Cocaine-Induced Stroke

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Brain infarcts among crack cocaine users may be secondary to large cerebral artery vasospasm with secondary intravascular thrombosis (with or without distal embolization).

A 30-year-old woman with past medical history of hypertension, hyperlipidemia, type 1 diabetes mellitus (T1DM), chronic kidney disease (CKD), and poly-substance abuse presented to the emergency department with a sudden change in mental status, weakness of the right side of the body, slurring of speech, and new-onset dysphasia. On examination, the patient’s vital signs were: temperature, 37.8°C (100°F); pulse, 98 beats/min; blood pressure, 190/95 mm Hg; and, respiratory rate, 12 breaths/min. Findings from cardiovascular, GI, and respiratory examinations were normal. Results of complete blood cell count were: hemoglobin and hematocrit, 11 g/dL and 38%, respectively; white blood cell count, 4500/µL; platelet count, 230,000/µmol. Results of a complete metabolic panel included sodium, 138 mEq/L; potassium, 4.1 mEq/L; blood urea nitrogen and creatinine, 3.5 mg/dL and 3.5 mg/dL, respectively; and blood glucose, 180 mg/dL. Variations from normal were attributed to her CKD and T1DM. Values for folic acid, vitamin B12, thyroid-stimulating hormone, and rapid plasma reagin were normal. Results of a test for HIV were negative. Findings on a CT scan of the head were normal.

Neurology was consulted, and diffusion-weighted MRI (DW-MRI) of the brain with and without contrast was performed. DW-MRI showed patchy areas indicating increased signal in the pons on both sides (Figure, above*) and otherwise normal brain parenchyma. MRA showed no evidence of stenosis or luminal irregularity to indicate intimal tears. Repeated MRA and DW-MRI were obtained 2 days later. DW-MRI showed diffusion abnormality restricted to the pons, which was diffusely involved (Figure, below*). Moreover, the observed abnormality did not extend into the anterior, middle, and posterior cerebral circulation, which ruled out embolic disease. The repeated MRA was normal.

The patient’s stroke was attributed to hypertension caused by heavy cocaine abuse. She was started on a regimen of aspirin and simvastatin (20 mg daily), and blood pressure and blood glucose were closely monitored and controlled. Over several days, her mental status, slurred speech, and dysphagia improved. After a week, the patient was transferred to physical rehabilitation. Her
weakness gradually improved and she was discharged home after 30 days. She was also provided
education on cocaine abuse.

Discussion
Cocaine became available for recreational use in the United States approximately 2 decades ago. The drug is a powerfully addictive stimulant that directly affects the brain. More than 30 million Americans have tried cocaine once, and 5 million report regular use.\(^1\) Adults aged 18 to 25 years have a higher rate of current cocaine use than any other age group.\(^1\) Overall, men report higher rates of current cocaine use than women.

Routes of administration
The principal routes of administration are oral, intranasal, intravenous, and inhalation. Any route of administration can lead to absorption of toxic amounts of cocaine, possible acute cardiovascular or cerebrovascular emergencies, and seizures, all of which can result in sudden death.

Mechanism of action
Cocaine prevents the uptake of catecholamine from nerve terminals, making epinephrine available to interact with postsynaptic receptors. The resulting potentiation of adrenergic transmission can result in vasoconstriction and ultimately cause ischemia.\(^2,3\)

Cocaine is also known to block serotonin uptake.\(^2\) Elevated levels of serotonin, as with catecholamine, can potentiate vasoconstriction of the large and medium-sized arteries of the cerebral circulation.

Pathophysiology
Cocaine and its metabolites (norcocaine and benzoylecgonine) are potent vasoconstrictors. Long-term use of crack cocaine may lead to prolonged vasoconstriction as a result of the more protracted half-lives of active metabolites.\(^2\) This effect can be further attenuated in association with ethanol.\(^4\)

Numerous reports suggest that cocaine use may be associated with myocardial infarction (MI) and stroke. The effect of cocaine on vessel diameter may depend on the location of the vessel and the dose of cocaine used.\(^5\) Brain infarcts among crack cocaine users may be secondary to large cerebral artery vasospasm with secondary intravascular thrombosis (with or without distal embolization).\(^6\)

Hemorrhage into an infarct may reflect reperfusion after resolution of vasoconstriction or recanalization from dissolution of the thrombus.\(^6\) Cocaine use may also cause acute hypertension associated with stroke, by causing cerebral vasoconstriction.

In a large cohort study to explore the relationship between cocaine and stroke,\(^7\) 5142 records for adults admitted to a tertiary neurovascular service were screened for current or previous cocaine use. Of the 96 patients identified, 45 (47%) were given the diagnosis of ischemic stroke/transient ischemic attack (TIA); 26 (27%), of intracerebral hemorrhage (ICH); and 25 (26%), of subarachnoid hemorrhage.\(^7\) The authors concluded that ischemic stroke/TIA is a common neurovascular presentation in patients with a remote history of cocaine use, often as a result of atherosclerotic disease; neither vasculitis nor vasospasm was identified as a common cause. ICH was more common
among current users perhaps because of acute spikes in blood pressure.⁷

Of note—and a particular caution in the ED setting—the use of beta-blockers in patients with cocaine-associated chest pain or myocardial infarction or ischemia is not recommended.⁸ Unopposed alpha-adrenergic stimulation may provoke or exacerbate coronary artery vasoconstriction and systemic hypertension.⁷ In patients with MI unrelated to cocaine abuse, beta-blocker administration can reduce mortality. Given the low death rate from cocaine-associated MI, the risk-benefit ratio is greatly altered.⁸ Nitroglycerin and benzodiazepines show similar efficacy in reducing cocaine-associated chest pain.⁸ Calcium channel blockers are not recommended as first-line treatment but may be considered for patients who do not respond to benzodiazepines and nitroglycerin.

*Please note that the figures reflect the only images available for this patient.*

References


Links: