

The clinical presentation of bitemporal field loss with a third nerve palsy was considered typical of a pituitary adenoma and this diagnosis was compatible with the CT scan appearance. Involvement of the third nerve is common in large pituitary tumours due to either compression against the interclinoid ligament or invasion of the cavernous sinus.¹ The pathological finding was of an ependymoma involving the pituitary fossa. Although we cannot exclude the possibility that the tumour arose in the third ventricle and extended downwards into the sella, an intrasellar origin of an ependymoma may be possible.

Ependymomas are primary glial tumours presumed to arise from a cell related to the ependymal lining.² Although these tumours are usually related to the ventricular system, a connection with the ventricular ependyma could only be identified in six out of 14 cases in one series³ and primary ependymomas have been described in extra-axial soft tissue locations.^{4,5} The presence of either embryological remnants of the ependymal cleft within the sella or heterotopic ependymal lining cells may explain the unusual location of the ependymoma in our case. In a 10 week (45 mm) human fetus the neurohypophysis develops as an elongated out-pouching of neuroepithelial cells which encloses a cavity that is continuous with the cavity of the neural tube.⁶ The infundibulum at this stage consists of undifferentiated ependymal cell precursors. The ependymal cleft is readily identified at 13 weeks (60 mm) but recedes by 16 weeks (112 mm) and is not present at birth. Isolated ependymal cells could be left behind in the infundibulum and neurohypophysis, as rests or heterotopias and these may undergo subsequent neoplastic transformation.

We are not aware of any other similar examples of this very unusual presentation of a glial tumour.

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References

- 1 Jefferson G. The invasive adenomas of the anterior pituitary. *The Sherrington lectures III*. Liverpool: University Press, 1955.
- 2 Russell DS, Rubenstein LJ. *Pathology of tumours of the nervous system* 4th ed. London: Edward Arnold, 1977:204-5.
- 3 Mork SJ, Loken AC. Ependymoma: a follow up study of 101 cases. *Cancer* 1977;40:907-15.
- 4 Anderson MS. Myxopapillary ependymomas presenting in the soft tissue over the sacrococcygeal region. *Cancer* 1966;19:585-90.
- 5 Morantz RA, Kepes JJ, Batnitzky S, Masterson BJ. Extrapapillary ependymomas: report of three cases. *J Neurosurg* 1979;51:383-91.
- 6 Sloper JC. The hypothalamo-neurohypophysial system. In: Haymaker W, Adams RD, eds. *Histology and histopathology of the nervous system*. Springfield, Illinois: CC Thomas, 1982:2027-167.

Paroxysmal dysarthria and ataxia: associated MRI abnormality

Sir: Paroxysmal dysarthria and ataxia has been reported in multiple sclerosis,¹ and such episodes may be the presenting feature of this disease.² In common with a variety of different paroxysmal symptoms seen in multiple sclerosis, the episodes are characteristically sudden in onset, brief in duration, stereotyped for an individual patient and tend to remit over a period of time. We report a case of paroxysmal dysarthria and ataxia and the associated finding on magnetic resonance imaging (MRI).

A 31 year old physiotherapist presented with a two week history of episodes of incoordination of the left arm and left leg with simultaneous dysarthria. Each episode occurred without warning and lasted up to six seconds. The paroxysms occurred with increasing frequency (up to 20 episodes per day) until carbamazepine therapy was initiated six weeks after the onset of symptoms. General and neurological examination between attacks was completely normal. During an episode, ataxia of the limbs and dysarthria were observed, although full examination was not possible due to the brevity of the episodes.

