Concurrent Myocardial and Cerebral Infarctions After Intranasal Cocaine Use

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**Background and Purpose:** Cardiac and cerebrovascular complications associated with cocaine abuse have increasingly been reported, but concurrent development of cocaine-induced cardiac disease and stroke has rarely been reported.

**Case Description:** A 37-year-old man with a remote history of intravenous heroin and amphetamine use, cardiomyopathy, and recent cocaine use developed chest pain and ventricular tachycardia 30 minutes after intranasal cocaine hydrochloride use and jogging on a cold winter morning. Ventricular tachycardia was converted to atrial fibrillation. He was proven to have a small myocardial infarction. Within 6 hours of cocaine use he suffered a left hemisphere stroke. Cardiac electrophysiologic evaluation revealed inducible ventricular tachycardia.

**Conclusions:** To our knowledge, this is the first report of concurrent myocardial infarction, life-threatening ventricular arrhythmias, and cerebral infarction temporally related to cocaine use. It is probable that one mechanism by which cocaine use causes stroke is to trigger expression of a known cardiac source of embolism. (Stroke 1992;23:427-430)

**KEY WORDS** • cardiomyopathy, congestive • cerebral infarction • cocaine • myocardial infarction

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n recent years, cocaine abuse has been linked to a variety of serious medical disorders. Cardiovascular complications include hypertension, bradycardia, and ventricular tachycardia, coronary vasospasm, angina, myocardial infarction, chronic or reversible cardiomyopathy, sudden death, and aortic rupture. Acute cerebrovascular disease, including ischemic and hemorrhagic (both subarachnoid and intracerebral) strokes, have also been reported.

Although temporal associations exist between cocaine use and acute cardiovascular events, patients generally experience one type of complication. However, the occurrence of ischemic stroke complicating “crack” or intravenous cocaine–associated cardiomyopathy has recently been described. We describe a patient with chronic idiopathic dilated cardiomyopathy who suffered a myocardial infarction, life-threatening cardiac arrhythmias, and an ischemic stroke within 6 hours of intranasal cocaine hydrochloride use.

**Case Report**

A 37-year-old man developed dizziness, palpitations, and chest tightness while jogging on a cold winter morning in 1990. Symptoms resolved with rest but recurred with continued jogging. He was later found on the street, cold and semiconscious. Thirty minutes before symptom onset, he had snorted an unknown quantity of cocaine hydrochloride for the first time in a year. Initial blood pressure was 40 systolic by Doppler, and an electrocardiogram (ECG) revealed a wide complex tachycardia with a rate of 300 beats per minute (Figure 1A). Chest pain was treated with morphine sulfate. Cardioversion (100 W/sec) resulted in atrial fibrillation with a rapid irregular ventricular response (150 beats per minute) with frequent wide complexes. The patient became alert, oriented, conversant, and moved all extremities well. Six hours after onset of symptoms, he was aphasic.

In 1975, he began using intravenous amphetamines, followed by a $100/day heroin habit (using both intravenous and “skin-popping” routes). Three years before admission, he discontinued heroin but began regular intranasal cocaine hydrochloride use. This stopped approximately 1 year before admission due to incarceration. He drank alcohol on occasion but denied past or present heavy use. In 1982, he developed idiopathic congestive cardiomyopathy and chronic atrial fibrillation, which improved and stabilized with treatment (digoxin, diuretics, and enalapril). Echocardiography in 1989 revealed a mild increase in left atrial size (4.5 cm), a markedly dilated and poorly functioning left ventricle without evidence of intracardiac clot, inferior dyskinesis, and mild mitral and tricuspid regurgitation.

Blood pressure was 135/80 mm Hg, temperature was 98°F, pulse was 115 beats per minute and irregular, with unlabored respirations at 18 breaths per minute. There was no jugular venous distension, S3, rales, or edema. He was alert but oriented only to person. Nonfluent speech with paraphasic errors, mildly impaired comprehension, dysnomia, orobuccal-lingual apraxia, and agraphia were present. Moderate right lower facial weakness, tongue deviation to the right, right hemipare-
Cocaine affects multiple sites within the cardiovascular system. In the periphery, cocaine inhibits the reuptake and promotes release of norepinephrine at adrenergic nerve endings, prevents metabolism of circulating nor-epinephrine, and sensitizes tissues to the effects of catecholamines.1,8 In the central nervous system, cocaine's local anesthetic effect leads to excitation and increased peripheral sympathetic tone.8 Experimental work in conscious rats37 and dogs8 indicates that cocaine may also potenti ate responses to catecholamines by central mechanisms. Elevated catecholamine levels increase heart rate and myocardial contractility but also predispose to myocardial infarction by increasing platelet aggregation.

