Hemorrhagic cerebral vascular accident associated with cocaine consumption

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Abstract

The clinical case of a 37-year-old man, a cocaine consumer, who was admitted through the Emergency Department having suffered an intracranial hemorrhage, is described. The authors discuss briefly the differential diagnosis and complications associated with the use of cocaine.

Key words: cocaine, stroke.

Introduction

The amounts of cocaine seized have increased considerably in recent years (360 kg was seized in 1990 and 2,115 kg in 1995).[1] Thus, it appears that its consumption is on the increase, and with it, medical complications related to its toxicity.

Cocaine was introduced to the clinical practice in the second half of the 19th century, when it was used as a local anesthetic and as a stimulant in the treatment of morphine and alcohol addiction, and depression, among other ailments. Access to the drug was easy, and it was used as an additive in many commercially available products. Until 1903, it was part of the formula of Coca-Cola. Recognizing its capacity to create addiction, it was withdrawn from the market in 1914. It was deemed a drug inducing a maximum of addictive behaviour.[2,3,4]

The psychological effects, for which cocaine is consumed, are violent and unpredictable, of short duration, and bring feelings of euphoria, exaltation, lack of inhibition, excessive self-esteem and hyperactivity. The disappearance of the effects is followed by feelings of dysphoria, anergy, anxiety, drowsiness, depression and irritability that can last for several days.[3,5,6]

Coca (leaves of Erytroxylon coca or E. novgranatense) can be chewed, but this is not the form of consumption in Europe and the United States. The first step in cocaine production is obtaining a coca paste, which can be smoked mixed with tobacco and/or cannabis. This paste is then used to obtain cocaine, a water-soluble powder, available on the market in mixtures of 12 to 75% purity. In this form it can be aspirated (slower absorption through the nasal mucosa) or administered intravenously. A form derived from the alkalinization of cocaine, known as crack or cocaine base, which can be smoked through a water pipe, became popular in the 1980s. The intravenous administration of cocaine and the use of crack are the forms in which the drug reaches the systemic circulation and the brain faster, and in the latter form, with longer lasting effects.[3,5]

The half-life of cocaine is from 30 to 90 minutes, when it is metabolized in the liver and excreted in the urine. The two most important metabolites are ecgonine methyl ester and benzoylecgonine, with 3.5 to 6 hours and 5 to 8 hours of half-life, respectively. Due to its short half-life, the inability to detect the drug or its metabolites in the blood or urine should not rule out its recent use. Cocaine metabolites can be detected up to about two to five days after use.[2,7]

Case report

The patient is a 37-year-old Spanish male, resident in Portugal for about five years, a professional driver, admitted on the 16th January 1997 to the Emergency Room after having been found at his home in a depressed state of consciousness. He lived alone, and a neighbor, not having seen or heard him for two days,

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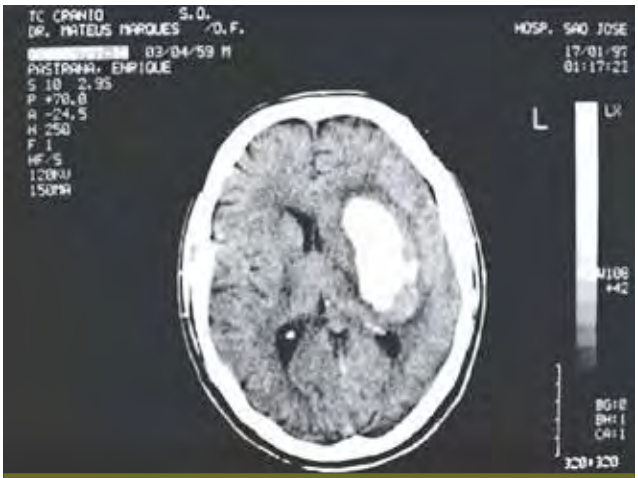
Received for publication on the 8th September 97



Computerized axial tomography scan taken upon the patient's admission.

FIG. 1

opened the door to the house, where he found the patient. The latter was a cocained user (smoked) and an alcoholic, especially in the last year, when episodes of acute intoxication were frequent. After one such incident of cocaine consumption, six months earlier, he had had to be taken to the Emergency Room, where he was placed under surveillance due to an episode of generalized convulsive crisis. There was no history of high blood pressure or use of intravenous drugs.

On admission, the patient presented paralysis of the seventh pair of cranial nerves on the right side, of the central type, global aphasia, right hypotonic hemiplegia, and painful right hemihypoesthesia. Cranioencephalic CT scan performed on the day of admission revealed "Left nucleocapsular hematoma". The test for cocaine metabolites in the urine was negative. The clinical evolution was favorable, presenting an improvement of the phasic deficit at the end of one month, maintaining difficulty of expression and using a walking aid. Although there was no history of high blood pressure, the patient underwent an echocardiogram, which did not show any abnormalities, and an examination of the fundus oculi. The summary urine examination was normal. The angioresonance did not show any vascular lesions suggestive of arteriovenous malformations, aneurisms or vasculitis. The antinuclear antibodies and anti-DNA were negative, as was the VDRL test. Complement fractions C3 and C4 were within the reference ranges.

Comments

We believe, with the supplementary diagnostic tests carried out, that the diagnosis of underlying vascular disease is unlikely. The location of the hematoma (subcortical) is suggestive of the concomitant existence of chronic high blood pressure. However, this is the site also described in cases of intracerebral hematoma associated with cocaine consumption.[10]

Cocaine has been described as associated mainly with cardiac pathologies (ischemia, left ventricular hypertrophy, arrhythmias, myocarditis, dilated cardiomyopathy),[8,9] but also with cerebral ones (convulsive crises, ischemic cerebral vascular accidents, hemorrhagic accidents, subarachnoid hemorrhage),[10,11,12] pulmonary diseases (pneumothorax, pulmonary edema)[13] and other pathologies (renal ischemia manifestations, gastrointestinal manifestations). The mechanisms by which these associations would be explained are not yet fully clarified. Cocaine has a sympathomimetic effect, as it inhibits the presynaptic recapture of catecholamines, increasing their post-synaptic availability. This effect is translated, specifically, by the increase in blood pressure, increase in cardiac frequency, vasoconstriction and stimulation of the central nervous system. The vasoconstrictor effect of cocaine, however, does not appear to be totally dependent on the sympathomimetic effect.[14] It is also believed that cocaine may have a platelet aggregation and procoagulant effect.[8,14] The issue of whether this drug plays any part in the development of cerebral vasculitis is still controversial. [15,16,17]

The toxicity of cocaine most frequently described is the temporal effect associated with its consumption. In this case, both the episode of convulsive crisis and intracranial haemorrhage were temporally associated with cocaine consumption. As stated above, the fact that the metabolites are not determinable in the urine does not rule out recent use of the drug.

The consumption of cocaine should be another factor to bear in mind when faced with a cerebral vascular accident in a young patient. ■

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