş dönmesi, terleme ve göğüs ağrısı gelişen hasta acil servise kardiyopulmoner arrest olarak getirildi. On dakika süren başarılı yaşama döndürme sonrası elektrokardiyografide akut inferiyor miyokart enfarktüsü örneği saptandı. Acil servise alerjik semptomlara eşlik eden göğüs ağrısı ile başvuran hastada alerjik miyokart enfarktüsü akla gelmelidir. Bu hastalarda akut koroner sendromu ekarte etmek için elektro-kardiyografi mutlaka çekilmelidir.

Summary– Allergic symptoms accompanied by myocardial ischemic symptoms are defined as Kounis syndrome. Etofenamate is a safe and effective non-steroidal antiinflammatory drug that has widespread utilization. We hereby present a 71-year-old man with Kounis syndrome. Following intramuscular 1 g etofenamate injection, the clinical presentation when admitted to the emergency department (ED) was erythematous rash, pruritus, nausea and vomiting, dizziness, diaphoresis, and chest pain resulting in cardiopulmonary arrest. After 10 minutes of successful cardiopulmonary resuscitation, the electrocardiogram revealed acute inferior myocardial infarction. In patients who are admitted to the ED with allergic symptoms accompanied by chest pain Kounis syndrome should be considered for prompt management. Electrocardiographic exami-nation should be an essential part of the initial evaluation in such patients.

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Abbreviations:

ACS Acute coronary syndrome
MI Myocardial infarction
CK Creatine kinase
CKMB creatine kinase MB-fraction

Allergic myocardial infarction was defined in 1991 by Kounis and Zarvas as a type of angina pectoris or allergic MI syndrome in which inflammatory mediators released from allergic reaction play a role. In allergic angina syndrome also defined as 'Kounis syndrome' allergic symptoms, and also myocardial ischemia findings are detected. Activation of mast cells suggestively plays a role in the pathophysiologic mechanism of the event.[1,2] Previously, cases with allergic acute syndrome (ACS) have been reported related to many drugs used in daily life such as antibiotics, analgesics, antineoplastic drugs, contrast materials, and also various conditions as food allergy, asthma, angioedema, ant bites, bee sting, and snake bites, exposure to latex and ivy leaves.[3] Case reports are related to various age groups including pediatric age group.[4]

Herein, a case of acute inferior MI developed in a 71-year-old male patient following etofenamate injection was presented.

CASE PRESENTATION

Nearly half an hour after 1 g IM etofenamate injection, a 71-year-old male patient consulted to a county state hospital with complaints of generalized erythematous rash all over his body, lassitude, dizziness, shortness of breath, feeling of tightness on his chest, nausea-vomiting, general decline in his health state. His complaints were not relieved, on the contrary his health state deteriorated after administration of an antihistaminic, and a steroidal drug. Then he was referred to our hospital. His electrocardiogram (ECG) obtained in the county state hospital revealed a heart rate with a normal sinus rhythm. He developed cardiorespiratory arrest on admittance to the emergency service of our hospital, and after 10 minutes of successful cardiopulmonary resuscitation his ECG revealed atrial fibrillation pattern, and a nearly 4 mm-ST-segment elevation in DII, DIII, and aVF. (Figure 1). He received 300 mg oral acetylsalicylic acid. Dopamine infusion was started for his hypotension at a dose of 15 μg/kg/min. In the emergency department he had suffered from four episodes of ventricular fibrillation which necessitated electroshock therapy with biphasic, direct current (DC) using 200 J, and antiarrhytmic treatment was initiated (lidocaine infusion: loading dose of 1 mg/kg, and then maintenance dose of 2mg/min; amiodarone infusion: loading dose of 200 mg, and then maintenance doses of 1mg/min for 6 hrs, followed by 0.5 mg/min for 18 hrs). An intratracheal respiratory tube was inserted, and he was connected to a mechanical ventilation device. Afterwards, he was hospitalized in

