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Postinfectious meningoencephalitis complicating *Mycoplasma pneumoniae* in a child

Sir: *Mycoplasma pneumoniae* is the primary non-bacterial pathogen causing lower respiratory tract infections among school-age children.^{1,2} Of the non-respiratory manifestations, neurological disorders are the most common. They include Guillain-Barre's syndrome, meningitis, transverse myelitis and encephalitis.^{3,4} The prognosis is generally favourable which may be illustrated by the following case report.

A 12-year-old, right-handed boy was admitted to hospital because of right-sided hemiparesis. Nine days prior to admission he had stayed home from school with a headache. During the next few days he had suffered mild upper respiratory symptoms with fever, his temperature rising to 38.5°C and he seemed confused at night. The boy was already starting to recover when he noticed a weakness in his right leg which, by the next day, had evolved into a right-sided hemiparesis, resulting in admission. His temperature was 38°C, pulse rate 104/min, blood pressure 110/70 mm Hg. On neurological examination, the boy was bradyphrenic but alert. He had a mild expressive aphasia, and right-sided hemiparesis. Complete blood count, ESR, chemistry profile and urine analysis were normal. CSF contained 102 WBCs/mm³, protein 86 mg/dl and glucose 3.8 mmol/l. The CSF cytogram showed polymorphonuclear leucocytes, monocytes, many lymphocytes, plasma cells, and macrophages. A chest radiograph demonstrated increased bronchial markings. A CT scan revealed white matter hypodensity in the left parietal region. An EEG showed diffuse marked slowing of background activity on the left side and no abnormalities on the right side.

During the next three days the aphasia increased, right-sided hemianopia became apparent and the hemiparesis developed into a hemiplegia. Four days after admission the boy started to vomit and became comatose. His left pupil was dilated but still responded to light. Bilateral decorticate rigidity developed suddenly, with equally wide and fixed oval pupils. A repeat CT (fig a) demonstrated marked finger shaped white matter hypodensity in the left hemisphere. An Ommaya device was inserted. Treatment was then continued with dexamethasone, hyperventilation under etomidate, repeated mannitol infusions and external CSF drainage with monitoring of CSF pressure. Serological data indicated active *Mycoplasma pneumoniae* infection: complement fixation 1:64, immuno-

Fig (a) CT (after intravenous contrast). Low density areas in the white matter, predominantly in the left hemisphere. Shift to the right. Left-sided cortical ribbon enhancement. (b) Four days later. Progression of white matter hypodensities, with compression of the left lateral ventricle. Drain from Ommaya reservoir in situ.

fluorescent antibody 1:5120 and Elisa IgM positive.^{5,6} CSF tests for *Mycoplasma pneumoniae* were negative. Tentative treatment with doxycycline was initiated. Serodiagnosis for adenovirus, mumps, Q-fever, Coxsackie A and B, ECHO, measles viruses, *Chlamydia tr.*, *legionella pneumoniae*, HHV, VZV, CMV and for IgM/IgG Epstein-Barr virus was negative as were routine and mycobacterial cultures. CT eight days after admission showed progression of white matter hypodensity (fig b). Bilateral carotid angiography showed no abnormalities. Ten days after admission CSF pressure remained spontaneously below 20 mm Hg. The CSF at that time was clear with 15 leucocytes/mm³.

In the weeks that followed, the boy made a remarkable recovery. Only the homonymous right-sided hemianopia remained unchanged. A CT performed 33 days after admission demonstrated left occipital infarction and slight dilation of the left lateral ventricle, but was otherwise normal. Neurological examination two months later showed, apart from the hemianopia, no other abnormalities but slight facial asymmetry. Neuropsychological examination three months after discharge revealed an overall WISC IQ of 109 (verbal 101, performance 118, memory 97), with hardly any signs of cerebral dysfunction. Five months after discharge antibodies to *Mycoplasma pneumoniae* were low: CF 1:8, IF 1:80, Elisa IgM negative.

As *Mycoplasma pneumoniae* infections are usually non-specific and often even asymptomatic we feel that their role in neurological disorders may be underestimated. The diagnosis of *Mycoplasma pneumoniae* infection is usually based on a fourfold increase in CF blood titre. As this may lead to false positive results^{7,8} it is advisable to add the more specific *Mycoplasma pneumoniae* IgM and IgG class immunofluorescent antibody and Elisa assays.

The pathogenesis of *Mycoplasma pneumoniae* associated neurological manifestations is unclear but is probably of an immunological origin.⁹ For example, the pathological changes in the brain described by Fisher *et al*¹⁰ indicate an immune-mediated mechanism: demyelination and perivenous oedema, perivenular mononuclear infiltrate surrounded by macrophages and reactive gliosis with relative sparing of neurons. The clinical and CT findings in our patient are compatible with this picture of postinfectious leukoencephalitis. Reports indicate transient depression of cell-mediated immunity^{11,12} in the early phase of *Mycoplasma pneumoniae* infection. The beneficial effects of plasma exchange in a patient with *Mycoplasma pneumoniae* associated transverse myelitis and with antibodies to nervous tissue,¹³ leave room for speculation on the role of autoimmunity. The components of the immune response of patients with

Mycoplasma pneumoniae associated neurological disease should be studied in detail.

