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## Bonsai-induced Kounis Syndrome in a young male patient

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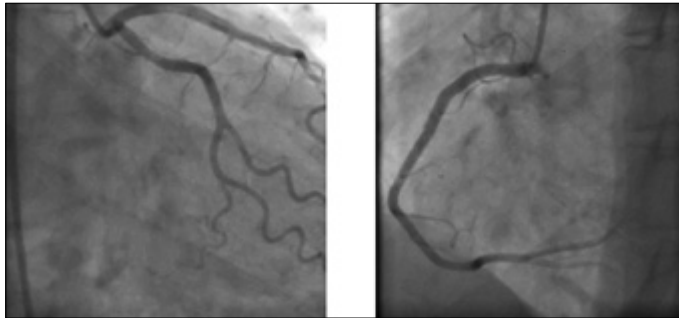
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### **Introduction**

The use of cannabis and its synthetic derivative, bonsai, has recently increased, and it has become an important health problem (1). Kounis



**Figure 1. ST elevation in inferior leads on 12-derivation ECG obtained in the emergency department**



**Figure 2. Demonstrating RCA, LMCA, LAD, and LCx on coronary angiography**

syndrome develops by the activation of mast cells, and it is an acute coronary syndrome (ACS) related to allergies, hypersensitivity, anaphylaxis, or anaphylactic reactions (2, 3). Bonsai-induced Kounis syndrome has not been reported in literature. The present study presents the case of a 27-year-old patient who arrived at the emergency clinic with chest pain 6 h after the use of bonsai.

## Case Report

A 27-year-old male patient arrived at the emergency clinic with sudden-onset retrosternal pain in the left arm, vomiting, and sweating. The chest pain was characterized by pressure and burning and lasted for 6 h. The patient did not have any known atherosclerosis risk factor and reported bonsai use for the first time in his life. He expressed that he had used a great amount of bonsai 1 h before the onset of chest pain. All vital signs of the patient were stable. His electrocardiographic (ECG) investigation revealed mild bradycardia and ST segment elevations in the inferior derivations (D2, D3, and AVF) (Fig. 1). The patient was referred to the coronary intensive care unit after the diagnosis of acute inferior MI was made. Bedside echocardiography revealed inferior and septal hypokinesia. Thrombolytic therapy was planned but was then disregarded as the recently recorded ECG showed ST-segment elevations returning to the isoelectric line. Troponin I value showed a typical increase (4 h)–decrease (24–36 h) (peak value 10.722 ng/mL). Mild leukocytosis and eosinophilia (4.9%) were present. The immunoglobulin E level was high (150 mg/L). The patient was referred to a more advanced center for coronary angiography (CAG). CAG indicated that all coronary arteries were patent (Fig. 2). The fibrinogen and homocysteine levels and antithrombin activity were all within normal ranges. The patient was followed-up for three days without any complications and was then dismissed from the hospital with prescriptions for 100-mg aspirin, 90-mg diltiazem, and 40-mg atorvastatin.

## Discussion

To our knowledge, this patient is the first bonsai-induced Kounis syndrome case in literature. Kounis syndrome, in other words allergic MI, has two types depending on the pathophysiology, or the presence of coronary artery disease. In type I, patients exhibit coronary vasospasms induced by allergic mediators such as histamine, thromboxane, and leukotrienes without the presence of atherosclerosis risk factors or coronary artery disease. In type 2, ACS develops due to coronary vasospasms, plaque erosion, or plaque rupture induced by these mediators in patients with atherosclerotic coronary artery disease. Recently, the fact that there are eosinophil and mast cells in the thrombus material excised from some patients in whom stent thrombosis developed after stent implantation with drug release makes us consider hypersensitivity reactions in these patients. This situation is accepted as the type III variant of Kounis Syndrome (4). With these findings, our case is in accordance with the type I variant of Kounis syndrome.

Cardiovascular and psychological problems are frequently reported to be associated with the use of bonsai. The main pathophysiology of Kounis syndrome is the release of many allergic mediators as a result of mast cell activation induced by allergic stimulants. It has been demonstrated in experimental studies that some endogenous cannabinoids suppress inflammation by decreasing mast cell activation via receptors; however, some endogenous cannabinoids trigger mast cell activation independent from receptors (5). In our patient, coronary arteries were revealed to be completely patent, and this may cause us to consider that a coronary vasospasm was the reason that was caused via mediators released by the bonsai-induced activation of mast cells. The main cardiovascular effects are coronary vasoconstriction, increase in the synthesis of tissue factor, thrombocyte activation, dysrhythmia development induced by various mechanisms, and plaque erosion (6, 7). In a patient considered to have Kounis syndrome, in addition to appropriate ACS management, the determination of serum histamine, specific IgE antibodies, and complement proteins and investigation of eosinophilia aid in the diagnosis (2). Leukocyte, eosinophil, and total IgE levels were increased in our patient, but other analyses could not be performed due to technical limitations.

## Conclusion

We hoped to emphasize the consideration of the use of bonsai-type synthetic drugs in a young patient with acute MI signs but without any risk factors.

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