

A Rarely Seen Type 1 Kounis Syndrome Caused By Tetanus Vaccine

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ABSTRACT

The pathogenesis mechanism of the syndrome is defined by mast cell degranulation that has been amplified to induce acute myocardial infarction in susceptible individuals, which is termed as Kounis syndrome. Herein, we reported a case of Kounis syndrome presented with acute coronary syndrome after a tetanus vaccine. Additionally, all possible other etiologies, especially ischemia were excluded.

Keywords: Allergic myocardial infarction, Kounis syndrome, tetanus vaccine

Oldukça Nadir Görülen Tetanoz Aşısının Neden Olduğu Tip 1 Kounis Sendromu

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ÖZET

Patolojik mekanizması mast hücrelerinin degranülasyonuna bağlı hassas kişilerde görülen akut myokardiyal enfarktüsü Kounis sendromu olarak tanımlanır. Biz burada tetanoz aşısı sonrası akut koroner sendrom ile başvuran bir Kounis sendromu bildirdik. Ek olarak, tüm olası sebepler özellikle de iskemi dışlandı.

Anahtar Kelimeler: Allerjik kalp krizi, kounis sendromu, tetanoz aşısı

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INTRODUCTION

Acute coronary syndrome with the activation of mast cells is induced by hypersensitivity, or allergic and anaphylactic or anaphylactoid reactions have not frequently been reported. Firstly, Kounis explained it as "hypersensitive angina syndrome" advancing to "allergic myocardial infarction." Hence, it was referred to as "Kounis syndrome" recently [1, 2]. In this case, we reported a patient who developed Kounis syndrome following an allergic reaction to a tetanus vaccine. We also described its possible clinical implications and pathophysiological mechanisms.

CASE REPORT

A 35-year-old man was admitted to our hospital's emergency department because of a typical chest pain and moderate pruritic skin rashes. His symptoms had begun roughly half an hour in the wake of ingesting a tetanus vaccine for a minor injury. He was admitted to our department for duration of 45 minutes after the onset of the tetanus vaccine's side effects. Besides, he had no risk factor for coronary artery disease. While after the confirmation, his electrocardiogram demonstrated ST elevations in leads d-II, d-III, and aVF and reciprocal changes in anterior leads (V 1-4) which was reflecting inferior myocardial infarction (Figure1 A). Subsequently, the patient was taken to our coronary angiography unit. We gave 300 mcg intravenous glycerol trinitrate because of his chest pain before the coronary angiography. His chest pain was resolved after the glycerol trinitrate. However, the left and right selective coronary angiography was normal (Figure 2 A-B). Then we took him to our coronary care unit, and his electrocardiogram was normal (Figure 1B). Despite this, Troponin-I level was 27 ng/mL (reference esteem: 0.015 ng/mL) and peak Creatine kinase-MB fraction was 107 U/L at the peak of his subsequent period, individually. Additionally, a moderate increase in leukocyte ($15.4 \times 10^3/\mu\text{L}$) and eosinophil (4.9 %) were also observed. The immunoglobulin E level was significantly higher (180 mg/L) (reference esteem: 150 mg/L). According to these, an allergic reaction was thought since his chest pain started immediately after a tetanus vaccine. Therefore, the patient received an oral antihistamine therapy as a measure against allergic reactions. Lastly, the patient was discharged on the fourth day of admission.

DISCUSSION

According to our understanding, this case seems very rarely Kounis syndrome induced by a tetanus vaccine in the literature. Kounis syndrome, also known as allergic myocardial infarction, has three types. In the first category, patients display coronary vasospasms actuated by mediators of allergic reaction, for example,

