Case Report

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Kounis Syndrome That Recurs in A Short Time Period: A Case Report

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Abstract

This case report is unique for the occurrence of a quickly recurring Kounis Syndrome (KS) due to re-exposure to the same agent. A 40-year-old male was brought to our ED with a diagnosis of non-ST-segment elevation myocardial infarction. He stated that he had taken one dose of amoxicillin-clavulanate 1,000 mg at 03.00 AM. After taking the drug, chest pain and vomiting began. ECG which was taken at the the rural hospital, revealed a normal sinus rhythm with no ischemic changes. The value of cardiac troponin I 0.34 ng/ml in the rural hospital. The patient was consultated to the cardiology clinic with a pre-diagnosis of KS. Percutaneous coronary intervention showed that coronary arteries were normal and no plaque formation was found. The patient, who was diagnosed with type I KS, left the hospital at his own request at 14:12 PM. The patient presented to our ED again at 22:30 PM with chest pain and shortness of breath after accidentally using the same allergenic drug ~eight hours after leaving our hospital. ECG showed > 0.5 cm ST-segment elevation in leads DII, DIII, and aVF. Quickly recurring KS was due to accidental reuse of the same agent may be more severe than the first occurrence.

Keywords: Kounis syndrome, acute coronary syndrome, allergic infarction, recurring Kounis syndrome

Introduction

Kounis syndrome (KS) is defined as the concurrent occurrence of an acute coronary syndrome (ACS) with a hypersensitivity reaction (1, 2). KS is an entity that develops coincidentally after exposure to allergen, that can affect all races and age groups and that can be seen in many geographical regions (3). Food, drugs and especially environmental agents are frequently encountered etiological causes of this entity (4). When the high risk of causing allergic reactions is considered, non-steroidal anti-inflammatories and antibiotics are the drugs that are most frequently associated with this syndrome (5).

Kounis syndrome is a lesser-known entity (3). However, in the literature, there are many cases of KS that have developed due to different agents. But 'recurrent' KS that has developed as a result of recurrent use of the same agent has been seen quite rare (5). In this case report, we aimed to present a recurrent case of KS with two different types of ACS, which developed in a short time after reuse of the same drug.

Case Report

A 40-year-old male patient was brought to our emergency department (ED) by an ambulance from a rural hospital with a diagnosis of non–ST-segment elevation myocardial infarction (NSTEMI). The past medical history of the patient was unremarkable in terms of coronary disease risk. There was no known allergy history. The patient stated that he had taken one piece of the prescribed medicine containing Amoxicillin-Clavulanate 1000 mg (Klavon 1000 mg - Husnu Arsan Drug Co., Turkey) at 03.00 AM. He went to the hospital with complaints of chest pain and vomiting which had started 10 minutes after the drug intake. He described his pain in the form of pressure, starting from the retrosternal region and spreading to the left arm and chin. The chest pain was accompanied by nausea, vomiting, and abdominal pain. Vital findings were recorded as follows: blood pressure 81/55 mmHg and pulse 106/min. His physical examination was unremarkable. The electrocardiogram (ECG) taken at the 15th minute of his admission revealed a normal sinus rhythm with no ischemic change. The value of cardiac

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troponin I (cTnI) was determined as 0.34 ng/ml (reference < 0.3 ng/ml) in the rural hospital. Other laboratory tests were within normal limits.

The patient was admitted to our ED as NSTEMI around 06.00 AM. He had no complaints on admission. The vital signs and physical examination were normal. No significant ischemic change was observed in his ECG (Figure-1). The cTnI values studied in our ED were above the normal limit (2.96 ng/ml). Therapy for ACS was started in the ED. The present status of ACS in the patient was associated with anaphylaxis that had developed due to drug use. The patient was consulted to the cardiology clinic with a pre-diagnosis of KS. The coronary arteries were considered normal in percutaneous coronary intervention (PCI). No plaque formation was found. The patient, who was diagnosed with type I KS, left the hospital at his own request while he was on follow-up (14:12 PM).

