Stroke After Heavy Marijuana Smoking

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I examined two young men who developed cerebral infarction associated with heavy marijuana smoking. Both were light tobacco smokers, but they did not drink alcohol or use other street drugs. Diagnostic work-up for nonatherosclerotic causes of stroke was unremarkable. I postulate that marijuana-associated alterations in systemic blood pressure resulted in vaso-spasm, leading to strokes in these patients. (Stroke 1991;22:406-409)

Stroke associated with drug abuse has been reported frequently. Although the incidence and prevalence of cerebral infarction and intracranial hemorrhage associated with drug abuse is uncertain, case series have provided extensive documentation of stroke occurring with the use of street drugs, especially cocaine, amphetamines, "T's and blues," anabolic steroids, lysergic acid diethylamide, barbiturates, alcohol, and heroin. However, an acute neurological deficit following heavy marijuana smoking has been reported infrequently. I examined two young men who had cerebral infarction during heavy marijuana smoking.

Case Reports

Case 1

A previously healthy 34-year-old right-handed white man experienced the sudden onset of dizziness, left arm and leg weakness, and slurred speech while smoking a marijuana cigarette. There was no headache, and he denied the use of other street drugs and alcohol. He had smoked less than one pack of tobacco cigarettes and up to seven marijuana cigarettes per day for 15 years. Recently, he had increased his marijuana smoking from seven to 14 cigarettes per day. There was no family history of stroke, heart disease, hypertension, diabetes mellitus, migraine headache, lupus erythematosus, autoimmune disorders, or sickle cell disease.

This patient's blood pressure was 164/110 mm Hg and his pulse was 104 beats/min on admission. Neurological examination performed ≤2 hours after the onset of symptoms showed severe dysarthria, right-left disorientation, and acalculia. Cranial nerve examination revealed left lower facial weakness. Strength of the left leg was 0/5, strength of the left arm was 1/5, and there was loss of pinprick and temperature sensation on the left side.

Results of the following laboratory studies were within normal ranges: complete blood count; platelet count; fibrinogen level; prothrombin time; partial thromboplastin time; concentrations of serum glucose, electrolytes, cholesterol, high density lipoprotein, low density lipoprotein, and triglycerides; liver and renal function tests; concentrations of metanephrine and catecholamines; erythrocyte sedimentation rate; presence of antinuclear antibodies and rheumatoid factor; VDRL; concentrations of C3 and C4; platelet function tests; lupus anticoagulant, anticardiolipin antibody, and protein C; human immunodeficiency virus titer; and concentration of antithrombin-3. The results of 24-hour Holter monitoring and echocardiography were normal. The results of renal angiography, done because of the patient's hypertension, were also normal. He did not consent to cerebral angiography. The results of non-invasive carotid Doppler and duplex studies were normal. A computed tomogram (CT scan) of the brain with and without contrast on the first hospital day was normal. A repeat CT scan with contrast 3 days later showed right basal ganglia and periventricular infarcts (Figure 1). On the sixth hospital day, single-photon emission computed tomography with delayed redistribution scan revealed right frontoparietal lobe and right basal ganglia infarcts. Analysis of the marijuana cigarettes revealed pure Cannabis without contaminants.

After hospital discharge the patient refused to stop smoking marijuana in spite of being strongly advised to do so because of its harmful effects; he consented to an experiment testing the effect of marijuana on his platelet function. He also agreed to have the case written and published. The in vivo test involved the collection of blood samples before and 0.5 and 2 hours after the patient smoked marijuana as witnessed by his brother. Platelet function studies were...
performed using four concentrations of adenosine diphosphate, collagen, epinephrine, arachidonic acid, and ristocetin. There was no significant difference in platelet function among the different samples of blood. The in vitro test involved collection of blood samples 0.5 and 2 hours after the patient smoked marijuana; Δ⁹-tetrahydrocannabinol (THC) in the range of 0.1–10 μg/ml was added to the samples. There was no significant difference between platelet function in these two different samples.

Physical therapy resolved this patient’s dysarthria and sensory deficit. Left-sided paresis remained and worsened and was associated with palpitations and dizziness whenever he smoked marijuana. He needed antihypertensive medications to control his blood pressure. One month after the stroke, the patient discontinued his marijuana smoking. His blood pressure then returned to normal without medical therapy, and his hemiparesis improved to some extent.

