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MULTIPLE INTRACEREBRAL HAEMORRHAGE IN A YOUNG HEALTHY PATIENTS AFTER SMOKING CRACK COCAINE.

Medicine

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ABSTRACT

After smoking "crack" cocaine and consuming large quantities of ethanol, a 28-year old man developed multiple cerebral hematomas. He was hypertensive for several days, but angiography revealed no evidence of vascular malformation or vasculitis. The multifocality of the hematomas and lack of underlying disease suggest that the hemorrhages resulted from cocaine-induced acute hypertension or arterial spasm, being possibly potentiated by heavy ethanol consumption. This literature suggests that cocaine use should always be considered as a possible cause of stroke, especially in the younger age group, and in patients who lack other known vascular risk factors. Toxicology screening should, therefore, be included alongside standard investigations in this group of patients, or when a history of substance misuse is suspected.

KEYWORDS

INTRODUCTION:

It has been recognised for some time that many disorders such as vascular malformations, hypertension, bacterial endocarditis, collagen vascular diseases, tumours, eclampsia, and blood dyscrasias can cause non-traumatic intracerebral haemorrhage(ICH) in adults. However, with the epidemic in the misuse of cocaine, ecstasy and amphetamine, primarily among young people, traditional aetiological factors for haemorrhagic stroke are becoming overshadowed.

A relationship between amphetamine consumption and ICH was first reported by Gericke in 1945, and it has become increasingly recognised in recent years. Previous reports of intracerebral haemorrhage and subarachnoid haemorrhage, so far could not find any evidence of an underlying vascular abnormality on the basis of microscopic examination of the cerebral vessels. The haemorrhages were explained by a necrotising angitis related to amphetamine misuse associated with hypertension. In 1996, a necropsy study of fourteen patients with ICH related to cocaine use found no evidence of an abnormal cerebral vasculature. A more intense investigation to seek an underlying cause was thought to be unnecessary.

The growing pandemic of cocaine use in South –Asain countries is providing increasing evidence of its association with intracerebral haemorrhage. There is a relative preponderance of haemorrhagic strokes associated with methamphetamine use in young population, and methamphetamine-related stroke is associated with poor outcomes clinically. Mechanisms of methamphetamine-associated stroke include hypertension, direct vascular toxicity, vasculitis and vasospasm. In a period of rising worldwide methamphetamine use, the incidence of methamphetamine-related stroke is bound to increase, which will consequently increase the burden of disease contributed by such events.

CASE DESCRIPTION:

A 28 Year, young healthy male patient after returning back from his friends party suddenly started complaining of headache , abdominal pain and nausea .He vomited twice following which his wife saw there was stiffness and irregular movement of the patients body . He was immediately brought to the emergency of our hospital . On arrival the patient was awake but combative and disoriented. His medical history, which was obtained from relatives, was normal. Although he was taking no prescribed medications, he had a history of alcoholism and drug abuses. Examination revealed a thin man with a temperature of 98.8° F, pulse of 104/min, blood pressure of 150/90 mm Hg, and respiratory rate of 20/min. There were no signs of scalp or facial trauma such as contusions, abrasions, or lacerations. The lungs were clear, and there were no cardiac murmurs. He occasionally vocalized but did not follow commands or respond to questions and occasionally described as having "periodic whole body trembling.". There was early bilateral papilledema; the right pupil measuring 4 mm in diameter, and the left measuring 3 mm in diameter. He had a left hemiparesis with intermittent decorticate posturing of the right side and bilateral Babinski signs.

Table 1: Arterial blood gas analysis.

