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Head and Neck

Acute carotid thrombosis and ischemic stroke following overdose of the synthetic cannabinoid K2 in a previously healthy young adult male

Raihan Faroqui BA, Peter Mena MD, Allen R. Wolfe MD, MPH, Joseph Bibawy DO, George A. Visvikis MD, Michael T. Mantello MD**

Department of Radiology, Richmond University Medical Center, 355 Bard Ave, Staten Island, NY 11310, USA

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ABSTRACT

With the popularity of synthetic cannabinoid street drugs such as "K2 and Spice," a number of serious neurologic adverse events are coming to light. This case is a 36-year-old African American man, with no significant medical history, who presented with extensive left cervical and intracranial internal carotid artery occlusion and subsequent ischemic stroke. The patient endorsed smoking K2—a synthetic cannabinoid (SC) with structural similarity to cannabis. The mechanism by which SC abuse induces a prothrombotic state leading to ischemic neurovascular sequelae is currently unclear, although a temporal association in the absence of other stroke risk factors suggests a causal relationship. Our case highlights the need for emergent neuroimaging upon suspected SC overdose. Practitioners should be vigilant in recognizing that ischemic stroke and unexplained neurologic deficit can arise after SC abuse, especially in younger populations with few stroke risk factors and who are prone to chronic cannabis use.

Introduction

Synthetic cannabinoids (SCs), with street names such as K2, Spice, Bliss, Black Mamba, and among others, are smokable herbal mixtures that contain synthetic psychoactive compounds that mimic tetrahydrocannabinol (THC) found in street cannabis. This novel class of drugs has emerged as an increasingly popular and cheaper alternative to marijuana, mostly sold at smoke shops or online as sealed bags of potpourris labeled "not for human consumption" or as "incense," although they are regularly used for illicit purposes especially in teenagers and younger adults who undergo regular drug screening [1]. SC is undetectable on routine drug screens—a key selling point. Federal law classifies SC as a Schedule I controlled substance [2]. SC can potentially be up to 100 times more potent than THC. SCs are full nonselective agonists of the cannabinoid receptor (CB) class CB1 and CB2 receptors as opposed to THC substances, which are only partial agonists [2]. More than 11,000 patients per year consult emergency department (ED) services

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* *Corresponding authors.*
E-mail addresses: rfaroqui@gmail.com (R. Faroqui); egleason@rumcsi.org (M.T. Mantello).

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because of the side effects of SC [1]. There is a false perception among the lay public that SCs are "safe"; however, at least 40 deaths have been attributed to SC overdose in the United States [2]. In recent years, several cases have emerged linking SC abuse to neurovascular complications [1]. Although myocardial infarction is a well-reported adverse event, cases of ischemic stroke (IS) are few in the literature. An even smaller cohort of IS cases is verified with extensive neuroimaging. Takematsu, Freeman, Moeller, and others have reported ischemic stroke after SC use with radiological depth [3–7]. However, all these cases involved infarcts in the cerebral circulation, most commonly in the middle cerebral artery (MCA). We will describe, for the first time, a patient with a carotid circulation infarct, specifically acute thrombosis of the left internal carotid artery (ICA) causing an IS. Complete occlusion of the ICA is never asymptomatic and presents with typical stroke symptoms, such as facial droop, arm weakness, and slurred speech. Appropriate and timely diagnosis and treatment is essential in preventing severe complications.

