

Acute Cocaine Abuse Associated With Cerebral Infarction

We present the case of a 29-year-old patient who developed a cerebral infarction, without hemorrhage, after using cocaine. A computed tomography scan of the brain at the time of initial presentation was entirely normal; a repeat CT scan on the fifth hospital day showed a basal ganglion infarction. No evidence of intracranial bleeding was detected. Theories of cocaine-induced cerebral infarction, including promotion of thrombogenesis and vasospasm, are reviewed. [Seaman ME: Acute cocaine abuse associated with cerebral infarction. Ann Emerg Med January 1990;19:34-37.]

INTRODUCTION

Cocaine abuse leads to a variety of central nervous system complications, including toxic psychosis, seizures, intracranial bleeding, and subarachnoid hemorrhage.¹ Reports of ischemic cerebral infarction temporally associated with cocaine abuse also have been described.²⁻⁷ The mechanism by which cerebral infarction occurs has not yet been clearly defined; however, a variety of studies supports several possible mechanisms by which infarction can occur and includes promotion of thrombogenesis, vasospasm, impaired cerebral autoregulation, and adverse effects on cerebral metabolism. The case of a patient who abused cocaine and then acutely developed an ischemic cerebral infarction is presented.

CASE REPORT

A 29-year-old woman smoked 1 g of "crack" cocaine during approximately one hour and then had a tonic-clonic seizure of one minute duration that was witnessed by a friend. The patient initially was disoriented but quickly became conversant and oriented. She had no history of seizure, stroke, or hypertension. Her medical history was notable for a dilated cardiomyopathy with a left ventricular ejection fraction of 24% documented eight months before the patient's current admission; the etiology of the patient's cardiomyopathy was uncertain but could have been a postpartum cardiomyopathy. The patient admitted to using cocaine at least once a week. She was a former cigarette smoker but reported she had stopped smoking "several years previously." She did not use birth control pills but did use furosemide, digoxin, potassium, folate, and captopril.

Vital signs showed a blood pressure of 129/93 mm Hg; pulse, 108; respirations, 30; and temperature, 37.4 C. On examination, the patient was alert but disheveled. No evidence of head trauma was found. Pupils were of equal size and reactive; the fundi were benign. Extraocular movements were intact. Auscultation of the neck showed no bruit. Few bibasilar rales were noted on auscultation of the chest. A II/VI systolic murmur was noted at the cardiac apex with radiation to the axilla. No rub or gallop was detected. Extremities showed no clubbing, cyanosis, or edema. Neurologic examination was notable for a left central seventh cranial nerve deficit; the remainder of the cranial nerves were intact. Motor testing showed a flaccid paralysis in the left upper extremity and $\frac{3}{5}$ strength in the left lower extremity. Diminished reflexes were noted on the left side. Sensation was intact. Babinski reflexes were flexor on both sides.

An initial noncontrast computed tomography (CT) scan of the brain was normal, with no evidence of cerebral infarction or hemorrhage (Figure 1). An arterial blood gas on room air showed a pH of 7.43; PO_2 , 78 mm Hg;

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FIGURE 1. CT scan of brain at time of admission showing no evidence of initial bleed or infarction.

and PCO_2 , 31 mm Hg. An ECG showed sinus tachycardia at 120; T-wave inversions in leads V_5 and V_6 were noted but were unchanged from previous ECGs. Electrolyte analysis showed a bicarbonate of 16 mEq/L; otherwise, all values were normal. The hemoglobin was 11.2 mg/dL; hematocrit, 32.1 mL/dL; and white blood cell count, 8.2×10^3 cells/ μL . Toxicologic screening (Emit® st®, Syva Co, Palo Alto, California) was positive for cocaine but negative for all other drugs tested (ie, amphetamine, barbiturates, benzodiazepine, cannabinoids, alcohol, opiates, phencyclidine, and cyclic antidepressants).