our cardiology clinics, and heparin infusion was started (loading dose: 80 U/kg, and maintenance dose 60 U/kg) so as to attain an activated partial thromboplastin time (aPTT) of 50-70 msec. A nitroglycerine preparation was not given because of the presence of inferior MI, and hypotension. Biochemical test results demonstrated increments in cardiac enzymes (troponin I, CK, CKMB) and in compliance with the diagnosis of MI, their levels peaked and then declined sharply. His hematological test results were as follows: WBC: $14.6x10^3/\mu l$, eosinopohils: $0.1x10^3/\mu l$, and basophils: $0.7x10^3/\mu l$. IgE, and tryptase levels could not be assessed in our hospital laboratory. On echocardiograms, an ejection fraction of 50 %, and hypokinetic left ventricular inferior, lateral, and apical segments were noted. On electrocardiographic follow-ups, abnormal Q waves were observed in D II, D III, and aVF. On the 2. day of his hospitalization inotropic support was discontinued, and his breathing tube was removed. Then he was discharged on the 10. day of his hospitalization because of his concomitant diseases. Based on the information obtained from his intimates, he had been given frequent doses of analgesics, and non-steroidal antiinflammatory drugs (NSAIDs) (i.e. etofenamate injections) because of his pains secondary to his lumbar discal hernia. His medical history revealed presence of hypertension, diabetes, chronic bronchitis, nearly 40 –pack-years of smoking, and his coronary angiographic records displayed noncritical narrowings of the coronary arteries. The patient was discharged because of his expressed disapproval of the recommended coronary angiographic examination.

DISCUSSION

Kounis syndrome is a life-threatening condition with its induced allergic reactions, and ACS manifestations. In a patient with allergic reactions, and clinical symptom of ACS, Kounis syndrome should be taken into consideration.[5] Histamine, and leukotriene released when the patient is exposed to an allergen can induce allergic reactions, and spasmodic contractions of coronary artery smooth muscles. Resultant release of proteases such as tryptase, and chymase is thought to cause plaque erosion, and rupture.[6] Although skin reactions caused by etofenamate have been reported in the literature, the drug is recognized as a safe, and an effective NSAID.[7]

Previously, Kounis syndrome had been classified as 2 types in the literature, however in recent years a third type has been defined. In Type I Kounis syndrome any underlying cardiac abnormality does not exist. Coronary vasospasm develops in normal, and intact coronary arteries. In Type II, an underlying atherosclerotic heart disease exists. Vasospasm, allergic reaction, and released mediators induce erosion, and eventually rupture of the

plaque leading to the sudden emergence of acute MI.[2] In recent years, stent thrombosis developed with manifestations of allergic reactions especially following implantation of drug eluting stents is termed as Type II Kounis syndrome.[8]