The patient re-presented to our ED at 22.30 PM, complaining of chest pain and shortness of breath. He had accidentally used the same allergenic drug once again about 8 hours after leaving the hospital. He stated that his complaints had started shortly after the drug use. The chest pain was similar to his previous pain. But the patient stated that the pain was more severe. The vital signs and physical examination findings were normal. His ECG showed > 0.5 cm ST-segment elevation in leads DII, DIII, aVF, and > 0.5 cm reciprocal ST-segment depression in leads DI, aVL (Figure-2). The cTnI value was 9.12 ng/ml. Treatment for anaphylaxis and ACS was started in the patient who was considered to have KS and an associated STEMI. He was discharged from the cardiology clinic after a 3-day follow-up and treatment and was informed about the need to avoid drugs containing amoxicillin-clavulanate.

Discussion

There is a quite wide literature on KS and each new case opens up new horizons for allergens that play a role in aetiology. This case, reported by us, can be considered quite ordinary if considering only the etiological agent (6). However, what makes our case special is that it is a recurrent KS that recurs in a very short time due to re-exposure to the same agent. Recurrent KS that has developed due to the same agent is a quite rare condition in the literature (5, 7, 8, 9, 10). KS with a quite short recurrence time is another important feature that differs from the literature in our case. Gunaydin et al. mentioned a case of KS that developed one year apart due to bee sting (7).

