

# Cocaine-induced stroke in a young patient

## Acidente vascular cerebral induzido por cocaína em paciente jovem

Taianne Maria da Cruz Rocha<sup>1</sup> , Luanny de Andrade Cardoso Fragoso<sup>1</sup> , Vítor Nunes Rodrigues<sup>1</sup> , Claudio José dos Santos Júnior<sup>2,3</sup> , Euclides Mauricio Trindade Filho<sup>1,2</sup> 

### ABSTRACT

The case report presented here addresses the relationship between cocaine use and stroke. 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. Thus, this work points to an eventual relationship between cocaine use and the case in question.

**Keywords:** Cocaine, Stroke, Young adult, Epidemiology.

### RESUMO

O relato de caso apresentado neste trabalho, aborda a relação do uso da cocaína e o acidente vascular cerebral após um paciente adulto jovem chegar a uma unidade especializada com déficits neurológicos e histórico de uso de drogas ilícitas de forma aguda e de longa data, sem outras comorbidades constatadas. Além disso, apresentou exames laboratoriais e sorológicos sem alterações significativas. O único fator de risco identificado no caso em tela para o acidente vascular foi, portanto, o uso de cocaína. Assim, neste trabalho, enfatiza-se eventual relação entre o uso da cocaína com o caso em tela.

**Palavras-chave:** Cocaína, Acidente vascular cerebral, Adulto jovem, Epidemiologia.

## INTRODUCTION

Stroke is one of the leading causes of death and physical and mental disability in men and women worldwide<sup>1</sup>. Cocaine abuse carries an approximately two-fold higher risk of hemorrhagic and ischemic stroke and subarachnoid hemorrhage<sup>2</sup>. The prevalence of users of psychostimulants and other drugs aged 15-64 years has increased by 23% in the last five years, and Brazil ranks second in the world in the use of cocaine<sup>3</sup>. The increasing number of young adults among these users is worrying because in the absence of other triggering factors, cocaine use shows an important relationship with cerebrovascular events. Such relationship, however, has been poorly investigated during the the provision of care to this public.

The alarming growth of both the number of cocaine users and the incidence of stroke, in addition to the association with worse outcomes, is a cause of concern. Users are at risk of fatal consequences<sup>4</sup>. The United Nations points to figures of about 200 million users of illegal drugs worldwide and stresses that these substances cause major social and health problems, especially in view of the increasing consumption among young people<sup>5</sup>. The situation in Brazil is worrying because this country accounts for 20% of the consumption of cocaine worldwide and has the largest market of drugs in the world<sup>6</sup>. In addition, stroke is the second leading cause of death in the world, responsible for 5.7 million deaths each year, and this number is expected to be approximately 7.8 million by 2030<sup>5</sup>.

The case analyzed in the present study raised a question about the relationship between

<sup>1</sup> CESMAC University Center, Maceió, (AL), Brazil.

<sup>2</sup> State University of Health Sciences of Alagoas, Maceió, (AL), Brazil.

<sup>3</sup> University of São Paulo, (SP), Brazil.



cocaine use and the cerebrovascular condition presented by the patient. The patient was a young male adult aged 49 years, without comorbidities or associated genetic factors or previous history of surgeries or cardiovascular and cerebral events. However, he was a chronic user of cocaine and presented focal deficits suggestive of stroke hours after using the drug. It is known that acute use of cocaine increases by 6.4 times the incidence of stroke within 24 hours after use in relation to the situation in which this drug is never used<sup>5</sup>.

The patient had used cocaine concomitantly with alcohol and tobacco, which may have potentiated the toxicity due to possible causal associations such as the release of inflammatory factors<sup>7</sup>. Convulsive episodes may be present in these cases, as in the patient studied here, according to information provided by the emergency unit. Seizure has a prevalence of 0.9-10.4% among cocaine users and, together with stroke, constitutes the main source of neuropsychiatric damages caused by the use of psychostimulants<sup>2</sup>. These data stress the importance of the identification of seizures and their management in the presence of this causal effect. Here we will describe a case of stroke in a young adult without previous comorbidities whose only risk factor was cocaine use.

