We are reporting a case of reversible cerebral angiitis and intracerebral hemorrhage in a methamphetamine abuser who had grand mal seizures. The characteristic radiographic changes were demonstrated by selective cerebral angiography and computerized tomography. The vascular abnormalities were no longer present after 1 month of treatment with prednisone and continued abstinence from drugs. The pertinent literature is reviewed.

Case Report

This 16-year-old right-handed white male high school student was in good health until the morning of admission. He went to school that morning as usual, where he developed nausea and vomiting. He was sent home. He had a generalized seizure with a complete loss of consciousness at home, as witnessed by his mother. He had a second grand mal seizure on the way to the emergency room. In the emergency room he had a third seizure, lasting a couple of minutes.

Examination. Approximately 30 minutes after the third attack, examination in the emergency room showed the results that follow. His blood pressure was 140/90 mm Hg. Respiration was shallow and regular. There was sinus tachycardia, with a heart rate of 100 beats/min. He was a well developed, well nourished boy with no external evidence of injuries. His neck was supple, his lungs were clear, and the abdomen was soft. His liver and spleen were not palpable. There was a scar in the lower back from a previous excision of a nevus, about a year earlier. Neurological examination was conducted while he was lying in bed with restraints. He looked drowsy but arousable. His face was flushed. At times he was slightly confused. He spoke coherently when awake and followed simple verbal commands. Funduscopic examination was unremarkable. Pupils were briskly reactive to light. External ocular movements were full. There was no facial weakness. He moved all four extremities purposefully. Deep tendon reflexes were very brisk and symmetrical. Plantar responses were flexor. There was no ankle clonus. Double simultaneous stimulation, stereognosis, and optokinetic nystagmus were normal.

Hospital Course. Intravenous fluid infusion was started with 5% dextrose/0.45% NaCl in the emergency room, and the patient was given Dilantin (phenytoin). Lumbar puncture revealed an opening pressure of 110 mm H2O. Cerebrospinal fluid (CSF) was grossly clear, with normal protein and sugar. Microscopic examination revealed 30 red blood cells and no white blood cells. Complete blood count, urinalysis, and chemical profile were normal. Serological tests were normal. Culture of CSF revealed no growth. Toxicology screening for barbiturate, ethanol,
salicylate, glutethimide, and meprobamate was negative. No further seizures developed during the subsequent hospitalization. The patient was gradually mobilized. In the meantime, he complained of mild frontal and retro-orbital headache.

Computerized tomography of the brain revealed an increased density in the left anterior parietal lobe, with some surrounding edema. This finding was consistent with acute intracerebral hemorrhage (Fig. 1). Selective left internal and external carotid arteriography was carried out via a transfemoral approach. The left internal carotid examination showed diffuse pronounced irregularity of flow, indistinctness, and beading of the anterior and middle cerebral arteries (Fig. 2 left). The selective left external carotid angiogram showed the superficial temporal artery to be normal. A renal angiogram was also performed and was normal. Despite the normal external carotid angiographic picture, a temporal artery biopsy was carried out and found normal. Further laboratory work, including testing for erythrocyte sedimentation rate, antinuclear antibody, lupus erythematosus, and rheumatoid factor were normal. Serum complement levels were normal. Serum protein electrophoresis and immunoglobulin electrophoresis were normal. Echocardiogram was normal. These separate blood cultures showed no growth.

The patient admitted that he had abused methamphetamine, LSD, and marijuana, at least for the preceding 6 months. Subsequently, he was placed on prednisone, 20 mg three times daily, and continued an uneventful hospital course, with the headache gradually subsiding. He was discharged on Dilantin, 100 mg three times daily, as well as prednisone, which was continued for an additional 1 month. The patient has denied further drug abuse since his hospitalization. Six weeks after discharge the repeat selective internal carotid arteriogram was entirely normal (Fig. 2 right). Clinical follow-up review over a 1-year period has been uneventful.

Discussion

Even though it is rather uncommon to see abuse of a single drug, the literature suggests that metham-

![Fig. 1. Computerized tomography scan showing an acute intracerebral hemorrhage in the left anterior parietal lobe.](image)

![Fig. 2. Left: Selective left internal carotid arteriogram showing diffuse pronounced irregularity, indistinctness of the vessel outline, and beading of the anterior and middle cerebral arteries. Right: Repeat carotid arteriogram after 1 month treatment with prednisone showing normal anterior and middle cerebral arteries.](image)
Angiitis and cerebral hemorrhage in drug abuse

Amphetamine is the most common denominator in multiple-drug abuse cases when vascular insult and/or hemorrhage result. Citron, et al., reported 14 cases of necrotizing angiitis associated with drug abuse. The drugs abused varied from narcotics and hallucinogens, to stimulants and sedatives. Among these drugs, methamphetamine was the most common. The characteristic features of the vascular pattern of necrotizing angiitis observed included microaneurysms, indistinctness of vessel outlines, segmental luminal irregularities, and thrombosis of the vessels of the lung, kidney, liver, pancreas, and small bowel. These changes took place mainly in the medium-sized and small arteries in most body organs and in the arterioles in the brain. Fibrinoid necrosis was apparent in the media and intima, with cellular infiltration. These changes were not distinguishable from those of periarteritis nodosa. They were different from hypersensitivity angiitis, which frequently involves small arteries, capillaries, and venules. Margolis and Newton described changes of the branches of the middle and anterior cerebral arteries seen after intravenous administration of methamphetamine. These changes were characterized by pronounced irregularity and beading of these arteries.

Our patient was involved in approximately 6 months of constant methamphetamine abuse. Selective external carotid angiography revealed the superficial temporal artery to be entirely normal. As expected, the biopsy of superficial temporal artery turned out to be normal in view of normal angiographic findings. But selective internal carotid angiography showed characteristic findings of diffuse pronounced irregularity of flow and beading of the anterior and middle cerebral arteries, as shown by Margolis and Newton. Arterial narrowing (spasm) was also observed. These findings were reversible changes, as clearly shown on the subsequent repeat selective internal carotid angiography after a month of prednisone and the end of the drug abuse.

It is interesting to note that these arterial changes were associated with the presence of an intracerebral hemorrhage. Delaney and Estes examined the literature for cases of intracranial hemorrhage associated with amphetamine abuse. The incidence of intracranial hemorrhagic complications associated with amphetamine abuse was higher in males by two to one. Seven patients died as a result of this complication. The mortality rate associated with this complication was nearly 50%.

In spite of the absence of the histological confirmation, the evidence suggests that our patient had drug-induced arteritis of the cerebral vessels. This, in turn, resulted in the intracerebral hemorrhage. The vascular changes seen in the carotid angiography were reversible after 1 month of treatment with steroid, while the patient denied resumption of drugs. While this paper was being prepared, a case of massive intraventricular and subarachnoid hemorrhage in a phencyclidine (PCP)-intoxicated patient was reported.

References


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