Case report

A 36-year-old African American man with no significant medical history presented to the ED with a 1-day history of right-sided weakness and aphasia. According to family members, he was witnessed the previous night crouching over his bed with knees on the floor and his right arms leaning on the bed. The patient's mother heard a loud noise in the morning and found the patient on the floor making strange noises and having difficulty speaking. He reported taking K2 (a well-known local street drug of the SC class) the night before symptom onset and smoking marijuana often in the past. The patient also stated he smoked tobacco and drank alcohol socially. He denied the use of any other street drugs. He used no daily medications. He denied previous stroke or transient ischemic attack, muscle weakness, paresthesia, seizure, headache, visual abnormality, or other neuromuscular insult. The patient had no personal or family history of coagulopathy or blood disorders. On presentation in the ED, the patient's vital signs were remarkable only for borderline hypertension (158/90 mm Hg), and electrocardiography revealed sinus bradycardia at 51 bpm. Physical examination was remarkable for an expressive aphasia. Also present was a dense, right central facial palsy with a dense right hemiparesis; power 0/5 in the right arm and 2/5 in the right leg. Also remarkable was a loss of sensation to touch in the right upper extremity. Right lower extremity and right side of the face touch sensation were intact. Noncontrast computed tomography (CT) of the head demonstrated hypodensity in the left basal ganglia and a left hyperdense MCA consistent with an acute ischemic infarct involving the left MCA distribution secondary to a thrombotic event (Fig. 1). Intravenous tissue plasminogen activator (t-PA) was not administered as the patient presented 13 hours after the last known normal. Computed tomography angiography (CTA) of the neck showed a large filling defect extending from the origin of the left ICA into the intracranial portions of the ICA—findings consistent with an extensive thrombosis (Figs. 2 and 3). The patient was not a candidate for endovascular therapy. Noncontrast magnetic

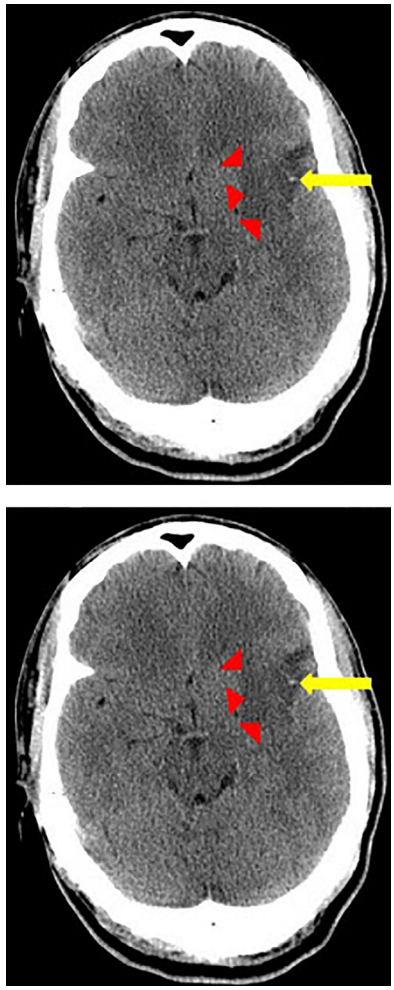

Fig. 1 – Axial computed tomography image demonstrates indistinct low density (red arrows) involving the left basal ganglia and external capsule, consistent with an acute ischemic infarct involving the left middle cerebral artery (MCA) vascular territory. Additionally, a hyperdense left MCA (yellow arrow) is present, concerning for a thrombotic event.

resonance (MR) imaging of the head confirmed the findings of acute large left-sided infarct along the MCA vascular distribution, whereas MR angiography demonstrated absent flow in the left ICA and diminished flow in the left MCA territory, confirming the neck CTA finding of occlusion of the left ICA (Fig. 4). There were no radiographic signs of vasculitis or other arterial disease. Transesophageal echocardiography was unremarkable and without evidence of a patent foramen ovale, aortic debris, valve vegetations, or other abnormalities. Urine drug screen was positive only for cannabis. A special SC laboratory panel was sent, testing for the most popular strains of these designer street drugs, including K2 compounds such as JWH-018, JWH-073, JWH-200, CP 47, 497, and CP47, 497 C8 homologue. The test came back negative—no active compounds were detected. The company stated the test had a "sensitivity greater than 90%," raising the possibility of a false negative, especially as the compounds in these drugs are ever evolving. As stated previously, the patient verbally endorsed