



Bilateral haemorrhagic infarction of the globus pallidus after cocaine and alcohol intoxication

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Abstract

Cocaine is a risk factor for both ischemic and haemorrhagic stroke. We present the case of a 31-year-old man with bilateral ischemia of the globus pallidus after excessive alcohol and intranasal cocaine use. Drug-related globus pallidus infarctions are most often associated with heroin. Bilateral basal ganglia infarcts after the use of cocaine, without concurrent heroin use, have never been reported. In our patient, transient cardiac arrhythmia or respiratory dysfunction related to cocaine and/or ethanol use were the most likely causes of cerebral hypoperfusion.

Key words: Cocaine; stroke; infarction; globus pallidus.

Introduction

Cocaine is a risk factor for both ischemic and haemorrhagic stroke. We present a 31-year-old man with bilateral ischemia of the globus pallidus after excessive alcohol and intranasal cocaine use.

Case report

A 31-year-old man without medical history or treatment was found unresponsive six hours after excessive alcohol and intranasal cocaine use. Blood pressure was 13/8.5 mmHg. Glasgow Coma Scale was 8 (in absence of focal neurological deficit), requiring intubation and mechanical ventilation. Electrocardiogram was normal. Urine toxicological screening was strongly positive for cocaine, and negative for other drugs (heroin, amphetamines, cannabis, and tricyclic antidepressant drugs). Blood ethanol level was 0.96 mg/ml, cardiac enzymes and blood carboxyhaemoglobin levels were normal, as were blood count, CRP, sedimentation rate, renal and liver function tests, ANF, lupus anticoagulant,

anticardiolipin antibodies, ANCA, and serology of HIV, syphilis, hepatitis B and C. MRI showed acute bilateral ischemia of the globus pallidus and the vascular watershed zones (Figure). Lumbar puncture, transesophageal echocardiography, and intra- and extracranial computed tomography angiography were normal. Consciousness improved progressively and made place for abulia and akinesia. The patient was extubated 4 days later. The following months, further clinical improvement was seen although mental slowing, executive dysfunction, hypophonia, and verbal fluency deficit persisted.

Discussion

Cocaine is a risk factor for both ischaemic and haemorrhagic stroke (Lange and Hillis, 2001; Treadwell and Robinson 2007). Cocaine is most often associated with haemorrhagic stroke (with possible etiological mechanisms including acute hypertension, failure of cerebrovascular autoregulation, underlying aneurysm or arteriovenous malformation, coagulopathy, vasculitis, and early haemorrhagic transformation of infarction). Potential mechanisms involved in cocaine induced ischemic stroke include vasospasm, vasculitis, enhanced platelet aggregation, cardiac arrest, cardioembolism, hypertensive surges associated with altered cerebral autoregulation and cerebral blood flow, and cocaine-related excitotoxicity. The earlier reported patients with cocaine-related ischaemic stroke involved the following arteries: anterior cerebral artery, middle cerebral artery, posterior cerebral artery, vertebrobasilar arteries, spinal cord arteries, and retinal arteries (Devenyi *et al.*, 1988; Mody *et al.*, 1988; Levine *et al.*, 1990; Sawaya and Kaminski, 1990; Daras *et al.*, 1991; Brown *et al.*, 1992; Daras *et al.*, 1994; Nanda *et al.*, 2006). Drug-related globus pallidus and

