

Cocaine-Induced Stroke in A Young Patient

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Received: 02 Mar 2024; **Accepted:** 30 Mar 2024; **Published:** 07 Apr 2024

Citation: Mohamed Amine Mnaili, Ahmed Bourazza. Cocaine-Induced Stroke in A Young Patient. J Med - Clin Res & Rev. 2024; 8(4): 1-3.

ABSTRACT

Cocaine consumption has increased over the last decade. The potent sympathomimetic effects of the drug can lead to serious neurovascular complications in the form of ischemic stroke (IS), intracerebral hemorrhage (ICH), or subarachnoid hemorrhage (SAH). A young adult patient arrived at a specialized unit with neurological deficits and a history of acute and long-term illicit drug use and presented no other comorbidities. In addition, he presented laboratory and serological tests without alterations. The only risk factor identified for stroke was, therefore, cocaine use.

Keywords

Cocaine, Stroke, Young adult.

Introduction

Cocaine is one of the most commonly used illicit drugs worldwide and is associated with a wide range of neurological complications, including ischaemic stroke. Cocaine-induced strokes are ischemic in nature, likely due to vasospasm of the cerebral arteries caused by excess neurotransmitters at the synaptic cleft [1]. In addition, cocaine may promote thromboemboli through enhanced platelet aggregation and potentiated endothelial dysfunction [1]. The case report presented here addresses the relationship between cocaine use and stroke

Case Presentation

A 28-year-old man was found unconscious by his father in his bedroom in the morning drinking alcohol and using cocaine during a night out with his friends. In the emergency room, a convulsive episode was observed. On arrival at the emergency department he presented dysarthria with heaviness of the left hemibody.

The patient's initial vital signs were as follows: body temperature of 37.1°C, heart rate of 59 to 67 beats/minute and blood pressure of 103/63 mmHg. He also had normal respiratory pattern and pulse oximetry. His first medical examination revealed that he was confused and very agitated and was not following commands.

Additionally, his neurological examination result was negative for muscle rigidity, nystagmus, or diffuse hyperhidrosis. However, his examination revealed dysrthria, left central facial paralysis and total left hiplégia. He scored 18 in the National Institutes of Health Stroke Scale (NIHSS). Head CT scan demonstrated Indirect signs of stroke: focal brain edema, obliteration of cortical sulcus and spontaneous hyperdense artery in the territory of left middle cerebral artery.

Chest x-ray and electrocardiogram were normal. Routine laboratory tests disclosed mild leukocytosis. Other tests such as renal function, C-reactive protein, glycemia and serology for HIV, syphilis and hepatitis did not present abnormalities. The result of her urine toxicology was positive for cocaine.

The patient was admitted to the Stroke Unit for neurological monitoring. On the third day of hospitalization, Control Head CT scan showed (Figure 2).

Transthoracic and transesophageal echocardiography, carotid and spinal Doppler ultrasound revealed no abnormalities. The patient's condition showed a partial improvement with a progression towards moderate dysarthria associated with a disproportionate left hemiparesis with brachial predominance. His NIHSS score improved to 9 as he had 2/0 for facial palsy, 3/0 for upper limb motor control, 2/0 for lower limb motor control and 2/0 for dysarthria.

