Cocaine Abuse: An Unusual Association

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Abstract
Cocaine addiction is a common problem in the adolescent and the young adults, pharmacologic interventions to reverse the effects of which do not exist. Neurological complications of cocaine abuse, such as seizures, headache, ischemic or hemorrhagic stroke, or subarachnoid hemorrhage, can be disastrous as a result of uncontrolled vasoconstriction and vasculitic damage. The lone occurrence of subdural hematoma in the absence of any other intracranial hemorrhagic complication is rarely seen in patients of cocaine abuse. We wish to share our experience of one such patient who presented to us with an unusual combination of the widespread cerebral infarction and subdural hematoma.

Introduction
Cocaine is one of the commonly abused psychostimulant drugs in contemporary times. Chemically, cocaine is a local anesthetic, with potent vasoconstrictor properties and whose addictive effects appear to be related to activation of dopaminergic neurons in the mesolimbic system. Its abuse may lead to a number of adverse health consequences and a dramatic increase in deaths is reported due to neurological and cardiovascular complications. Neurological complications are the most common complaints among cocaine abusers presenting to the emergency department and appear to be particularly devastating.¹ These include seizures, headache, ischemic or hemorrhagic stroke, and subarachnoid hemorrhage.

We, herein, present a case of cocaine abuse with a very unusual combination of neurological complications in the form of acute infarction and subdural hematoma, but without any evidence of an intracerebral hemorrhage.

Case Report
A 22-year-old right-handed student presented to university, a tertiary care referral facility, in an altered sensorium for 2 days. He was addicted to cocaine both by intravenous as well as intranasal route, for the past 2 years. There was no history of fever, headache, trauma to head or body, any intercurrent illness, diabetes mellitus, hypertension, or previous vascular disease.

On examination, he was afebrile with a blood pressure of 110/70 mm Hg and a pulse rate of 64 beats per minute; the Glasgow Coma Scale score was 8/15 (E3,M5,V0). There was marked paucity of movements involving the right half of the body; the deep tendon jerks were brisk and the plantar response was extensor bilaterally. There were no signs of meningeal irritation and the oculocephalic reflex was normal.

Investigations revealed a normal hemogram, renal function tests including electrolytes, liver function tests, urine analysis, chest X-ray and echocardiogram. Prothrombin time, activated partial thromboplastin time, arterial blood gas analysis, protein C, protein S, and homocysteine levels

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Fig. 1: Axial sections (A, B, C) of non-contrast computed tomography of the head depict an acute subdural hematoma (arrows), falcine hyperdensity (arrow-heads) and hypodense involvement (asterisk) of the medial part of left occipital lobe. There is effacement of sulci of the left hemisphere and suggestion of mass-effect.

were also found to be within normal limits. He was non-reactive to HIV, hepatitis-B and hepatitis-C assays. Cranial computed tomography done at the time of presentation (outside our facility) was suggestive of an acute infarct involving the left temporo-parieto-occipital region and a subdural hematoma, that matched with the density of the skull rendering it almost inconspicuous (Figure 1 A-C). Magnetic resonance imaging of the brain done on the 3rd day revealed an extra-axial concavo-convex lesion displaying signal intensity alterations in the left fronto-temporo-parietal region, suggestive of subdural hematoma, with mass effect, in the form of effacement of cortical sulci, Sylvian fissure, ipsilateral lateral ventricle, and a midline shift of approximately 8 mm towards the right side. Signal intensity alterations suggestive of an acute infarct were observed in the posterior part of body and sphenoid of corpus callosum, bilateral frontal, left medial temporal and parasyllian, left occipito-parietal regions and the right cerebral peduncle (Figure 2 A-L). Magnetic resonance angiography did not reveal any abnormality.

A neurosurgical consultation was sought after which the patient was managed conservatively with measures to reduce the intracranial pressure. During the hospital stay, the patient became alert and responsive, and his GCS improved to 11/15 (E4M6V1); he was discharged on the 17th day of admission. At 12 weeks of follow-up, he was independent for his activities of daily living with a residual hemiparesis (MRC grade 4/5).

