

BILATERAL LESIONS OF THE GLOBUS PALLIDUS IN A YOUNG WOMAN

R. Kadi, A. Romy, T. Stadnik, M. Cannie, C. Mabilia, L. Divano¹

We report a case of stroke due to cocaine abuse in a 30-year-old woman. The initial examinations pointing to this diagnosis were CT and MRI. Magnetic Resonance Imaging revealed bilateral globus pallidus infarction characterized by restricted Diffusion with low ADC values. There was also a partial hemorrhagic component confirmed on T2* sequence. After gadolinium injection there was a ring enhancement consistent with a blood brain barrier leakage. Similar lesions have been described in post hypoxic-anoxic injuries such as following cardiac arrest, severe blood loss and CO intoxication.

Key-words: Cocaine – Drugs, abuse.

Cocaine is a risk factor for both ischemic and haemorrhagic stroke. We present a 30-year-old woman with bilateral ischemia of the globus pallidus after excessive intranasal cocaine abuse.

Case report

A 30-year-old woman patient with no particular medical history was found unconscious. She had a cardio-respiratory arrest and was resuscitated by invasive intubation and ventilation.

Her toxicological blood report showed high levels of cocaine, positive for benzodiazepines, amphetamines, and methadone but was negative for heroin and cannabis. The toxicological analysis also showed a high concentration of ethanol and carboxy-hemoglobin.

Computed-tomography (CT) of the brain showed a dedifferentiation of the globi pallidi (infarction) with ring enhancement after contrast injection (rupture of the blood brain barrier) surrounded by low density area (edema) (Fig. 1).

Magnetic resonance imaging (MRI) of the brain, performed 1 day later, demonstrated bilateral globus pallidus infarction characterized by restriction on Diffusion Weighted Imaging with low apparent diffusion coefficient (ADC) value suggesting ischemic brain injury (Fig. 2). There was also a partial hemorrhagic component confirmed by heterogeneous low intensity deposits on a T2* sequence. After gadolinium injection there was a ring enhancement due to

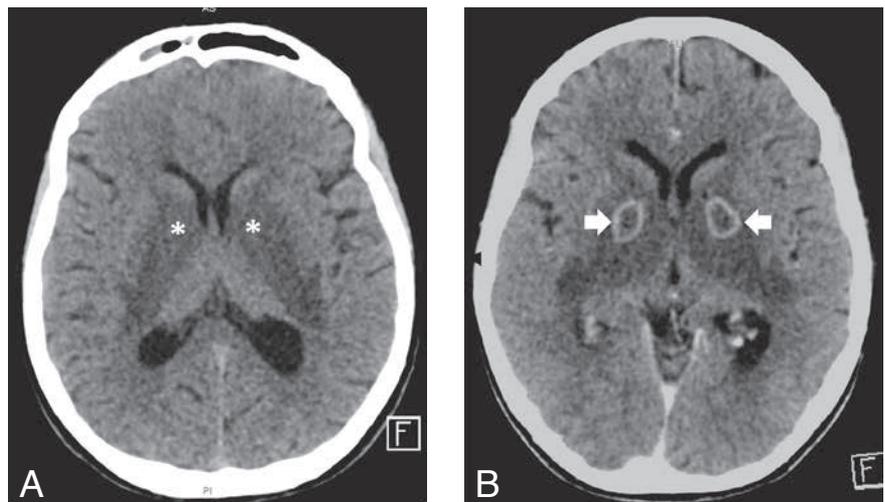


Fig. 1. — Computed tomography of the brain pre (A) and post (B) IV contrast: dedifferentiation (asterisk) of the globus pallidus (infarction) surrounded by hypoattenuated area of edema (A). Ring like enhancement (arrow) of the globus pallidus representing a rupture of the blood brain barrier (B).

blood brain barrier leakage. MRI also showed a vasogenic edema surrounding these lesions as well as few ischemic lesions in the subcortical white matter (Fig. 2). The presence of signs of acute sinusitis in the right fronto-ethmoido-maxillary sinuses along with thickening of the nasal fossa on the same side are typical for the patients with chronic cocaine abuse (Fig. 3).