## CASE REPORT

A 49-year-old male was found unconscious on the street early in the morning after he had drunk alcohol and used cocaine in a city in the countryside of the state. It was estimated that the last time he was well was at dawn. In the emergency unit, a convulsive episode was observed. Upon arriving at a hospital that has one of the stroke units of the capital, he presented dysarthria, deviation of lip rhyme to the left, hemiplegia in right dimidium and decreased superficial and deep sensitivity in the right dimidium of sudden onset, without other findings. At admission, his blood pressure and heart and respiratory rates were in the normal ranges. He also had normal respiratory pattern and pulse oximetry. He was alert, with a Glasgow coma scale score of

15, and isochoric and photoreactive pupils. There were no changes in the evaluation of the cranial nerves. He scored 10 in the National Institutes of Health Stroke Scale (NIHSS) because he presented 1/0 for facial palsy, 3/0 for motor command in the upper limbs, 4/0 for motor command in the lower limbs, 1/0 for sensitivity and 1/0 in the evaluation of dysarthria. After evaluation of the other systems, excoriations were found in the right forearm region, without other alterations or complaints.

When questioned, the patient reported ingestion of beer and hard liquor in addition to the use of cocaine by inhalation hours before the onset of the condition. He also admitted he had been a user of illicit drugs on a weekly basis for more than ten years and a long-time smoker. He informed no comorbidities or previous history of stroke and Acute Myocardial Infarction (AMI), use of medications, recent surgeries and previous hospitalizations. He had no family history of stroke.

After initial laboratory tests, he presented leukocytosis (14,010 per  $mm^3$ ) without deviation, increased TGO (120 IU), uric acid (9.30mg/dL), lactic dehydrogenase (799 mg/dL), total cholesterol (147mg/dL), and triglycerides (332mg/dL). Other tests such as evaluation of renal function, electrolytes, TGP, C-reactive protein, alkaline phosphatase, glycemia and serology for HIV, syphilis and hepatitis did not present abnormalities. In a computed tomography (CT) of the skull, an area of cortico-subcortical hypoattenuation was found on the left side, suggesting acute ischemic infarction, affecting the insula, lentiform nucleus, posterior portion of the internal capsule and M2, in a territory of partial irrigation of the middle cerebral artery, with Alberta Stroke Program Early CT Score (ASPECTS) of 6. Arterial angiogram of the intracranial and cervical vessels showed no significant changes. Transthoracic echocardiography showed an ejection fraction of 68% and moderate diastolic dysfunction of the left ventricle. No change was found in the electrocardiogram.