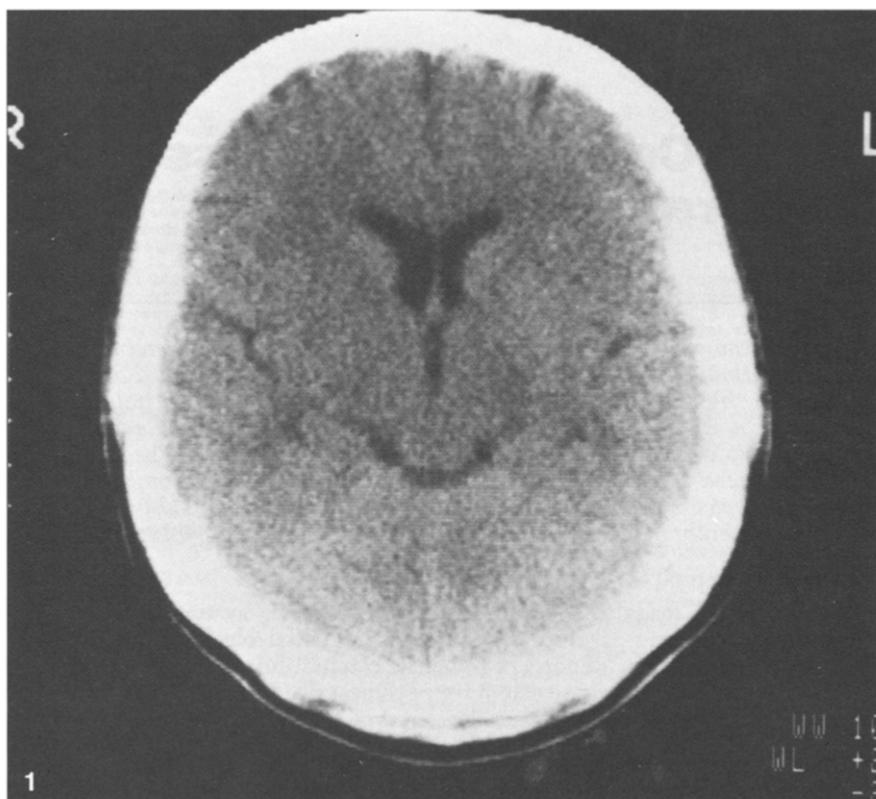
Further workup on the second hospital day included an echocardiogram, which showed no valvular vegetations or thrombus. Holter monitoring on the third hospital day showed rare premature ventricular contractions and several short runs (four to seven beats) of ventricular tachycardia. A repeat noncontrast CT scan of the brain on the fifth hospital day showed a cerebral infarction in the right basal ganglion (Figure 2).

The patient remained hospitalized for three weeks with improvement in left leg strength but no improvement in the left arm motor deficit.

DISCUSSION

Since 1986, there have been reports of at least 11 patients who developed ischemic strokes proven by CT scan after abusing cocaine.²⁻⁷ Ten were men. One was a newborn whose mother abused cocaine. The ages of the other patients ranged from 23 to 38 years with a mean age of 31 years. Most patients reported smoking cocaine,^{3,6,7} although IV use⁵ and inhalation of aerosolized cocaine⁴ also have been associated with cerebral infarction. (Our patient had smoked cocaine.)

The onset of symptoms has been variable, with some infarcts occurring during use;^{4,6,7} some patients are not aware of their hemiparesis until many hours after cocaine use.^{3,7} In the group of ten adults with cerebral infarction proven by CT, cerebral angiography was performed on five patients; evidence of arterial sten-



osis or occlusion was seen in four studies, and vascular spasm was induced in one patient.^{3-5,7}

In contrast to the recent reporting of cerebral infarction, there have been multiple other reports of hemorrhagic cerebral vascular accidents associated with cocaine abuse, including intracerebral bleeding and subarachnoid hemorrhages.⁸⁻¹⁸ One patient has been reported to have developed cerebral vasculitis associated with cocaine abuse,¹⁹ although the validity of the diagnosis has been questioned.²⁰

Evidence supports a variety of factors contributing to cocaine-induced cerebral infarction, but the precise mechanism of ischemic cerebral infarction has not been firmly established. Several articles describe mechanisms of cerebral injury associated with cocaine, including vasospasm, thrombogenesis, impaired cerebral vascular autoregulation, cocaine-induced cerebral metabolic abnormalities, and other possible factors.