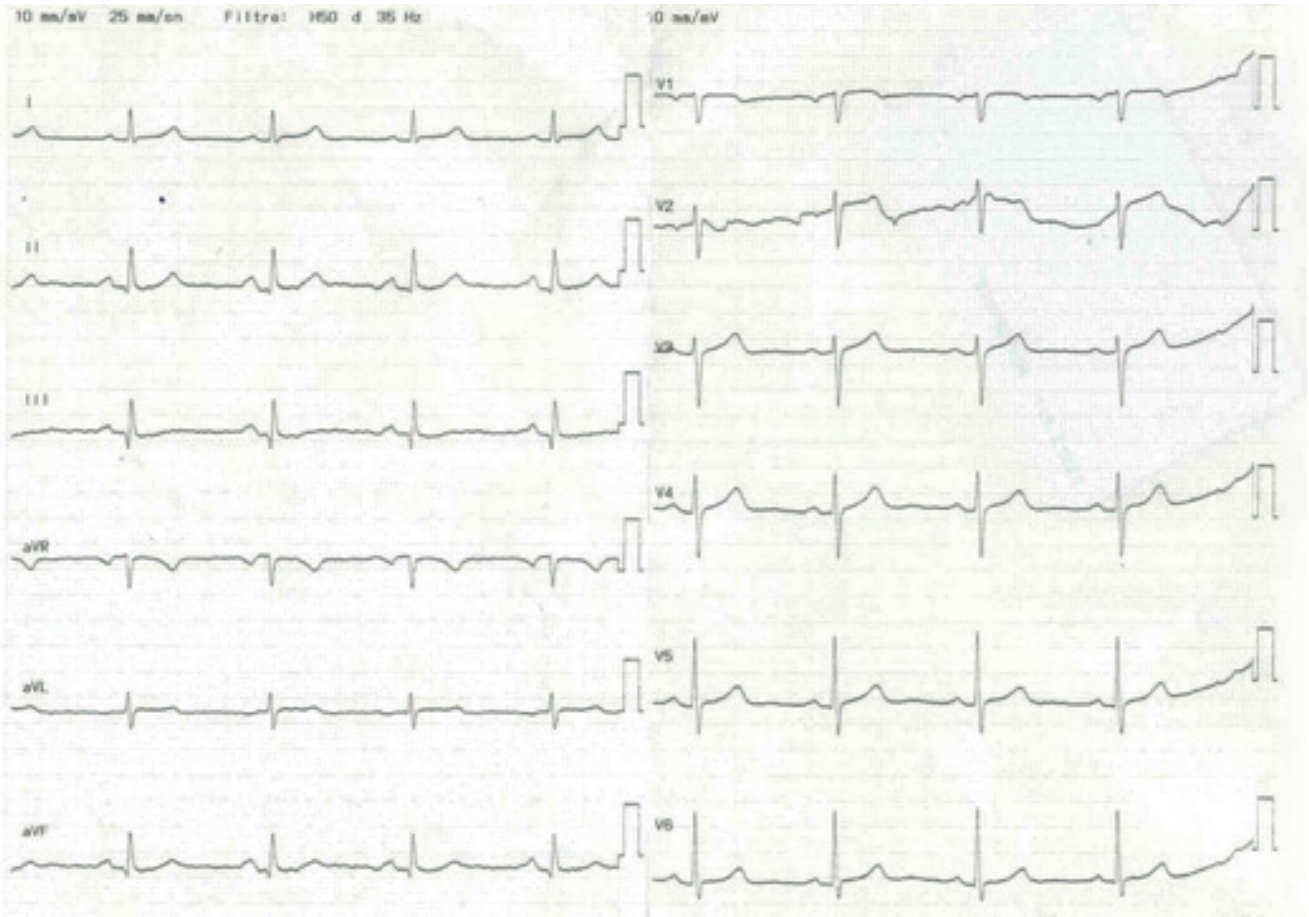

Figure 1. Initial ECG in ED revealed no significant ischemic changes

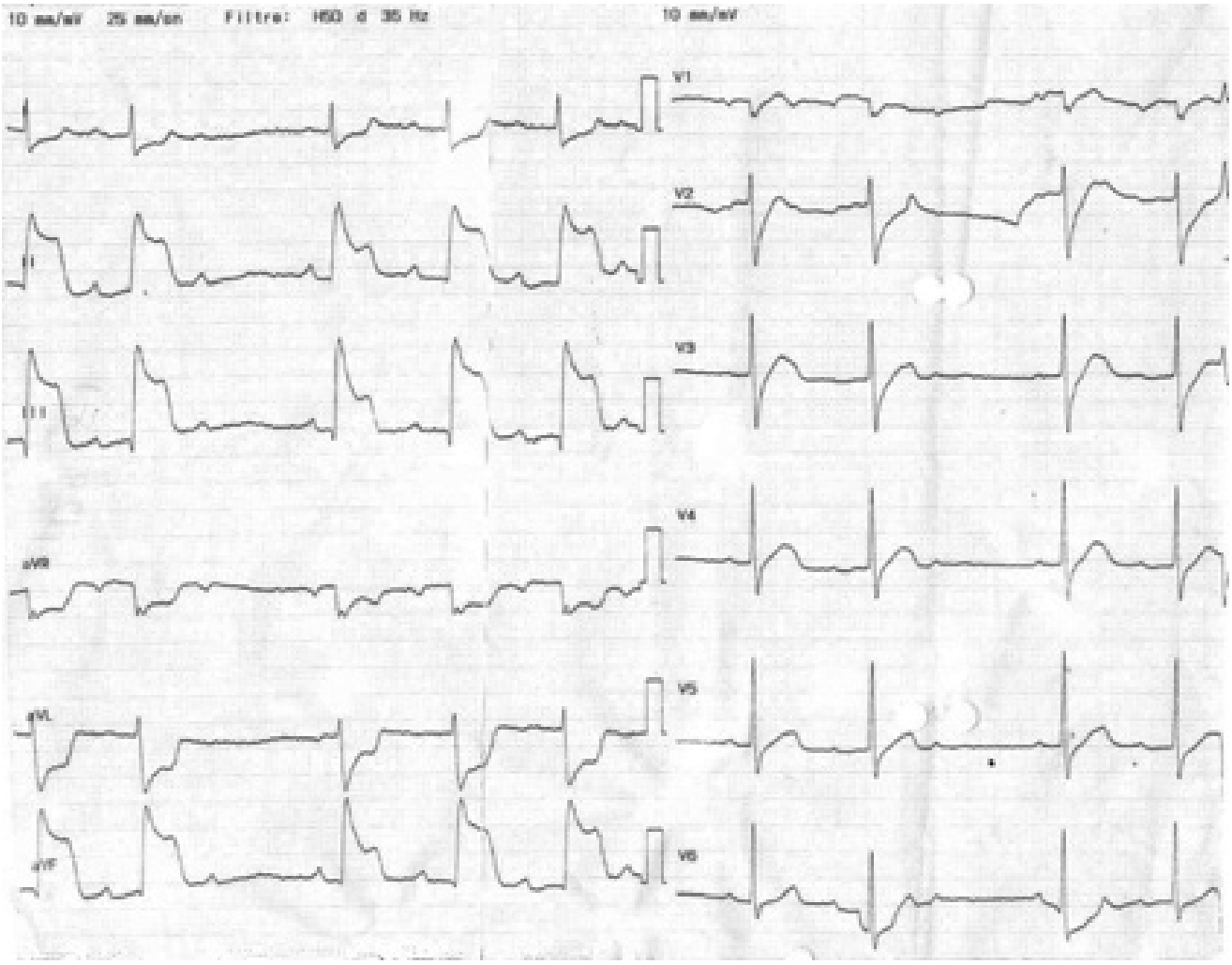

Figure 2. ECG showed > 0.5 cm ST-segment elevation in leads DII, DIII, and aVF and > 0.5 cm reciprocal ST-segment depression in leads DI and aVL

Celiker et al. reported a case of KS that occurred at 8-months interval due to pseudoephedrine use (5). In another case report, a case of KS that developed due to general anaesthetics that were given at 4-week intervals was reported (9, 10). Patane et al. presented a case of allergy and allergy-related ACS as a result of clopidogrel use. In this case, recurrence developed as a result of reuse of the same drug after three days (8). In our case, KS recurrence time is approximately 18 hours. To the best of our knowledge, this time period is much shorter than those so far reported in other cases.

KS can be presented with many different clinical pictures. However, this syndrome should definitely come to mind in the presence of allergic symptoms accompanying chest pain (11, 12). The clinician's suspicion is the most important step in diagnosis. Diagnosis begins with a detailed medical history. It is confirmed based on symptoms and signs, ECG changes, laboratory findings, echocardiography, and angiography at the timed of admission (4, 5). In our case, both the medical history and accompanying ECG and laboratory findings were the clues that allowed us to think about KS in preliminary diagnosis. The most important point that should not be forgotten in a diagnosis is that clinical findings may not always reflect KS fully. While silent angina may be seen in some patients, in others, as in our case, the clinical manifestations of allergy may not be clear (2). In some cases, the presence of hypotension without skin manifestations may be the only clue in the diagnosis of anaphylaxis (7).

Three different types of KS have been described: Type 1 coronary vasospasm, Type 2 native plaque destabilization and Type 3 stent thrombosis (7, 12). In the type I variant, there is no risk factor for coronary artery disease and the coronary arteries are angiographically normal. Coronary vasospasm developing due to an allergic reaction is responsible for the disease. If vasospasm progresses when cardiac enzymes are normal, it may increase due to myocardial damage (2, 7, 13). Our case also had no risk factors for AKS and the coronary angiography was normal. Therefore, the case was considered as KS Type 1 variant. In addition, our case was also compatible with the literature, which states that KS Type 1 is more common in cases of KS induced by amoxicillin than other variants (14).

ECG changes in KS can range from ST segment elevation to depression, and from any degree of heart block to cardiac arrhythmias (15). The first admission ECG of our

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case was normal, however, ECG showed elevation of the ST segment in the inferior leads in the second admission. Although it is thought that the clinical status in the second reaction to the allergen will be worse, it can be is seen that the clinical symptoms are similar in recurrent KS cases in the literature (5, 8, 10). However, since this situation may not be valid for all cases, these patients who develop KS should be sufficiently informed and the list of drugs that are to be avoided should be stated.

As a result, KS can be seen for the second time in a very short time due to the accidental use of the same agent.

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