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A case of Kounis syndrome induced by food allergies

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Abstract: A 78-year-old male felt abdominal discomfort and nausea after eating lunch with 350 ml of beer. He went to the toilet due to diarrhea and subsequently felt faintness and called his family with a handy-phone. When his family assessed him, he was found to be in an unconsciousness state. Upon arrival, he demonstrated disorientation with words, with a blood pressure of 76/42 mmHg, heart rate of 82 BPM, and displayed both erythroderma and dysarthria. A chest roentgenogram and electrocardiogram were all negative. He received a rapid infusion of 500 ml of lactate ringer, and his blood pressure showed a transient increase; however, reducing the speed of the infusion resulted in repeat hypotension. A repeat electrocardiogram indicated elevation of ST at the aVR lead. An urgent coronary angiogram showed mild diffuse spastic changes in both right and left coronary arteries, which resolved with the infusion of Nicorandil. As he remained in a hypotensive state with erythroderma, an anaphylactic reaction was suspected. Administration of Chlorpheniramine had a dramatic effect, and the patient demonstrated normo tension with a normal color skin. The troponin T level increased to 0.337 (0.032 >) ng/ml on the second hospital day. Finally, he received a diagnosis of coronary vasospasms secondary to food allergies. Special attention should be paid to anaphylactic complaints and the findings of real-time electrocardiograms, as well as the levels of cardiac markers, in order to ensure a correct diagnosis and a good outcome of treatment.

Keywords: food allergies; variant angina; Kounis.

INTRODUCTION

The onset of allergic angina and allergic myocardial infarction is termed Kounis syndrome [1, 2] most causative factors for Kounis syndrome are drugs. Rarely, insect stings, cold temperatures or foods may induce Kounis syndrome [3, 4]; we herein report a rare case of Kounis syndrome induced by food allergies.

CASE PRESENTATION

A 78-year-old male with hypertension, diabetes mellitus, gout and chronic renal failure felt abdominal discomfort and nausea after eating lunch with 350 ml of beer. He went to the toilet due to diarrhea after embarking on a ship, where he subsequently felt faintness and called his family with a handy-phone. When his family assessed him, he was found to be in an unconsciousness state and was therefore transferred to our hospital via a physicianstaffed helicopter. The patient had a habit of drinking approximately 1,500 ml of beer each day. Upon arrival, he demonstrated disorientation with words, with a blood pressure of 76/42 mmHg, heart rate of 82 BPM, respiratory rate of 16 BPM, 100% SpO2 under 10L/minute of oxygen delivered via a mask and body temperature of 38.2 Celsius. He displayed both erythroderma and dysarthria. A chest roentgenogram, electrocardiogram (Figure 1a), cardiac sonography scan

and whole body plain computed tomography scan were all negative. The main abnormal results of a biochemical analysis of the blood were as follows: white blood cell count of 10,800/µl, hemoglobin level of 10.5 g/dl, platelet count of 12.0 x 104 /µl, glucose level of 220 mg/dl, blood urea nitrogen level of 28.3 mg/dl, creatinine level of 1.66 mg/dl, C-reactive protein level of 0.3 mg/dl and troponin T level of 0.020 (0.032 >) ng/ml. As the patient exhibited hypotension with a normal cardiac function, he was thought to be in alcoholic intoxication with a vagal reflex due to an unknown cause. He therefore received a rapid infusion of 500 ml of lactate ringer, and his blood pressure showed a transient increase; however, reducing the speed of the infusion resulted in repeat hypotension. Electrocardiographic monitoring showed depression of the ST segment in the II lead, while a repeat electrocardiogram indicated elevation of ST at the aVR lead and depression of the ST segment at the II, III, aVF, V4, V5 and V6 leads, without any chief complaints. In addition, an urgent coronary angiogram showed mild diffuse spastic changes in both right and left coronary arteries, which resolved with the infusion of Nicorandil, following normalization of the electrocardiogram findings (Figure 2). The patient was treated with an indwelling intra-aortic balloon pump and admitted to the coronary care unit. As he remained

