

Cocaine-Induced Bilateral Basal Ganglia Ischemia Presenting with Unilateral Wrist Drop

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Received Jan. 17, 2023; Accepted for publication Feb. 23, 2023; Published online April 24, 2023
<https://doi.org/10.17161/kjm.vol16.19114>

INTRODUCTION

Cocaine, an alkaloid extract from a plant, acts as a central nervous system stimulant by binding and blocking monoamine (dopamine, norepinephrine, epinephrine, and serotonin) and reuptake transporters thus creating euphoria with increased energy and motor activity.¹ The available forms of cocaine in the market can be either inhaled or smoked. Given the mode of action of cocaine, its abuse is associated with central nervous system (CNS) complications including hemorrhagic and ischemic strokes, seizures, toxic encephalopathy, and vasculitis.² Its toxicity can manifest in an array of ways of which some are more common than others. For that purpose, it is important to shed light on the rare complications that can go unnoticed in the emergency setting.

Basal ganglia infarcts are uncommon types of strokes that are seen in patients with carbon monoxide poisoning, methanol intoxication, ischemic brain injury, bibasilar artery occlusion and heroin use.³ While it is not rare to see ischemic strokes with heroin, it is uncommon to have bilateral basal ganglia infarcts. Since bilateral basal ganglia infarcts were not well-documented in the literature, the following case is presented.

CASE REPORT

A 40-year-old Hispanic male with a past medical history of alcohol use disorder, substance use, and hypertension, presented to the emergency department for right gluteal pain, right wrist-drop, and neck pain that started one day prior. He endorsed having mild headaches and generalized fatigue. He also mentioned having darker urine on the morning of his admission. When asked about history of drug use, he admitted to snorting cocaine every morning for the past two years. He denied having any visual changes, nausea, vomiting, fever, or chills.

The patient's vital signs showed elevated blood pressure but were otherwise within normal limits. On physical exam, he was alert and oriented to time, place, and person. He had normal deep tendon reflexes and gait. However, his motor exam showed right arm weakness at the level of the wrist and finger extensors. Similarly, sensory exam showed decreased pinprick sensation of the right hand.

A computed tomography (CT) scan of his head without contrast was done for further workup of his neurologic symptoms. It showed bilateral hypodensities involving the globus pallidus. Labs were drawn and the results showed elevated AST and ALT (both > 2000 U/L), serum creatinine of 3 mg/dl, and a creatine phosphokinase (CPK) greater than 18000 U/L. The patient was admitted to the hospital for treatment of his acute kidney injury secondary to rhabdomyolysis, and to investigate the above findings further.

As workup of the patient's neurologic symptoms, CT angiography of the head and neck showed intact intracranial circulation with no

evidence of large vessel occlusion or high-grade stenosis, patent dural venous sinuses, and no pathologic intracranial enhancement. Again, the low attenuation in the globus pallidus was observed. Furthermore, fat stranding and edema of the left posterior paraspinal musculature was seen and thought to be related to patient's rhabdomyolysis. Brain magnetic resonance imaging (MRI) showed small areas of diffusion restriction involving the bilateral globus pallidus regions and focal areas involving the bilateral putamen regions, focal punctate areas of diffusion restriction involving the posterior right frontal lobe centrum semiovale, right occipital lobe, left parietal lobe, and possible involvement of the posterior left occipital lobe region (Figure 1). MRI of the cervical spine showed edema within the posterior neck musculature. No spinal stenosis or abnormal findings in the spinal cord were observed. Carotid duplex ultrasound was normal.

Given the patient's multi-organ involvement including the brain, kidneys, and liver, as well as history of cocaine use, he was diagnosed with cocaine-induced multi-organ damage. For his rhabdomyolysis, he was started on aggressive IV hydration. His CPK level trended-down and he had adequate urine output. As for hepatic injury, the patient's liver enzymes were trended and markedly improved with supportive management. His wrist-drop, however, persisted. Upon stabilization of his acute condition, the patient was discharged with instructions to follow-up with neurology for electromyography (EMG) studies to characterize the cause and extent of his motor weakness, and cardiology for loop recorder insertion as outpatient. While the patient's acute renal failure and hepatotoxicity resolved with supportive management, his neurological symptoms persisted. He was offered physical therapy as inpatient and outpatient to help with his rehabilitation.

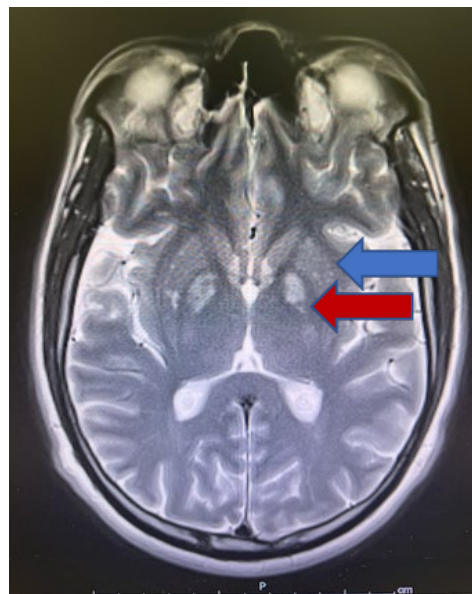


Figure 1. The cross-sectional brain MRI shows hypointensity of the bilateral basal ganglia (globus pallidus in red arrow and putamen in blue arrow).

DISCUSSION

This case highlighted the wide range of clinical findings associated with cocaine use. In this case, a young and relatively healthy patient presented to the emergency department with vague neurologic symptoms and findings of ischemia on head CT, as well as evidence of multi-organ failure. The deleterious effects of cocaine on the body have shown to harm the central nervous system by causing stroke,⁴ the renal system with rhabdomyolysis,⁵ the cardiovascular system with endocarditis, coronary atherosclerosis and arrhythmia,^{6,7} the gastrointestinal system with liver injury and mesenteric ischemia,⁸ the pulmonary system with interstitial lung disease and pulmonary hypertension,⁹ not to mention the damage it can impose on a person's quality of life due to its strong addictive capability. In our case, its effects seemed to involve the CNS, kidneys, and liver.

Unfortunately, the long-term effects of cocaine on this patient's motor function were unknown. While this case was treated as an acute presentation, there could be a possibility of chronic effect of cocaine use on the central nervous system. Furthermore, the patient was referred to follow with neurology and the prognosis of his wrist drop remains unclear. Given that our patient presented with rhabdomyolysis, the possibility that his wrist drop could have been caused by a muscular injury cannot be ruled out. In the grand scheme of this clinical presentation, and especially given the MRI changes (Figure 1), the likelihood of his weakness being a CNS insult remains a high probability.

CONCLUSIONS

In this case report, a rare neurologic finding was highlighted that was triggered by a long history of cocaine use. Being aware of the common as well as the uncommon presentations and findings of substance-induced brain injury can aid the diagnosis and management of patients, especially in the acute settings. Basal ganglia infarct is rare, and its etiologies can vary. Thus, it is essential to obtain a thorough history and consider drugs like cocaine as offending agents affecting the CNS in ways physicians might not be familiar.

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Keywords: cocaine-related disorders, basal ganglia cerebrovascular disease, radial neuropathy, brain ischemia, central nervous system