Investigations revealed normal visual, auditory and somatosensory evoked potentials. The cerebrospinal fluid (CSF) contained three lymphocytes/m³ and a total protein content of 0.5 g/l. Oligoclonal bands were present on CSF protein electrophoresis. CT brain scan was normal, but MRI of the brain revealed a solitary lesion in the deep white matter of the left cerebellar hemisphere, abutting without deforming the

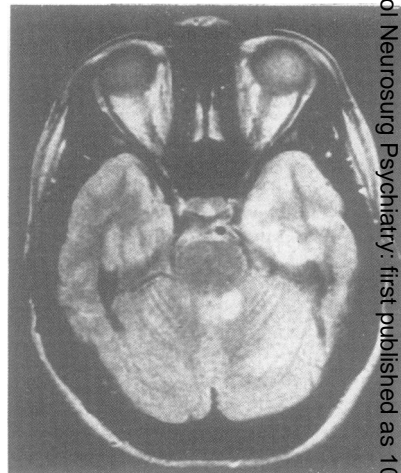


Fig MRI showing high signal from lesion in left cerebellar hemisphere.

fourth ventricle and dorsal aspect of the pons. No other abnormality was seen. (fig 1)

MRI of the brain was repeated three months after the first scan and the solitary lesion involving the left middle cerebellar peduncle was unchanged.

Carbamazepine (100 mg tds) was started one month after presentation and further paroxysms of dysarthria and ataxia occurred at a rate of only one per day. On discontinuing the carbamazepine, the number of paroxysms increased to the previous frequency of up to 20 per day. Reintroduction of the drug completely suppressed the attacks.

Using the classification proposed by Poser *et al*,³ a diagnosis of multiple sclerosis cannot be confirmed in this patient, despite the supportive evidence of CSF oligoclonal bands—there are to date no symptoms, clinical nor paraclinical evidence (as defined by Poser *et al*) of a second lesion in the CNS of this patient. Nevertheless, paroxysmal dysarthria is so characteristic of multiple sclerosis, and has been described as pathognomonic for this disease,⁴ that an alternative diagnosis is unlikely.

The symptoms responded successfully to treatment with carbamazepine but the dramatic response of paroxysmal attacks in multiple sclerosis to carbamazepine has been previously well described.⁵ The MRI lesion in our patient, which probably represents a solitary plaque of demyelination, demonstrated anatomically a correlation with symptoms of paroxysmal dysarthria and ataxia.

The mechanism of ephaptic conduction is widely believed to be the cause of paroxysmal attacks. When Ekbohm *et al*⁶ reported

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tonic seizures associated with contralateral paraesthesia, they postulated transversely spreading activation between axons in the spinal cord as the mechanism. Similarly, Matthews⁷ proposed that the underlying pathophysiology in paroxysmal attacks in multiple sclerosis is abnormal lateral axonal spread of excitation in plaques. A normally transmitted impulse, on reaching a plaque of demyelination, depolarises a neighbouring demyelinated axon. This ephaptic conduction leads to the attack, the nature of which is dependent on the afferent or efferent axon involved. Osterman and Westerberg⁸ explain how transversely spreading activation of axons within a partially demyelinated plaque accounts for various paroxysmal phenomena by describing in detail the relevant anatomy of the spinal cord and brain stem.

Thus, paroxysmal attacks in multiple sclerosis are considered to be due to axonal discharge rather than neuronal discharge through usual anatomical and physiological connections. Although the beneficial effect of the anticonvulsant drug carbamazepine seems to suggest that paroxysmal attacks in multiple sclerosis might be due to focal epilepsy, carbamazepine has effects on the axonal membrane thereby blocking ephaptic conduction⁹ as well as its better known effects on neuronal discharge.

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References

- 1 Matthews WB, Acheson ED, Batchelor JR, Weller RO. *McAlpine's Multiple Sclerosis*. Edinburgh: Churchill Livingstone, 1985; 111-6.
- 2 Twomey JA, Espir MLE. Paroxysmal Symptoms as the first manifestations of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1980;43:296-304.
- 3 Poser CM, Paty DW, Scheinberg L, et al. New diagnostic criteria for multiple sclerosis: guidelines for research protocols. *Ann Neurol* 1983;13:227-31.
- 4 Wolf P, Assmus H. Paroxysmale Dysarthrie und Ataxie: ein pathnomisches Anfallssyndrom bei multipler Sklerose. *J Neurol* 1974;208: 27-38.
- 5 Espir MLE, Millac P. Treatment of paroxysmal disorders in multiple sclerosis with carbamazepine. *J Neurol Neurosurg Psychiatry* 1970;33:528-31.
- 6 Ekbohm KA, Westerberg CE, Osterman PO. Focal sensory-motor seizures of spinal origin. *Lancet* 1968;i:67.
- 7 Matthews WB. Paroxysmal symptoms in multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1975;38:617-23.
- 8 Osterman PO, Westerberg CE. Paroxysmal attacks in multiple sclerosis. *Brain* 1975;98:189-202.
- 9 Schauf CL, Davis FA, Marder J. Effects of carbamazepine on the ionic conductances of myxicola giant axons. *Pharmacol Exp Ther* 1974;189:538-43.

