Cerebral Infarction Associated with Cocaine Use

Iliana Meza
Carlos A. Estrada
Justo A. Montalvo
Walter N. Hidalgo
Jennie Andresen

Follow this and additional works at: https://scholarlycommons.henryford.com/hfhmedjournal

Part of the Life Sciences Commons, Medical Specialties Commons, and the Public Health Commons

Recommended Citation
Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol37/iss1/15

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons.
Cerebral Infarction Associated with Cocaine Use

Iliana Meza, MD,* Carlos A. Estrada, MD,† Justo A. Montalvo, MD,* Walter N. Hidalgo, MD,* and Jennie Andresen, MD*

We report the case of a young man with an acute infarction of the left putamen and caudate nucleus, whose symptoms appeared six hours after intranasal use of approximately 0.5 g of cocaine hydrochloride. It seems probable that in this patient cocaine consumption played a role in the development of stroke. (Henry Ford Hosp Med J 1989;37:50-1)

The clinical picture of cocaine intoxication is well known (1). However, medical complications from its abuse have only recently been reported (1,2). These include acute myocardial infarction, cardiac arrhythmias, and central nervous system involvement (1-8). We report the case of a young adult man with cerebral infarction following cocaine snorting.

Case Report

A 31-year-old male normotensive chronic cocaine user was admitted with a history of dysarthria, tonic-clonic motor seizures of the right upper limb, and right-sided hemiparesis appearing six hours after intranasal use of approximately 0.5 g of cocaine hydrochloride and heavy alcohol consumption (witnessed by his wife). He did not complain of headache. The patient's history included a similar hemiparesis episode six months earlier which resolved spontaneously in 20 minutes. There was no history of intravenous drug abuse. He suffers from chronic asthma but was taking no medications. His father died from acute myocardial infarction at age 55, and a coronary bypass graft was performed on his brother at age 46. There was no family history of cerebrovascular disease or homocystinuria.

On physical examination his blood pressure was 130/90 mm Hg (supine), heart rate was 78 beats/min, and temperature 37°C (98.6°F). General physical examination was normal. He was alert and oriented but had slurred speech. Right-sided flaccid hemiparesis was present, with brachial predominance. He also had paresis of the right 6th and 7th (central type) cranial nerves, and hyperreflexia was present on the right side. The funduscopic examination was normal.

Laboratory studies revealed a hematocrit of 0.50, WBC count of 9.9 × 10⁹/L, erythrocyte sedimentation rate of 2 mm/h, blood glucose level of 5.5 mmol/L (100 mg/dL), and serum creatinine of 88.4 μmol/L (1 mg/dL). Serum cholesterol was 5.82 mmol/L (225 mg/dL), and serum triglycerides were 0.34 mmol/L (30 mg/dL). Chest x-ray and electrocardiogram were normal. Computed tomography (CT) of the head (Figure) showed an acute infarct of the left putamen and head of the caudate nucleus surrounded by edema. The patient was treated with phenytoin sodium, steroids, and aspirin. His neurologic status improved substantially except for right upper limb motor activity, and he was discharged five days later. No urine drug screen for cocaine or other drugs was done. The patient refused further workup such as lumbar puncture or cerebral angiography.

Comments

Neuropsychiatric effects of cocaine use include euphoria, hyporexia, increased creativity and physical performance, paranoid ideation, tremors, and seizures (1). However, in recent years, acute cerebrovascular events have been reported to occur in association with cocaine use (2-8).

We have identified in the English literature seven published cases of cerebral infarction associated with cocaine use. Chasnoff et al (4) described a newborn infant whose mother had used cocaine intranasally for three days before delivery; on the day of delivery she used approximately 1 g of cocaine. This infant had an acute infarction in the distribution of the left middle cerebral artery. A second patient had snorted cocaine one day prior to developing a cerebral infarct (5). A third patient smoked two vials of “crack” four hours before the stroke (6). A fourth patient injected himself intramuscularly with cocaine one to two hours before the cerebrovascular event (7); in this patient, neither CT nor angiography was performed. Three recently reported patients had cerebral infarction also documented by CT and angiography; two had used cocaine intranasally (3,8), and one had smoked “crack” (2).

Possible mechanisms of cerebral infarction in our patient include vasospasm and intracerebral thrombosis (2), as proposed...
for young patients who develop an acute myocardial infarction with no identifiable cardiovascular disease (1). This patient may also have an increased risk for vascular disease in view of his family history. Ethanol consumption may also be associated with an increased risk of cerebral infarction in young adults (9). Other possible causes of stroke in this patient such as a minor aneurysmal leak or homocystinuria are unlikely. Strokes due to rupture of the minor aneurysms located in the patient’s area of neurologic defect are usually associated with hypertension (10). Moreover, cerebral infarction associated with aneurysms results from posthemorrhage vasospasm of the arteries, and symptoms usually appear four to 14 days after the hemorrhage (11). The patient had neither hemorrhage nor history of hypertension. The clinical picture of homocystinuria includes mental retardation, osteoporosis, failure to thrive, severe myopia, glaucoma, malar flush, marfanoid appearance, and recurrent thrombotic events (12,13). Our patient had none of these characteristics.

The clinical findings deserve further comment, given the location of the lesion on the CT scan. Paresis of the 6th cranial nerve is sometimes a false localizing sign due to a transient increase of intracranial pressure (14), but its etiology may remain unknown in up to 30% of the cases (15). Also, the presentation with seizures is a known, but rare, clinical finding in subcortical lesions. The mechanism of such events is unclear (16,17).

Stroke associated with cocaine use is a clinical entity that is being reported with increased frequency. It is uncertain if cocaine causes or precipitates stroke. The history of a transient ischemic attack and subsequent documented infarct following nasal cocaine intake in this patient supports a causal relationship between cocaine use and cerebral infarction.

References