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Cocaine-Induced Acute Fatal Basilar Artery Thrombosis: Report of a Case and Review of the Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 75
Final Diagnosis: Locked in syndrome
Symptoms: —
Medication: Aspririn
Clinical Procedure: MRI brain
Specialty: Neurology

Objective: Rare disease

Background: Cocaine use is a well-known contributing factor for both ischemic and hemorrhagic stroke; however, basilar artery thrombosis due to cocaine use is a rare entity with few cases reported in the literature.

Case Report: A 75-year-old African-American man with history of hypertension and cocaine use presented to the emergency room with coma. Neurological examination revealed asymmetrical dilated pupils and preserved oculocephalic and gag reflexes. The patient was noted to have semi-rhythmic jerking movement of the right arm and extensor posturing in response to noxious stimuli. Non-contrast computed tomography (CT) of the brain showed hyperdense basilar sign consistent with acute thrombosis. On brain magnetic resonance imaging (MRI), he was found to have bilateral pons acute ischemic stroke with early petechial hemorrhagic conversion. His laboratory work-up was unremarkable except for positive cocaine in the urine toxicology screen test.

Conclusions: Cocaine is a common global illicit drug that may trigger acute basilar artery thrombosis leading to a catastrophic neurological outcome.

MeSH Keywords: Basilar Artery • Cocaine-Related Disorders • Magnetic Resonance Imaging • Quadriplegia

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/894565

Background

Cocaine is the second most popular illegal recreational drug in the United States and United Kingdom, behind marijuana [1]. The National Survey on Drug Use and Health reported that in 2008, cocaine was used by 36.7 million people in the USA [2]. The European Commission on Drug Abuse has stated that 13 million adults have used cocaine at least once in their lifetime [3]. In the United States only, an estimated 1.7 million American have used cocaine between 2007 and 2012 [4].

Cocaine is a powerfully addictive stimulant drug made from the leaves of the *Erythroxylon coca* plant native to South America.

The main pharmacological mechanism of action is the inhibition of norepinephrine reuptake by sympathetic neurons, and the reuptake of 5-hydroxytryptamine (5-HT) and dopamine. Almost half of the cocaine-induced strokes are ischemic in nature, likely due to vasospasm of the cerebral arteries. Nevertheless, it has been postulated that cocaine may cause cerebral thrombosis due to the above-mentioned mechanism, which potentiate endothelial dysfunction and enhanced platelets aggregation [9].

With the increasingly widespread global use of cocaine, a broad spectrum of central nervous system potential hazards related to cocaine is emerging. Herein, we present an overview of the presentation, pathophysiology, literature review, investigation, and suggested treatment of this uncommon entity. This case illustrates the complex presentation of this rare subset of stroke, most likely due to cocaine use, which can result in grim neurological outcomes.

Case Report

A 75-year-old African-American man with history of hypertension and long-standing cocaine was brought to the emergency room after being found unresponsive for unknown period of time by a family member, as the patient lived alone. Upon arrival, his vital signs were remarkable for elevated blood pressure of 190/70 and he was gasping, with rapid and shallow breathing. His electrocardiogram (EKG) showed sinus tachycardia only. He was intubated immediately for airway protection. Neurological examination before intubation revealed a comatose patient with asymmetrical pupils; the right pupil was 5 mm and the left was 3 mm. His oculocephalic reflex and gag reflex were both preserved. He was noted to have a right upper extremity semi-rhythmic jerking movement and extensor posturing in response to sternal rub. He received intravenous glucose and naloxone en route to the hospital, without any response. Initial blood tests – complete blood count (CBC), basic metabolic panel (BMP), and cardiac enzymes – were all unremarkable except for acute kidney injury with elevated creatinine

