Letter to Editor

Distal internal carotid artery dissection after consumption of synthetic cannabinoid “Bonzai”

Dear Editor,

Since synthetic cannabinoids are easily accessible and cheap, they have increasingly become popular in Turkey as well as all over the world in the recent times. There are many studies reporting an association between cardiovascular and cerebrovascular events and cocaine, heroin and amphetamine use. “Bonzai”, known as “K2” or “spice” in the Europe, is the most popular synthetic cannabinoid in Turkey [1]. A few ischemic stroke cases have been reported following the use of Bonzai [1-3]. Herein, we discussed an unusual patient, who developed distal internal carotid artery dissection (DICAD) after consuming Bonzai or cannabis.

A 21-year-old male patient was brought to the emergency room for unconsciousness and right-sided weakness on the second day of onset of these symptoms. His anamnesis revealed cannabis use for 3 years and Bonzai use for 2 months. We learned from his friend that he took Bonzai on the day of the event and that he lost his consciousness immediately afterwards. Since the patient was brought to the emergency room on the second day of onset, we do not have clear evidence that if patient may have had a severe tonic clonic seizure from the bonzai from both exam and medical history. Patient did not look dysmorphic or very tall or very flexible to suggest connective tissue disease. There was no any other medications or over the counter drugs, hormones or pills history. There was no family history of stroke or history of stroke risk factors for young stroke such as hypercoagulability, congenital heart disease and cardiac arrhythmias. On admission to the emergency room, his blood pressure was 157/90 mmHg and heart rate was 92 beats/min. Neurological examination revealed lethargy, global aphasia and hemiplegia on the right side. The headache was not questioned because the patient was lethargic and had aphasia, and there was no finding of Horner’s syndrome. The laboratory analyses including biochemistry and complete blood count were all within the normal range. Computed Tomography (CT) of the brain showed hypodense area in the vascular territory the left middle cerebral artery (Alberta Stroke Program Early CT score (ASPECTS) = 4/10). Diffusion-weighted imaging showed acute infarction in the vascular territory of the left middle cerebral artery. Digital subtraction angiography and Cone Beam CT revealed a double-lumen appearance compatible with dissection in the petrous segment of the left internal carotid artery extending to the cavernous and clinoid segments, as well as occlusion of the left proximal middle cerebral artery (Fig. 1). During the hospitalization Electrocardiogram, transthoracic and transesophageal echocardiogram were normal. Anti-cardiolipin and anti-phospholipid antibodies, protein C antigen and activity, protein S antigen and activity, antithrombin 3, prothrombin 20210, methylenetetrahydrofolate reductase gene mutations, factor V leiden, and lupus anticoagulant were unremarkable. The toxicological analysis showed only cannabis use. Anticoagulant and antiedema treatment was given; his modified Rankin Scale score was 5 at the time of hospital discharge and 2 during 3rd-month follow-up visit.

To the best of our knowledge, this is the first case of DICAD following the use of Bonzai or cannabis reported in the literature. The mechanism of DICAD following Bonzai is unclear. It was thought that dissection might have developed secondary to the patient’s likely fall due to the unconsciousness he developed after consuming Bonzai. However, no sign of trauma was detected either by physical or radiological examination performed in the neurointensive care unit. Dissection is also likely to have developed rather spontaneously than due to trauma.

DICAD can be considered as an uncommon etiological factor in all types of ischemic strokes, but it appears to be an etiological factor particularly under the age of 45 years. It accounts for 2% of ischemic strokes in all age groups and 20–25% under the age of 45 years [4]. Trauma is the most important factor in the etiology of DICAD. DICAD can occur also spontaneously, however, the etiology and the mechanism of spontaneous dissection remain unclear. In addition, autosomal dominant polycystic kidney disease, connective tissue diseases such as Ehler–Danlos syndrome, fibromuscular dysplasia and Marfan syndrome, α1 antitrypsin deficiency, osteogenesis imperfecta, cystic medial necrosis and hereditary hemochromatosis as well play a role in the etiology of spontaneous DICAD [5]. Beside the reported acute ischemic
stroke, seizure, and myocardial infarction, intracerebral hemorrhage has also been reported with the consuming of Bonzai [6]. Although Bonzai abuse has not been reported as the etiological factor for DICAD until now, the relationship between cannabinoid use and development of dissection would be better understood by increasing the number of case reports and by investigating this subject in detail.

**Informed consent**

Written informed consent was obtained by the patient who was participated in the study.

**Conflict of interest**

None declared.

**Acknowledgement and financial support**

None declared.

---

**Fig. 1** (A–C) Intracranial carotid artery dissection started from petrous segment to cavernous and clinoid segments. (D) Occlusion of left middle cerebral artery. (E) Anterior and middle cerebral artery flows were normal. (F) Infarction in middle cerebral artery territory on the left side was shown.

---

**REFERENCES**


Hasan Bilen Onan  
Cukurova University, Faculty of Medicine, Department of Radiology,  
Adana, Turkey

Fatma Ozge Salkin  
Sebnem Bicakci  
Cukurova University, Faculty of Medicine, Department of Neurology,  
Adana, Turkey

*Corresponding author at: Cukurova University, Faculty of Medicine, Department of Neurology, Adana 01330, Turkey  
E-mail address: tdemir@cu.edu.tr (T. Demir)