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later the patient was symptomless; CT and cerebral angiography were normal with complete resolution of the SSS and cortical veins thrombosis.

Unequivocal evidence of CVT involving the SSS and the cortical veins were present in our patient; clinical and radiological features were consistent with established criteria^{1,2,4} including so-called benign intracranial hypertension and the empty delta sign: axial CT sections of the SSS showed a triangular enhancement pattern surrounding a central hypodense area, which is considered as pathognomic of SSS thrombosis. Extensive investigations for other causes of CVT were negative, raising the question of its relationship with the concurrent viral infections. CMV primary infection was diagnosed on IgG seroconversion, presence of specific IgM and viraemia; CMV was not isolated from CSF. The diagnosis of HIV₁ primary infection was supported by the rise of IgG titres and the presence of specific IgM; HIV₁ antigenemia and reverse transcriptase activity were absent but they are not constant features in HIV₁ primary infection.⁶

Simultaneous CNS dual viral infection is increasingly reported in the acquired immunodeficiency syndrome^{7,8,9} but occurs very rarely in immunocompetent adults;¹⁰ in all these cases, CMV was involved. To our knowledge, the occurrence of CVT in CMV or HIV₁ infection has not been described. CMV causes a wide spectrum of neurological disorders¹¹ including Guillain-Barré syndrome, diffuse meningo encephalitis, brachial plexus neuropathy and possibly myelitis; although cephalalgia and elevated CSF pressure were recorded in some of these cases¹² there were no other characteristics of CVT present. Disseminated CNS vasculitis involving medium and small sized arteries and veins (the latter to a lesser degree) occurred in one immunocompromised patient.¹³ A limited number of neurological syndromes have been associated with seroconversion to HIV₁: meningitis,¹⁴ encephalopathy,¹⁵ neuropathy¹⁶ acute myelopathy.¹⁷ Arteritis has been described in two patients with well documented HIV₁ infection.^{18,19} In our patient, the exact causative link between HIV₁, CMV and CVT remains speculative. Clinical and serological evidence suggest CVT was caused by viral infection. Although intra-blood-brain-barrier IgG secretion was not measured, the presence of anti-HIV₁, but not anti-CMV, IgG, and the blood-to-CSF ratio of anti-HIV₁ IgG titres suggest that HIV₁ (preferentially to CMV) may be the causative agent.

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Supratentorial meningeal spread from brain-stem glioma

Sir: Brainstem gliomas commonly spread by infiltration of the brainstem, resulting in multiple cranial nerve deficits, pyramidal tract dysfunction, or cerebellar ataxia.¹ Extensive subarachnoid spread (meningeal gliomatosis) is a rare but well-described complication.¹⁻³ The spinal subarachnoid space seems to be the site of predilection for seeding.

We report a young woman in whom the initial clinical picture was caused by supratentorial meningeal involvement.

A 25 year old right-handed woman complained of headaches and nausea for three weeks. She was admitted with a sudden onset of dysphasia and right-sided weakness. On examination, she was alert but mute and showed right-sided neglect, but no signs of weakness or pyramidal features. Brain CT was performed and appeared to be normal. Lumbar puncture showed opening pressure of 250 mm CSF, 40 WBCs (mononuclear cells), 20 RBCs, a glucose level of 5.0 mmol/l and a protein level of 12.7 g/l. Six hours later she became confused and obtunded and started vomiting. Examination, at this time, showed a temperature of 39.5°C and bilateral Babinski sign. Her neck was stiff and Kernig and Brudzinski signs were positive. Repeat CT (contrast enhanced), again was negative. A second lumbar punc-

ture showed an opening pressure of 40 mm CSF, 120 WBCs, mostly mononuclear. Cerebral digital subtraction angiography was normal. Electroencephalography showed diffuse slow wave activity, with a left-sided occipital focus of delta activity. A presumptive diagnosis of herpes simplex encephalitis was made and the patient was started on acyclovir treatment. The next day she was alert and able to talk, but she had a retrograde amnesia extending over one month. The patient's condition remained clinically stable for one week, but then she again became obtunded, with dysarthria and right-sided hemiparesis. Repeat CT was normal. CSF showed an even higher protein level of 17 g/l with a cell count as previously mentioned. Two weeks after admission, she developed fluctuating cranial nerve involvement with impairment of the right oculomotor and both abducens nerves. There were also periods of apnoea, requiring intubation for a short time. A fourth lumbar puncture disclosed a few malignant cells of unknown origin. Seventeen days after admission a left temporal brain biopsy was performed. The biopsy specimen showed glial tumour cells in the leptomeninges. The cortex was not infiltrated. Thereupon the diagnosis of meningeal gliomatosis was made. She was treated with whole brain radiation therapy, but her clinical condition deteriorated. Her level of consciousness varied, she developed left-sided oculomotor and bilateral blindness. Subsequently, she started to complain of excruciating pain in the legs and developed progressive flaccid paraparesis and sphincter disturbances. After ten weeks of hospitalisation, she died of aspiration pneumonia.

Pathological findings

General necropsy only disclosed bilateral bronchopneumonia. The brain weighed 1260 grams after fixation. The pons varoli showed a deep infiltrating tumour mass with extensive spread to the leptomeninges of the cerebellum, brainstem and spinal cord. The cerebral leptomeninges were focally involved (see figure). The tumour enclosed the spinal cord from the cervical level to the cauda equina. Small tumour nodules were present in the lateral ventricles. Whole brain sections were made and the brainstem and spinal cord were studied at regular intervals. Microscopically, the tumour consisted of spindle-shaped astrocytes, positive for GFAP. Small foci of vascular proliferation and necrosis were also present. Mitotic figures were manifold and some tumour giant cells were found. These findings were compatible with a diagnosis of astrocytoma grade IV. There

Fig Subarachnoid tumour implants on the surface of the cerebrum (inset). The cerebellum and the brainstem are covered by subarachnoid tumour. There is marked enlargement of the pons.

was only a superficial infiltration in the spinal cord and the tumour did not infiltrate in the cerebral cortex.