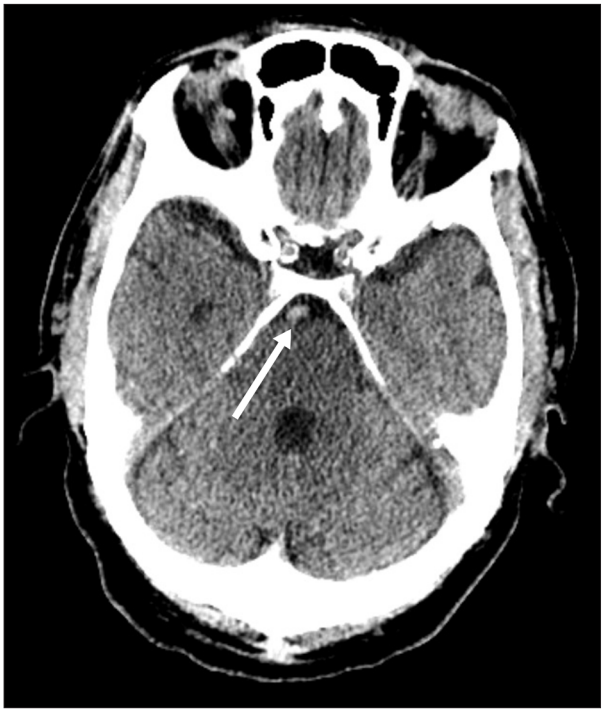

Figure 1. Axial image of brain CT scan, showing hyperdense basilar sign due to cocaine-induced basilar artery thrombosis (arrow).

level. An urgent non-contrast CT of the brain showed a hyperdense basilar sign consistent with basilar artery thrombosis (Figure 1). Subsequent brain MRI showed restricted diffusion in the pons bilaterally (Figure 2), and occlusive thrombus and filling defect of the basilar artery seen on the T1-weighted sequence with administration of gadolinium (Figure 3). Multiple foci of petechial hemorrhage (Figure 4) were seen as well. A transthoracic echocardiogram (TTE) showed normal ejection fraction and no evidence of cardiac thrombus or abnormal wall motion. Continuous electroencephalogram (EEG) obtained due to early concern for seizure versus status epilepticus showed no evidence of epileptiform discharges. Of note, his urine toxicology test was positive for cocaine. After discussion with the patient's power of attorney, the intensive care unit (ICU) team and the neuro-endovascular team, it was determined that the patient was found not a good candidate for an acute endovascular interventional procedure given the large size of his stroke, early hemorrhagic conversion, and poor neurological status. He was started on slow heparin infusion hoping to prevent further thrombosis while observing for evolving hemorrhagic conversion. The patient developed locked-in syndrome and he remained intubated. His endotracheal tube was replaced by a tracheostomy tube. He was started on enteral feeding through a nasogastric tube. One week later, he developed fulminant pneumonia and continued to worsen clinically, with absence of cortical somatosensory evoked responses. Unfortunately, he died shortly thereafter due to pneumonia.

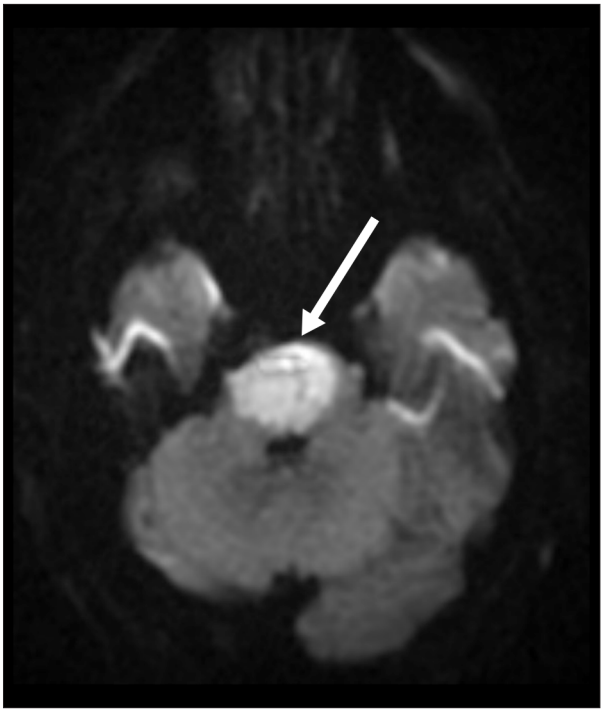

Figure 2. Increased signal of the diffusion-weighted imaging (DWI) in the bilateral pons consistent with acute ischemic stroke (arrow).

