Case Report

**Stroke Associated with Marijuana Abuse**

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**Key Words**
Marijuana · Stroke

**Abstract**
We present the case of a 15-year-old with a cerebellar infarct that involved multiple arterial territories. It was temporally related to, and probably caused by, heavy marijuana use. While the mechanism of marijuana-associated stroke is unclear, the drug is known to cause hypotension and to impair peripheral vasomotor reflexes. We suspect that the child had diminished cerebral autoregulatory capacity and developed the stroke during a period of hypotension.

Marijuana use is widespread. It is a drug commonly abused by young adults, not infrequently by adolescents and occasionally by children. There is a perception among these groups that short-term use of marijuana is safe, and that the consequences of overdose are minimal.

Over the past several years, a number of reports have been published documenting strokes in adults that were thought to be due to marijuana use [1–5]. Given the demographics of abuse, similar problems are likely to exist in the pediatric population, but perhaps go unrecognized. We recently cared for a 15-year-old boy who had a stroke during a marijuana binge.

**Case Report**

J.P. is a 15-year-old, previously healthy boy who presented to the emergency room with a 3-day history of headache, nausea and unsteady gait. His symptoms developed over a period of hours and had been continually present and of relatively constant severity since their onset. The boy was known to smoke marijuana, and had been doing so, essentially continuously, for several days prior to and including the day he developed the symptoms described. He denied other drug or alcohol use. His medical history was notable only for delayed puberty that was thought to be familial. No family members had recognized clotting disorders nor had any suffered a stroke or myocardial infarction. On examination, the child was awake but slightly lethargic. He complained of a headache and associated nausea. He was unable to perform heel to shin or finger to nose testing and had marked dysdiadochokinesia. These findings were present bilaterally, but were much severer on the right. He did not have nystagmus, other cranial neuropathy or myelopathy. He had no signs of trauma, systemic vasculitis or valvular disease. A urine drug screen was performed: only cannabinoids were found. A CT scan was obtained which demonstrated diffuse hypodensity in the right cerebellar hemisphere. MRI was consistent with a cerebellar stroke that involved the territories of several arteries (fig. 1). MRA demonstrated normal vasculature in the posterior fossa without evident vascular cut-off or vasculitis. A needle
biopsy was performed which confirmed the diagnosis of a cerebellar infarction.

J.P. had an extensive evaluation. His sickle cell screening study was negative. His erythrocyte sedimentation rate was 5 mm/h with a prothrombin time of 12.7 s and a partial thromboplastin time of 22 s. Cardiac echo was performed; a small patent foramen ovale (PFO) could be demonstrated with Valsalva maneuver, but there were no other abnormalities. Clotting parameters that were obtained included: protein C, protein S, antithrombin antibody, activated protein C resistance, lupus anticoagulant, antiphospholipid antibody, prothrombin 20210a, lipoprotein A and factor 5 Leiden mutation. These were all normal or not present. An angiogram was performed and the posterior fossa vasculature was shown to be normal with a clearly defined PICA, AICA and SCA each with a distinct point of origin. The child improved over several days and was discharged with obvious but improving cerebellar dysfunction. He was seen several weeks later and had only mild dysdiadochokinesia in his right hand.

**Discussion**

This child’s stroke was most likely caused by marijuana use. The ictus occurred during a period of extremely heavy marijuana exposure, and he has no other definable risk factors for early stroke. There is no history of thrombotic disease in the child or in other members of his family. Extensive evaluation of his clotting function showed it to be normal. He had no evidence of trauma, subarachnoid hemorrhage or vasculitis. He does have a PFO that can be demonstrated with a Valsalva maneuver. It is unlikely that the PFO is significant, in that it is unlikely that the stroke was embolic. The infarct involves the territories of the PICA, AICA and SCA, but does not involve any brainstem branches of the basilar artery or any vessels in the distal basilar distribution. The 3 involved arteries have distinct origins, so an embolus would have had to occlude all 3 without involving other basilar branches. Alternatively, 3 separate emboli could have occluded the involved vessels, but the chance of that happening seems extremely remote.

The mechanism of marijuana-induced stroke is not known: in fact, surprisingly little information is available about the cardiovascular effects of marijuana ingestion. The drug appears to be a peripheral vasodilator. Increased blood flow can be recognized in both skin and skeletal muscle after smoking marijuana [6]. Postural hypotension and tachycardia are also quite common [7]. These effects are thought to be mediated via β-adrenergic receptors since β-blockers ameliorate the response [6]. Interestingly, marijuana smoking also impairs peripheral vascular re-

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**Fig. 1. a** Long TR axial slice through the cerebellar hemispheres demonstrating abnormal T₂ prolongation of both anterior and posterior cortices. **b** Coronal fluid-attenuated inversion recovery image which shows the abnormal T₂ prolongation involving the superior and inferior cortices as well as the tonsil. **c** Axial short TR slices after contrast administration. The lesion shows T₁ prolongation, displacement of the fourth ventricle, mild hydrocephalus and no contrast enhancement.
flex responses [6]. Specifically, a stimulus, such as the application of ice, which would normally cause vasoconstriction, does not do so in subjects who have recently ingested marijuana.

The effects of marijuana on the blood vessels of the central nervous system have not been extensively studied. The information that does exist suggests acute exposure may slightly increase cerebral blood flow in experienced users while slightly decreasing flow in those with little prior exposure to the drug [8]. It is thought that heavy long-term use decreases cerebral blood flow; this change is at least partially reversible with abstinence [9, 10]. There is suggestive evidence that marijuana may impair vascular autoregulation, but this assertion cannot currently be proven [7]. The mechanism of these changes is not known.

It is difficult to draw conclusions about the mechanism of marijuana-induced stroke based on this child’s presentation. Our supposition is that he had impaired cerebral vasoregulatory capacity, and that his stroke was caused by a period of hypotension in that setting. It is hard to understand the distribution of his infarct if that was the case, however. Classic watershed areas were spared, and no obvious abnormality of vascular anatomy could be found to explain the relative susceptibility of the involved vessels. It is possible that he had localized vasospasm, but there is no evidence that marijuana causes spasm and no suggestion of localized trauma or subarachnoid hemorrhage that might cause a problem unrelated to his drug ingestion. We do not think that the cause of the stroke was an embolism for the reasons previously stated.

References
