CASE REPORT

Pulmonary embolism due to synthetic cannabinoid use: Case report

Sentetik kannabinoid kullanımına bağlı pulmoner emboli: Olgu sunumu

Gökhan Yırgın, M.D., İhsan Ateş, M.D., Bilal Katipoğlu, M.D., Burak Furkan Demir, M.D., Nisbet Yılmaz, M.D.

Ankara Numume Training and Research Hospital, Ankara, Turkey

Summary– A 29-year-old male patient was treated and followed up for a pulmonary embolism. The patient had no relevant medical history, other than the fact that he had smoked bonzai, a synthetic cannabinoid derivative, for 2 years. Hypercoagulability tests were normal. The use of synthetic cannabinoids is increasing in the young population and should be kept in mind among the causes of pulmonary embolism.

Özet– Yirmi dokuz yaşında erkek hasta pulmoner emboli nedeniyle kliniğimizde takip edildi. Hastanın iki yıldır bir sentetik kannabinoid türevi olan bonzai kullandığı öğrenildi. Hiperkoagülabilite açısından bakılan tetkikleri normaldi. Hastada sentetik kannabinoid kullanımına bağlu pulmoner emboli geliştiği düşünüldü. Sentetik kannabinoid kullanımı genç nüfusta giderek artmaktadır ve bu popülasyonda pulmoner emboli sebepleri arasında akılda tutulmalıdır.

Pulmonary embolism is a clinical condition with a high rate of morbidity and mortality. The most common causes are immobilization, surgery, trauma, congestive heart failure, oral contraceptive use, inherited thrombophilia, and autoimmune diseases. A rare cause of pulmonary embolism is the use of illicit substances.[1]

In this case report, a patient with a pulmonary embolism due to synthetic cannabinoid use is discussed.

CASE REPORT

A 29-year-old, previously healthy man presented at the clinic with complaints of dyspnea, cough, and weakness. There was no indication of systemic disease, prescription drug use, trauma, surgery, or allergy in the patient's medical history. It was determined that he had smoked bonzai for 2 years. His family history was uneventful for chronic disease. A physical examination revealed a moderate general condition. Tachycardia and tachypnea were present. Other systemic findings were normal and an electrocardiographic evaluation revealed sinus tachycardia. A chest X-ray indicated no pathological findings (Fig. 1a). Laboratory tests were requested. A complete blood count, levels of electrolytes and cardiac parameters, and liver and kidney function test results were normal. The D-dimer level was determined to be 1633 ng/mL. Hypoxia and hypocapnia were detected in an arterial blood gas test. Based on these findings, pulmonary embolism was considered. A thorax computed tomography angiography revealed a thrombus beginning at the main pulmonary arteries and extending to the lower lobes and bilateral segmental bronchi (Fig. 1b and 1c). An extensive workup was requested to investigate the etiology of the pulmonary embolism. Doppler ultrasonography of the lower and upper extremity deep veins revealed no thrombus. The ejection fraction was measured as 65% on a transthoracic echocardiography, and while no thrombus was observed in the heart cavities, a slight dilatation was observed in the right atrium. There was no significant finding in a hypercoagulability testing panel that included examination for antinuclear antibodies, homocysteine, cardiolipin antibodies, anti-double stranded DNA, antineutrophil cytoplasmic antibodies, lupus anticoagulant, proteins C and S, antithrombin, and factor V Leiden. A fundus examination was performed for

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Correspondence: Dr. Gökhan Yırgın. Ankara Numune Eğitim ve Araştırma Hastanesi, Ankara, Turkey.
Tel: +90 312 - 508 40 00 e-mail: gokhanyirgin@gmail.com

Behçet's disease, and no uveitis finding was observed. An HLA-B51 gene test was negative. Other causes of pulmonary embolism were excluded. The patient was diagnosed with a pulmonary embolism likely related to synthetic cannabinoid use. Anticoagulation treatment with enoxaparin was initiated and the patient was discharged.

DISCUSSION

Cannabinoids are the most commonly produced and consumed illegal substances in the world. Synthetic cannabinoids are produced as an alternative, and some have been marketed under names such as bonzai, spice, and K2 since 2004.