ACID/BASE 37.0 °C		
pH	6.904↓	
pCO_2	28.5↓	mmHg
pO_2	105.7	mmHg
HCO_3 act	5.5	mmol/L
HCO_3 std	7.9	mmol/L
BE(B)	-26.9	mmol/L
BE(ecf)	-27.3	mmol/L
$ctCO_2$	6.4	mmol/L
CO-OXIMETRY		
Hct	57	%
tHb	19.3↑	g/dL
sO_2	92.6	%
FO_2Hb	92.1↓	%
FCOHb	0.3↓	%
FMetHb	0.2	%
FHHb	7.4	%
nBili	<2	mg/dL
OXYGEN STATUS 37.0 °C		
BO_2	26.7	mL/dL
$ctO_2(a)$	25.0	mL/dL
ELECTROLYTES		
Na^+	145.8	mmol/L
K^+	3.67	mmol/L
Ca^{++}	1.09↓	mmol/L
Cl^-	110↑	mmol/L
AnGap	34.0	mmol/L
METABOLITES		
Glu	193↑	mg/dL
Lac	16.56↑	mmol/L

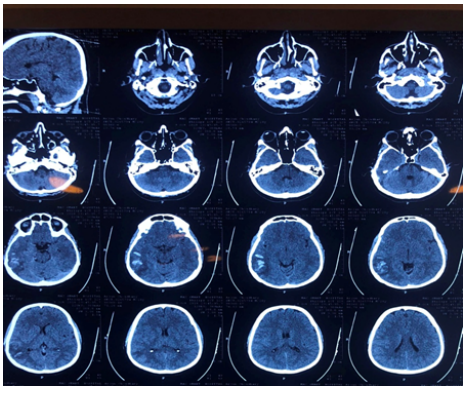

Figure 1: Computed Tomography (CT-Scan) was done which showed intracerebral haemorrhage in right temporo-occipital region and sub arachnoid haemorrhage in right parietal region.

Laboratory studies revealed the following: hemoglobin concentration 15.3 g/dl, hematocrit 45.7%, leukocyte count 8400/mm3 , platelet count 189,000/mm3 , prothrombin time 13.9 seconds (ratio 1.1), partial thromboplastin time 22.1 seconds, bleeding time 2.5 seconds, serum aspartate aminotransferase concentration 176 /ug/ml, serum alanine aminotransferase concentration 187 /ug/ml. Serum creatinine kinase – 240u/l. Normal studies included electrolytes, electrocardiography, chest x-ray, and echocardiography.

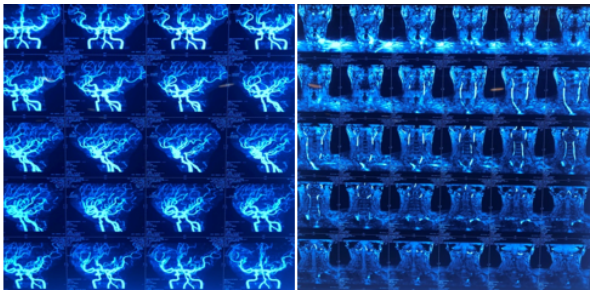

Figure 2: MRI-Angiography was done to rule out any underlying pathology which was normal

Urine Toxicology was done which was positive for Cocaine. Neurology and Neurosurgery reference was taken and treatment of the patient was started which included intravenous Levetiracetam, mannitol , dexamethasone and sodium bicarbonate. Patient was shifted to intensive care unit.

CONCLUSION:

Cocaine Toxicity can result in a variety of clinical manifestations including both ischaemic and haemorrhagic stroke. Contrary to past opinion, drug related intracerebral haemorrhage often seems to be related to an underlying vascular malformation., but potential mechanisms involved in cocaine-induced stroke include vasospasm, cerebral vasculitis, enhanced platelet aggregation, cardioembolism, and hypertensive surges associated with altered cerebral autoregulation. Cerebral computed tomography should always be performed when severe headache or altered consciousness, or both occur. Arteriography should be part of the evaluation of most young patients with non-traumatic intracerebral haemorrhage.

The growing number of case reports describing cocaine-related stroke, and the increasing evidence supporting this aetiological link, suggests that cocaine use should always be considered as a possible cause of stroke, especially in the younger age group, and in patients who lack other known vascular risk factors. A thorough medical history focusing on the use of illegal drugs and toxicological screening of urine and serum should be part of the evaluation of any young patient with a stroke.

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