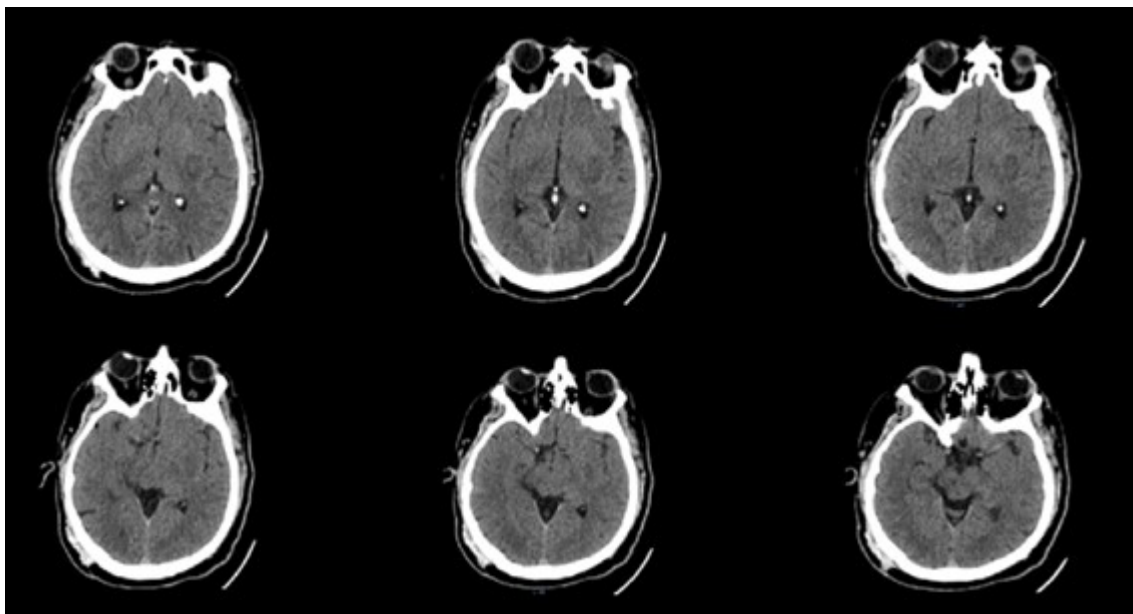
The patient was admitted to the Stroke Unit for neurological monitoring. The following measures were prescribed: absolute bed rest with the head of the bed elevated 30 degrees, cardiac monitoring, non-invasive pulse oximetry,

decubitus change every three hours, intravenous hydration with saline solution (SS) 0.9% 500 mL every twelve hours, omeprazole 40 mg (one ampoule) under fasting conditions, one 40 mg tablet of simvastatin after dinner, acetylsalicylic acid (ASA) 100 mg once a day, one 5 mg tablet of diazepam orally every eight hours, and one 200 mg tablet of carbamazepine orally every twelve hours, the latter two for monitoring of abstinence and seizure control, respectively. Multidisciplinary follow-up was also performed with occupational therapy, social work, speech therapy and physiotherapy, the latter performed only when absolute rest was not indicate any longer. No thrombolysis and/or thrombectomy were performed, as the patient arrived at the unit outside the established time for the application of these procedures.

On the second day of hospitalization, when a control cranial tomography showed a recent right parietal subgaleal hematoma without association with fractures remains, hematic collection in the reabsorption stage in the nuclei of the base on the left with perilesional edema measuring about 1.8 x 1.1 cm, and partial collapse of the ventricular chambers on the left, without deviation from

the midline of the brain, the medical team started to question whether an ischemic stroke (ischemic cerebrovascular accident [ICVA]) was progressing to a hemorrhagic stroke (hemorrhagic cerebrovascular accident [HCVA]) or whether it was the case of a HCVA since the beginning. Although literature data on the effects of inhaled cocaine are still inconclusive, it is known that the substance may have an impact on stroke and increase the risk of hemorrhagic events (by approximately 70%), and the use of this substance tends to lead to several systemic changes that predispose to the appearance of ischemic strokes. The latter are more often described in the literature, when evaluated the other routes of use of the drug. Since the cause of the event of the patient in the present study could not be determined, there was a need to suspend the use of acetylsalicylic acid to avoid further bleeding and damage to the patient's health. Phenobarbital 100 mg at night was prescribed and hydration was intensified to 500 mL of SS 0.9% every eight hours.

A new non-contrast-enhanced cranial CT of the skull was performed on the fifth day post-ictus to evaluate the evolution of the bleeding and



**Figure 1:** Computed tomography of the skull showing an area of cortico-subcortical hypoattenuation on the left, suggestive of acute ischemic infarction, affecting the insula, lentiform nucleus, posterior portion of the internal capsule and M2, in a territory of partial irrigation of the middle cerebral artery (ASPECTS 6).