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in a hypotensive state with erythroderma, anaphylactic reaction suspected was and Chlorpheniramine was administered. This treatment had a dramatic effect, and the patient demonstrated normo tension with a normal color skin. Finally, he received a diagnosis of coronary vasospasms secondary to food allergies. The troponin T level increased to 0.337 (0.032 >) ng/ml on the second hospital day and subsequently decreased to the normal range the following day. The post-angiogram course was uneventful, and the patient was discharged on foot on the eighth hospital ~ day. 3. Discussion only a few cases of coronary syndrome induced by an anaphylactoid reaction resulting from a food allergy have been reported [5-11]. In the current case, the differential diagnosis was a combination of carcinoid syndrome and variant angina [12]. The present patient had no history of previous recurrent episodes of flushing, secretory diarrhea, bronchospasms or hypotension. In addition, CT did not disclose any abnormal masses. Accordingly, the possibility of carcinoid syndrome with variant angina was minimized.

Meanwhile, the onset of hypotension, erythroderma and diarrhea after meals and the subsequent resolution of these symptoms following the administration of a H1 blocker are compatible with a diagnosis of anaphylaxis induced by foods. The release of mediators during allergic insults has been demonstrated to induce coronary artery spasms and/or atheromatous plaque erosion or rupture. Clinically, Kounis syndrome is characterized by the concurrence of acute coronary syndrome and conditions associated with mast cell activation, including allergic reactions hypersensitivity and/or anaphylactic oranaphylactoidinsults [1]. The disease is caused by the release of inflammatory mediators via mast cell activation. Hence, special attention should be paid to anaphylactic complaints and the findings of real-time cardiac sonography and electrocardiograms, as well as the levels of cardiac markers, in order to ensure a correct diagnosis and a good outcome of treatment. Conflict of Interest: We do not have any conflict of interests.

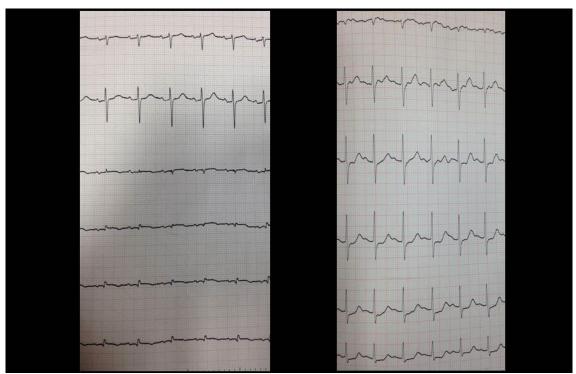


Fig-1a:

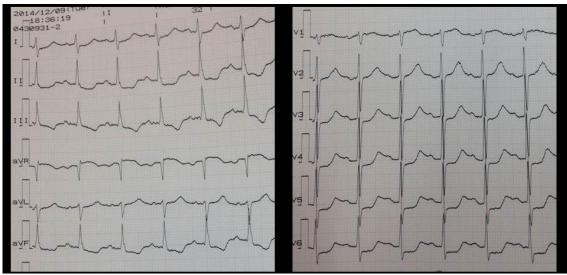


Fig-1b

Figure 1a, 1b: Electrocardiogram (ECG) findings on arrival (1a) and at one hour (1b) The initial ECG showed no specific changes in the ST segment (Figure 1a). However, a repeat electrocardiogram indicated elevation of ST at the aVR lead and depression of the ST segment at the II, III, aVF, V4, V5 and V6 leads (Figure 1b).



Figure-2: Coronary angiogram obtained after the ischemic changes noted on the electrocardiogram An urgent coronary angiogram revealed mild diffuse spastic changes in both the right and left coronary arteries (upper), which resolved with the infusion of Nicorandil (lower)

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