



Kounis Syndrome as a Result of Anaphylactic Reaction to Gold Dust: A Case Report

Öner Bozan, Asım Kalkan, Şeref Emre Atış, Bora Çekmen, Adnan Türk

Istanbul Okmeydanı Training and Research Hospital, Clinic of Emergency Medicine, Istanbul, Turkey

Abstract

Kounis syndrome (KS) is described as the provocation of an acute coronary syndrome through activation of mast cells resulting in allergy, hypersensitivity, anaphylaxis, or anaphylactoid reaction. The case is here presented of a patient who developed KS with retrosternal chest pain and ST-segment elevation myocardial infarction 30 minutes after the ingestion of gold dust. A 40-year-old male without a history of allergy or atopy was admitted to the emergency room with complaints of red, itchy skin, chest pain, and shortness of breath 30 minutes after exposure to gold dust. Arterial blood pressure was 129/72 mmHg, pulse rate was 94 bpm, and oxygen saturation was 98%. On physical examination, urticarial lesions were observed on the anterior chest wall without uvula edema and pulmonary bronchospasm. Electrocardiogram (ECG) showed ST elevation in the V1-V6 and aVL leads. High sensitivity Troponin I level was measured as 12.1 ng/L (reference=0-19.8 ng/L), and the Troponin I levels increased to 229.7 ng/L two hours later. KS was considered because of the findings of chest pain, urticaria on the body, and lack of cardiac disorder. KS should be kept in mind in cases that present with allergic reactions together with chest pain following exposure to agents to which the immune system may be allergic. Cardiac enzymes can either be normal or elevated in these patients. ECG usually has ST, and the T wave changes.

Keywords: Kounis syndrome, anaphylactic reaction, gold dust

INTRODUCTION

Kounis syndrome (KS) is described as the provocation of an acute coronary syndrome (ACS) by the activation of mast cells resulting in allergy, hypersensitivity, anaphylaxis, or an anaphylactoid reaction. Drugs, food, environmental factors (insect bite, bee sting, pollens, latex contact), and intracoronary stent placement can be there as on which trigger the allergic reaction (1). In this paper, the development of KS with retrosternal chest pain and ST segment elevation myocardial infarction after 30 minutes following the ingestion of gold dust exposure is discussed.

CASE REPORT

A 40-year-old male without a history of allergy or atopy was admitted to the emergency room with complaints of red, itchy skin, chest pain, and shortness of breath 30 minutes after exposure to gold dust. Arterial blood pressure was 129/72 mmHg, pulse rate 94 was bpm, and oxygen saturation was 98%. On physical examination, urticarial lesions were found on the

patient's anterior chest wall without uvula edema and pulmonary bronchospasm (Figure 1). His electrocardiogram (ECG) showed ST elevation in V1-V6 leads (Figure 2). High sensitivity Troponin I level was measured as 12.1 ng/L (reference=0-19.8 ng/L), and the Troponin I levels increased to 229.7 ng/L two hours later. KS was considered because of the findings of chest pain, urticaria on the body, and lack of cardiac disorder. The patient was consulted with cardiology. Echocardiography showed segmental cardiac wall abnormalities with 50% ejection fraction. Coronary angiography revealed no sign of atherosclerosis. The patient was diagnosed to have a KS type 1 variant, secondary to gold dust ingestion.

DISCUSSION

Allergic events can cause not only angina episodes but also acute myocardial infarction (2,3). KS was first described in 1991 and has a clinical spectrum ranging from chest pain with an acute or chronic allergic reaction to acute myocardial infarction (1).



Address for Correspondence: Öner Bozan, Istanbul Okmeydanı Training and Research Hospital, Clinic of Emergency Medicine, Istanbul, Turkey
Phone: +90 505 512 15 00 E-mail: onerbozan@gmail.com ORCID ID: orcid.org/0000-0002-4195-2601

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Figure 1. Patient's rash on anterior thorax and abdomen

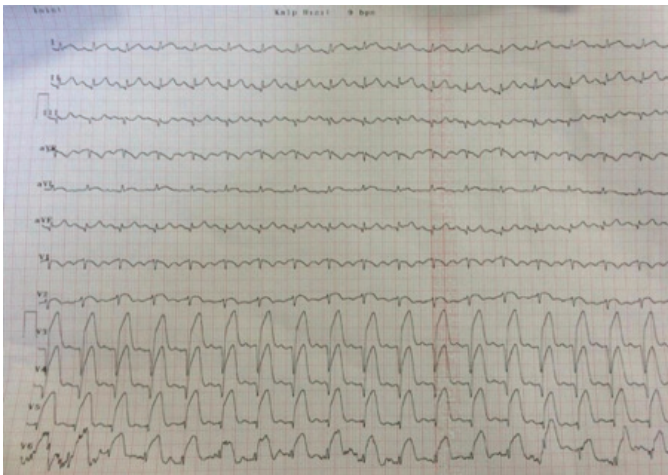


Figure 2. Patient's initial electrocardiogram when admitted to hospital
Vasospasm of the coronary arteries has been suggested to be the main pathophysiologic mechanism (4). In 1998, Braunwald (5) reported that allergic reactions could induce vasospastic angina with mediators such as histamine and leukotrienes acting on coronary vascular smooth muscle. Two types of KS have been described (6). In type 1 variant, patients have normal coronary arteries. An acute allergic event induces coronary artery spasm, resulting in chest pain and ischemic electrocardiographic changes. Cardiac enzymes can either be normal or elevated, which reflects progression to an acute myocardial infarction (2).

The mechanism responsible for this type would be endothelial dysfunction or microvascular angina (7). In type 2, patients have underlying coronary artery disease, and chest pain occurs during an acute allergic reaction (8). An acute allergic episode can induce plaque erosion or rupture manifesting as an acute myocardial infarction (2,9). We know that several antibiotics have been associated with allergic reactions and KS. An ACS with ST elevation after exposure to amoxicillin was reported by Vivas et al. (3). In the present case, the allergic reactions resulting in chest pain were seen 30 minutes after exposure to gold dust. According to our knowledge, this is the first case with gold dust induced by KS.

The primary treatment of KS is the management of ACS and regression of the allergic reaction. The regression of symptoms of the allergic reaction with steroids and antihistamines may be enough to resolve coronary vasospasm (10).

CONCLUSION

KS should be kept in mind in cases that present with allergic reactions along with chest pain following exposure to agents that may be allergic to the immune system. Cardiac enzymes can either be normal or elevated in these patients. ECG would have ST and T wave changes, which would be normal.

Ethics

Informed Consent: Was obtained.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Ö.B., A.K., Concept: Ş.E.A., Design: Ö.B., A.K., Data Collection or Processing: Ö.B., A.T., Analysis or Interpretation: Ş.E.A., Literature Search: B.Ç., A.T., Writing: A.K., Ş.E.A.

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