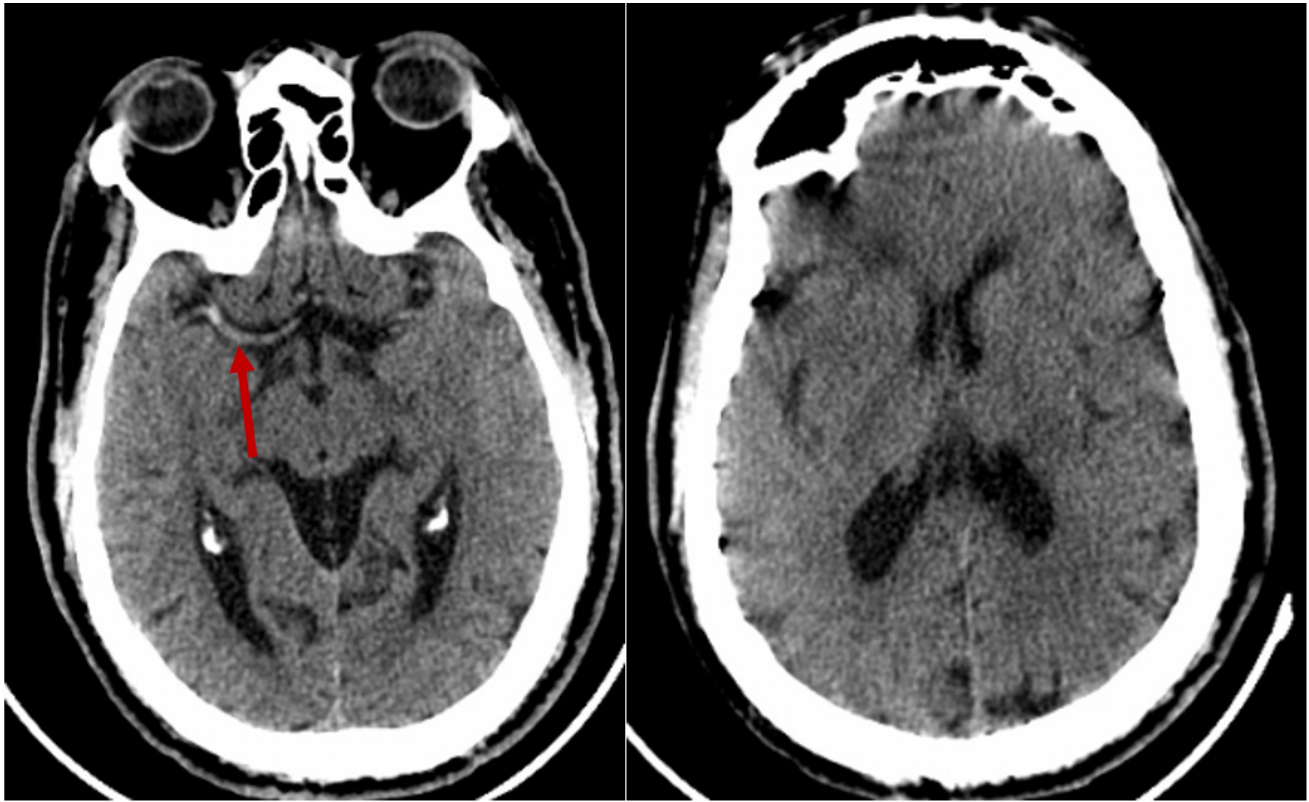


Figure 1: Head CT scan showed indirect signs of stroke: focal brain edema, obliteration of cortical sulcus and spontaneous hyperdense artery in the territory of right middle cerebral artery (arrow).