Development of HTLV-I associated myelopathy (HAM) in a seroconverted patient for antibody to HTLV-I

Sir: Seroconversion in recipients of transfused human T lymphotropic virus type-I (HTLV-I) antibody positive blood was reported by one of the authors (KO) in 1984.¹ Osame *et al* suggested that infection transmitted by blood transfusion could be a basis for the development of HTLV-I associated myelopathy (HAM).^{2,3} However, the development of HAM caused by blood transfusion has not been previously reported.

A 70 year old Japanese woman was admitted to our clinic on August 20 1988 in a lactic acidotic coma with hypoglycaemia caused by calcium-hopantenate.⁴ She had had an operation for a DeBakey type I dissecting aneurysm and had been given a blood transfusion on May 22, 1985. She had gradually developed spastic paraplegia six months to one year after the operation. From July 1986 she had been bed-ridden with complete paraplegia as well as sensory and sphincter disturbances. No orthopaedic abnormality was detected. On this admission, she recovered from the coma within nine days but the spastic paraplegia, sensory and sphincter disturbances remained unchanged. Magnetic resonance imaging (MRI) revealed no abnormality in the spinal cord. A high anti-HTLV antibody titre (1280) in the serum and cerebrospinal fluid (CSF) (320) was detected by an indirect immunofluorescence method,¹ indicating HAM. After checking the transfusion history, it was revealed that the patient had been in a follow up study for transfusion-transmitted infection of HTLV-I started in this hospital in 1981.

Anti-HTLV-I antibody was not detected before her operation but she became positive (antibody titre = 5 at one month, 80 at two months later). During her operation the patient received one unit of anti-HTLV-I antibody positive packed red cell (PRC, antibody titre = 640) and one unit of antibody positive fresh frozen plasma (FFP, antibody titre = 640), as well as antibody

negative 26 units of PRC and 10 units of FFP all of which were antibody negative. We believe this to be the first reported case of a patient developing HTLV-I HAM from a blood transfusion. To prevent similar occurrences a donor screening programme was set up in Japan in 1986.

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References

- 1 Okochi K, Sato H, Hinuma Y. A retrospective study on transmission of adult T cell leukemia virus by blood transfusion: Seroconversion in recipients. *Vox Sang* 1984;46: 245-53.
- 2 Osame M, Usuku K, Izumo S, et al. HTLV-associated myelopathy, a new clinical entity. *Lancet* 1986;i:1031-2.
- 3 Osame M, Izumo S, Igata A, et al. Blood transfusion and HTLV-I associated myelopathy. *Lancet* 1986;ii:104-5.
- 4 Noda S, Umezaki H, Yamamoto K, et al. Reye-like syndrome following treatment with the pantothenic acid antagonist, calcium hopantenate. *J Neurol Neurosurg Psychiatry* 1988; 51:582-5.

Parkinsonian symptoms in a patient with AIDS and cerebral toxoplasmosis

Sir: Extrapyramidal symptoms of bradykinesia, rigidity and tremor, have been reported as rare presentations of brain tumour,¹ subdural haematoma² and tuberculoma.³ We report the case of an AIDS patient with parkinsonian features due to bilateral basal ganglia toxoplasma abscesses.

In 1983 the patient, a 66 year old white female, had a resection of a right cystic hamartoma of the bile duct with incidental right adrenalectomy. During her stay in hospital, she required seven units of blood. She did well postoperatively until June 1984 when she developed a disseminated petechial rash. The platelet count was 4,000/mm³ and the laboratory investigations were consistent with an autoimmune thrombocytopenia. With high dose prednisone, the platelet count rose to 12,000/mm³. Incidental toxoplasma titres were drawn and revealed an IgG of 1:4096 and an IgM of 1:128. In