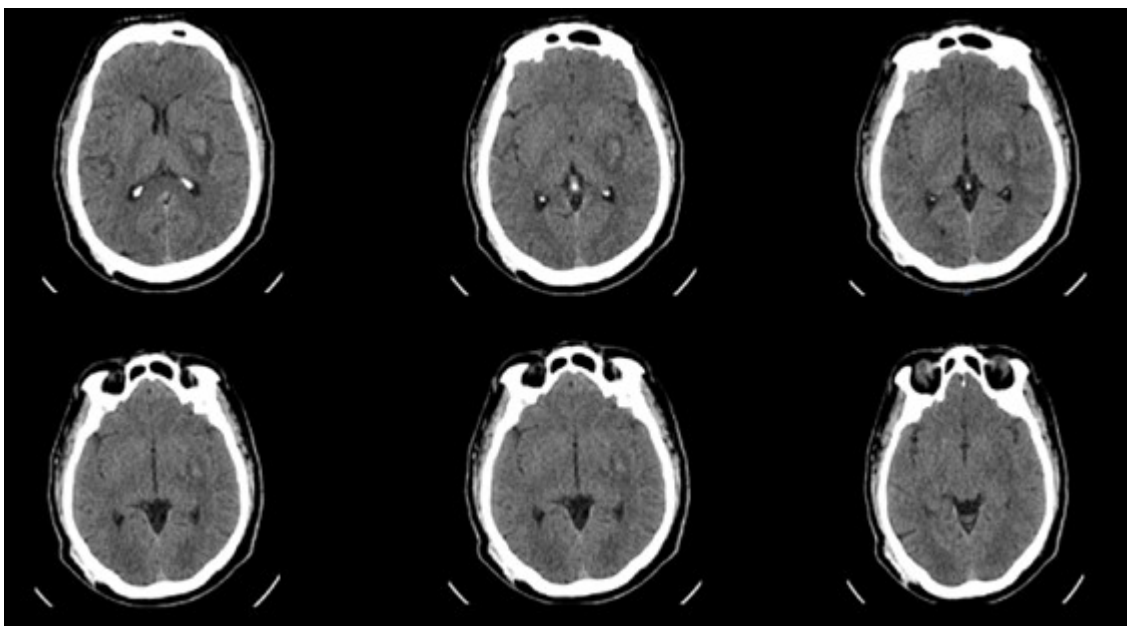
a hypoattenuating area was detected, compromising the left nucleocapsular and subinsular region, related to subacute ischemia, with a focus of conversion of hemorrhage into reabsorption, with persistence of a small central dense area. Other portions of the brain parenchyma presented normal morphology and attenuation. Cortical sulci and basal cisterns had preserved appearance for the age group. No signs suggestive of intracranial expansive formations and hemorrhage were detected.

The patient's condition presented partial improvement, as it was possible to observe an evolution to mild dysarthria and less deviation of lip rhyme to the left, in addition to a disproportionate right hemiparesis of brachial predominance. His NIHSS score improved to 6, as he presented 1/0 for facial palsy, 3/0 for motor command in upper limbs, 1/0 for motor command in lower limbs, and 1/0 for dysarthria. There were no changes in laboratory tests. The patient was alert and cooperative with the medical team and was discharged 12 days after hospitalization and referred to multiprofessional outpatient follow-up and external prescription.

## DISCUSSION

The prevalence of users of psychostimulants and other drugs aged 15-64 years has increased by 23% in the last five years, and Brazil ranks second in the world in the use of cocaine<sup>3</sup>. Acute intoxication by the use of this stimulant can have neurotoxic adverse effects. Due to the vasoconstrictive and thrombogenic properties of this drug, its chronic and indiscriminate use is associated with worse outcomes in cases of stroke<sup>2</sup> as well as with greater risk of both ischemic and hemorrhagic stroke, especially in individuals between the 3<sup>rd</sup> and 4<sup>th</sup> decade of life<sup>5</sup>. However, this association is rarely made during the assistance provided to this public. In this study, the absence of other risk factors triggering the cerebrovascular event in a young adult indicates that the relationship with cocaine use is highly likely.