Discussion

Few cases in the literature have reported basilar artery thrombosis due to cocaine use. However, causality of basilar artery thrombosis due to cocaine is probably underreported. The definitive diagnosis of basilar artery occlusion due to cocaine is sometimes challenging and often delayed, and the complex neurological presentation of the basilar artery thrombosis may be misinterpreted as myoclonus seizure or a variant of movement disorders [10]. The lack of collateral information regarding illicit drug use during the initial presentation, especially in the case of coma, also may make diagnosis difficult. An overview of these reported cases i presented in Table 1.

Cocaine-induced basilar artery thrombosis can have multiple underlying pathophysiological mechanisms, resulting in various complex neurological presentations. The main mechanism of cocaine action is the inhibition of norepinephrine reuptake by sympathetic neurons, as well as the reuptake of 5-hydroxytryptamine (5-HT) and dopamine, also known as triple reuptake inhibitor (TRI), resulting in massive sympathetic overactivity, leading to tachycardia, arrhythmia, severe hypertension, acute coronary syndrome, and even sudden cardiac death [5]. This mechanism may trigger transient cardiac ischemia resulting in partial or global ventricular wall abnormality and cardiomyopathy and thus may potentiate a cardiac thrombus source. Cocaine can cross the blood-brain barrier faster and

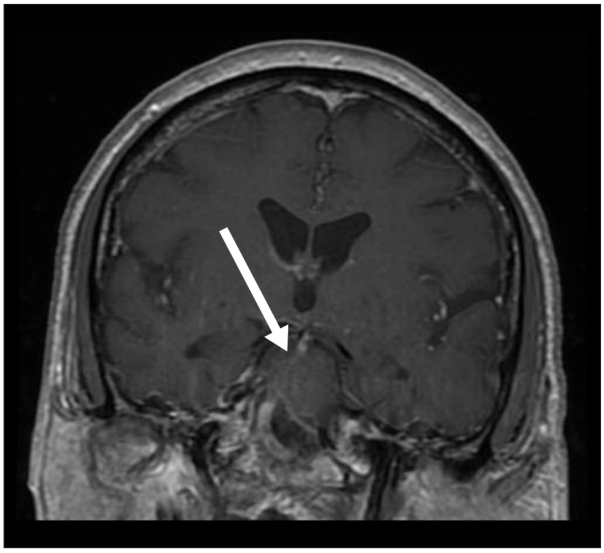

Figure 3. Coronal T1-weighted image after gadolinium administration shows a large filling defect consistent with occlusive thrombus of the basilar artery (arrow).

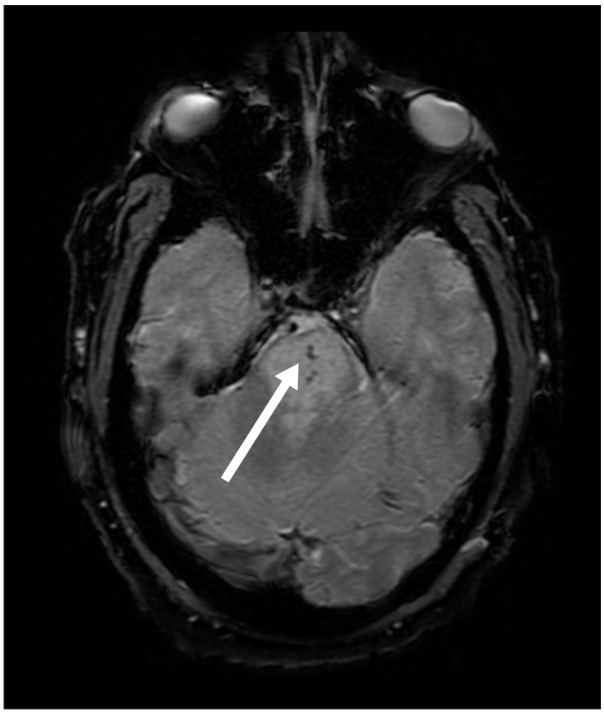

Figure 4. Susceptibility-weighted imaging (SWI) at the level of the pons shows hyperintense signal due to early hemorrhagic conversion (arrow).

