Pathological and radiological correlation of subarachnoid hemorrhage in phencyclidine abuse

Case report

OREST B. BOYKO, M.D., PH.D., PETER C. BURGER, M.D., AND E. RALPH HEINZ, M.D.

Department of Radiology, Indiana University School of Medicine, Indianapolis, Indiana, and Departments of Radiology and Pathology, Duke University Medical Center, Durham, North Carolina

Although hypertension can be associated with phencyclidine (PCP) use, subarachnoid hemorrhage (SAH) is a rare result. The radiological and pathological findings are reported of a patient with acute SAH who had chromatographic evidence of PCP in his blood. The occurrence of SAH in a patient who uses PCP may be caused by a disrupted arterial vessel wall and/or vasospasm due to the pharmacological action of the drug on the cerebral vasculature.

KEY WORDS • phencyclidine • subarachnoid hemorrhage • substance abuse

INCLUDED in the spectrum of cerebrovascular disease is an association between stroke and drug abuse. Cerebral complications in drug abusers have included intracerebral hemorrhage, subarachnoid hemorrhage (SAH), emboli, ischemic stroke, mycotic aneurysm, and hypertensive encephalopathy. It is rare for SAH to be a presenting finding in drug abuse unrelated to methamphetamine use, and it is even rarer for SAH to be associated with phencyclidine (PCP) abuse.

In this case report, we present the radiological and pathological findings in a patient who presented with acute SAH after PCP abuse. At autopsy, a 1-mm perforation of the ventral surface of the basilar artery was the anatomic cause for the SAH. There was no vasculitis. In two previous case reports of intracranial hemorrhage associated with PCP, the exclusion of vasculitis and the exact vascular etiology for the hemorrhage could not be determined.

Case Report

This 17-year-old boy presented to an outside hospital after having fallen off a bar stool at a video arcade while experiencing a sudden onset of headache and syncope. On admission, examination revealed the following pertinent findings: blood pressure 100/70 mm Hg, cyanosis, and tachypnea with a dilated right pupil. A chest radiograph demonstrated pulmonary edema. The clinical impression was probable aspiration pneumonia and noncardiogenic pulmonary edema; there was a question of myocardial infarction. The patient had a history of labile hypertension (210/100 mm Hg) discovered on a routine examination by his family physician, but he was normotensive on follow-up visits. His family reported that on occasion he had “fits of rage” and in the past month he had suffered two to three headaches over the vertex of his head, but they had no knowledge of drug or alcohol abuse.

The patient was treated with morphine, digoxin, furosemide, lidocaine, dopamine, and acetaminophen. An acute myocardial infarction was excluded, and he was transferred to our institution 24 hours after admission to the outside hospital.

Course. After transfer he became responsive to pain after receiving intravenous Narcan (naloxone hydrochloride). He was breathing spontaneously with papilledema in the left optic disc and minimally responsive pupils. Noncontrast computerized tomography (CT) showed subarachnoid and intraventricular hemorrhage. A focal circular area of increased attenuation surrounded the interpeduncular cistern portion of the basilar artery, suggesting the presence of a hematoma and this vessel as the site of SAH. Four-vessel cerebral angiography showed no extravasation of contrast material, aneurysm, arteriovenous malformation, vasculitis, or spasm.

Approximately 24 hours after the initial episode of headache and syncope, a drug screen performed by the toxicology laboratory using thin-layer chromatography demonstrated PCP in the blood. Lumbar puncture...
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yielded bloody cerebrospinal fluid (CSF) with 226,756 red blood cells and 303 white blood cells. Electrocardiography was normal.

The patient received Amicar (aminocaproic acid), 18 gm/12 hrs, and an intracranial pressure monitor showed an initial reading of 30 mm Hg with bloody CSF drainage. He required ventilator support, but was weaned off the respirator 5 days after admission; 2 days later he required reintubation after suffering an episode of unresponsiveness and respiratory distress. Repeat CT showed residual SAH with no new areas of bleeding. Angiography the same day revealed vasospasm of both supraclinoid internal carotid arteries and of the upper basilar artery but no site of bleeding. The patient was started on a course of Decadron (dexamethasone), with 5 mg of verapamil. His neurological status deteriorated and he was pronounced brain dead 11 days after admission.