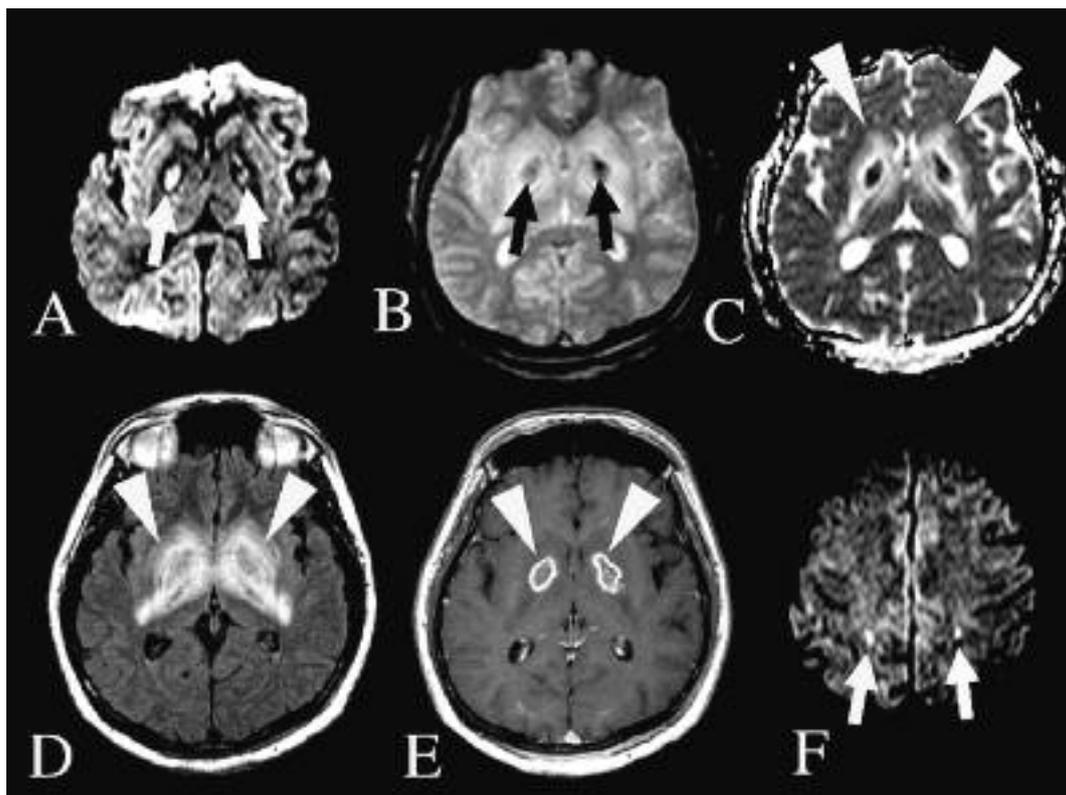


FIG. 1. — Three days after symptom onset, acute bipallidal infarction was seen (hyperintense on DWI imaging, A; hypointense on ADC sequences, C), partly haemorrhagic on echo-gradient T2-weighted imaging (asymmetrical decreased signal, B) and surrounded by vasogenic oedema (ADC imaging, C; FLAIR sequences, D). Pallidal enhancement was seen after gadolinium-enhanced T1 sequences (E). Borderzone infarction (between anterior and medial cerebral artery territory) was seen on DWI (F).

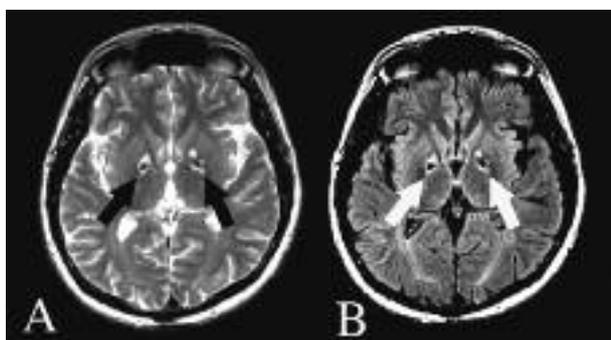


FIG. 2. — Six months later, vasogenic oedema disappeared and signs of haemorrhagic necrosis persisted in the globus pallidus (A, T2-weighted imaging; B, FLAIR sequences).

borderzone infarctions (similar to radiological abnormalities seen in carbon monoxide poisoning) are most often associated with heroin, probably due to opioid-related respiratory depression (Vila and

Chamorro, 1997; Andersen and Skullerud, 1999; Daras *et al.*, 2001).

Bilateral basal ganglia infarcts after the use of cocaine (without concurrent heroin use) have never been reported. One case of bilateral hippocampal stroke secondary to cocaine-associated cardiac arrest has been described (Bolouri and Small, 2004).

The risk of acute myocardial infarction related to cocaine use is clearly increased, especially during the 60 minutes after drug administration, probably unrelated to the amount ingested, the route of administration, and the frequency of use (Mittleman *et al.*, 1999). In addition, several types of cocaine-associated cardiac dysrhythmias and conduction disturbances have been reported (Levine *et al.*, 1990). Since no signs of myocardial ischemia were present in our patient, transient cardiac arrhythmia or respiratory dysfunction, directly or indirectly (e.g. associated with a generalized seizure) related to cocaine and/or ethanol use was to be the most probable cause of cerebral hypoperfusion.

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