Letters

- 7 Sharer LF, Kapila R. Neuropathologic observations in acquired immunodeficiency syndrome (AIDS). *Acta Neuropathol (Berl)* 1985;66:188-98.
- 8 Hooper DC, Pruitt AA, Rubin RH. Central nervous system infection in the chronically immunosuppressed. *Medicine* 1982;61:166-88.
- 9 Bishburg EB, Sunderam G, Reichman LB, Kapila R. Central nervous system tuberculosis with the acquired immunodeficiency syndrome and its related complex. *Ann Int Med* 1986;105:210-3.

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Magnetic resonance imaging in an HTLV-I antibody positive patient with tropical spastic paraparesis

Sir: Recently interest has been shown in the magnetic resonance imaging of patients with HTLV-I antibody positive tropical spastic paraparesis (TSP).¹⁻³ The MRI changes in TSP may only be mild and with the sensitivity of MRI in detecting lesions these may be fortuitous rather than disease associated.¹ We describe below the case of a young West Indian woman with a spastic paraparesis in whom there were extensive white matter lesions on MRI with an oligoclonal pattern to the CSF electrophoresis and circulating HTLV-I antibodies.

A 36 year old woman who had lived all her life in Grenada, presented with a 3 year history of dragging the left leg progressing to stiffness in both legs and unsteadiness when walking. In 1979, she had pulmonary tuberculosis, treated medically. There was no suggestion of a vasculopathy and she was a non-smoker. General physical examination was normal, except for a spastic paraparesis with bilateral extensor plantar responses. There was reduced vibration sense below the costal margins and position sense was impaired in the feet.

Routine blood tests were normal, as was full myelography. The CSF contained 2 white cells, 0.4 g/l protein and 3.2 mmol/l of glucose. Oligoclonal bands were present in the CSF but not in the serum. Serum angiotensin converting enzyme was 35 (16-53 mmol/minute/ml). Visual evoked potentials were normal. The CT brain scan revealed low attenuation lesions in the white matter, particularly in the periventricular region. MRI (fig.) showed multiple areas of abnormally increased signal in the white matter of both hemispheres and brain stem.

Fig MRI scan showing multiple areas of high signal

The clinical and laboratory findings in our patient would have suggested a diagnosis of multiple sclerosis except that she had lived her life in the West Indies where the disease is rare.⁴ The finding of HTLV-I antibodies in the blood is consistent with a clinical diagnosis of tropical spastic paraparesis.⁵ The clinical similarity of the two diseases has been dealt with elsewhere but the pathology is quite different with an inflammatory meningeal process with mononuclear cell infiltration predominating in TSP.¹ The extent of the white matter changes on MRI of our patient is unlikely to be fortuitous, age related or due to ischaemic or granulomatous pathology. It may well be that the degree of white matter involvement depends on the stage of the illness and the suggestion⁶ that the lesions in TSP are more sparse than in multiple sclerosis is not supported by this case.

As the pathological changes are so different in the two conditions, it is disappointing that the MRI findings are similar. This case, like others, demonstrates the need for full investigation of patients with spinal cord symptomatology and negative myelography. We are grateful to Dr A Dalglish and Dr K Cruickshank for performing the HTLV-I antibody screen on our patient.

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References

- 1 Newton M, Miller D, Rudge P, *et al.* Antibody to human T-lymphotropic virus type-I in West Indian born UK residents with spastic paraparesis. *Lancet* 1987;ii:415-516.
- 2 Mattson DH, McFarlane DE, Mora C, Zanirovic V. Central nervous system lesions detected by magnetic resonance imaging in a HTLV-I antibody positive symptomless individual. *Lancet* 1987;ii:2-49.
- 3 Tournier-Lasserre E, Gout O, Gessain A, *et al.* HTLV-I, brain abnormalities on magnetic resonance imaging, and relation with multiple sclerosis. *Lancet* 1987;ii:249-50.
- 4 Cruickshank EK. A neuropathic syndrome of uncertain origin—review of 100 cases. *West Indian Med J* 1956;5:147-58.
- 5 Gessain A, Vernant JC, Maurs L *et al.* Antibodies to human T-lymphotropic virus type-I in patients with tropical spastic paraparesis. *Lancet* 1985;ii:407-10.
- 6 Miller DH, Newton MR, Rudge P, Cruickshank K. Magnetic resonance imaging in HTLV-I antibody positive patients. *Lancet* 1987;ii:514.

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Parkinsonism as first manifestation of lymphomatoid granulomatosis

Sir: Lymphomatoid granulomatosis (LG) was first described in 1972 by Liebow *et al.*¹ as an angiocentric and angiodestructive lymphoreticular and proliferative disorder of the lungs. LG has a distinctive organ distribution most frequently involving lung, skin, renal interstitium and central nervous system (CNS). Although initially described as a vasculitis, it is now considered that LG is a lymphoproliferative lesion composed predominantly by T-lymphocytes.² Two-thirds of the patients die with a median survival of 14 months despite therapy, and a definable lymphoma is found to have developed in 12% of the patients. The time interval between initial diagnosis and lymphoma range from weeks to several years.³

CNS dysfunction has been reported in 20% and peripheral neuropathies in 15% of patients.⁴ However, extrapyramidal involvement has been rarely described. We report a case of a woman with pulmonary LG and extrapyramidal manifestations.

In December 1986, a 67 year old woman was admitted to hospital because of cough, fever, weight loss and inability to walk. In 1984 she had developed a mood disorder and she was treated with tricyclic compounds without any improvement. In April 1985, the