The patient was extubated after 5 days and gradually recovered her consciousness, however the patient remained with a psychomotor impairment characterized by apathy and hypothyroidism.

Discussion

Cocaine and its metabolites including cocaethylene, amongst all, is a powerful vasoconstrictor which also affects the cerebral vascularization (1, 2). Cocaine abuse results in a high rate of ischemic or hemorrhagic strokes of the central nervous system (CNS) classically in men in their third decades (1, 3). All the vascular territory can potentially be affected (1). The risk of rupturing of a pre-existing aneurysm is equally elevated (1).

The high intensity lesions on diffusion and T2 imaging have been observed in acute stage of cocaine abuse typically in the hippocampal area and in the globus pallidus (1, 4). Similar lesions have been noted in the deep white matter in cases of chronic abuse of cocaine but rarely after an acute intoxication (1, 5).

These anomalies have been interpreted previously as secondary demyelination due to intense vasoconstriction. The chronic exposure to

From: 1. Department of Radiology of the University Hospital Brugmann, Brussels, Belgium.

Address for correspondence: Dr R. Kadi, M.D., Dpt of Medical Imaging, CHU-UVC Brugmann, Place Van Gehuchten 4, 1020 Brussels, Belgium.
E-mail: Redouane.kadi@gmail.com

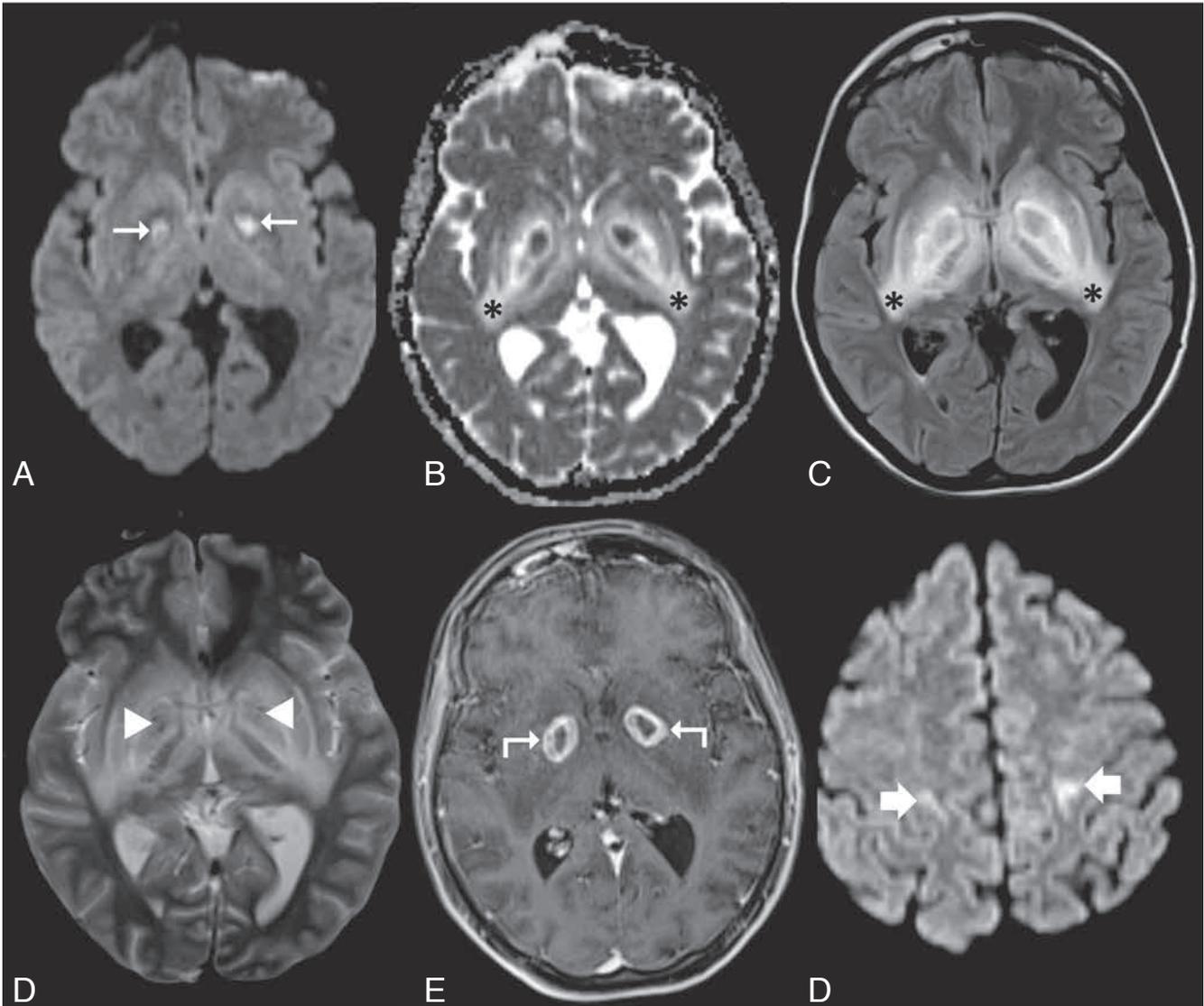


