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Marijuana induced Reversible Cerebral Vasoconstriction Syndrome

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Introduction

Reversible cerebral vasoconstriction syndrome (RCVS) is a transient multifocal arterial vasoconstriction and dilatation. The term was proposed by a panel of experts[1] as a unifying term for a variety of a syndromes characterized by cerebral vasospasms and resultant clinical manifestations of cerebral ischemia. These syndromes include Call–Fleming syndrome, drug-induced cerebral angiopathy, benign angiopathy of the CNS, postpartum cerebral angiopathy, and migrainous vasospasm. Drug abuse, especially cannabis, is an important etiologic factor for RCVS. It is one of the most widely used recreational substances in the world, considered by many consumers as a relatively safe drug with few significant side-effects.[2] Cannabis contains 9-tetrahydrocannabinol (THC), which is rapidly absorbed when smoked, reaching a peak plasma concentration at 10–30 min, and is lipid soluble with a large volume of distribution.[3] Cannabinoids bind to cannabinoid receptors, CB1 in the central nervous system and CB2 in the immune system.

Case Summary

A 59-year-old left-handed African–American male with a past medical history of diabetes and hyperlipidemia presented with an episode of acute aphasia, right hemiparesis, and left gaze deviation. A noncontrast CT scan of the head showed microvascular ischemic white matter changes and CT angiography showed patent cerebral vasculature with diffuse narrowing of cerebral vessels (Figure 1). Coagulation profile was normal. Urine toxicology was positive for THC. Additional parameters, such as a urine drug screen, hypercoagulable panel, and alcohol screen, were negative. An MRI of the brain without contrast demonstrated acute infarcts in the left thalamus and left parietal lobe posteriorly (Figure 2).

On admission, the patient was started on aspirin and received physical therapy. He showed marked improvement with mild residual dysarthria. The patient continued to improve and was discharged on Hospital Day 4 with a National Institutes of Health Stroke Scale of 0. A repeat CT angiography at discharge showed patent cerebral vasculature with improved flow in the bilateral higher convexities (Figure 3).

Discussion

Cardiovascular side effects of marijuana have been documented, including tachycardia, hypertension, postural hypotension, and increased levels of carboxyhemoglogin.[4] There have been several reports of stroke following cannabis use.[5] Since 1987, fewer than 25 cases of ischemic stroke associated with cannabis smoking have been published.[6–8] Postulated mechanisms for such stroke include cerebral vasospasm,[9–11] vasculitis, and postural hypotension with impairment in cerebral autoregulation of the brain blood flow.[8, 12]

In addition to the direct central nervous system action, marijuana may augment stroke onset via other mechanisms, such as causing cardiovascular effects, which include tachycardia,[13] hypertension, and postural hypotension.[14] Furthermore, smoking marijuana increases carboxyhemoglobin, which reduces oxygen transportation capacity and leads to the onset of stroke.[15] Additional causes of stroke associated with cannabis are due to an increased risk of myocardial infarction and paroxysmal atrial fibrillation.[5] Concomitant alcohol consumption or unusually high consumption of cannabis has been regarded as possible precipitating factors for acute ischemic strokes.[7,8] ‘Cannabis arteritis’ that pathologically resembles Buerger’s disease is found in patients that are regular cannabis users and only moderate tobacco smokers.[14]

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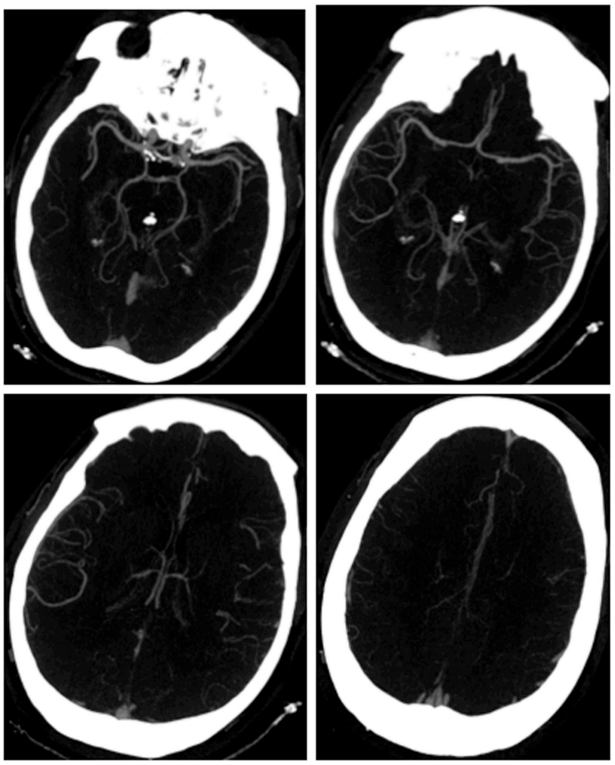

