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 paraphrasic 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]

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1 Levine SR, Brust JC, Futrell N, Ho KL, *et al.* Cerebrovascular complications of the use of the "crack" form of alkaloid cocaine. *NEJM* 1990;323:699–704.
2 Volkow ND, Mullani N, Gould KL, Adler S, Krajewski R. Cerebral blood flow in chronic cocaine users: a study with positron emission tomography. *Brit J Psych* 1988;152:641–8.
3 Lauritzen M, Lassen NA, Pawlson OB. Regulation of regional cerebral blood flow during and between migraine attacks. *Ann Neurol* 1983;14:569–72.
4 Liptor RB, Choy-Kwong M, Solomon S. Headaches in hospitalised cocaine users. *Headache* 1989;29:225–8.
5 Satel SL, Gawin FH. Migraine like headache and cocaine use. *JAMA* 1989;261:2995–6.

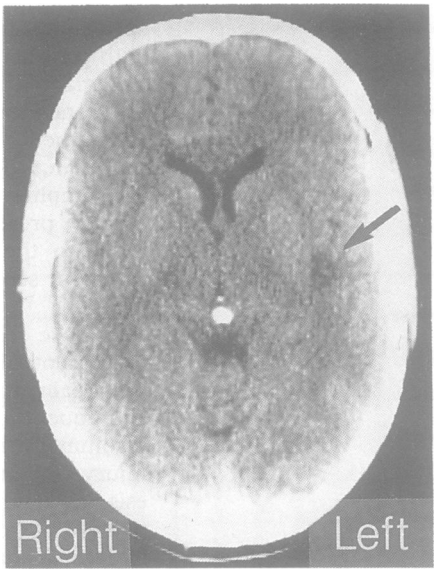

Figure CT scan showing a small left temporal lobe infarction. An arrow delineates the lesion.

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

brain stem. In our case the lesion was involving the pons and medulla.

Pathological laughter may be a rare and unusual presentation of a brain stem lesion (glioma) that has not been reported earlier.

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1 Poeck K. Pathological laughter and crying. In: Vinken PJ and Bruyn GW, eds. *Handbook of clinical neurology*, Vol 45. Amsterdam: North Holland, 1985;219–26.

Post radiation monomelic amyotrophy

Lamy *et al* recently reported three cases of post radiation lower motor neuron syndrome presenting as monomelic amyotrophy.[1] I would like to report a further case.

A thirty six year old man presented in 1989 with weakness abducting his left hip. Nineteen years before he had been treated for a left testicular seminoma with an orchidectomy and radiotherapy. The abdominal radiotherapy field dispensed a prescribed tumour dose of 32·5 Gy, and the para-aortic fields increased the dose to 50 Gy in twenty fractions. The fields included the lower 6 cm of the spinal cord, the whole cauda equina and the lumbrosacral roots and plexuses. The initial weakness of the left leg has gradually progressed to weakness and wasting of all muscle groups with ankle dorsiflexion, hip flexion and abduction being more severely affected. He now walks with a stick. The limb is areflexic with a flexor plantar response. There is no sensory loss or sphincter involvement. The right leg is not involved. Six months of treatment with prednisolone (20 mg per day) was of no benefit. Electrophysiological examination showed advanced denervation confined to the muscles of the left leg, with large amplitude motor unit potentials. An abnormal axon reflex and absent F wave in extensor digitorum brevis suggests a lesion at the root or anterior horn cell level. Motor and sensory conduction velocities and distal latencies were normal. General examination, laboratory investigations including CSF analysis and a myelogram were all normal.

The 17 year latency in this case, and the nine and twelve year latencies reported by Lamy *et al*,[1] as well as the previously mentioned electrophysiological data point to an anterior horn cell disorder reminiscent of the postpolio syndrome.[2] It is plausible that the radiotherapy damaged a critical number of motor neurons and that surviving neurons sprout to reinnervate more muscle fibres than normal. This process produces large motor units that may stress the cell body. After a number of years these hyperfunctioning motor units may not be able to maintain the metabolic demands of all their sprouts and a deterioration of individual terminals may result. Eventually enough nerve terminals are destroyed and enough reserves are diminished for weakness to appear. This would be consistent with the focal nature of post radiation lower motor neuron syndrome and its slow, stepwise and unpredictable progression.

As Lamy *et al* state, the cause of this disorder seems unpredictable. My patient continues to deteriorate. Maier *et al*[3] report that three of their 15 patients had a monomelic amyotrophy, but give no information on clinical course. Vibeke Schiodt and Kristenson[4] report that two of their patients had monomelic amyotrophy—one had "slight subjective weakness of the left leg" which had improved by six months and the other case had non-progressive right leg weakness.