histamine, thromboxane, and leukotrienes without the availability of risk factors that causes atherosclerosis or coronary artery syndrome. While in the second type, there is an acute coronary disorder, which arises because of coronary vasospasms, plaque disintegration, or rupture of plaque incited by these arbiters in patients with atherosclerotic coronary artery syndrome. It is also a fact that thrombus material contains eosinophil and mast cells extracted from several patients who have stent thrombosis after the stent implantation with medication discharge that makes us consider excessive hypersensitivity responses in those patients individually. Besides, this circumstance is acknowledged as a type III variation of Kounis Syndrome [3-5]. Hence, based on these findings, our case is as per the type I Kounis syndrome. Increased degranulation of mast cell which induces coronary artery spasm, as well as myocardial infarction, was defined as the primary pathophysiological mechanisms of Kounis syndrome. The degranulation of mast cells particularly is going to have an effect on people who are more vulnerable to coronary artery spasm. On the other hand, during the mast cell degranulation, a few vasoactive molecules such as leukotrienes, serotonin and histamines, and collagen-degrading compounds such as neutral proteases are increased in the peripheral circulation. All these mediators can cause a vasospasm in the coronary arteries. Additionally, the platelets which trigger thrombosis are activated by Histamine. This mediator can also contribute to the progressing of acute coronary syndrome by initiation and provocation of plaque erosion, rupture or coronary vasospasm [6, 7]. In this case, the patient's coronary arteries were completely normal, and this might make us think that a coronary vasospasm was the reason that came into existence due to the release of mediators by tetanus vaccine. The primary cardiovascular effects of coronary vasoconstriction are plaque erosion, thrombocyte activation, dysrhythmia development, induced by various mechanisms and increment in the synthesis of tissue factor [8]. However, a patient who is suffering from Kounis syndrome, in addition to appropriate acute coronary syndrome management, the determination of specific IgE antibodies, eosinophilia, serum histamine, and complement proteins help in the identification of this disease [9]. Moreover, eosinophil, total IgE, and leukocyte levels were increased in our patient. It can be concluded that the vaccination of tetanus can be a core reason for acute coronary syndrome. Clinical findings and laboratory diagnosis might provide a suggestion that Kounis syndrome should be taken into consideration while diagnosing acute coronary syndromes. This case shows the importance of clinical knowledge of acute coronary syndromes. The physicians need to be aware of this effect and take note of it in the diagnosis of myocardial infarction.

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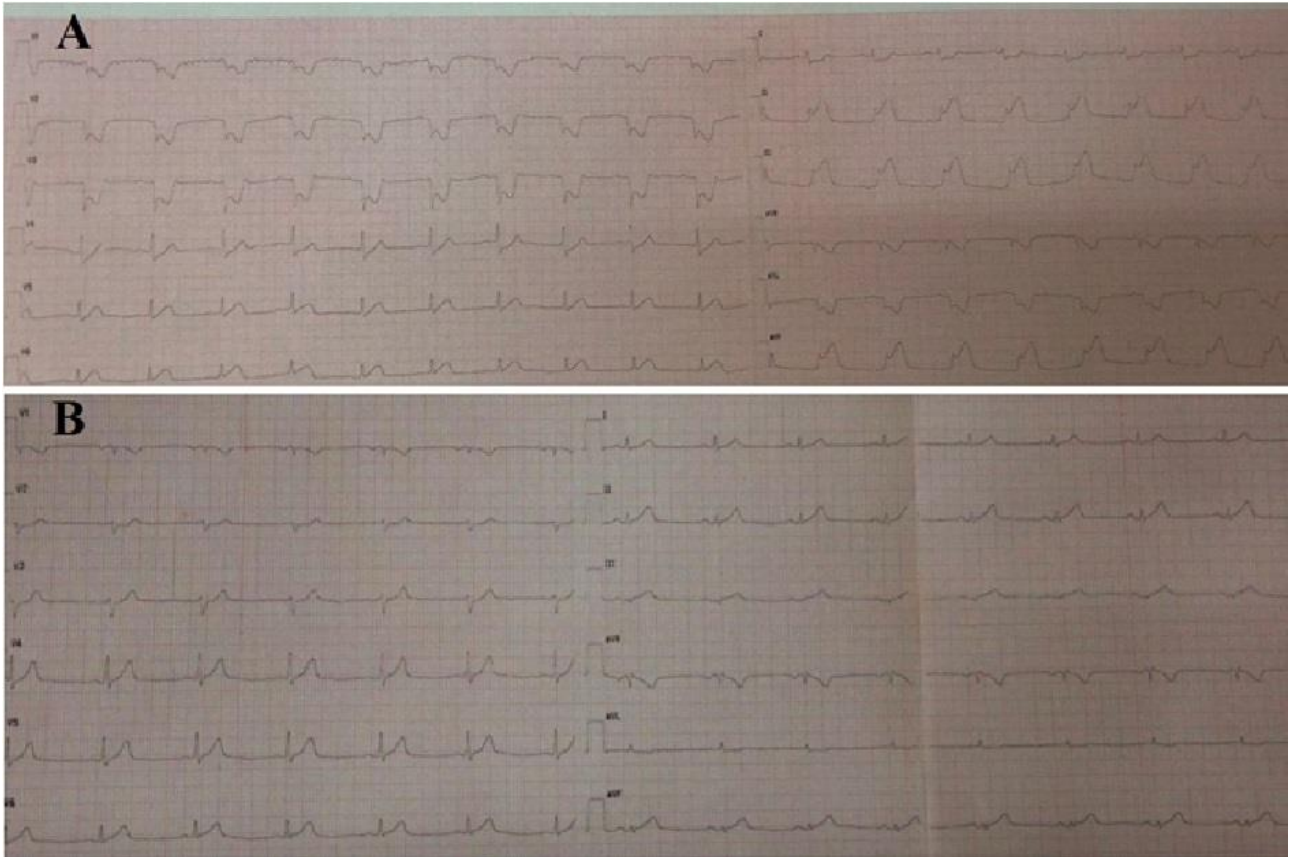
Figure Legends:

