Embolic Stroke After Smoking
“Crack” Cocaine

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A 39-year-old woman had an embolic upper division middle cerebral artery branch occlusion 3 hours after smoking the free base of cocaine (“crack”). Radionuclide ventriculography demonstrated cardiomyopathy, and echocardiography documented a left atrial thrombus. This case demonstrates that embolism is one mechanism of ischemic stroke after cocaine use, and that cardiomyopathy, possibly cocaine induced, may be the source of emboli. A cardiac source of embolus should be sought in patients with cocaine-associated cerebral infarction. (Stroke 1990;21:1632–1635)

Accompanying the current American cocaine epidemic are increasingly frequent reports of myocardial infarction, myocarditis, cerebral infarction, cerebral hemorrhage, and subarachnoid hemorrhage in young adult users.1–7 Proposed, but as yet undocumented, mechanisms of cocaine-related cerebral infarction include increased platelet aggregation and cerebral vasospasm due to increased sympathetic activity and acute elevation of blood pressure.1 We recently encountered a patient who had a cardioembolic stroke after smoking the free base of cocaine (“crack”). Evaluation demonstrated a left atrial thrombus in the setting of a cardiomyopathy that may have been cocaine induced.

Case Report

A 39-year-old left-handed black woman was admitted to the Neurological Institute of New York for acute left hemiparesis. She had no history of hypertension, hypercholesterolemia, heart disease, alcohol abuse, intravenous drug use, syphilis, or collagen vascular disease. She had smoked one pack of cigarettes daily for 5 years before admission and had taken oral contraceptives in the past. Her father died from stroke of unknown mechanism at age 37, her mother had a myocardial infarction at age 51, and there was a family history of polycystic kidney disease. The patient had smoked crack cocaine almost daily for the previous 2 years. During the week before admission, she had a brief episode of numbness in the right leg, and another brief episode of slurred speech, tingling in the right face and arm, and imbalance.

The day before admission, she smoked crack at 6 PM. Three hours later, she briefly experienced numbness and tingling on the right side of her face. Thirty minutes later, she noticed left face, arm, and leg weakness plus slurred speech. These symptoms persisted, and the following morning she came to the hospital. Blood pressure was 140/80 mm Hg, pulse was 86 beats/min and regular, respiratory rate was 24 per minute, and temperature was normal. She had scattered rales and ronchi and an S3 heart sound. There was no pedal edema. She was alert and oriented with dysarthria, but there was no language abnormality, hemineglect, anosognosia, or constructional disturbance. Visual fields and eye movements were normal. Her tongue deviated to the left, and her lower face and arm were nearly paralyzed, but her leg had normal strength at the ankle and was only slightly weak at the hip. Sensation to light touch, pinprick, and vibration were decreased on the left side of the body; proprioception was impaired in the left hand but not in the left foot. There was an extensor response in the left toe.

A computed tomography (CT) scan of the brain was normal on admission. Duplex Doppler examination demonstrated no abnormality of either carotid bifurcation. Transcranial Doppler demonstrated no evidence of middle cerebral artery stem occlusion or stenosis. Cerebrospinal fluid was acellular with normal opening pressure, protein, and glucose, and negative routine cultures and cryptococcal antigen. Electrocardiogram demonstrated sinus rhythm with occasional premature ventricular contractions, an
intraventricular conduction defect, left atrial enlargement, and inverted T waves in leads II, III, aVF, and V₂ through V₆. A 24-hour Holter monitor showed sinus rhythm with many repetitive ventricular premature complexes. Echocardiogram demonstrated mild left ventricular hypertrophy, a nondilated left ventricle with marked global hypokinesis and ejection fraction of 26% (reduced), a mildly dilated left atrium and a moderately dilated left atrial appendage with a mobile 1.3x1.5 cm mass felt to represent a thrombus, mild-to-moderate mitral regurgitation, moderate tricuspid regurgitation, and an enlarged right atrium and right ventricle. A chest radiograph demonstrated cardiomegaly and pulmonary vascular congestion. Cocaine metabolites were detected in the urine. The following blood laboratory examinations were normal: glucose, electrolytes, cholesterol, erythrocyte sedimentation rate, complete blood count, platelet count, prothrombin time, partial thromboplastin time, blood cultures, lupus anticoagulant, antithrombin III activity, protein C activity, free protein S activity, serum protein immunofixation electrophoresis, antinuclear antibody, syphilis serology, hepatitis B antigen, cryoglobulins, angiotensin converting enzyme, thyroid function tests, screening test for sickle cells, CPK-MB fraction, lactic dehydrogenase isoenzymes, and T-cell ratio.