far better than any other stimulant drugs due to its unique lipophilic efficiency [6]. Cocaine also induces apoptosis in the cerebral vascular smooth muscles, leading to ischemia and activation of endothelin-1-dependant receptors and subsequent cerebral vasoconstriction. Cocaine use is well known to induce various types of brain and cardiac vasculature damage,

Table 1. Overview of all case reports of cocaine-induced basilar artery thrombosis.

Case report	Patient age	Initial clinical presentation	Imagining modality before intervention	Presence of early hemorrhagic conversion	Time from presentation to intervention	Intervention modality	Outcome
Vallee et al. (2003)	25 years	Confusion/ dysarthria/ right hemiplegia	Brain CT MRI brain (DWI,FLAIR) MRA head	No	30 hours	Thrombaspiration	Favorable
MacEwin et al. (2008)	40 years	Writhing movements of limbs/slurred speech/ diplopia	Brain CT	No	11 hours	Mechanical aspiration plus intra-arterial thrombolysis	Favorable

including cerebral vasculitis, accelerated atherosclerosis, cocaine-induced cardiomyopathy, intracranial bleeding, and ischemic stroke [7,8].

Cocaine may produce a prothrombogenic state, including cerebral thrombosis, which induces and potentiates endothelial dysfunction and enhanced platelets aggregation especially if there are underling cerebrovascular risk factors [9]. This mechanism is likely responsible for the basilar thrombosis in our patient, given his older age, history of arterial hypertension, and long-standing cocaine use, which all might translate into an increased overall risk of basilar artery thrombosis.

The main aim of cocaine-induced stroke therapy is to establish reperfusion therapy promptly in cases of confirmed thrombotic or thromboembolic stroke within the standard window of time. However, in cases of basilar artery thrombosis outside the standard time window, intra-arterial thrombolysis and mechanical retrieval thrombectomy are suggested. Given the grim prognosis of complete basilar artery occlusion without treatment and the presence of many vascular collaterals, which can delay the development of irreversible damage, the window of time for thrombolysis is often longer in posterior circulation strokes. However, MRI diffusion and perfusion-weighted imaging should be obtained prior such decision to assess the ischemic penumbra and identify threatened but salvageable tissue that might benefit from thrombolysis or thrombectomy [11]. Vallee et al. and MacEwin et al. described successful intra-arterial thrombolysis and aspiration in patients with basilar artery thrombosis induced by cocaine [5,12]. In both case reports, the patients were young, had no reported significant medical history, and were not comatose at initial presentation. In addition, there was no early hemorrhagic conversion seen in the brain images. In such patients, intra-arterial thrombolysis with or without mechanical thrombectomy can be considered as a therapeutic option. In our case, the patient's age and presence of early hemorrhagic conversion suggested that further interventions would be unsuccessful. With the rapidly growing neuroendovascular field, more evidence and cases are expected to validate this view.

Conclusions

It is important to understand that cocaine use is still a significant health hazard with complex cardiac and neurological presentation. Various pathophysiological mechanisms are responsible for cocaine-induced basilar thrombosis. This case report illustrates the complex presentation of basilar artery thrombosis enhanced by cocaine use. Cocaine use should be routinely investigated by obtaining a urine toxicology screen for all patients with altered mental status. Brain MRI is essential tool and very helpful in planning management. Early diagnosis and treatment of basilar artery thrombosis in such situations is crucial due to potential persistent disabilities or even death. Healthcare workers should consider more aggressive management of basilar artery thrombosis if no early hemorrhagic conversion is seen in MRI or CT scans.

Conflict of Interest

None reported.

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