Case Reports

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

In recent years, cocaine abuse has been linked to a variety of serious medical disorders.1-7 Cardiovascular complications include hypertension,1,8 bradyarrhythmias and ventricular tachyarrhythmias,1,8-12 coronary vasospasm,13-17 angina,18-21 myocardial infarction,1,3-18-24 chronic or reversible cardiomyopathy,25-31 sudden death,1,32 and aortic rupture.1 Acute cerebrovascular disease, including ischemic5,29,33-35 and hemorrhagic (both subarachnoid and intracerebral)4,5,33,35,36 strokes, have also been reported.

Although temporal associations exist between cocaine use and acute cardiovascular3,10,18-24,32 or cerebrovascular3,28,34,35 events, patients generally experience one type of complication. However, the occurrence of ischemic stroke complicating “crack”29,30 or intravenous8 cocaine–associated cardiomyopathy has recently been described. We describe a patient with chronic idiopathic dilated cardiomyopathy who suffered a myocardial infarction, life-threatening cardiac arrhythmia, 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 dyskinesia, 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, S 3, 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-
patients (proximal upper extremity 2–3/5, distal upper extremity 4–5, proximal and distal lower extremity 3/5), diffuse hyperreflexia, and right Babinski's sign were evident. There was loss of pinprick, temperature, vibratory, and joint position sense on the right.

Routine chemistries, computed tomographic scan, and magnetic resonance imaging scan were normal. Chest x-ray showed a globular heart. Serial creatine kinase reached a peak of 431 units/dl with a total isoenzyme MB fraction of 33 units/dl (7.7%), consistent with a small acute myocardial infarction. Routine chemistries, computed tomographic scan, and magnetic resonance imaging scan were normal. Chest x-ray showed a globular heart. Serial creatine kinase reached a peak of 431 units/dl with a total isoenzyme MB fraction of 33 units/dl (7.7%), consistent with a small acute myocardial infarction. Routine chemistries, computed tomographic scan, and magnetic resonance imaging scan were normal. Chest x-ray showed a globular heart. Serial creatine kinase reached a peak of 431 units/dl with a total isoenzyme MB fraction of 33 units/dl (7.7%), consistent with a small acute myocardial infarction.

Cardiac electrophysiological testing, before and after propranolol administration, demonstrated inducible sustained monomorphic ventricular tachycardia leading to hemodynamic compromise (Figure 1B). A loading regimen of amiodarone was administered, and speech and right hemiparesis gradually improved. He was transferred to a rehabilitation facility 1 month after admission.

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 electrophysiological study showed that ventricular tachycardia was inducible 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 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.4-8 In the central nervous system, cocaine's local anesthetic effect leads to excitation and increased peripheral sympathetic tone.9 Experimental work in conscious rats9 and dogs10 indicates that cocaine may also potentiate responses to catecholamines by central mechanisms. Elevated catecholamine levels increase heart rate and myocardial contractility but also predispose individuals to cardiac arrhythmias.1,8,14,16,17 Experimental14,16,17 and clinical13,14,16 studies demonstrate that cocaine may induce coronary artery vasoconstriction and myocardial ischemia, particularly with subchronic treatment9,10 or at sites of preexisting atherosclerotic stenosis.15 Acute myocardial infarction has been induced in patients with or without preexisting coronary artery disease.13,14,15 In some cases, cocaine-induced constriction of a normal coronary artery has culminated in thrombosis and infarction,16-23 perhaps facilitated by enhanced responsiveness of platelets to arachidonic acid, increased thromboxane production, and platelet aggregation.24 In patients who die from cocaine intoxication, histological examination has revealed myocardial contraction band necrosis, coronary intimal hyperplasia, myocarditis, and cardiomyopathy.24-43 The presence of cardiomyopathy or contraction bands may add to the cocaine user's susceptibility to cardiac arrhythmias.42

Cocaine has multiple effects on the cerebral vasculature. Experiments in rats,44,45 cats,46 and dogs,47 and evaluation of acute48 or chronic42 cocaine users by single-photon emission computed tomography49 or positron emission tomography50-52 suggest that cocaine and cocaine metabolites benzoylglycine46 and norcocaine46 may cause transient53 or prolonged54 vasoconstriction. Involved vessels include cortical pial arteries, arterioles, veins, and venules.43,54 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.5,55 The mechanism of cerebral ischemia in a susceptible individual may be multifactorial, consisting of multiple actions of cocaine (vasoconstriction56-57 and platelet aggregation58), drug interaction (cocaine and ethanol arrhythmias and prothrombotic tendencies59,60), or effects on preexisting stroke risk factors and vascular disease.

The circumstances surrounding our patient's temporally related myocardial and cerebral ischemic events are complex (Figure 2). The etiology of his cardiomy-opathy is uncertain, but patients who abuse multiple drugs, including heroin and amphetamines, are susceptible to myocardial injury.50 After cocaine use, coronary...
artery vasoconstriction\textsuperscript{13,14} may have initiated ischemia, perhaps potentiated by the increased myocardial oxygen demand due to cold exposure\textsuperscript{51} and exercise.\textsuperscript{52,53} The combination of these effects, as well as platelet aggregation,\textsuperscript{40} can readily explain the occurrence of myocardial infarction.\textsuperscript{1,3,34} Underlying myocardial damage\textsuperscript{1,2,25,28,38} and myocardial infarction\textsuperscript{1,3,22–24} may serve as the anatomic substrate for the electrophysiologic abnormalities, including life-threatening reentrant ventricular tachyarrhythmias.\textsuperscript{1,2,38,42}

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,\textsuperscript{2,41–42} heroin,\textsuperscript{50} or amphetamine\textsuperscript{41,50} 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.\textsuperscript{12,49} Ineffective left ventricular contraction associated with sustained ventricular tachycardia may have led to intracardiac stasis and, with increased platelet aggregation,\textsuperscript{40} to 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.\textsuperscript{29,31} Alternatively, slow absorption of cocaine from nasal mucosa\textsuperscript{44} may have produced delayed extracranial or cerebral pial artery vasoconstriction\textsuperscript{44,47} and platelet aggregation,\textsuperscript{40} 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,\textsuperscript{29} life-threatening ventricular arrhythmias,\textsuperscript{1} and major cerebrovascular events.\textsuperscript{28–31} Because the association between cardiovascular and cerebrovascular disease complicating cocaine use has been recently described,\textsuperscript{29–31} 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\textsuperscript{29} 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\textsuperscript{31} 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.\textsuperscript{35}

Patients presenting with cocaine-associated cerebral infarction should be evaluated for evidence of a cardiac source of embolism,\textsuperscript{29–31} 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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