**Discussion**

On the background of cocaine abuse, our patient presented with a bizarre combination of subdural hematoma with widespread ischemic foci, in the absence of subarachnoid or parenchymal hemorrhage. The various reported neurological complications of cocaine are transient cerebral ischemia, cerebral and spinal cord infarction, ischemic optic neuropathy, seizures, intracerebral and subarachnoid hemorrhage. Of the vascular complications, subdural hematoma is rare and has not been observed in patients with cocaine abuse in the absence of an aneurysm or an arteriovenous malformation.

Several mechanisms have been suggested for cocaine-induced neurological insult, which include transient hypertension, migraine, vasospasm and vasculitis. Volkow et al compared the relative distribution of cerebral blood flow (CBF) in a group of chronic cocaine users with the normal people using positron emission tomography. They found areas of deranged CBF as evidenced by patchy regions of defective isotope accumulation throughout the brains of cocaine users, especially in the prefrontal cortex. This was considered to be the effect of vasospasm in cerebral arteries of brains that were chronically exposed to cocaine and its vasoconstrictor effects.

Saleh et al reported a case of spontaneous acute subdural hematoma and the mechanism explained in the absence of a definite vascular lesion was cocaine-induced vasospasm. The acute increase in blood flow following the vasospasm led to the rupture of vessel wall which was already damaged by vasospasm-induced ischemia. This mechanism probably explains the occurrence of acute spontaneous hemorrhages of arterial origin which may occur in the superficial arteries of arachnoid matter and organize to form subdural hematoma. However, they failed to identify any classical mechanisms described earlier for the pathophysiology of cortical artery bleeding.

Fredericks et al reported a
patient with acute encephalopathy following intravenous and intranasal administration of cocaine whose brain biopsy was suggestive of cerebral vasculitis.\(^5\) Angiographic narrowing of the supraclinoid portion of the internal carotid artery, without any presence of vasculitis, has been shown in a gentleman presenting with right middle cerebral territory infarct; it was suggested that cocaine-induced vasospasm due to sympathomimetic actions of cocaine was the reason for infarction.\(^6\) The absence of vasculitis producing contaminants (amphetamine) in cocaine and the immediate complaint of headache post-cocaine use strongly favors vasospastic pathogenesis of cocaine rather than vasculitic process in amphetamine poisoning. The vasospasm induced by cocaine is due to inhibition of norepinephrine uptake at peripheral sympathetic terminals.

Cocaine abuse should be considered in young adults in the differential diagnosis of non-traumatic acute subdural hematoma. Alves et al reported a case of spontaneous acute subdural hematoma caused by cocaine abuse.\(^7\) Spontaneous acute subdural hematoma without the presence of intraparenchymal or subarachnoid hemorrhage is a rare occurrence. The various non-traumatic causes of spontaneous acute subdural hematoma reported in the literature are ruptured intracranial aneurysm, ruptured cortical artery, hypertensive cerebral hemorrhage, malignancy, hematologic disorders, anticoagulant and thrombolytic therapy, cerebral amyloid angiopathy, dural arteriovenous fistulas and acquired immunodeficiency syndromes.\(^8\) One of the rare reasons is aneurysmal rupture causing subdural hematoma that is usually confined to subarachnoid hemorrhage and intracerebral hemorrhage. Koebrel et al was the first one to report an internal carotid artery bifurcation aneurysm manifesting as acute subdural haematoma.\(^9\) The mechanism proposed was high pressure leading to pia-arachnoid rupture and extravasation of blood into the subdural space. This appears to be more common in superficially located aneurysms which are predisposed to bleed in subdural space.

**Conclusion**

This case report demonstrates the unusual combination of the adverse effects of cocaine abuse in the form of cerebral infarction as well as the subdural hematoma. The patient had presented in an altered sensorium and on CT an infarct was seen, probably as an effect of cocaine induced vasospasm; however a subtle subdural hematoma was missed on CT and was later confirmed on MRI. The lone presence of SDH, without the presence of the subarachnoid hemorrhage and intracranial hemorrhage, makes this case unique in its occurrence. Further, the absence of aneurysm on MR angiography attests the role of cocaine abuse as the cause of isolated SDH.

**References**