Cerebral vasospasm resulting from cocaine has been proposed as one mechanism of cerebral infarction.⁴ Cocaine is known to block reuptake of the potent cerebral vasoconstrict-

tors norepinephrine and serotonin.²¹⁻²⁵ Additional thrombotic complications associated with cocaine abuse have been described, including coronary thrombosis^{26,27} and renal artery thrombosis.²⁸ Muscle and skin infarction also have been reported.²⁹ Cocaine has been shown to inhibit prostacyclin and increase platelet aggregation *in vitro*;³⁰ these effects may contribute to thrombogenesis. Lupus anticoagulant was found in one user of IV cocaine and believed to have promoted thrombogenesis in a previous case.⁵

Hypertension associated with acute cocaine abuse could be associated with failed cerebral autoregulation of blood flow. It has been suggested that the effect of acute hypertension on cerebral autoregulation can lead to brain edema and cell death.³¹⁻³³ Ventricular dysrhythmias have been associated with cocaine abuse;¹ cerebral hypoperfusion associated with a ventricular dysrhythmia could cause a cerebral infarction. Cocaine also has been shown to depress cerebral metabolism,³⁴ produce cellular structural abnormalities, and diminish neural cell viability;³⁵ these effects also may contribute to cerebral infarction in patients who

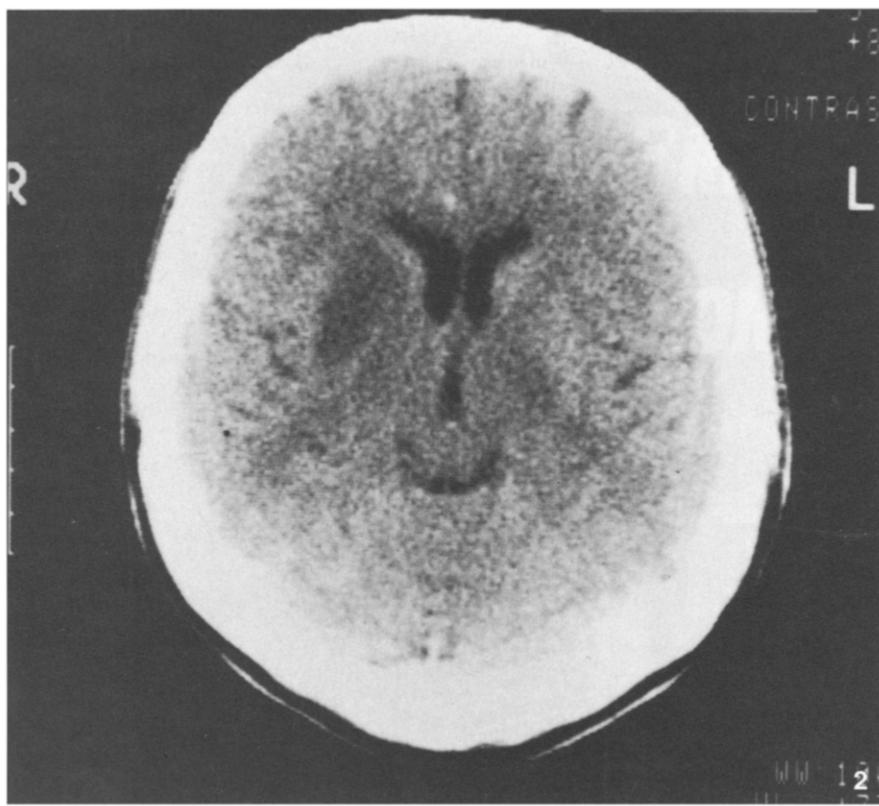


FIGURE 2. CT scan of brain on fifth hospital day showing infarction of right basal ganglion with impingement on internal capsule.

abuse cocaine.

Our patient was young but could be considered at risk for a cerebrovascular accident on the basis of her underlying cardiomyopathy. However, there was no evidence of mural thrombus in the ventricles. The temporal relationship of cocaine abuse to the onset of the patient's ischemic cerebral infarction points to cocaine as the most likely precipitant of this event.

SUMMARY

The case of a 29-year-old woman who smoked cocaine and then developed a cerebral infarction is discussed. This case emphasizes that neurologic deficits after cocaine abuse may be due not only to intracranial bleeding or subarachnoid hemorrhage but also to cerebral infarction. Cocaine may promote cerebral ischemia and infarction by a variety of possible mechanisms, including promotion of thrombogenesis, vasospasm, impaired cerebral vascular autoregulation, and depression of cerebral metabolism.

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