Fig. 2. — Brain MRI (pre and post gadolinium injection) performed 17 days after onset of symptoms: Acute bipallidal infarction (arrow) showing a hyperintense on DWI imaging (A) and hypointense on ADC sequences (B). Surrounded by vasogenic edema (asterisk) hyperintense ADC imaging (B) and FLAIR sequences (C). The hemorrhagic component (arrowhead) can be recognized by its heterogenous decreased signal on echo-gradient T2*-weighted imaging (D). Ring bipallidal enhancement (curved arrows) was seen after gadolinium-enhanced T1 sequences representing the blood brain barrier leakage (E). Few ischemic lesions in the subcortical white matter (thick arrow) were seen on DWI (F).

cocaine leads to a reduction of volume not only in the grey matter and in the cerebellar hemispheres but also in the frontal and temporal cortex and the thalamus (1, 6).

The cocaine is responsible for the infarction of the globi pallidi and also of the borderzone, showing similar radiological lesions as compared to those seen during an acute intoxication by carbon monoxide: restricted

Diffusion weighted imaging with low ADC value and hyperintense lesion on FLAIR suggesting ischemic brain injury. These lesions are most commonly associated with heroine abuse, probably due to the suppression of the respiratory center by the opium derivatives (7, 8).

The isolated infarction of the globus pallidus and of the hippocampus (3) has been also reported

without any association with heroine abuse.

The risk of myocardial infarction due to acute consumption of cocaine is also very high (9). Moreover cardiac rhythm disorders due to conduction disturbances have also been reported (10).

Therefore, the ischemic lesions in the globi pallidi in our patient can be explained by a direct vasoconstriction

