

helper/inducer T cells and with Leu-2a (Beckton Dickinson) to detect suppressor-cytotoxic T cells. Peripheral blood mononuclear cells were separated through a Ficoll-Paque gradient and characterised by an indirect immunofluorescence assay as previously described.<sup>3</sup> Cytological preparations were made from fresh CSF samples with a Shandon Cytospin. The cells were stained with haematoxylin and immunocytochemistry with monoclonal antibodies was performed using the Vectastin ABC kit (Vector Laboratories). At least 100 cells were counted per slide.

The table shows the values for T cell subsets and the helper/suppressor ratio for each patient. The lowering of the ratio of T helper to suppressor T lymphocytes in PBL from a mean of 2.20 in control subjects to 0.46 in patients with AIDS is also observed in the CSF, with a decrease from a mean of 2.18 in other neurological disorders (2 polyradiculoneuritis, 1 meningoradiculoneuritis, 5 low back pain, 1 cerebrovascular disease and 1 dementia) to 0.33 in patients with AIDS or AIDS-related complex. These differences are statistically highly significant ( $p < 0.001$ ) in both the CSF and the peripheral blood. The marked decrease in the percentage of T helper cells found in AIDS is often accompanied by an absolute or a relative increase in suppressor cells and this was observed in most of the patients. One patient had a Burkitt lymphoma with meningeal involvement at the time of examination and over 80% of the cells in the CSF reacted with the B cell antigen Leu 12.

The finding of severely reduced percentage of T helper cells in the CSF of AIDS patients and in the AIDS-related complex is of interest aside from its possible diagnostic implications. It has been shown that the AIDS

virus (HIV) was detected in the brain of AIDS patients and it has been suggested that HIV could be implicated as a possible cause of AIDS encephalopathy.<sup>4</sup> Furthermore, the presence within the blood-brain barrier of a specific immunoglobulin to HIV in patients with neurological complications of the AIDS or the AIDS-related complex has been documented.<sup>5</sup> Though the decrease in T helper lymphocyte in the CSF may reflect an altered ratio in the peripheral blood, it may also suggest actual destruction of the helper subset in the brain. Migration and sequestration of activated T cells from peripheral blood into the central nervous system could lead to a persistent virus infection. This could play an important role in relation to long term immuno-pathological consequences. It is of interest to note that many viruses involved in CNS infections also infect lymphocytes and that lymphotropic viruses have recently been implicated in the pathogenesis of multiple sclerosis,<sup>6</sup> a chronic neurological disease associated with disturbances in the relative proportions of T cell subpopulations.

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Hiccups and vomiting as initial manifestations of multiple sclerosis

Sir: Hiccups and vomiting are such rare features of multiple sclerosis that they are not mentioned in standard texts on the disease,<sup>1,2</sup> although they have been described in isolated case reports.<sup>3,4</sup> We report a case in which both hiccups and vomiting were the presenting features of the illness, though this was not recognised at the time.

A 28 year old woman from the Dominican Republic was seen at various emergency rooms and clinics in Rhode Island for a period of 6 months prior to admission, for complaints of intermittent sustained hiccuping and mild vomiting for periods lasting up to 16 days. Physical examination, complete blood count and electrolytes were all normal. She returned to the Dominican Republic, where a neurological evaluation was normal. Two months prior to admission she developed headache and diplopia in association with worsening and now continuous hiccups, all of which resolved following a course of ACTH given for an unsubstantiated diagnosis of adrenal insufficiency.

She was admitted to the Roger Williams General Hospital with recurrent diplopia and a severe headache located behind the eyes, but without hiccups or vomiting. Other symptoms included diminished sensation on the left side of the face and difficulty chewing on the left side. Past medical history was remarkable for hypothyroidism treated with thyroid replacement, and an episode of "diet pill" toxicity. Cranial nerve examination was

Table

Clinical stage	CSF cells/mm <sup>3</sup>	% of total cells			
		suppressor cells	helper cells	helper/suppressor ratio*	
AIDS, Burkitt lymphoma with meningeal involvement	441†	CSF	8	4	0.50
		PBL	55	38	0.69
AIDS, cryptococcal meningitis	74	CSF	55	13	0.24
		PBL	53	20	0.38
AIDS, encephalopathy	3	CSF	64	0	0.00
		PBL	70	10	0.14
AIDS-related complex	8	CSF	7	4	0.57
		PBL	31	20	0.65

\*Mean ± SD for normal in peripheral blood is 2.20 ± 0.17 (n = 44) and mean ± SD for other neurological disorders in CSF is 2.18 ± 0.25 (n = 10).  
†Over 80% of the cells in the CSF reacted with the B-cell antigen Leu 12.

