Neurological manifestations and mycoplasma pneumoniae infection

We report three patients presenting with acute neurological symptoms associated with mycoplasma pneumoniae infection.

Case reports

Case 1—A 31-year-old man developed an influenza-like illness with a cough productive of yellow sputum seven days before hospital admission. He had no obvious spastic paraparesis, extensor plantar responses, sensory level at D4, and retention of urine. Haemoglobin was 14.3 g/dl, white cell count 19.5 x 10^9/l (polymorphs 84%), erythrocyte sedimentation rate (ESR) 12 mm in 1 hour. A chest radiograph showed consolidation of the right upper lobe. A myelogram was normal. Cerebrospinal fluid (CSF) contained 25 polymorphs and 12 lymphocytes/μl and no organisms were seen or cultured. Serum mycoplasma antibody titres were raised (see table). He was treated with tetracycline and physiotherapy. His paraparesis improved gradually and he recovered completely within eight months.

Case 3—A 46-year-old man developed an influenza-like illness with headache and myalgia. Two weeks later he became dysarthric and unsteady on walking with a tendency to fall to his right. On admission he was afebrile and fully conscious but dysarthric and had inco-ordination of both arms and legs with definite ataxia of gait. There was no meningism and examination was otherwise normal. Haemoglobin was 16.9 g/dl, white cell count 7.7 x 10^9/l, ESR 15 mm in 1 hour. A chest radiograph was normal. CSF protein concentration was 0.85 g/l and there was no pleocytosis. Serum mycoplasma antibody titres were raised (see table). No specific treatment was given and his cerebellar signs improved gradually. Six months after the onset he had only slight residual ataxia.

Comment

These three patients presented with acute neurological syndromes, the first affecting the spinal cord, the second the cerebral hemispheres, and the third the cerebellum. Although each had an initial influenza-like illness, there were no other specific clinical features to indicate a primary mycoplasma infection. The neurological complications of mycoplasma pneumoniae include meningoencephalitis, cerebellar syndromes, cranial and spinal polyradiculonucleritis, and transverse myelitis. Few such cases have been reported in Britain and only one with transverse myelitis.

Antecedent or concurrent chest symptoms are not invariable. Nevertheless, a causal relationship between mycoplasma pneumoniae and the neurological complications seems likely either with direct infection of the nervous system or possibly with a secondary immunological reaction. Although death and persistent neurological deficits have been reported, the prognosis is often favourable.

Our experience supports the view that tests for mycoplasma pneumoniae should be included in the investigation of patients.

Mycoplasma antibody titres in the three patients (measured by complement fixation test)

<table>
<thead>
<tr>
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<th>One month later</th>
<th>Four months later</th>
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</thead>
<tbody>
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<td>4096</td>
<td>2048</td>
<td>256</td>
</tr>
<tr>
<td>2</td>
<td>Encephalitis</td>
<td>1080</td>
<td>640</td>
<td>80</td>
</tr>
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Sialochemistry in evaluating bromhexine treatment of Sjögren's syndrome

Frost-Larsen et al1 found that lacrimal gland secretion, measured by the Schirmer test, increased during bromhexine treatment for Sjögren's syndrome (SS). They did not find that bromhexine had any effect on salivary gland function, but their methods of estimating salivary secretion "were crude and of doubtful value." Changes in the composition of saliva are a sensitive indicator of salivary gland disease in SS. Significantly raised concentrations of Na, IgA, and IgG in saliva have been reported.2,3 We therefore decided to study the effect of bromhexine on the quantity and quality of saliva in patients with SS.

Patients, methods, and results

Twenty patients under the age of 60 were divided into two groups. Group 1 (SS group) consisted of five patients with sicca syndrome who had no associated disease and who had been followed up for at least 18 months, and seven patients with Sjögren's syndrome associated only with seropositive rheumatoid arthritis. Group 2 (control group) consisted of eight patients with seropositive rheumatoid arthritis without sicca complex. The criteria for sicca and Sjögren's syndrome were decreased tear flow to less than 5 mm/min by Schirmer's test, staining of the cornea with rose bengal dye, diminished salivary flow, and abnormal salivary composition. All patients with rheumatoid arthritis fulfilled the American Rheumatism Association's criteria for either definite or classical rheumatoid arthritis.

Bromhexine 16 mg three times daily was given for four weeks. The medical treatment remained unchanged during this period. Saliva was collected before and at the end of the course of bromhexine. Total mixed unstimulated saliva was collected for 10 minutes. The rate of flow was measured and the saliva analysed for Na, IgA, and IgG as described.4 Student's t test was applied for statistical analysis. Symptoms were alleviated to a varying degree during bromhexine treatment. They recurred when bromhexine was discontinued and improved when bromhexine was again given. None of the control group had sialectomas or excessive lacrimation. No side effects were recorded even during prolonged treatment. The SS patients had significantly higher initial concentrations of Na, IgA, and IgG when compared with the controls, whose saliva was normal (table). Bromhexine had no effect on salivary composition in the control group, but in the SS group concentrations of Na, IgA, and IgG were significantly reduced towards normal without an increase in salivary flow. Concentrations were not always uniformly reduced in all patients. In a few the concentration of only one component was significantly lower. Four SS patients took bromhexine continuously for months and a further gradual lowering of Na, IgA, and IgG concentrations was noted. Two SS patients took part twice in the trial, with a two-weeks interval between courses of treatment. The concentrations of Na, IgA, and IgG had returned to their original levels after the interval. Further treatment lowered them, as before.

Comment

The change in salivary composition towards normal without a significant increase in salivary flow raises the question whether the clinical improvement with bromhexine treatment could be due to the change in the quality of the saliva. Bromhexine reduces sputum viscosity in chronic bronchitis.4 A reduction in sodium concentration may affect salivary viscosity, which changes with the cationic concentration.2 How bromhexine alters salivary composition in SS is unknown. Since it lowers the concentrations of IgG and IgA perhaps it inhibits the local transformation of B lymphocytes.

We thank Ikapharm-Pharmacare Ltd for supplying the bromhexine (Solvex) tablets.


(Accepted 11 July 1979)

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Effect of PUVA on serum 25-OH vitamin D in psoriasis

The action of ultraviolet radiation (UVR) on 7-dehydrocholesterol in the epidermis is one of the main sources of vitamin D in man. It is important to know whether treatment of psoriasis with PUVA (8-MOP) and ultraviolet light (UV-12) may lead to excessive production of vitamin D and to toxic concentrations in the blood.

Patients, methods, and results

Twenty-five patients with chronic plaque psoriasis were studied. They had never had PUVA treatment and had not recently had UVR. They were irradiated two hours after taking the 8-methoxypsoralen (8-MOP), when the peak concentrations of the drug are believed to occur in blood and skin. Further details of treatment, which was given three times a week till the rash was clear and approximately weekly after that, are described elsewhere.1

<table>
<thead>
<tr>
<th>Rate of flow (ml/min)</th>
<th>Na (mmol/(mg/l))</th>
<th>IgA (mg/l)</th>
<th>IgG (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Control group (n=8)</td>
<td>0.30</td>
<td>0.31</td>
<td>4.6±1.4</td>
</tr>
<tr>
<td>SS group (n=12)</td>
<td>0.08</td>
<td>0.07</td>
<td>22.6±17.1</td>
</tr>
</tbody>
</table>

Statistical analysis

1) Pretreatment control pretreatment SS: Na P<0.01; IgA and IgG P<0.001 (Student's t test).
2) Pretreatment: no significance. B SS group—Na and IgA P<0.001; IgG (n=6) P 0.01; rate of flow, no significance (t test for paired comparison).