that there is a considerable shortfall in specialist neurological services in the United Kingdom. The question arises as to whether or not the urgent situation of patients in a factory is surprising that over 50% of new referrals were given a priority classification. This high figure may reflect the known long waiting time for “routine” patients, rather than a truly perceived seriousness of the medical condition. It is worth noting that some patients with serious disease were put on the non-urgent list, thus indicating that in some cases at least the initial priority category was inappropriate. The information retrieval letters might assist consultants to classify patients appropriately.

In conclusion, this study highlights particularly: 1) the predominance of the diagnostic role of the neurology outpatient consultation; 2) the small proportion of patients referred with serious disease; 3) the unacceptably long waiting time, and the inappropriate priority classification of some patients.

Mrs V A Wood gratefully acknowledges the help she received from Dr D T Wade, Consultant Neurologist, Rivermead Hospital, Oxford; Dr Pamela Endicott, Consultant Psychiatrist, Frimley Health Authority, and Mr H Rothman, Reader in Management, Bristol Polytechnic, who advised and supervised her through her MPhil degree. Acknowledgement and thanks is also extended to Mrs Judith Weeks and Mrs Susan Janes, Higher Clinical Officer, Department of Neurology, Frenchay Hospital, who kept a vigilant surveillance of medical case notes.

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We would be pleased to consider for publication short letters describing similar audit of outpatient practice in other countries.

Ed


HTLV-I infection: the clinical spectrum widens

A neurological condition causing spastic paraparesis has long been recognised in the West Indies but it has only in recent years that the association between tropical spastic paraparesis (TSP) and human T-cell lymphotropic virus type 1 (HTLV-I) has been confirmed.1,2 Serological tests for HTLV-I can now support a diagnosis of TSP in patients with atypical clinical features. Patient 1 was born in Jamaica and came to the United Kingdom at the age of 38 years. At the age of 61 years she presented with pain in her left shoulder, a three year history of difficulty raising her arms above her head, fatigue and inability to walk long distances. On examination there was no evidence of spasticity, and there were no features suggesting of any other scapulæ weakness in the deltoids, triceps, biceps, without fasciculations. Distal upper limb musculature was normal. The biceps and supinator jerks were absent and there were normal and the triple and biceps plantar responses. Sensory testing was normal. The patient was mildly elevated and muscle histology revealed neurogenic changes. Myelography and CSF examination were normal. There was a polyclonal increase in the serum immunoglobulins.

At first a diagnosis of motor neuron disease was considered but there was no change in her condition during the following four years and reinvestigation revealed serum antibodies against HTLV-1 in a titre of 1 in 6400. In TSP, pain, weakness, or preceeding a specific event of weakness but is characteristically confined to the lower limbs and lumbar spine. This patient is also unusual in the severity of the muscle wasting which in TSP is seldom prominent and usually confined to the intrinsic hand muscles.

Patient 2 was born in Jamaica and came to the United Kingdom aged 42 years. He was first seen aged 63 years following a single generalised convulsion; his right plantar response was extensor but there were no other neurological signs. CT brain scan and EEG were normal. Tendon reflexes were normal (VDRL negative, TPHA positive, FTA IgG positive, IgM negative) without a history of previous venereal disease or yaws. Lumbar CSF was acelluar with normal protein concentration and the tests for syphilis were all negative; Link’s IgG index was 0.78 (upper limit of normal 0.58) suggesting intrathecal immunoglobulin synthesis. He was treated with a course of intramuscular procaine penicillin and regular phenytoin. At the age of 70 years he was seen again for reassessment of his epilepsy. Only on direct questioning did he admit that his legs had become weak sooner one month after moving to New York and had difficulty rising from a low chair but otherwise his gait was normal. There was a mild spastic paraparesis with brisk lower limb reflexes and the right plantar response was extensor as before. A CT brain scan was again normal. MRI scan showed several small puncate white matter lesions in both cerebral hemispheres but no abnormality of the spinal cord. Antibodies to HTLV-I were detected in the serum in a titre of 1 in 8000.

Patient 3 was born in British Guyana and came to the United Kingdom at the age of 34 years. At the age of 59 years she developed bilateral uveitis and was found to have positive treponemal serology (VDRL negative; TPHA positive; FTA IgG positive, IgM negative) without a past history of venereal infection or yaws. CSF contained 32 lymphocytes/mm³ and the protein concentration was raised (0.85 g/ℓ); CSF tests for VDRL, TPHA and FTA were negative. Her vision returned to normal over a few months but she took a one month course of oral doxycycline. Six months later she developed weakness and sensory loss in the legs which progressed over a year. At that time she had a spastic paraparesis with a sensory level at T10 and normal position sense. The upper limbs and cranial nerves were normal. Myelography was normal but was followed by urinary retention requiring catheterisation. The CSF on this occasion contained no cells but the protein concentration remained raised (1 0 2/1)

A diagnosis of neurosarcoidosis was considered but the patient showed no response to oral corticosteroid therapy; subsequent bronchoscopy, bronchial biopsy and Kveim test were found to be normal.

At follow up two years later her gait had deteriorated slightly, she had developed mild bilateral nerve deafness and there was muscle wasting in both hands. Antibodies against HTLV-1 were detected in the serum in a titre of 1 in 6400. The blood film was normal but there was a polyclonal increase in the serum immunoglobulins.

The initial detection of HTLV-1 antibodies was by gel particle agglutination assay and confirmed by more specific methods (ELISA, indirect immunoelectrophoresis, IgG antibody capture radio-immunoassay and Western blot techniques).3

These patients illustrate many of the recognised features of TSP. Indeed, this unusual illness in each of them had been tentatively attributed to another cause: motor neuron disease (patient 1), parasagittal tumour or neurosyphilis (patient 2), neurosarcoidosis or neurosyphilis (patient 3). Their neurological presentations were pectoral pain and amyotrophy (patient 1), late-onset epilepsy (patient 2) and uveitis (patient 3). Testing for HTLV-I antibody confirmed the diagnosis on these patients and should be performed in all West Indian patients with spastic paraparesis and with other unexplained neurological syndromes. If effective treatment for HTLV-I infection becomes available early diagnosis will be necessary to identify patients before severe, irreversible neurological damage has occurred. This will require greater awareness of the diverse ways in which TSP may present. The full spectrum of HTLV-I infection remains to be defined.

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Temporal lobe phenomena during the aura phase of migraine attacks

I report a patient who often experienced temporal lobe phenomena during the aura phase of his migraine attacks. A 27 year old right handed computer
HTLV-1 infection: the clinical spectrum widens.

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J Neurol Neurosurg Psychiatry 1991 54: 371
doi: 10.1136/jnnp.54.4.371

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