Case 2

A previously healthy, 32-year-old right-handed black man experienced the sudden onset of right arm and leg weakness and slurred speech ≤0.5 hour after smoking a marijuana cigarette. He denied the use of alcohol or other street drugs. He had smoked one half pack of tobacco cigarettes per day for 9 years and marijuana heavily during the preceding 14 years. He had increased his marijuana smoking during the 2 weeks prior to the onset of symptoms. There was a medical history of undifferentiated personality disorder. His family history was unremarkable.

This patient’s blood pressure was 155/105 mm Hg on admission. There was right hemiparesis, dysarthria, and lower facial weakness. Results of studies identical to those performed for case 1 were also within normal ranges. Results of magnetic resonance imaging (MRI), angiography, and duplex studies of his carotid system were normal. Toxicological evaluation of the patient’s urine and plasma showed only cannabinoids. An initial CT scan of the brain was normal. A contrast CT scan of the brain (Figure 2) obtained ≤1 week after the stroke demonstrated a left basal ganglia infarct and a small left parietal lobe infarct. Brain MRI revealed a left basal ganglia infarct. Cerebral angiography was not performed due to the patient’s refusal. On a low-sodium diet the patient’s blood pressure returned to normal within 1 week, and after 3 months of physical and occupational therapy there was marked improvement of his neurological deficits.

Discussion

Marijuana use became popular in the United States in the 1960s and remains the most commonly used illicit recreational drug. It is usually smoked in cigarette form but may be consumed orally. Pharmacokinetic studies indicate that smoking is almost equivalent to intravenous administration except that
lower peak plasma concentrations of THC are attained. In addition to its psychotropic effects, marijuana may induce hypotension, tachycardia, an increase in the concentration of carboxyhemoglobin, nausea, hunger, conjunctival congestion, and dryness of the mouth and throat. Heavy smoking may be associated with chronic bronchitis, airway obstruction, and squamous metaplasia of the respiratory tract.

Stroke symptoms have been described after marijuana smoking in only four prior cases. While the neurologological deficits were carefully documented, supporting studies such as CT, MRI, toxicology, and angiography were not carried out. Thus, the underlying pathophysiological mechanism of neurological deficit in these cases is obscure.

Both of my patients had large, deep cerebral infarcts associated with heavy marijuana use and a recent increase in marijuana smoking. Although both patients smoked tobacco cigarettes daily, since they were light smokers (<10 cigarettes/day) the relative risk of stroke from this factor may not be significant according to the Framingham Study. There was no concurrent use of alcohol or other drugs, and short of cerebral angiography (though MRI angiography was done) there was no evidence of an atherosclerotic cause for stroke. Interestingly, in one patient repeat marijuana smoking was associated with worsening of the neurological deficit.

I postulate that marked swings in blood pressure caused the strokes. Intravenous administration of THC produces systemic and ocular hypotension. Fluctuations in blood pressure have been well documented in healthy volunteers and marijuana users, and marijuana in particular can produce inconstant changes in pulse rate and blood pressure. Both of my patients were hypertensive without other risk factors for stroke at the time of hospital admission, although their blood pressure readings returned to normal in the weeks following the stroke with no medical treatment and abstinence from marijuana. I propose that the elevations of systemic blood pressure in my patients, who had both recently increased their amount of marijuana smoking, could be a reaction to cerebral vasospasm preceded by hypotension, which led to cerebral infarction. It is also possible that tobacco smoking could have added to this problem. Brain damage has not been confirmed in humans, although some suggestions of ultrastructural damage have been found in animals. Crawford and Merritt found that maximal decreases in systolic pressure (15–22%) and diastolic pressure (13–20%) were evident 60 minutes after THC inhalation. Afterward, the systolic and diastolic pressures increased simultaneously during 90–120 minutes to control levels.

Based on these cases, no definitive conclusions can be drawn as to the etiology of the strokes. Similarly, conflicting reports have been published relating the effects of marijuana on adenylate cyclase activity, sympathetic nervous system stimulation, and parasympathetic nervous system blockage. More studies are needed to determine if marijuana is a risk factor for stroke, especially in combination with tobacco smoking.

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References


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