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1 Lamy C, Mas JL, Zeigler M, De Recondo J. Postradiation lower motor neuron syndrome presenting as monomelic amyotrophy. *J Neurol Neurosurg Psychiatry* 1991;**54**:649–9.
2 Munsak TL, ed. *Post polio syndrome*. Boston: Butterworth-Heineman, 1991.
3 Maier JG, Perry RH, Taylor N, Sielak MH. Radiation myelitis of the dorsolumbar spinal cord. *Radiology* 1969;**93**:153–60.
4 Vibeke Schiodt A, Kristenson O. Neurological complications after irradiation of malignant tumours of the testis. *Acta Radiologica Oncology* 1978;**17**:369–78.

Sellar tuberculoma

In Asian countries, before the advent of chemotherapy, tuberculomas accounted for about 30% of all intracranial mass lesions.[1] They remain a major problem even though they are now less common due to antituberculosis drugs and improved living conditions. Intrasellar tuberculomas, not uncommonly, seen at post mortem examinations, rarely present clinically.[2–4] Only five surgically verified cases have been reported to date.

A 40 year old man, resident of an area where tuberculosis is endemic, presented in January 1988 complaining of an intermittent, dull, generalised headache of two years duration, and progressive diminution of vision in both eyes over a period of six months. He was in good general health with no clinical signs of endocrinopathy. Visual acuity was reduced (right eye—finger counting 3 m; left eye—hand movement perception at one meter). Perimetry showed constricted field of vision in the right eye, the left was unascertainable. He had bilateral optic atrophy, but no other neurological abnormality. The clinical diagnosis was a sellar tumour. Haematological and biochemical investigations were unremarkable except for an erythrocyte sedimentation rate (ESR) of 90 mm in the first hour. Radiographic examination revealed a normal chest and an enlarged sellar on *x* ray of the skull. CT scan (figure) showed a uniformly hyperdense enhancing sellar mass with a suprasellar extension.

An operation was performed on 14 January 1988 using a transnasal transsphenoidal route. The sellar floor and dura mater were intact. The dura mater was tough and thickened and when it was opened, a greyish white, tough nonsuckable tumour was revealed. It was adequately decompressed under intraoperative pneumoencephalography.

Microscopic examination showed that pituitary tissue had been partially replaced by granulomas comprising of epitheloid cells, Langhan type of giant cells surrounded by lymphocytes, and plasma cells. Minimal caseation was present in some granulomas. Tuberculoma of the pituitary gland was diagnosed. A postoperative Mantoux test was positive but sputum culture for acid fast bacilli was negative. Treatment was started with isonex, rifampicin and ethambutol. The patient developed hepatotoxicity to rifampicin, but isonex and ethambutol were continued for nine months. He was in good health, had no headache, and vision in the left eye had improved (finger counting—3 m). The right eye vision showed no improvement in the constricted field (finger counting—6 m).

A pituitary tuberculoma is extremely rare but usually presents as a chiasmal syndrome. In two reported cases the lesion was successfully removed subfrontally.[2 3] An exclusively intrasellar tuberculoma[5] was approached transsphenoidally and treated with isonex only for three months. In our patient the lesion was intrasellar with a suprasellar extension. It could be treated, however, by a transnasal transsphenoidal approach. There was little reason to suspect a pituitary tuberculoma before the operation except for the raised ESR. The transnasal transsphenoidal approach allowed a subtotal removal of the tuberculoma while avoiding CSF contamination by tuberculous material.

Tuberculous meningitis occurs in the majority of surgically treated intracranial tuberculomas without antituberculous chemotherapy.[1–3] Chemotherapy should be given in a three drug combination for three months, followed by a two drug combination for a further 15 months.

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1 Dastur HM. Tuberculoma. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*, Vol 18. New York: American Elsevier, 1975:413–26.
2 Brooks HB, Dumalo JS, Bronsky D, Waldstein SS. Hypophysial tuberculoma with hypopituitarism. *Am J Med* 1973;**54**:777–81.
3 Coleman CC, Meredith JM. Diffuse tuberculosis of the pituitary gland simulating tumor with postoperative recovery. *Arch Neurol Psychiatry* 1940;**44**:1076–85.
4 Eckland DJA, O'Neill JH, Lightman SL. A pituitary tuberculoma. *J Neurol Neurosurg Psychiatry* 1987;**50**:360–1.
5 Esposito V, Fraioli B, Ferrante L, Palma L. Intrasellar tuberculoma. Case report. *Neurosurgery* 1987;**21**:721–3.

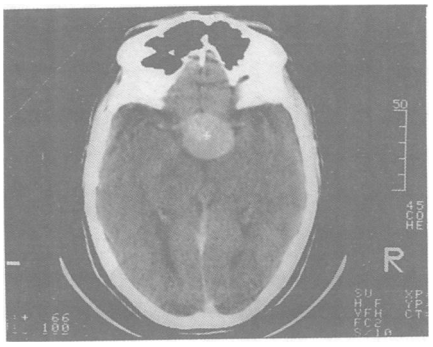

Figure CT scan showing sellar and suprasellar hyperdense lesion.