Multiple Intracerebral Hemorrhages After Smoking “Crack” Cocaine

Richard M. Green, MD, Kevin M. Kelly, MD, PhD, Trygve Gabrielsen, MD, Steven R. Levine, MD, and Chris Vanderzant, DO

After smoking “crack” cocaine and consuming large quantities of ethanol, a 36-year-old man developed multiple, bilateral, deep, and superficial cerebral hematomas. He was hypertensive for several days, but angiography revealed no evidence of vascular malformation or vasculitis. The multifocality of the hematomas and lack of underlying disease suggest that the hemorrhages resulted from cocaine-induced acute hypertension or arterial spasm, possibly potentiated by heavy ethanol consumption. (Stroke 1990;21:957–962)

Subarachnoid or intracerebral hemorrhage has been temporally related to cocaine abuse in 65 previously reported cases. We describe multiple bilateral intracerebral hemorrhages in a patient with a history of cocaine and ethanol abuse.

Case Report

A 36-year-old right-handed black man smoked several vials of “crack” cocaine and drank large quantities of vodka and beer over several hours while visiting friends. He developed headache, abdominal pain, and nausea, and as he attempted to leave the apartment, his friends saw him slump to the hallway floor, then slide down a flight of 14 steps without apparent injury. He was admitted to a local hospital in a stuporous state, where examination revealed a temperature of 96.1°F, pulse of 80/min, blood pressure of 148/88 mm Hg, and respiratory rate of 16/min. He later awoke but was combative and disoriented and was also described as having “periodic whole-body trembling.” His serum ethanol level was 249 mg/dl, and the urine drug screen was positive only for cocaine. The next morning he was transferred to another local hospital. He was drowsy but answered questions appropriately. He demonstrated a left hemiparesis and bilateral Babinski signs. Computed tomography (CT scan) revealed multiple bilateral intracerebral hemorrhages (Figure 1).

The patient was transferred to our institution. His medical history, which was obtained from relatives, was normal. Although he was taking no prescribed medications, he had a history of alcoholism and drug abuse, including heroin and cocaine by nasal, freebase, and intravenous routes. He had not used intravenous drugs for several months but had smoked crack at least twice a month for the past year. Examination revealed a thin man with a temperature of 98.8°F, pulse of 104/min, blood pressure of 152/90 mm Hg, and respiratory rate of 20/min. There were no signs of scalp or facial trauma such as contusions, abrasions, or lacerations. Old needle tracks were evident on the volar surface of the left forearm. The lungs were clear, and there were no cardiac murmurs. He occasionally vocalized but did not follow commands or respond to questions. There was early bilateral papilledema and anisocoria; the right pupil measured 4 mm in diameter, and the left was 3 mm. He had a left hemiparesis with intermittent decorticate posturing of the right side and bilateral Babinski signs. Laboratory studies revealed the following: hemoglobin concentration 14.7 g/dl, hematocrit 44.2%, leukocyte count 11,000/mm³, platelet count 125,000/mm³, prothrombin time 13.9 seconds (ratio 1.1), partial thromboplastin time 22.1 seconds, bleeding time 2.5 seconds, fibrinogen concentration 286 μg/ml, fibrin split products ratio 8, serum aspartate aminotransferase concentration 176 μg/ml, serum alanine aminotransferase concentration 187 μg/ml. Normal studies included electrolytes, arterial blood gases, electrocardiography, chest x-ray, and echocardiography. The electroencephalogram was diffusely slow with right-sided emphasis.

Treatment of the patient’s increased intracranial pressure included intubation, hyperventilation, and intravenous mannitol and dexamethasone. Triple antibiotic treatment was started empirically for pos-
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FIGURE 1. Computed tomograms without contrast enhancement showing multiple bilateral intracerebral hemorrhapses 12 hours after the ictus.

Possible endocarditis. Sodium nitroprusside was infused for 48 hours to control blood pressures rising as high as 200/120 mm Hg. Antibiotics were discontinued when the first four blood cultures were negative. Four-vessel cerebral angiography on the third day of illness revealed some vessels displaced by the known hematomas, but was otherwise normal. Lumbar puncture revealed an opening pressure >370 mm; the cerebrospinal fluid was xanthochromic with a protein concentration of 101 mg/dl, a glucose concentration of 79 mg/dl, an erythrocyte count of 29,000/mm³, and a leukocyte count of 7/mm³. All cerebrospinal fluid smears, cultures, and bacterial and fungal latex agglutination titers were negative. Serologic workup revealed hepatitis B core and surface antibody positivity; HIV antibody was negative. On the fourth hospital day his temperature rose to 102°F. Blood cultures grew Escherichia coli in one aerobic
bottle. All indwelling catheters were changed, and he received a 14-day course of intravenous cefazolin. Blood pressures returned to the normal range by the fifth hospital day, and the platelet count rose to 310,000/mm$^3$ by the end of the first week. Brain CT on the twentieth hospital day revealed the expected evolution of the hematomas but no evidence of infarction. Plasma cholinesterase was 16.7 IU/ml (normal range 7.0–19.0).