Three months later, he drank an unknown quantity of rum and developed dizziness, palpitations, nausea, left chest pressure, and syncope. Moderate right hemiparesis was unchanged. Cardiac enzymes and toxicological screen were negative. Repeat cardiac electrophysiolog ical study showed that ventricular tachycardia was in ducible only during isoproterenol infusion. Metoprolol was added to his medical regimen, and warfarin sodium (Coumadin) was administered for stroke prophylaxis. Three months later, he experienced severe bradycardia (39 beats per minute) and chest pain. Myocardial infarction was ruled out. Repeat noninvasive cardiac evaluation showed that the ejection fraction was 30–35%. Cardiac catheterization revealed normal right heart pressures and coronary arteries. Metoprolol was discontinued. Thirteen months later, the patient had no new cardiovascular or neurological problems.

Discussion

Cocaine has multiple effects on the cerebral vasculature. Experiments in rats,64,45 cats,46 and dogs,43 and evaluation of acute67 or chronic62 cocaine users by single-photon emission computed tomography49 or positron emission tomography,46 suggest that cocaine and cocaine metabolites benzoylecgonine46 and nor cocaine44 may cause transient45 or prolonged47 vasoconstriction. Involved vessels include cortical pial arteries, arterioles, veins, and venules.44 Increasing concentrations of cocaine hydrochloride may cause capillary rupture,44 and patients who use intravenous cocaine hydrochloride may be more likely to suffer a hemorrhagic stroke.5,6 The mechanism of cerebral ischemia in a susceptible individual may be multifactorial, consisting of multiple actions of cocaine (vasoconstriction45-47 and platelet aggregation48), drug interaction (cocaine and ethanol arrhythmias and prothrombotic tendencies12,49), or effects on preexisting stroke risk factors and vascular disease.

The circumstances surrounding our patient's temporarily related myocardial and cerebral ischemic events are complex (Figure 2). The etiology of his cardiomyopathy is uncertain, but patients who abuse multiple drugs, including heroin and amphetamines, are susceptible to myocardial injury.50 After cocaine use, coronary

Rhythm strip of spontaneous ventricular tachycardia performed by paramedics at time patient was found in semiconscious state. Panel 1b: Surface lead aVF and intracardiac electrograms from right ventricular apex recorded during programmed stimulation studies. Studies are performed by right heart catheterization with multipolar pacing leads. Stimulation protocol consisting of up to multiple ventricular extrastimuli, and rapid, short-burst pacing was used. Reproducible induction of sustained monomorphic ventricular tachycardia is a positive response and indicative of underlying arrhythmic substrate. In response to a six-beat pacing burst, sustained monomorphic ventricular tachycardia was induced. Paper speed for both figures, 25 mm/sec.
The combination of these effects, as well as platelet aggregation, can readily explain the occurrence of myocardial infarction. Underlying myocardial damage and myocardial infarction may serve as the anatomic substrate for the electrophysiologic abnormalities, including life-threatening reentrant ventricular tachyarrhythmias.

In our patient, reproducible induction of sustained monomorphic ventricular tachycardia in a drug-free setting is consistent with the presence of an anatomic substrate, possibly created by prior cocaine, heroin, or amphetamine exposure. The existence of a chronic increased susceptibility to arrhythmias is further supported by induction of ventricular tachycardia during catecholamine exposure and the later development of symptoms after alcohol use. Ineffective left ventricular contraction associated with sustained ventricular tachycardia may have led to intracardiac stasis and, with increased platelet aggregation, formation of friable new clot. With restoration of a more effective cardiac rhythm (atrial fibrillation), this fresh clot may have dislodged and caused an embolic cerebral infarction. Alternatively, slow absorption of cocaine from nasal mucosa may have produced delayed extracranial or cerebral pial artery vasoconstriction and platelet aggregation, leading to later thrombotic occlusion of a cerebral artery. However, we have no evidence to support this mechanism for stroke.

Our case illustrates that patients with underlying structural heart disease who continue cocaine abuse are at risk for myocardial infarction, life-threatening ventricular arrhythmias, and major cerebrovascular events. Because the association between cardiovascular and cerebrovascular disease complicating cocaine use has been recently described, it is probable that one mechanism by which cocaine use causes stroke is to trigger expression of a known cardiac source of cerebral embolism with its cardiac stimulatory and platelet aggregation effects. Petty et al have recently described a 39-year-old woman with cardiomyopathy and left atrial thrombus who had an embolic stroke 3 hours after smoking crack. Sauer described a 39-year-old man with cardiomyopathy and apical thrombi who had an embolic stroke 1 hour after intravenous cocaine use. Our patient is unique in that he was proven to have had a stroke 6 hours after myocardial infarction and life-threatening cardiac arrhythmias associated with intranasal cocaine hydrochloride use. A similar mechanism of drug-associated stroke has been recently described after phencyclidine use.

Patients presenting with cocaine-associated cerebral infarction should be evaluated for evidence of a cardiac source of embolism, acute myocardial infarction, and cardiac arrhythmias. Patients who enter treatment programs and forsake drug use may enjoy improved cardiac function and be considered as candidates for long-term antithrombotic therapy to prevent stroke.

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References


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