She was anticoagulated with heparin and then warfarin. Her left hemiparesis and dysarthria slowly improved. A follow-up CT scan done 6 days after admission demonstrated a right opercular and lower rolandic infarction (Figure 1). On the 15th hospital day she had a 15-minute episode of right facial weakness, which completely resolved. Prothrombin time was 13.8 seconds (control, 11.8 seconds). Repeat CT scan with contrast injection did not demonstrate a new infarction or hemorrhage. Treatment with digoxin, diuretics, and angiotensin-converting enzyme inhibitors resulted in improvement in her cardiac function. Repeat echocardiogram 3 weeks after admission demonstrated marked improvement in left ventricular function with mild global hypokinesis, ejection fraction of 45% (mildly decreased), and only trace mitral regurgitation. The previously documented thrombus in the left atrial appendage was not detected. A radionuclide ventriculogram demonstrated an ejection fraction of 40% (mildly decreased), increased left atrial size, normal left ventricular size, and inferolateral posterior hypokinesis. She was discharged to a drug rehabilitation center.

**Discussion**

This patient had an embolic right middle cerebral artery upper division branch occlusion after smok-
ing crack cocaine. The embolic source was a cardiomyopathy with thrombus in the left atrial appendage. To our knowledge, her stroke represents the only report of embolic cerebral infarction occurring after cocaine use. This case underscores the importance of searching for a source of embolus in this setting. Although angiography was not performed to exclude intracranial stenosis as a possible mechanism of her stroke, her young age, the clinical profile of the event, the topography of the stroke as demonstrated by CT, the normal middle cerebral artery transcranial Doppler findings, and the documented cardiac source of embolus make embolic middle cerebral artery upper division branch occlusion the most likely mechanism for her stroke. Of note is that her stroke was preceded by two transient ischemic attacks during the week before admission, and that she experienced one other transient ischemic attack 2 weeks into her hospital course. Although embolism is not typically preceded by transient ischemic attacks, fully 13% of embolic cerebral infarctions in the Stroke Data Bank of the National Institute of Neurological and Communicative Disorders and Stroke were preceded by transient ischemia.9 Wójak and Flamm9 reported a 51-year-old patient with atherosclerotic heart disease hospitalized for cerebral hemorrhage after cocaine use who, a week after admission, developed a cerebral infarction in the setting of an acute myocardial infarction and atrial fibrillation. However, the presumed right middle cerebral artery occlusion in their patient was temporally remote (1 week) from the episode of cocaine use, and it was unclear if the infarct was of cardiac origin. In contrast, our patient’s cerebral infarction was temporally related (3 hours) to the cocaine use, and she had echocardiographically documented impaired myocardial function and a left atrial thrombus. The 3-hour delay between her last cocaine use and the onset of her stroke does not exclude a direct drug effect. The elimination half-life of cocaine is reported to be 30–90 minutes,10 and it has been suggested that it may be a two-compartment drug with significant levels persisting in the body for 2 weeks or more.11 Moreover, benzylecgonine, a pharmacologically active metabolic product of cocaine, usually is detectable in urine for several days, and even longer in chronic heavy users.10,12 Cocaine-induced arrhythmia may have dislodged an embolus from the atrium just before the time of stroke onset. We cannot exclude the possibility that vasospasm or platelet aggregation, two postulated mechanisms for occlusive stroke in cocaine abuse,1,2 may have played a role in her stroke, but we have no objective evidence to support this notion.

Because our patient did not undergo cardiac catheterization or biopsy, the mechanism of her cardiomyopathy is uncertain. She had smoked cigarettes for 5 years and had a family history of stroke and myocardial infarction, but her young age and the absence of historical and objective evidence of myocardial infarction make it unlikely that atherosclerotic heart disease was the etiology of her cardiomyopathy. Although we cannot exclude the possibility that she had a so-called “cryptogenic cardiomyopathy,” we propose that her cardiomyopathy may have been the result of chronic cocaine abuse. Cocaine can cause myocardial injury by mechanisms other than infarction. Tazelaar et al4 have reported a significantly higher incidence of contraction band necrosis in hearts of patients with cocaine-associated death compared to patients dying of sedative-hypnotic overdose. They acknowledged that contraction band necrosis is a nonspecific finding, possibly reflecting calcium- and catecholamine-related metabolic abnormalities within the heart, but suggested that this abnormality might provide an “anatomic substrate” for arrhythmia.4 In another autopsy study, Virmani et al5 found myocarditis (mononuclear infiltrate) in 20% of patients with cocaine-associated death compared to 3.7% of patients dying suddenly of trauma. They proposed that cocaine might cause myocarditis via microvascular injury, in some patients perhaps superimposed on underlying atherosclerosis. A number of clinical case reports13–18 and anatomical studies19,20 support the notion that cocaine may indeed cause symptomatic dilated cardiomyopathy. If chronic cocaine use does cause cardiomyopathy, our patient certainly would be at risk for such a complication by virtue of her daily use of cocaine for 2 years before her stroke.

Whatever the mechanism of her cardiomyopathy, our patient illustrates that embolus is one mechanism of cocaine-associated cerebral infarction. Patients who have ischemic stroke after using cocaine should be evaluated for a cardiac source of embolus, because treatment with anticoagulants would be considered if one is found.

References


**Key Words** • cerebrovascular disorders • cocaine • embolism
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