## Letters

remarkable for bilateral internuclear ophthalmoplegia, bilateral third nerve paresis with normal pupils, bilateral sixth nerve paresis, and diminished fifth nerve sensation and strength. The remainder of the cranial nerves, motor and sensory systems were normal. There was a mild gait ataxia. Pertinent laboratory data included a white cell count of  $10.3 \text{ mm}^{-3}$ , normal electrolytes, normal thyroid functions, and sedimentation rate of  $6 \text{ mm/1 l}$ . Cerebrospinal fluid contained no red cells and 8 white cells, of which 92% were lymphocytes and 8% were monocytes. CSF protein was  $58 \text{ mg/dl}$  and the glucose  $52 \text{ mg/dl}$ . Cytology was negative. Oligoclonal bands were absent and myelin basic protein was normal. Computed tomography of the head with intravenous contrast demonstrated a non-enhancing hypodense area in the right temporal lobe, and no abnormalities in the brainstem. An EEG was abnormal because of the presence of intermittent excessive slow waves emanating from the right temporoparietal region. Brainstem auditory evoked potentials were normal. Somatosensory evoked potentials were normal on the right, but suggested an abnormality between the brachial plexus and dorsal column/medial lemniscus on the left. The visual evoked potential revealed no response to pattern shift visual evoked response in either eye. Magnetic resonance imaging of the head demonstrated multiple T2 weighted abnormalities diagnostic of a demyelinating process (see fig) affecting the right temporal horn, the paraventricular areas, the corpus callosum, and brainstem along the fourth ventricle in the pons and medulla.

Vomiting and hiccups were the initial manifestations of multiple sclerosis in this patient. We have found only one previous report<sup>3</sup> describing three cases of multiple sclerosis associated with intractable hiccups. The authors suggested that a lesion such as a plaque may either interfere with normal descending inhibition, or serve as an irritative lesion. The lesions responsible for hiccups are presumably in the brainstem, but the exact location remains unknown.

Vomiting is an uncommon manifestation of multiple sclerosis.<sup>4</sup> The "vomiting centre" is situated in the dorsal portion of the lateral reticular formation of the medulla at the level of the dorsal nucleus of the vagus, and a chemoreceptor trigger zone is located in the floor of the fourth ventricle in the area postrema.<sup>5</sup>

We believe this patient had both vomiting and hiccups as part of the symptom complex of multiple sclerosis. Magnetic resonance imaging revealed demyelinated plaques in the appropriate areas of the brainstem to

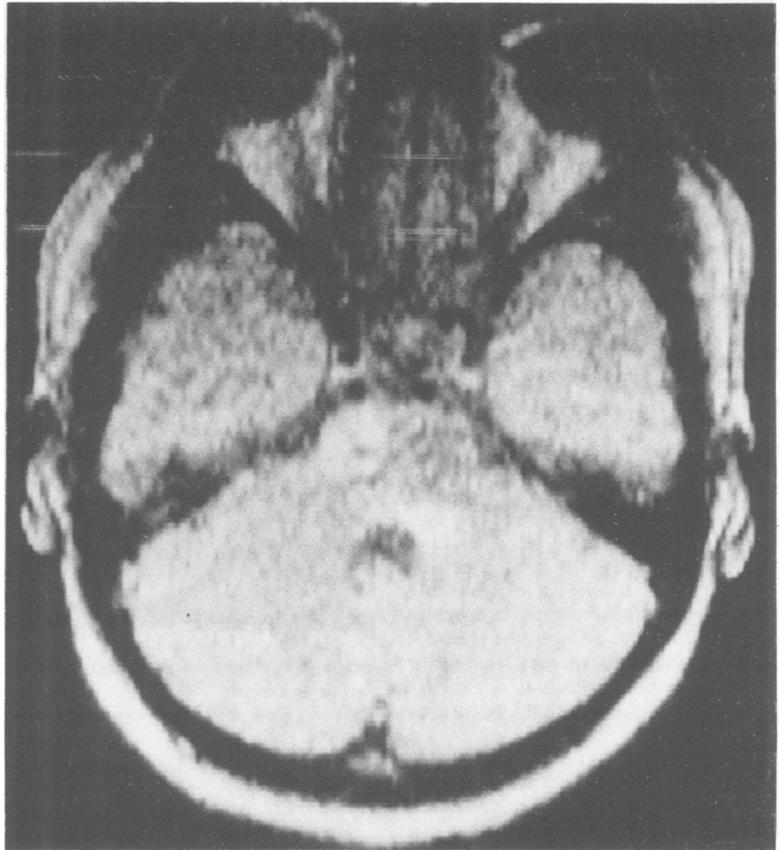


Fig Bright areas along fourth ventricle indicate demyelinated plaques, presumably explaining patient's vomiting and hiccups.

explain all the symptoms. Both the protean manifestations of multiple sclerosis and the power of magnetic resonance imaging are well-illustrated in this case.

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