Cannabinoids have 2 types of receptors: CB1 and CB2. The CB1 receptors are primarily located in the central nervous system, which is responsible for the psychotropic effects of cannabinoid use. The CB2 receptors are mainly located in the cells of the immune system. The side effects associated with use of synthetic cannabinoids are similar to those associated with cannabis use, but the effects associated with use of synthetic cannabinoids are more severe.[2] All known synthetic cannabinoids are lipophilic. This leads to a large distribution volume and a cumulative effect after repeated use.[3] Side effects can include nausea, vomiting, shortness of breath, hypertension, tachycardia, chest pain, susceptibility to thrombosis, muscle twitches, acute renal failure, anxiety, agitation, psychosis, suicidal thoughts, and cognitive impairment.[4] It is not clear how synthetic cannabinoids cause thrombosis. Vasospasm, plaque rupture, and platelet aggregation have been considered in the pathophysiology.[5] It is thought that they stimulate the inflammatory response by providing phospholipase A2 activation through the CB2 receptors.[6] According to another hypothesis, cannabinoids exert direct prothrombotic action by binding to their receptors on the platelet membrane.[7]

To the best of our knowledge, only 1 previous case of pulmonary embolism due to synthetic cannabinoid use has been reported. Raheemullah et al.[8] reported the case of a 32-year-old female patient who had a pulmonary embolism due to synthetic cannabinoid use. She had 2 renal infarcts, 1 pulmonary embolism, and an ischemic stroke, on separate occasions. A hypercoagulability panel of that patient was negative.

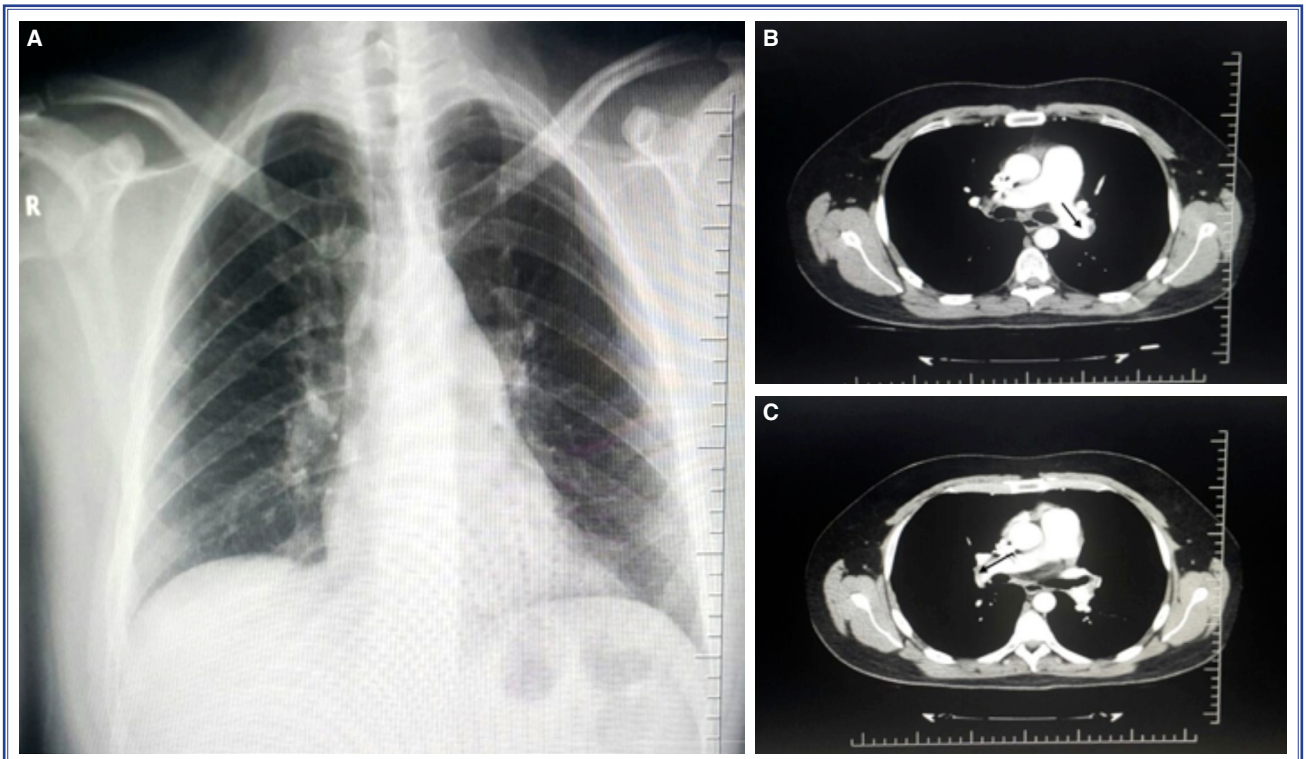

Figure 1. **(A)** A chest X-ray revealed no pathological findings. **(B and C)** Computed tomography thoracic angiography indicated defective filling of the bilateral pulmonary arteries consistent with a thrombus.

Presently described is the case of a patient who had only a pulmonary embolism. Other causes of a pulmonary embolism were excluded, and it was thought to be due to synthetic cannabinoid use. He was discharged with enoxaparin treatment. No study was conducted to determine whether anticoagulant regimens differ in cannabinoid-induced thrombosis therapy.

The use of synthetic cannabinoids is increasing in the young population and should be kept in mind among the potential causes of pulmonary embolism. Further investigation is warranted to clarify the relationship between synthetic cannabinoids and pulmonary embolism.

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