

LETTERS TO THE EDITOR

Cocaine abuse simulating the aura of migraine

Cocaine abuse is a growing problem in the United Kingdom but the association of cocaine use with neurological problems is less well recognised than the social and economic consequences. We report a patient with a past history of migraine who developed migraine-like symptoms with aura 24-36 hours after use of intranasal cocaine. A subsequent CT scan showed cerebral infarction and haemorrhage.

A 46 year old right handed professional man had used intranasal cocaine at regular intervals for some years. He had last used LSD 20 years previously and currently smoked marijuana and up to 40 proprietary cigarettes a day. He had no previous neurological symptoms with cocaine. There was a history from childhood of frontal, sometimes severe but infrequent, migraine headaches (once every two years) with vomiting, photophobia and hyperacusis, without any aura. The headaches usually resolved with sleep. One week before admission to hospital he developed a kaleidoscopic effect of flashing lights in the peripheral vision of his left visual field. This effect drifted towards the centre of vision and drifted out again over a period of 30 minutes. There was a slight, dull left frontal headache for one to two hours without nausea. The following morning, when he attempted to speak to a friend he "couldn't say what my brain was thinking". He made noises he did not recognise but had no difficulty uttering expletives. His speech became normal within 24 hours including the paraphrastic errors and mixing up of words. Two weeks previously he had had a similar episode lasting only 45 seconds. On both occasions he had smoked cocaine within the preceding 24-36 hours, using similar amounts and achieving a comparable effect with that of previous use.

On examination, within a few hours of his speech disturbance, he was dysnomic but with an otherwise normal neurological examination. When referred at 48 hours there was no speech disturbance with normal reading and similarly normal neurological and cardiac examination without carotid bruits. His blood pressure was 140/80 mm Hg.

A CT scan showed a small area of haemorrhage in the right frontal region and a small area of low attenuation in the left temporal region, consistent with infarction (figure). An MRI performed two weeks later showed persistent signal change in the area of infarction only. Four vessel cerebral angiography, performed at two weeks, was normal without any vasospasm. Extensive screening for vascular disease risk factors was negative with a normal ECG.

In the absence of risk factors for vascular disease, apart from cigarette smoking, this man's two dysphasic episodes were attributed to the use of cocaine within the preceding 24-36 hours. Cocaine related cerebrovascular events are well described,¹ occurring at the time of administration or within a similar

time interval as our patient. There is no good explanation why the manifestation of cocaine induced vasospasm should be delayed. Decreased relative cerebral blood flow, presumed to reflect the effect of vasospasm, has been found in chronic users of cocaine up to ten days post withdrawal.² The combination of a history of migraine in our patient and the delay in the development of vascular events after the use of cocaine is of particular interest as patients experiencing migraine with aura have impaired carbon dioxide reactivity after headache.³ In this case, the initial symptoms may have been a migrainous aura. The combination of reduced cerebral blood flow in migraine with impaired hypercapnic vasodilation, with the addition of cocaine, may have caused a cumulative effect sufficient to result in cerebral infarction.

Alternatively, his recent symptoms may have all been due to cocaine, as migraine-like symptoms in cocaine users who are admitted to hospital are no more common than in the general population. Patients with previous migraine, with or without aura, may be at an increased risk for a migraine attack with cocaine.⁴ Another patient who experienced typical migraine headaches, waking him at 4am, were more regular following recreational use of intranasal cocaine during a preceding evening. The relationship was sufficiently apparent for both patients to give up the drug with symptomatic relief. Patients with migraine should be advised that cocaine is potentially dangerous.

Patients without a previous history of migraine may develop migraine-like symptoms with cocaine, with or without aura.^{4,5} Migraine without aura is probably not associated with vasospasm. In this instance, the migraine induced by cocaine may have a different mechanism, including inhibition of re-uptake of serotonin, an effect mediated by chronic use of cocaine and of possible relevance to a lowered pain threshold in migraine.⁵

Cocaine-related cerebrovascular disease is not as well recognised in the United Kingdom as it is on the other side of the Atlantic. It was only after a casual remark by a friend of the patient that the relationship of his symptoms to cocaine was noted. The importance of inquiring about drug abuse in all non-elderly patients with cerebrovascular symptoms, headaches and migraines, underlines a realisation of the association of cocaine with neurological symptoms and prompted our patient and similar patients to stop using this drug.^{4,5}

S S MOSSMAN
P J GOADSBY
National Hospital for Neurology
and Neurosurgery, Queen Square,
London, UK

Correspondence to: Dr Mossman, Wellington Hospital, Private Bag, Wellington South, New Zealand.

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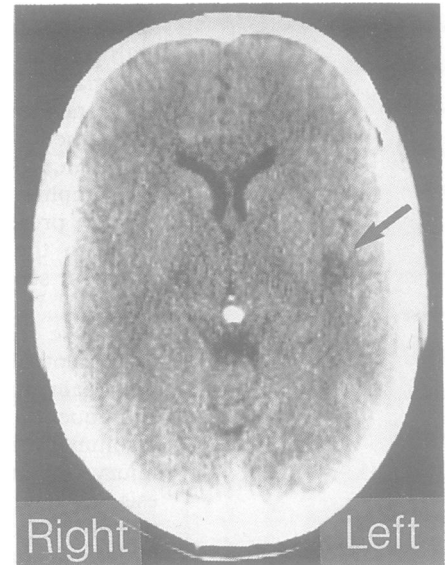


Figure CT scan showing a small left temporal lobe infarction. An arrow delineates the lesion.

Pathological laughter and brain stem glioma

An 18 year old girl presented with a two months history of progressive gait ataxia, left hemiparesis and uncontrollable laughter. On examination, the patient was well oriented and the optic fundi and extraocular movements were normal. The corneal reflexes were absent bilaterally, there was wasting of the right masseter and a left lower motor neuron facial palsy and bilaterally absent gag reflexes were present. She had a grade 4 hemiparesis on the left with bilaterally exaggerated deep tendon reflexes (left more than right) and left-sided incoordination of limbs. The most peculiar feature noted, however, was that the patient broke into irrepressible bouts of laughter on any attempted movement of the facial muscles.

CT of the brain showed an irregular isodense contrast-enhancing lesion in the ponto-medullary region causing widening of the brain stem and effacement of the brain stem cisterns. There was no hydrocephalus. A CT-guided stereotactic biopsy of the lesion was reported as astrocytoma Grade II. The patient was treated with conventional external beam radiation with 55 Gys and started on chemotherapy with procarbazine, CCNU and vincristine. At follow up after eight months, the pathological laughter had reduced, and she was able for the first time to communicate verbally with the examiner. A repeat CT scan of the brain still showed the isodense contrast enhancing lesion in the ponto-medullary region.

Laughter is termed pathological when it is continuous and inappropriate. It is not a disorder of emotions, but due to a dysfunction of the motor components of affective expression. It is commonly seen in conditions such as pseudobulbar palsy, motor neuron disease involving the facial musculature, as part of an epileptic seizure, or rarely in the syndrome called "Fou rire prodromique".¹

In a necropsy study of 30 cases, Poeck and Pilleri found that the lesion commonly involved the genu of the internal capsule and adjacent basal ganglia. The lesions occurred above the structures of the middle and lower