He was discharged to a rehabilitation facility after 5 weeks, and he returned home 5 weeks later. When he was seen in the clinic 3 months after his ictus, he was alert, conversant, and ambulatory. Examination revealed mild left-sided spatial neglect, constructional apraxia, bilateral spastic hemiparesis that was worse on the left side, and brisk reflexes bilaterally.

**Discussion**

This case is unusual because of the large number of deep and superficial hematomas in both cerebral hemispheres. Multiple simultaneous intracranial hematomas are uncommon, and only double lesions are usually described. Freytag$^{23}$ found more than one hematoma in 13 of 393 (3%) autopsied cases of fatal hypertensive brain hemorrhage. McCormick and Rosenfield$^{24}$ found multiple hematomas in 16 of 144 (11%) autopsied patients with massive nontraumatic brain hemorrhage. In Weisberg’s series$^{25}$ of consecutive cases of nontraumatic intracerebral hematoma diagnosed by CT, only 12 of 600 (2%) cases had multiple hematomas.

Our patient’s multiple simultaneous hemorrhages were associated with abuse of crack cocaine and ethanol. The differential diagnosis of multiple simultaneous intracerebral hematomas includes trauma, hypertension, leukemia or bleeding diathesis, metastatic or primary neoplasms, venous sinus thrombosis, vasculitis, septic or aseptic emboli, amyloid angiopathy, phenylpropanolamine ingestion, ataxia-telangiectasia, and no identifiable etiology. Head trauma is a frequent cause of intracranial bleeding, but the clinical and radiographic features of this case are not consistent with trauma. Our patient was not observed to strike his head, and there was no scalp or facial injury, skull fracture, or epidural or subdural blood. After closed head injury, purely intracerebral hematomas are much less common than either epidural or subdural lesions. Traumatic intracerebral hematomas are most common in the frontal and temporal lobes, often associated with cortical contusions or lacerations, or situated beneath skull fractures. Contrasting with our patient’s CT findings, traumatic hematomas tend to be irregular in contour, poorly demarcated, and nonuniform in density. Our patient had no history of hypertension, which is rarely implicated as the sole cause of multiple intracerebral hemorrhages. Blood pressures soon after his ictus were only mildly elevated, and the subsequent higher elevations paralleled the course of his increased intracranial pressure. A bleeding diathesis was excluded by a normal platelet count and coagulation parameters. Bacterial endocarditis was excluded by his clinical findings, diagnostic evaluation, and hospital course.

We know of 65 previously reported cases$^{1–22}$ of subarachnoid or intracerebral hemorrhage that were temporally related to cocaine abuse (Table 1). Subarachnoid hemorrhage occurred in 29 patients, and intracerebral hemorrhage occurred in 36 patients. For nine of these patients only a few clinical details were reported, and these were excluded from the following analysis. Thus, the following clinical values are for 27 patients with subarachnoid hemorrhage and for 29 patients with intracerebral hemorrhage.

The mean age of patients with subarachnoid hemorrhage was 31.4 (range 19–63) years; 14 of 27 were men. The mean age of those with intracerebral hemorrhage was 34.2 (range 22–57) years; 20 of 29 were men. The clinical presentations of subarachnoid and intracerebral hemorrhage related to cocaine have been similar: headache, altered mental status, lateralized deficits, and seizures, in varying combinations. Sudden death was also a presenting feature.

Angiographic or autopsy data were reported for 26 of the 27 cocaine-related cases of subarachnoid hemorrhage. A saccular aneurysm was identified in 21 of the 27 (78%) cases. Of these, nine were of the anterior communicating artery, six of the posterior communicating artery, and one each of the following arteries: superior cerebellar, middle cerebral, anterior cerebral, anterior cerebral–middle cerebral junction, circle of Willis, and an unspecified location.

Angiographic or autopsy data were reported for 25 of the 29 cases of intracerebral hemorrhage. An underlying lesion was found in 12 of the 25 (48%) cases: arteriovenous malformation in seven, saccular aneurysm in two, arterial “irregularities” and “beading” in two, and an astrocytoma in one. In six of the remaining 13 patients, who experienced intracerebral hemorrhage but no apparent vascular lesion, the hematoma was situated in the subcortical white matter. In seven of these 13 patients, three patients had basal ganglionic hemorrhages, three had massive, poorly localized hemorrhages, and one had a thalamic hemorrhage. The high percentage of lobar hematomas is not typical of hypertensive intracerebral hemorrhage, which most commonly involves the basal ganglia and internal capsule. In series of lobar cerebral hemorrhage, hypertension was identified as a potential etiologic factor in approximately one third of the cases. Many cases of lobar intracerebral hemorrhage of undetermined etiology may be due to small cryptic vascular malformations.