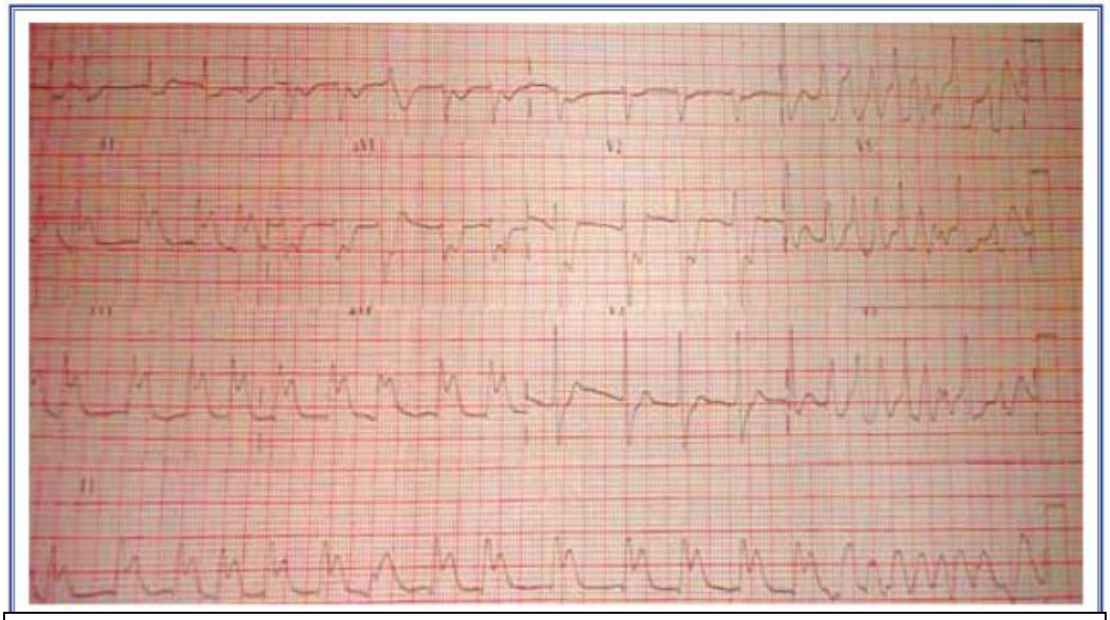

Figure 1. Electrocardiogram demonstrating tracings of an episode of atrial fibrillation. Consistent with the diagnosis of acute myocardial infarction nearly 4 mm-ST-elevation, and ventricular fibrillation were observed in DII, DIII, and AVF

Even though, cases coursing with myocardial ischemia induced by various allergenic agents have been reported so far, we haven't encountered any case of Kounis syndrome triggered by etofenamate in our literature review. Manifestations of our patient who had been angiographically diagnosed as atherosclerotic heart disease fits in the definition of Type II Kounis syndrome.

As is the case in many drugs, life-threatening conditions like MI can develop related to NSAIDs frequently used in daily life. ACS should be considered among potential complications which can develop when using NSAIDs. Even more importantly, electrocardiograms should be obtained, and allergic MI should be ruled out in patients who consult to the emergency service with allergic manifestations accompanied by symptoms resembling especially chest pain, and angina pectoris.

Conflict of Interest: None declared

REFERENCES

1. Kounis NG, Zavras GM. Histamine-induced coronary artery spasm: the concept of allergic angina. Br J Clin Pract 1991;45:121-8.
2. Nikolaidis LA, Kounis NG, Gradman AH. Allergic angina and allergic myocardial infarction: a new twist on an old syndrome. Can J Cardiol 2002;18:508-11.
3. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): a natural paradigm? Int J Cardiol 2006;110:7-14.
4. Biteker M, Duran NE, Biteker F, Civan HA, Gündüz S, Gökdeniz T, et al. Kounis syndrome: first series in Turkish patients. Anadolu Kardiyol Derg 2009;9:59-60.
5. Cevik C, Nugent K, Shome GP, Kounis NG. Treatment of Kounis syndrome. Int J Cardiol 2010;143:223-6.
6. Kounis NG, Grapsas ND, Goudevenos JA. Unstable angina, allergic angina, and allergic myocardial infarction. Circulation 1999;100:e156.
7. Guevara-López U, Uscanga-Sánchez S, Márquez J, Bárcenas-Olivares J, Martínez-Arenas A, Palma-Aguirre JA. Comparative clinical multicenter study to evaluate analgesic effectiveness of intramuscular etofenamate and diclofenac in patients with post-surgical pain. Cir Cir 2004;72:483-90.
8. Biteker M. A new classification of Kounis syndrome. Int J Cardiol 2010;145:553.

Anahtar sözcükler: Anjina pektoris; kardiyovasküler hastalıklar; koroner vazospazm; miyokart enfarktüsü.

Key words: Angina pectoris; cardiovascular diseases; coronary vasospasm; myocardial infarction.