

Cocaine-Related Acute Spinal Cord Infarction

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ABSTRACT

We report a rare case of anterior spinal artery syndrome in the setting of acute cocaine use. A 31-year-old man presented to the hospital unarousable with leukocytosis and a positive toxicology screen for opioids, cocaine, benzodiazepines and cannabis. He was placed on intravenous naloxone. As the patient regained consciousness, he was found to have paraplegia, sensory loss below the level of T5, and urinary retention. MRI findings showed a signal intensity abnormality from the level of T1-4, highly suggestive of an acute ischemic spinal cord infarct.

KEYWORDS: cocaine, anterior spinal artery syndrome, spinal cord, spinal cord infarction

INTRODUCTION

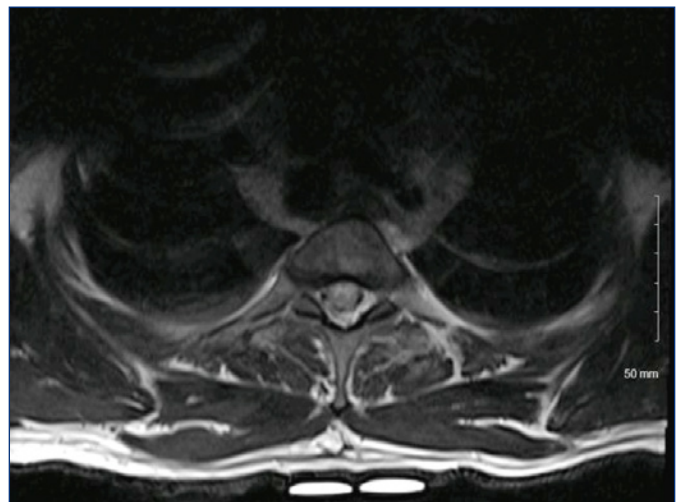
Case Report

A 31-year-old man with no known medical history presented to the hospital after being found unresponsive at his home. He was somnolent and received 10mg naloxone by emergency medical services (EMS). On initial presentation, he had a temperature of 36.6 degrees C, heart rate of 108 beats per minute, respiratory rate of 25 respirations per minute, blood pressure 118/97 mmHg and oxygen saturation 100% on room air. He was arousable only to loud voice. He moved all extremities, and had erythema with evidence of track marks in his left antecubital fossa. A CT of his head was unremarkable. His laboratory workup was remarkable only for white blood cells $21.5 \times 10^3/\text{mCL}$. His urine toxicology screen was positive for cocaine, benzodiazepines, opiates, and cannabis. He was admitted to the intensive care unit for polysubstance overdose and was started on intravenous naloxone. The next day, the patient became increasingly alert and was found to have bilateral decreased lower extremity sensation and paraplegia. A Foley catheter was placed due to severe urinary retention. CT angiography of the chest, abdomen and pelvis revealed a normal aorta, although the Artery of Adamkiewicz was not visualized. MRI of the lumbar spine was within normal limits except for edema and contrast enhancement of the left erector spinae musculature. Thoracic spine MRI revealed a signal intensity abnormality from the T1-T4 (Figure 1). The patient was paraplegic, with pinprick sensory loss below the level of T5, leg

Figure 1. MRI of thoracic spine illustrating area of infarction.



Figure 2. Axial MRI



hypertonia and absent plantar reflexes. Deep tendon reflexes were absent in the lower extremities bilaterally. Proprioception was preserved. Sagittal MRI demonstrated anterior spine infarction, while axial MRI was not definitive (Figure 2). The findings were attributed to acute ischemic stroke of the Artery of Adamkiewicz secondary to acute cocaine use. The patient was treated supportively and was discharged to a rehabilitation center.

DISCUSSION

This case presents a rare incidence of anterior spinal artery infarction secondary to cocaine use. Our patient presented with decreased pinprick sensation and motor loss below the level of T5, with preserved proprioception, indicating preservation of the posterior columns of the spinal cord consistent with an anterior spinal cord infarction.

Although cocaine use is associated with neurological complications, they are most commonly intracranial pathologies¹, such as intracranial hemorrhage, subarachnoid hemorrhage, cerebral vasculitis, and seizures² and only few instances of spinal cord infarction have been reported.^{1,2,3,4,5,6} Of the cases of spinal cord infarction reported, all appear to involve the anterior spinal artery.¹

The pathophysiology of cocaine-induced spinal infarction is unknown. Proposed mechanisms include vasospasm, vasculitis, vasoconstriction, embolus due to ventricular fibrillation, and thrombosis secondary to increased platelet aggregation.⁷

Cocaine prevents the uptake of neurotransmitters at the nerve terminal, which may increase sensitization of the nerves to epinephrine and norepinephrine, resulting in vasospasm². By potentiating the effect of monoamines and delaying their reuptake, cocaine induces hypertension and thus increases the risk for cerebrovascular events.⁹ Additionally, cocaine has been shown to increase platelet production of thromboxane, and therefore increase platelet aggregation, which may also account for cocaine-induced infarction². Likewise, cocaine is also associated with ventricular fibrillation and other cardiac arrhythmias, which could cause thromboemboli.

It is impossible to distinguish cocaine-induced spinal cord infarction from other causes of ischemia. There is no specific test to determine the exact etiology,⁴ therefore clinical presentation should alert the physician. While cocaine-induced spinal cord infarction is indeed rare, it should remain in the differential diagnosis of acute spinal cord pathology in the setting of a non-traumatic etiology.

CONCLUSION

Our report presents a rare case of cocaine-induced spinal infarction. While cocaine has been known to cause neurologic pathology, the majority of these cases involve the brain⁹. Clinicians should be aware of the anterior spinal artery infarction secondary to cocaine use.

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