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Case Report

Intracerebral hemorrhage in a middle-aged cocaine user despite normal blood pressures ${}^{\bigstar}$

Increasing evidence supports the relationship between cocaine and hemorrhagic stroke in young patients, and the type of formulation plays an important role. High blood pressure has been reported to be a common important risk factor for intracerabral hemorrhage. We report the case of a 40-year-old woman who did not declare at admission her 4-year history of intranasal cocaine hydrochloride and developed an intracerebral subcortical spontaneous hemorrhage. She documented a normal blood pressure, and computer tomography angiography did not show vascular abnormalities and excluded the presence of arterial aneurisms. We performed a urine toxicology screen test revealing that she was positive for cocaine. Various mechanisms are involved in the etiologies of intracerebral hemorrhage in cocaine users. In the presence of normal blood pressure without vascular abnormalities, an endothelial dysfunction cannot be excluded.

Therefore, we strongly suggest that a toxicological drug screening test be performed in young patients with normal blood pressure and with spontaneous subcortical hemorrhagic stroke in the presence of negative anamnesis for drug abuse.

Cocaine use seems to have a strong association with stroke, particularly in young people [1-3], and it is usually related to the development of aneurysmal subarachnoid hemorrhage [4]. The type of cocaineformulation plays an important role in the development of hemorrhagic stroke [2,3,5]. In fact, crack is associated with both ischemic strokes and hemorrhage strokes, and cocaine hydrochloride with intracerebral and subarachnoid bleeding [2]. However, high blood pressure is a common risk factor for intracerebral hemorrhage [6]. In this case, we report a young woman, a cocaine hydrochloride user, who developed intracerebral hemorrhage in the presence of normal blood pressure.

A 40-year old woman who did not declare at admission her 4-year history of intranasal cocaine hydrochloride abuse presented to our observation in emergency department for facial paralysis on the left side of her body, mild dysarthria, and hemiplegic upper limb and lower limb left, with the positivity to Babinski sign on the left. The remainder of the physical examination was unremarkable. History revealed that she had migraine with vomiting and photophobia in 2013 that resolved with sleep but excluded the presence of other systemic diseases, medical or surgical history, and alcohol abuse. Clinical evaluation documented a normal blood pressure (115/70 mm Hg; heart rate, 85 beats/min).

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Noncontrast computer tomographic (CT) scan revealed the presence of a supratentorial, small intracerebral (intraparenchymal) hemorrhage originated within the right basal ganglia (FigureA). Computer tomographic scan repeated in 2 and 6 days was unchanged with respect to the admission. A CT angiography at the time of admission and repeated 2 days later did not show vascular abnormalities and excluded the presence of arterial aneurisms (FigureB). Electrocardiography excluded the presence of heart diseases and biochemical and hematologic tests were in normal range. We performed a urine toxicology screen test revealing that she was positive for cocaine and lorazepam used (at the final dosage of 1 mg/d) to treat anxiety symptoms. No other drugs were taken by the patient at the time of this study. Clinical conditions improved in 24 hours and clinical evaluation confirmed hemiplegic upper limb and lower limb, left side, with the positivity to Babinski sign on the left and regression of facial paralysis on the left and mild dysarthria. During hospitalization, she has been subjected to pressure monitoring in a stroke unit, and mean blood pressure values were 119/76 mm Hg (heart rate, 83 beats/min). Six days later, she was transferred to the rehabilitation unit with hemiplegic manifestation on the left side of the body.

We report the development of hemorrhagic stroke in a not-declared cocaine user. Several mechanisms are involved in the etiologies of intracerebral hemorrhage in cocaine users including rapid increases in blood pressure, aneurysmal rupture, and vasculitis [2]. Drug abuse can also represent a risk factor of intracerebral hemorrhage in patients with underlying vascular malformation [2]. Several authors suggested that spikes in blood pressure represent an important etiological factor in the development of intraparenchymal hemorrhage [7,8]. Martin-Schild et al [9] studying patients with intracerebral hemorrhage documented that cocaine users have higher blood pressure with respect to cocaine nonusers, and it may be related to the effects of cocaine on reuptake of catecholamines at presynaptic nerve endings [2,10]. A possible mechanism may be also that cocaine use causes spikes in blood pressure in patients with preexisting hypertensive cerebral vasculopathy in the subcortical areas leading to intracerebral hemorrhage [8,9]. In our patient, history excluded the presence of blood hypertension, whereas CT scan revealed a subcortical location of hemorrhagic lesions without vascular abnormality. We cannot exclude that intracerebral hemorrhage was related to endothelial dysfunction.

In conclusion, we strongly suggest that a toxicological drug screening test be performed in young patients with normal blood pressure and with spontaneous subcortical hemorrhagic stroke in the presence of negative anamnesis for drug abuse at admission. The mechanisms of cocaine-induced intracerebral hemorrhage in presence of normal blood pressure also require further study.

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ARTICLE IN PRESS

A. Siniscalchi et al. / American Journal of Emergency Medicine xxx (2016) xxx-xxx

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Figure. A, CT scan showing a small right subcortical (right basal ganglia) intracerebral hemorrhage in a cocaine user. B, CT angiography image of the circle of Willis.

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