Our patient's initial clinical picture (that is, dysphasia and hemi-inattention) and CSF findings suggested meningo-encephalitis. A meningeal syndrome, consisting of fever, nuchal rigidity and CSF pleocytosis has been reported in patients with intracranial glioblastoma multiforme.⁴

The meningeal irritation is attributed to chemical inflammation resulting from the release of lipid-containing necrotic tissue

into the CSF or to an immunological cellular response of the CSF to tumour cells. Cytological examination of CSF is negative in about one-third of the patients reported with meningeal gliomatosis.⁵ In our patient only a few tumour cells were observed after repeated lumbar puncture. GFAP-staining was not performed and might have indicated its glial origins.⁵ The correct diagnosis of cerebral leptomeningeal gliomatosis was only established after brain biopsy. Since neither the cortex nor the arteries supplying the left hemisphere were invaded by tumour,

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we do not have a satisfactory explanation for our patient's left cerebral hemisphere dysfunction. In the final stage of the disease, the clinical picture was dominated by radicular pain in the legs and flaccid paraparesis, also indicating spinal subarachnoid seeding. Postmortem examination not only confirmed this, but also disclosed that the primary tumour was located in the pons. The incidence of leptomeningeal dissemination of brainstem gliomas is difficult to determine since only few series of patients have been described.^{3,4} Moreover, the diagnosis is often made by postmortem examination. However, Packer *et al* were able to diagnose meningeal gliomatosis antemortem in five of 15 brainstem glioma patients (33%).¹

All previously reported patients were found to have extensive spinal subarachnoid seeding. To the best of our knowledge, there have been no well-documented reports on brainstem gliomas spreading to the supratentorial meninges.

We are grateful to Professor A J M van der Werf for performing the brain biopsy.

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Accepted 7 March 1989

CSF oligoclonal IgG bands in a patient with torsion dystonia

Sir: The CNS changes underlying idiopathic torsion dystonia are unknown. Immunological changes have not been recognised in this condition but we report here a case in which oligoclonal IgG bands were identified repeatedly in the cerebrospinal fluid.

A 38 year old male patient was referred for investigation of his abnormal gait in 1983. His walking was disturbed by gross abnormal movements. Sustained dystonic muscular contractions provoked inturning, twisting and high stepping of the right foot with a tendency to hold the left leg stiffly. An exaggerated lordosis, twisting of the trunk, and wide swinging of the left arm accompanied these movements.

Dystonic posturing of both legs, and particularly the left arm, were exaggerated by voluntary activity. Finger-nose testing on the left was disrupted by these movements although there was no ataxia, alteration in tone, weakness or sensory loss in the limbs. Dystonic tongue movements made the patient's speech dysarthric. Visual acuities were 6/60 right and 6/6 left. There were no Kayser-Fleischer rings and cranial nerve examination was otherwise normal. Limb reflexes were present symmetrically, and both plantar responses were flexor. General physical examination was otherwise normal.

The patient had been born at 38 weeks gestation by caesarean section, birth weight 2.97 kg, his mother having had hypertension in pregnancy. He was healthy at birth but developed pertussis at 6 weeks of age. This illness required hospital admission for 10 weeks and he was said to have had a number of seizures during this period. He could walk at 15 months, and at this time was noted to have a squint affecting the right eye.

His abnormal gait was first brought to medical attention by his mother at the age of 6 years, and gradually deteriorated throughout childhood. Clinical records suggest a "... jerky, clumsy gait, ... with normal balance, co-ordination and reflexes (6 years) ...", "... spastic muscular inco-ordination (7 years) ..." and "... a curious gait in running, a habit (8 years). Examination at 8 years is recorded as showing no evidence of organic disorder or spasticity. The condition progressed gradually to abnormal posturing of the legs and left arm at rest. Dystonic movements of the tongue, jaw and neck were noted when the patient was seen with back pain aged 36 years.

The patient's family were non-Jewish and there was no family history of any abnormal movement disorder. The patient did not

smoke, drank alcohol only occasionally, and was taking no drugs when seen. He had never received neuroleptic or other drugs associated with abnormal involuntary movements.

Investigations showed a normal full blood count and film, urea and electrolyte estimation, plasma glucose, B12, folate, thyroid and liver function tests. Serum copper 16.2 µmol/l, (laboratory normal range 13-24 µmol/l), and caeruloplasmin 190 mg/l, (laboratory normal range 150-600 mg/l). Serum antinuclear factor, auto-antibody screen, VDRL, and urine amino acid chromatography were negative. Peripheral motor and sensory nerve conduction studies, chest radiograph, CT and magnetic resonance imaging of the brain were normal. The presence of Kayser-Fleischer rings was excluded by slit lamp examination.

Cerebrospinal fluid (CSF) examination in 1983 showed a normal pressure and glucose level, less than 1 white cell/mm³, protein 0.650 g/l, IgG 0.089 g/l, IgG index 1.36 and multiple oligoclonal bands detected by isoelectric focusing. Repeat CSF examination in 1987 showed CSF protein 0.544 g/l, albumin 0.237 g/l, IgG 0.108 g/l, IgG index 1.78, oligoclonal bands strongly positive in

Fig Immunoblot showing multiple discrete IgG bands in unconcentrated CSF(C) but not serum(S).