In these previous studies,$^{1–20}$ a predisposing vascular lesion has been identified in 78% of cocaine users with subarachnoid hemorrhage and in 48% of those with intracerebral hemorrhage. Cocaine presumably induces an acute rise in blood pressure or vasoconstriction, rupturing the aneurysm or vascular malformation. The pathogenesis of cocaine-induced hemorrhage in cases without a vascular lesion is less certain. Despite the predominantly lobar location of hematomas in these cases, acute hypertension could still be an
### Table 1. Features of Intracranial Hemorrhage With Cocaine Abuse

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Route</th>
<th>Presentation</th>
<th>Other drugs or factors</th>
<th>Brain CT findings</th>
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<td>M</td>
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<td>Ruptured R SCA aneurysm</td>
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<td>Alcohol</td>
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<td>R PCoA aneurysm</td>
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<tr>
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<td>34</td>
<td>F</td>
<td>?</td>
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<td>?</td>
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<td>Not performed</td>
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<td>R PCoA aneurysm</td>
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<td>SAH</td>
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<td>Morphine</td>
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<td>43</td>
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<td>HTN</td>
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<td></td>
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<td>Caplan et al¹⁸</td>
<td>22</td>
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<td>L posterior frontal AVM</td>
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<td>HA, SZ</td>
<td>Angina</td>
<td>R frontal hem</td>
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TABLE 1. Features of Intracranial Hemorrhage With Cocaine Abuse (continued)

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<tr>
<th>Author</th>
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<th>Route</th>
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<th>Other drugs or factors</th>
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<td>Lowenstein et al</td>
<td>32</td>
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<td>Intranasal</td>
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<td>Coitus before ictus</td>
<td>R BG and IC hem</td>
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<td>Mody et al</td>
<td>34</td>
<td>M</td>
<td>?</td>
<td>Not reported</td>
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<td>R parieto-occipital hem</td>
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<tr>
<td></td>
<td>30</td>
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<td>L parietal hem</td>
<td>Abn L pericallosal and L PCA</td>
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<tr>
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<td>M</td>
<td>Oral</td>
<td>Not reported</td>
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<td>R frontoparietal AVM</td>
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<td></td>
<td>28</td>
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<td>?</td>
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<td>Not performed</td>
<td>L parietal hem*</td>
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<td>Benzodiazepine</td>
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<td>45</td>
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</tbody>
</table>

CT, computed tomogram; M, male; R, right; SCA, superior cerebellar artery; F, female; HA, headache; ACoA, anterior communicating artery; PCoA, posterior communicating artery; SAH, subarachnoid hemorrhage; IVH, intraventricular hemorrhage; SZ, seizure; ACA, anterior cerebral artery; MCA, middle cerebral artery; L, left; HTN, hypertension; SDH, subdural hemorrhage; LOC, loss of consciousness; CoW, circle of Willis; hem, hemorrhage; AVM, arteriovenous malformation; PCA, posterior cerebral artery; vent, ventricular; BG, basal ganglia; IC, internal capsule; abn, abnormal; phenylprop, phenylpropanolamine; ICH, intracerebral hemorrhage.

Tuchman et al evaluated five men aged 21-40 years with intracranial (two with subarachnoid, three with intracerebral) hemorrhage related to cocaine. Nalls et al reported four cases of basal gangliocerebral hemorrhage related to cocaine. In these reports, other clinical features were not described.

*Autopsy data.
important etiologic factor. Alternatively, intracerebral hemorrhage may occur in some cases after an initial phase of ischemia. Severe cerebral ischemia or infarction may occur during cocaine-induced vasospasm. Hemorrhage might then result from an acute increase in blood flow once the vasospasm subsides, rupturing vessel walls that were subjected to ischemic damage.26

Other sympathomimetic agents, especially phenylpropanolamine, ephedrine, and methamphetamine, have also been implicated in intracranial bleeding. Phenylpropanolamine, even in a single dose, has been associated with multiple bilateral intracranial hemorrhages.27,28 Angiography often demonstrates “beading” and focal narrowing in large- and medium-sized vessels, suggesting cerebral vasculitis.28 However, there is no pathologic evidence that cocaine causes a cerebral vasculitis.

Our patient was also abusing ethanol at the time of his hemorrhage. Ethanol may potentiate the hypertensive and vasoconstrictive effects of cocaine, increasing the risk of intracranial hemorrhage. Ethanol has a pressor effect that may contribute to acute and chronic rises in blood pressure29 and also exerts a direct action on cerebrovascular smooth muscle, causing vasospasm.30 Acute ethanol ingestion may also reduce the hepatic metabolism of cocaine and thereby prolong its pressor effects.31 Ethanol intoxication is a risk factor for subarachnoid hemorrhage,32 and population studies demonstrate that regular consumption of alcohol is associated with an increased incidence of intracranial hemorrhage.33 Ethanol has direct toxic effects causing thrombocytopenia and impaired function of circulating platelets.34

Thus, several mechanisms potentially contribute to the intracranial hemorrhages associated with cocaine and ethanol abuse. Rupture of an aneurysm or vascular malformation accounts for many intracranial hemorrhages related to cocaine abuse, but severe hemorrhages such as those suffered by our patient may occur in the absence of a predisposing vascular lesion.

References
34. Key Words • cerebral hemorrhage • cocaine • alcohol drinking • hypertension
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