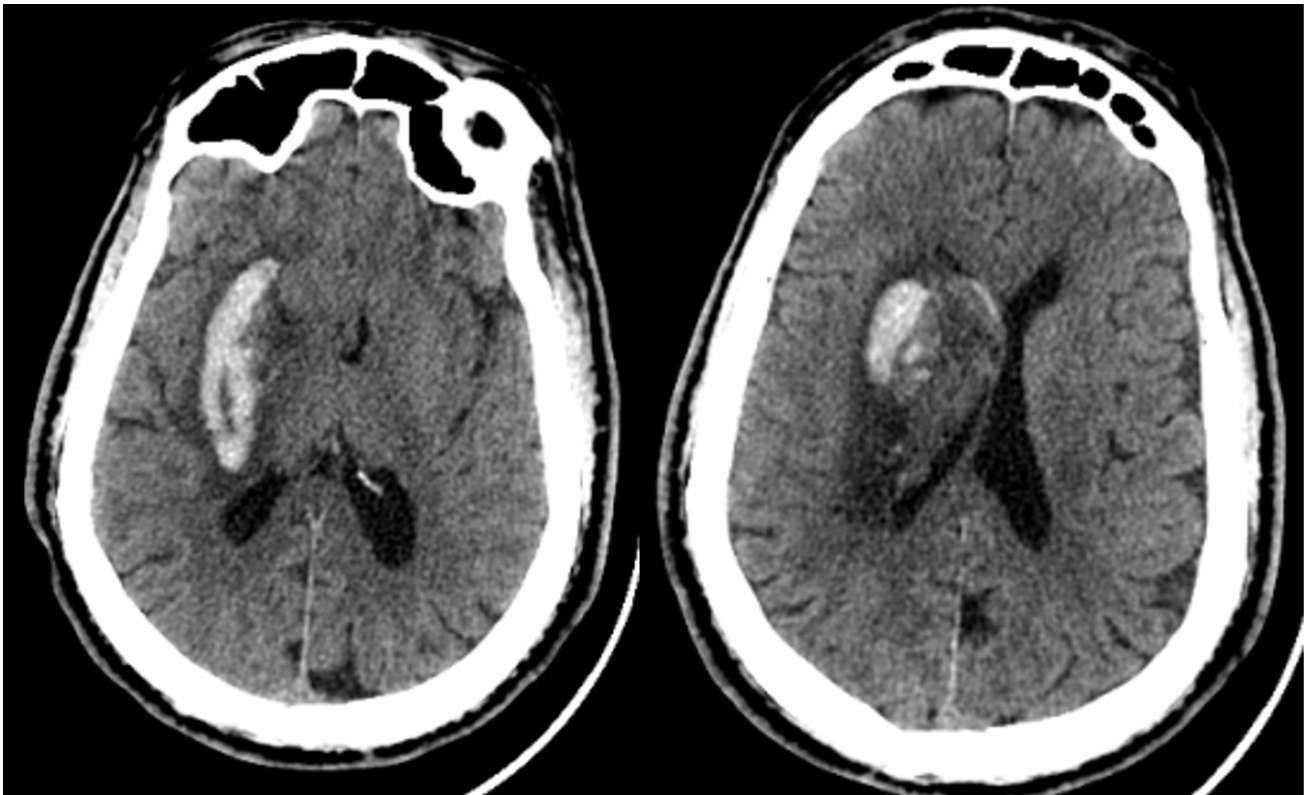


Figure 2: Head CT scan after 3 days of hospitalization showing ischemic stroke with hemorrhagic transformation in the deep territory of the right middle artery.

The patient was alert and cooperative with the medical team and was released 20 days after his hospitalization and oriented towards multi-professional outpatient follow-up.

Discussion

Ischemic brain damage secondary to cocaine is a well-recognized phenomenon. In young to middle aged adults, cocaine use is associated with a relative risk of ischaemic stroke six to seven times higher than age-matched controls in the 24 h after use [2]. In our case, the absence of other risk factors triggering the cerebrovascular event in a young adult indicates that the relationship with cocaine use is highly likely.

The concomitant use of cocaine, alcohol and tobacco may potentiate the toxicity due to possible causal associations, such as the release of inflammatory factors [3]. The patient reported no use of cannabis. With the use of cannabis, the prevalence of ischemic and hemorrhagic stroke corresponds to 1.2% and 0.3%, compared to the prevalence of 0.8% and 0.2% in non-users, respectively; the influence of the time of exposure with these rates is not well documented [4]. Imagery must be requested immediately after admission and after 24 hours for monitoring. The latter, in the reported case, showed changes that raised a discussion regarding the possibility of a transformation of ischemic into hemorrhagic stroke or of an hemorrhagic stroke from the beginning.

Additional measures that can be adopted include toxicological examination and serology for HIV-1, hepatitis C and syphilis for the exclusion of differential diagnoses. The patient in the present case did not present significant laboratory changes in serologies and other tests. Thus, other causal associations were ruled out. The exact mechanism of cocaine related stroke remains unclear and there are likely to be a number of factors involved. The issue is complicated further by the fact that contaminants such as procainamide, quinidine and antihistamines, which are often mixed with the cocaine, may contribute to the effects seen and influence the underlying pathophysiology [5]. Cocaine is a potent vasoconstrictor due to its sympathomimetic action, preventing the reuptake of noradrenaline, serotonin and dopamine at pre-synaptic nerve terminals. However, it also has direct effects on calcium channels, promoting intracellular calcium release from the sarcoplasmic reticulum in cerebral vascular smooth muscle cells [6]. Cerebral vascular thrombosis may occur secondary to vasospasm, and it has been suggested that vasospasm results in endothelial injury and platelet aggregation with subsequent release of smooth muscle growth factor and obstructive intimal hyperplasia. Advanced atherosclerosis has been observed in the renal arteries and aorta of cocaine users [7]. Another potential mechanism for ischaemic stroke associated with cocaine use is cardioembolism. Cerebral emboli with subsequent infarction can originate from cardiac thrombi which form during cocaine induced myocardial infarction, and case reports have also documented embolic stroke secondary to cocaine related cardiomyopathy [8,9].

The management of stroke in patients experiencing acute psychostimulant intoxication is similar to the protocol for stroke with no apparent cause. The actions are determined according to the time of onset of symptoms [10]. The patient in the present case was not eligible for reperfusion therapies proven to be effective such as intravenous thrombolysis and mechanical thrombectomy.

Conclusion

This case report illustrates the need for routine investigation by obtaining a urine toxicology screen for all patients with altered mental status and the importance of awareness of the serious complications of cocaine use.

In our patient, we had only identified cocaine use as risk factor. The clinical situation, therefore, calls attention to the harm that the use of psychostimulants, especially cocaine, can bring to young adults, including increased risk of ischemic or hemorrhagic stroke. Potential mechanisms involved in cocaine-induced stroke include vasospasm, cerebral vasculitis, enhanced platelet aggregation, cardioembolism, and hypertensive surges associated with altered cerebral autoregulation.

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