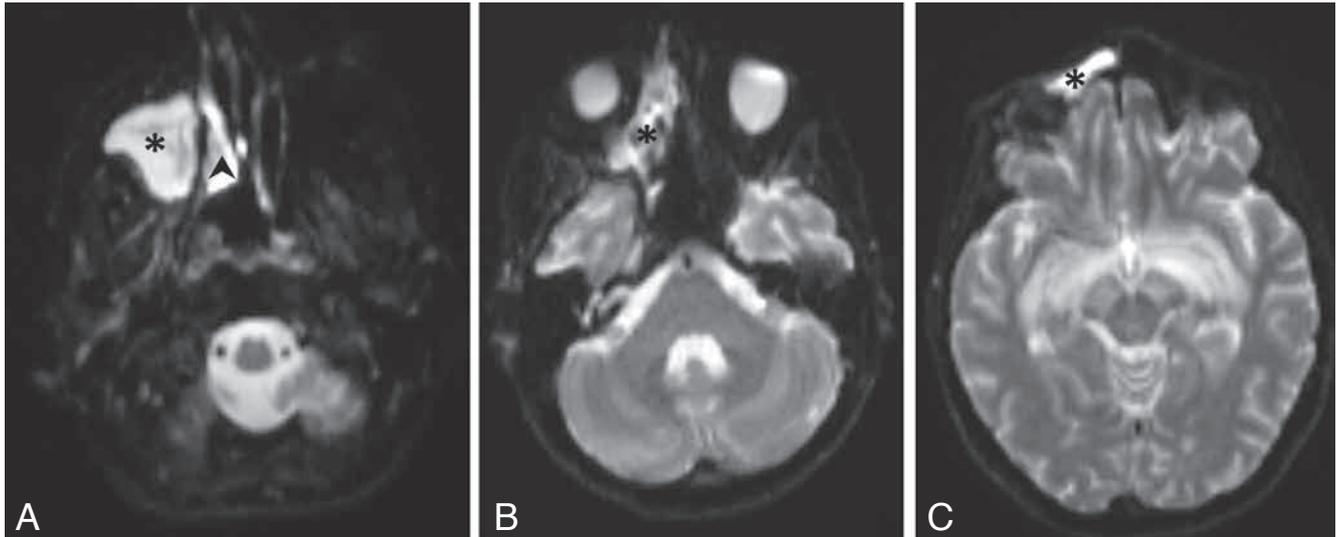


Fig. 3. — Signs of acute sinusitis (asterisk) of the frontal, ethmoidal and maxillary sinus on the right side with thickening of the nasal fossa (arrowhead) noted at the diffusion imaging at B0 secondary to cocaine inhalation (A, B, C).

effect of cocaine and also by subsequent to the cardiac arrest.

Conclusion

The ischemic lesions affecting the globus pallidus is nonspecific and can be seen not only in post hypoxic-anoxic injury (cardiac arrest, severe blood loss and CO poisoning) but also in cocaine abuse due to strong vasoconstriction leading to a high incidence of ischemic stroke.

Drug related globus pallidus infarctions are most often associated with heroin. Bilateral basal ganglia infarcts after cocaine abuse, without concurrent heroin use, are rarely reported in the literature.

References

1. Hantsona P., Duprez T.: Imagerie cérébrale et intoxications. *Réanimation*, 2009, 18: 598-605.
2. Lange R.A., Hillis L.D.: Cardiovascular complications of cocaine use. *N Engl J Med*, 2001, 345: 351-358.
3. Treadwell S.D., Robinson T.G.: Cocaine use and stroke. *Postgrad Med J*, 2007, 83: 389-394.
4. Boulouri M.R., Small G.A.: Neuroimaging of hypoxia and cocaine-induced hippocampal stroke. *J Neuroimaging*, 2004, 14: 290-291.
5. De Roock S., Hantson P., Laterre P.F., Duprez T.: Extensive pallidal and white matter injury following cocaine overdose. *Intensive Care Med*, 2007, 33: 2030-2031.
6. Sim M.E., Lyoo I.K., Streeter C.C., Covell J., Sarid-Segal O., Ciraulo D.A., et al.: Cerebellar gray matter volume correlates with duration of cocaine use in cocaine-dependent subjects. *Neuropsychopharmacology*, 2007, 10: 2229-2237.
7. Vila N., Chamorro A.: Ballistic movements due to ischemic infarcts after intravenous heroin overdose: report of two cases. *Clin Neurol Neurosurg*, 1997, 99: 259-262.
8. Andersen S.N., Skullerud K.: Hypoxic/ischaemic brain damage, especially pallidal lesions, in heroin addicts. *Forensic Sci Int*, 1999, 102: 51-59.
9. Mittleman M.A., Mintzer D., Maclure M., Tofler G.H., Sherwood J.B., et al.: Triggering of myocardial infarction by cocaine. *Circulation*, 1999, 99: 2737-2741.
10. Levine S.R., Brust J.C., Futrell N., Ho K.L., Blake D., et al.: Cerebrovascular complications of the use of the "crack" form of alkaloidal cocaine. *N Engl J Med*, 1990, 323: 699-704.