Postmortem Examination. Autopsy demonstrated subarachnoid and intraventricular hemorrhage, with a 1-mm longitudinal perforation in the ventral surface of the basilar artery surrounded by a hyperemic border (Fig. 1). This segment of basilar artery was serially sectioned, and histological analysis revealed complete perforation of the ventral wall of the basilar artery (Fig. 2). As evidence for antemortem rupture, there was hemorrhage on the adventitial surface and a layering of fibrin along the transection line (Fig. 2). Within the transected vessel wall, only a rare inflammatory cell representing a degenerating polymorphonuclear leukocyte was present. No histological defects of the internal elastic layer could be demonstrated with special stains in other areas of the basilar artery. Microscopic examination showed no vasculitis in the basilar artery or in the internal carotid, vertebral, posterior cerebral, or middle cerebral arteries. No cerebral aneurysms were identified. Examination of the cardiovascular system was remarkable only for the presence of mild cardiomegaly (heart weight 465 gm). Microscopically, other organs demonstrated no evidence of chronic intravenous drug abuse.

Discussion

Phencyclidine hydrochloride, 1(1-phenylcyclohexyl)piperidine (PCP) hydrochloride, is a popular illicit recreational drug. This drug has been noted to have serious psychotomimetic and pressor side effects. 6 In a review of 1000 patients with PCP intoxication, 13 intracranial hemorrhage was not reported even though 106 patients were brought to medical attention in coma. To our knowledge, intracranial hemorrhage associated with PCP abuse has been reported in only two previous cases, 7 and SAH was present in only one of these. In both cases the exact vascular etiology for the hemorrhage was never conclusively demonstrated and vasculitis was not ruled out histologically.

Our case demonstrates that a perforation in the ventral surface of the basilar artery occurred antemortem and was the cause of SAH (Figs. 1 and 2). Vasculitis was not present and therefore does not appear to be a necessary component for the mechanism of SAH associated with PCP abuse. This suggests that the hypertensive pharmacological effect of PCP 6 could have been the cause of SAH. The only pertinent pathological finding in examination of the cardiovascular system was mild cardiomegaly of unknown origin.
Autopsy findings in one of the previously reported cases of intracranial hemorrhage with PCP included hemorrhage involving the left internal capsule and striate bodies in the distribution that would be expected of a hypertensive bleed of the left middle cerebral artery (Office of the Medical Examiner of Wayne County, Michigan, personal communication, 1983). The fact that the patient was a 13-year-old boy even further implicated the pressor effect of PCP. All three cases of intracranial hemorrhage with PCP abuse reported to date have occurred in males who were aged 20 years or younger.

The spectrum of cerebrovascular disease secondary to drug abuse has included such mechanisms of vascular injury as endocarditis, direct toxic injury, embolization of foreign matter, immunological mechanisms, and pharmacologically mediated vascular changes. Both cocaine and lysergic acid diethylamide (LSD) mediate their effects through their pharmacological properties. This is in contrast to methamphetamine abuse, where changes in the cerebral vasculature associated with necrotizing angiitis and vasculitis are found. Our case lends further support for the categorization of cerebrovascular disease associated with PCP to be secondary to its pharmacological properties and not to vasculitis. Recently, the use of verapamil in the reversal of cerebral vasospasm presumed to be secondary to PCP intoxication has been reported.

The basilar artery as the site of SAH in our case offers interesting possibilities concerning pathogenesis, especially in view of the recent demonstration of PCP receptors in the basilar and middle cerebral arteries of canines. The area of rupture on the ventral surface of the basilar artery closely corresponds to the anatomic location of the embryological development and regression of the primitive trigeminal artery. and a congenital structural weakness might have been left. Usually, the primitive trigeminal artery will anastomose with the basilar artery below the origin of the superior cerebellar artery and above the origin of the posterior inferior cerebellar artery. The point of anastomosis occurs on either side of the basilar artery rather than on the ventral surface. Aneurysms of the persistent trigeminal artery have been reported previously.

Additional considerations for the susceptibility of the ventral surface of the basilar artery to rupture would include a medial defect of congenital origin of the basilar artery wall as outlined by Forbus, or an acquired breach in the internal elastic layer, termed “Reuterwall’s tears.” Reuterwall’s tears of the basilar artery have been described as occurring both in longitudinal and transverse directions, singly or in multiples, and they have been found in patients less than 20 years of age. Such a lesion was not found postmortem in our case, although a small tear could have preceded the fatal rupture. Reuterwall’s tears were not found in any of the other cerebral vessels studied histologically.

In summary, our case lends further evidence for the need to consider the sympathomimetic and contractile response of cerebral vessels to PCP when patients who abuse this drug present with neurological sequelae.

References

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Address reprint requests to: Orest B. Boyko, M.D., Ph.D., Department of Radiology, 926 West Michigan Street, Indiana University School of Medicine, Indianapolis, Indiana 46223.