Recurrent Embolic Stroke and Cocaine-Related Cardiomyopathy

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Ischemic stroke temporally related to cocaine abuse has become increasingly common in young adults. Despite this relation, however, the pathogenesis of infarction in many of these patients remains obscure. I report the case of a 39-year-old man who developed occlusion of the frontopolar branches of the left middle cerebral artery 1 hour after intravenous cocaine use. Eleven days later he developed occlusion of the superior division of the right middle cerebral artery. In this case the mechanism of infarction was clearly cardiogenic embolization. Chest radiograph and echocardiogram revealed dilated cardiomyopathy with left ventricular thrombi. No cause other than cocaine abuse was found for his cardiomyopathy. This is the second reported case of cocaine-related cardiomyopathy presenting as embolic stroke and associated with intracavitary thrombus. Such an association may be more common than previously thought. Thorough cardiac evaluation in all patients with ischemic stroke related to cocaine abuse is appropriate. (Stroke 1991;22:1203-1205)

In the past decade, coincident with the dramatic increase in cocaine abuse in this nation, an increasing number of cases of stroke temporally related to cocaine use have been reported.1 Cocaine abuse has also been associated with heart disease, including myocardial infarction, myocarditis, and endocarditis.2-3 Recently, in this journal, a case of embolic stroke due to cocaine-related cardiomyopathy was reported.4 I report a second, remarkably similar case that was associated with recurrent, fatal embolic infarction.

Case Report

A 39-year-old right-handed black man was admitted to the Regional Medical Center, Memphis, Tenn., because of right hemiparesis. He had been in excellent general health with the exception that 2 weeks prior to admission he was diagnosed and treated for mild diastolic hypertension. He had abused cocaine, chiefly by the intravenous route, for >12 months. He was a half pack per day smoker. Recent alcohol use was denied.

He was well until the morning of admission. Approximately 1 hour after the intravenous injection of an unknown quantity of cocaine, he suddenly became mute and was unable to move his right arm. When admitted several hours later, vital signs were normal except for a blood pressure of 150/100 mm Hg. General examination as well as cardiac examination were normal. Neurological examination revealed that, although fully alert, he could speak only occasional single words and was totally unable to repeat. He could follow only simple, one-step verbal commands. Motor examination revealed right lower facial weakness, a flaccid paralyzed right arm, and moderate (3/5) strength of his right leg. The right plantar response was extensor.

Admission laboratory data revealed normal complete blood count, erythrocyte sedimentation rate, platelet count, prothrombin time, activated partial thromboplastin time (aPTT), blood urea nitrogen, glucose, electrolytes, creatine phosphokinase-MB fraction, and lipid profile. Four blood cultures were sterile. Urine drug screen was positive for cocaine metabolites. Fluorescent antinuclear antibody titer and rheumatoid factor were both negative. An unenhanced cranial computed tomogram (CT scan) on admission was normal, but a repeat CT scan performed 1 week later with contrast revealed cortical enhancement consistent with infarction in the distribution of the frontopolar branches of the left middle cerebral artery. A carotid Doppler ultrasonogram was normal. Cerebrospinal fluid examination was unremarkable.

An electrocardiogram on admission revealed only nonspecific ST-T wave changes, but chest roentgenography revealed cardiomegaly with a prominent left ventricular shadow. Echocardiography on the second hospital day revealed a dilated, severely hypokinetic left ventricle. No valvular lesions were identified. Two moderate-sized nonmotile thrombi were seen in
the left ventricular apex. A cardiology consultant concurred that the patient had a dilated cardiomyopathy, probably related to cocaine use. Intravenous heparin was begun, and the aPTT was maintained between 1.5 and 2.0 times control.

During the first week of hospitalization, there was significant improvement in the patient’s hemiparesis and aphasia. Oral warfarin was added. On the 11th hospital day, however, he abruptly became confused, agitated, and belligerent. A repeat cranial CT scan was unchanged. No toxic or metabolic cause for his delirium was found. Two days later he gradually became stuporous, and left arm weakness was noted for the first time. A cranial CT scan at that time revealed a large area of hypodensity in the distribution of the superior division of the right middle cerebral artery with secondary hemorrhagic transformation (Figure 1). Anticoagulant therapy was withheld. A neurosurgical consultant did not believe that surgery was indicated. The patient failed to improve, and after multiple medical complications, in particular pneumonia, he died 4 weeks after admission.

Discussion

Although angiography was not performed in this case, there is little doubt that the mechanism of infarction was cardiogenic embolization. In addition, serial cranial CT scans revealed evidence of infarction in the distribution of branches of both middle cerebral arteries. Although the patient was an intravenous drug abuser, four blood cultures were sterile and no valvular lesions were detected by echocardiography. The etiology of his underlying cardiac disease, then, was clearly dilated cardiomyopathy with embolization from left ventricular thrombi.

This is the second report of embolic cerebral infarction temporally related to cocaine abuse and associated with cardiomyopathy. Both patients were young adults with long-standing histories of cocaine abuse who developed branch occlusion of a middle cerebral artery ≤3 hours after cocaine administration. In addition, both patients had clinical and echocardiographic evidence of cardiomyopathy with intracavitary thrombi. The mechanism by which cocaine use may have triggered embolization in these patients is speculative. A significant amount of cocaine and its metabolites, however, would have been present in both patients at the time of embolization. It is conceivable that cocaine-induced hypertension or arrhythmia may have acted to dislodge an intracavitary thrombus.

Compared with myocardial infarction, only a relative handful of patients with cardiomyopathy linked to cocaine abuse have been reported. While it is conceivable that the patient described in this report may have had an alternative cause for his cardiomyopathy, he had no electrocardiographic or enzymatic evidence of myocardial infarction, and he had no preceding viral illness or vasculitis. In addition, the clinical, radiological, and echocardiographic features of his illness were quite consistent with prior reports of cocaine-related cardiomyopathy. The similarity of this case to that of Petty et al, as well as to other cases of cardiomyopathy, suggests that cocaine-related cardiomyopathy is a distinct, although rare, clinical entity.

This patient’s course was complicated by the development of agitated delirium on the 11th hospital day. Subsequent cranial CT scans documented recurrent embolic infarction in the distribution of the superior division of the right middle cerebral artery. While agitated delirium is a syndrome of diverse cause, it is a common complication of right middle cerebral artery distribution infarction, which, in turn, is often secondary to cerebral embolization. It is of interest that the patient described by Petty et al developed transient right facial weakness on the 15th hospital day, presumably due to recurrent cerebral embolization. In both cases “therapeutic” anticoagulation failed to prevent late complications.

It is likely that a variety of mechanisms are responsible for ischemic stroke temporally related to cocaine abuse. A cardiac source for embolism, however, should be sought in each case. Because of the few reported cases of cocaine-related cardiomyopathy, the prevalences of intracavitary thrombi and embolic infarction are at present unknown. This case
and that of Petty et al., however, suggest that both complications may be relatively common. Despite the presence of hemorrhagic transformation in the present case, anticoagulant therapy is still indicated. The duration of therapy is largely empirical, but it would appear prudent to continue anticoagulation until serial echocardiography demonstrates the disappearance of intracavitary thrombus or until there is spontaneous improvement in cardiac function.

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References


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