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Selective increase in cerebrospinal fluid immunoglobulin G in a patient with Sydenham's chorea

Sir: Evidence exists to suggest that immunological mechanisms may be involved in the pathogenesis of Sydenham's chorea.¹⁻⁶ This evidence is led by the demonstration of tissue specific immunoglobulin G (IgG) antibodies to neurons of normal human caudate and subthalamic nuclei in the serum of 14 of 30 children with Sydenham's chorea.² Moreover, antibody titres correlated with disease activity and the antibody was absorbed out with membranes of group A streptococci, infection with which is a recognised antecedent to the development of Sydenham's chorea.⁷ The case for antibody

mediated responses playing a primary role in Sydenham's chorea would be strengthened were there evidence of excess IgG present in the cerebrospinal fluid (CSF) of these patients.⁸ Such a finding is not recorded in either a contemporary authoritative text⁹ or review article¹⁰ on the CSF, or in recent accounts in which the CSF of patients with Sydenham's chorea was studied (for example refs 11, 12). These observations prompted the following case report.

A 15-year-old black female was admitted to Kalafong Hospital on 12 August, 1985 with a 6-day history of abnormal movements. Her past medical history was remarkable for recurrent sore throats, the most recent of which occurred approximately 3 weeks before admission. There was no known family history of chorea and she denied drug ingestion or treatment with hormonal contraceptives. Examination revealed emotional lability, distractibility, restlessness, occasional snorts and grunts, mild dysarthria and generalised involuntary movements typical of Sydenham's chorea.¹³ The limb muscles were hypotonic and the patellar reflexes were "hung up". Voluntary movements were exaggerated and she was unable to walk without aid. The temperature was 38°C and pulse rate 110 beats per min. In the opinion of a cardiologist, the heart was clinically normal.

Results of pertinent investigations were as follows: Haemoglobin 12.7 g/dl, erythrocyte sedimentation rate 35 mm/first h, total white blood cells $7.7 \times 10^9/l$, serum IgG 29.6 g/l (normal 8.00-18.00), antistreptolysin O titre 400 Todd's units (<200), streptokinase haemagglutination antibody 1:2560 (<1:1280) and rapid plasma reagin (RPR) 1:128. The *Treponema pallidum* haemagglutination antibody (TPHA) and fluorescent treponemal antibody absorption (FTA-ABS) tests were positive in the blood, the IgG FTA-ABS test reactivity being 3+ (max 4+) and the IGM 1+. (These results prompted appropriate therapy with parenteral penicillin). An echocardiogram revealed evidence of stenosis and incompetence of the mitral valve. Normal or negative results were obtained for the following: Throat swab culture, C-reactive protein, Heller agglutination test, fluorescent anti-nuclear antibody test, total haemolytic complement, serum C₃, plasma C₄, circulating immune complexes, thyroid function tests, Kayser Fleischer rings, serum copper and ceruloplasmin, somatosensory evoked potentials (median nerves), electrocardiography and CSF RPR, TPHA and FTA-ABS tests.

An electroencephalogram on 26 August

showed an excess of 4-7 Hz and intermittent 2-3 Hz activity in a diffuse, bilateral distribution. Contrast enhanced computed tomography of the brain (10 September) was normal. Lumbar CSF on 22 August was acellular with a normal glucose concentration and a total protein of 0.46 g/l (0.15-0.45). The IgG was 0.146 g/l (0.002-0.028), 31.7 per cent (5-12%) of the total protein, and CSF/serum IgG ratio was 4.9×10^{-3} ($1.0-3.9 \times 10^{-3}$). Repeat CSF examination on 11 September was unremarkable, the IgG (0.018 g/l) being 4.6% of the total protein (0.39 g/l) and the CSF/serum IgG ratio 0.98×10^{-3} (serum IgG 18.3 g/l). The patient was discharged on 25 September free of psychologic and neurologic deficits and taking no medication other than oral penicillin.

Of the numerous nervous system diseases associated with a selective increase in CSF IgG,^{9 10 15} neurosyphilis has special relevance in this patient since blood test results indicated a recent response to *Treponema pallidum* infection.¹⁶ Results of the RPR, TPHA and FTA-ABS test in the CSF were negative, however, which, together with the absence of cells and normal total protein concentration, to all intents and purposes excludes¹⁶ syphilitic involvement of the nervous system.

Using the IgG index formula, Tourtellotte *et al.*¹⁷ reported excess CSF IgG in 9% asymptomatic normal individuals ($n = 56$), raising the possibility that the findings in the present case represent a fortuitous example of a normal phenomenon rather than the occurrence of a pathological immunoglobulin fraction. Tourtellotte *et al.*¹⁷ did not state whether an elevated IgG index was a persistent finding in their control subjects, precluding any relevant deductions from the presence of a normal IgG concentration in the later sample of CSF in this patient. At first glance, this normalisation of the CSF over a period of 3 weeks could cast doubts on the veracity of the initial result. However, not only were both readings double checked (Dr L Van Niekerk, personal communication), but also the second lumbar puncture was performed when the patient's chorea had virtually subsided, at which time Husby *et al.*² found that serum antineuronal antibodies had disappeared in cases studied with serial samples. Notwithstanding these circumstances, the observations of Tourtellotte *et al.*¹⁷ together with the constraints pertaining to the study of a single case, make it imprudent to draw inferences on the possible immunopathogenic basis for Sydenham's chorea from the CSF findings reported herein. With respect to further