Some risk factors are widely known to interfere with the outcome of stroke in each patient. Hypertension, atrial fibrillation, heart failure, smoking, alcohol consumption, low physical activity, overweight, and hypercholesterolemia are among the main ones<sup>8,9</sup>. Among cases of



**Figure 2:** Computed tomography of the skull after 24 hours of hospitalization showing the presence of recent right parietal subgaleal hematoma without association with fractures remains, hematic collection in the reabsorption stage in the nuclei of the base on the left with perilesional edema measuring about 1.8 x 1.1 cm.

stroke, 64% are overweight and 26% are obese patients<sup>10</sup>. The patient in the present case did not present altered Body Mass Index (BMI), comorbidities or associated genetic factors. However, he was a chronic user of cocaine and presented focal deficits suggestive of stroke hours after using cocaine. The use of this substance may lead to a worse prognosis and alter the coagulation state through mechanisms of platelet activation, endothelial injury and tissue factor expression<sup>11</sup>. The concomitant use of cocaine, alcohol and tobacco may potentiate the toxicity due to possible causal associations, such as the release of inflammatory factors<sup>12</sup>. 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<sup>13</sup>.

Cocaine abuse carries an approximately two-fold higher risk of hemorrhagic and ischemic stroke and subarachnoid hemorrhage<sup>2</sup>. Thus, the use of this drug should be investigated mainly in patients aged between 15 and 64 years because 4.8% of the users of cocaine worldwide correspond to people in this age range and this may be the main cause of cerebrovascular events in young adults without comorbidities. In the clinical case presented here, the 49-year-old patient used cocaine on a weekly basis for about 10 years, mainly inhaling it. The last time he had used the drug was a few hours before the episode. A 6.4-fold higher incidence of stroke within 24 hours after use is reported among people making acute use of cocaine when compared to people who never use cocaine<sup>5</sup>.

Widespread and increasing use of cocaine is a public health problem due to numerous physical and mental health harms associated with the use of psychostimulants<sup>2</sup>. Cocaine can cause neuropsychological deficits similar to cognitive disorders with alterations in areas of attention, language, memory, learning and executive functions. In addition, unwanted acute effects include agitation, sweating, vomiting, headache, delirium and hallucinations, as well as cardiovascular, neurological and cerebrovascular complications<sup>11</sup>. Convulsive episodes may also be present, as

observed in the present case when he was being assisted in the emergency unit. He may have experienced a decrease in the convulsive threshold due to the release of dopamine with subsequent depletion of serotonin and noradrenaline and suppression of gamma-aminobutyric acid due to cocaine use. He also showed loss of consciousness and deficits associated with the stroke.

Neurological evaluation and neuroimaging tests are mandatory for the diagnosis of stroke. The patient may present focal deficits and changes in CT such as the presence of a hypodense area in case of ischemic stroke. The tomography 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. Angiotomography may also be requested to evaluate, for example, possible lesions in the cerebral and cervical vessels. In addition, magnetic resonance imaging tests can be used if available in the medical unit. 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 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<sup>14</sup>. The patient in the present case was not eligible for reperfusion therapies proven to be effective such as intravenous thrombolysis and mechanical thrombectomy. Thus, initiating monitoring and damage control protocols was recommended. In the present case, after the appearance of a hemorrhagic collection, antiplatelet medication was discontinued and the need for surgical decompression was considered. After discharge, outpatient follow-up with multiprofessional support, periodic neurological evaluations and prophylaxis against new events was necessary<sup>15</sup>. Further, access to the substance should be restricted.

## CONCLUSION

In this study, we present a case of stroke in a young patient whose only identified risk factor was cocaine use. 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.

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Corresponding Author:  
Taianne Maria da Cruz Rocha  
[taianecruz@hotmail.com](mailto:taianecruz@hotmail.com)

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