Figure 1. Multiple cuts of the initial CT angiogram at presentation showing an evidence of vasospasm.

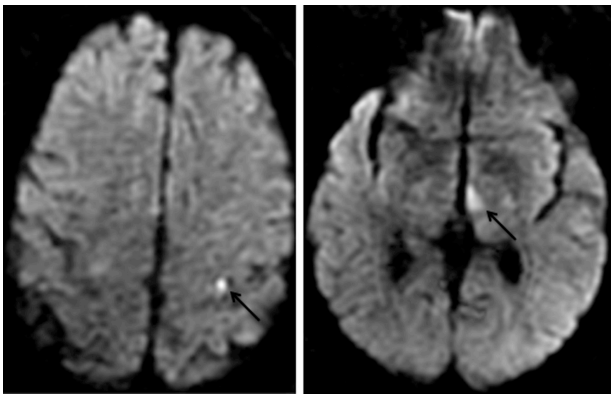

Figure 2. MR diffusion imaging demonstrates an hyperintense signals in the left thalamus and parietal lobe.

Ischemic stroke studies have suggested that cannabis associated strokes were more frequent in vertebrobasilar territory, suggesting susceptibility of posterior circulation. [6,8] Mesec et al reported arterial spasms on transcranial ultrasound and MRA in a patient with cannabis related stroke.[9] Ducros et al also reported RCVS with cannabis abuse.[11] Mathew et al used transcranial ultrasound

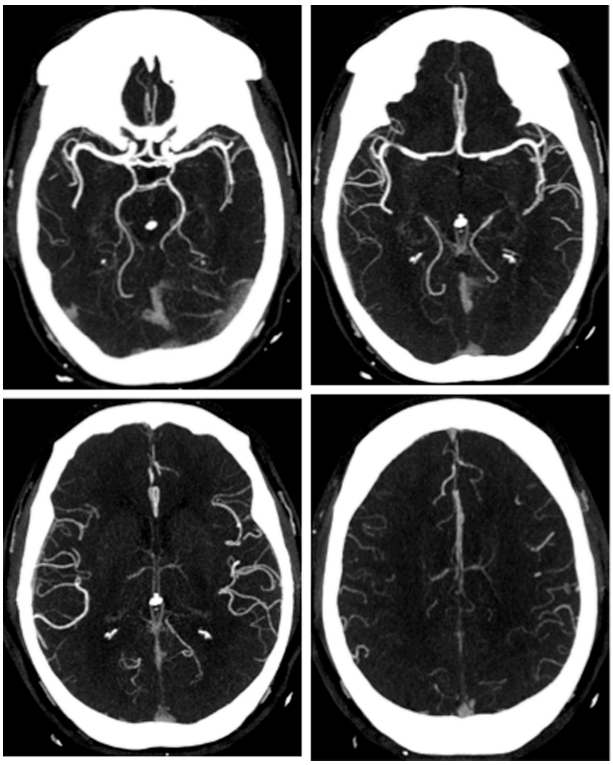

Figure 3. Multiple CT angiogram cuts after five days of the ictus showing increase density of the contrast in the vessel indicating partial resolution of the vasospasm.

to demonstrate reduced middle cerebral artery (MCA) velocities but normal systemic pressures after cannabis use, suggesting the possibility of impaired cerebral autoregulation.[16] Furthermore, Herning et al examined blood flow velocity in the anterior and MCAs using transcranial Doppler sonography in marijuana users (who were stratified into light, moderate, and heavy smokers).[12] It was found that the pulsatility index and systolic velocity were significantly increased in marijuana users when compared with control subjects and these increases persisted in the heavy marijuana users after a month of monitored abstinence.[12]

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