Figure 1: A: Electrocardiogram demonstrated ST elevations in leads d-II, d-III, and aVF and reciprocal changes in anterior leads (V 1-4), B: Normal electrocardiogram

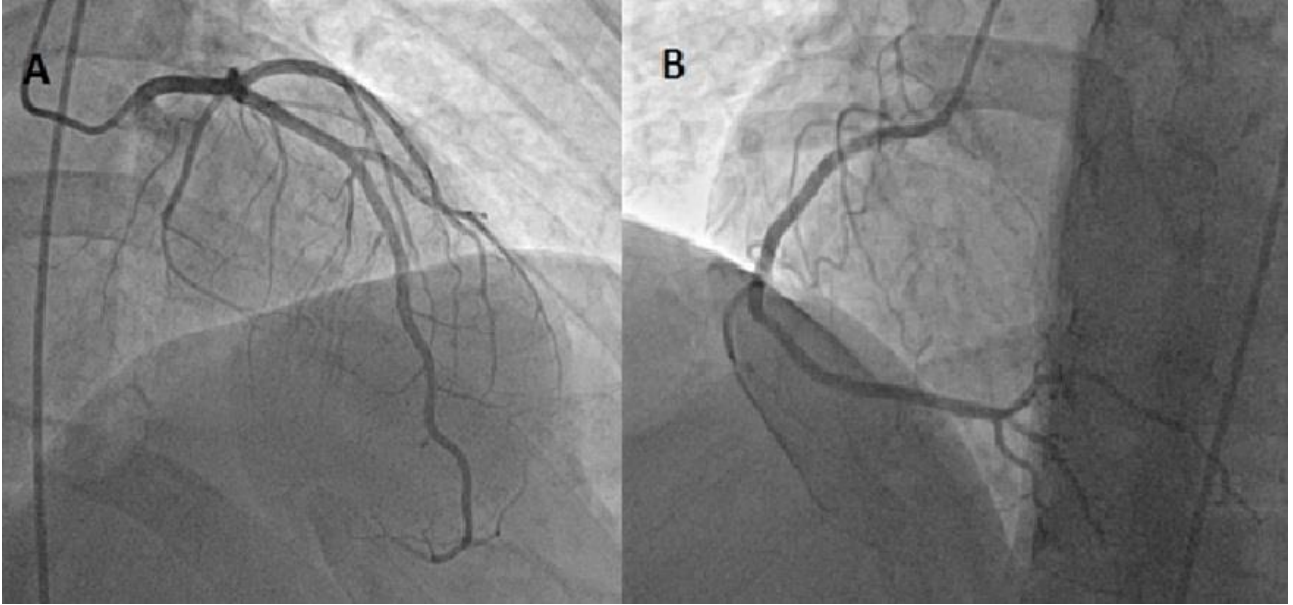


Figure 2: Normal coronary arteries, (A: left selective coronary angiography) (B: right selective coronary angiography)