the pathogenesis of meningococcal infections, where invasive disease correlates with the lack of meningococcal bactericidal activity² because of the presence of blocking antibody.³ Evidence for a similar phenomenon in the case of the gonococcus is supported by the finding that serum resistance, a characteristic of *N gonorrhoeae* in disseminated disease,⁴ is also related to the presence of blocking antibody.⁵

Our data suggest that the gonococcus may transiently colonise the pharynx without causing complications. Since we cannot distinguish between patients who will spontaneously become culture-negative and those who will develop sequelae, withholding routine treatment does not appear to be warranted. Further studies are needed to define the factors that play a determinant part in the host-gonococcal interaction.

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Enterotoxigenic *Escherichia coli* causing diarrhoea in travellers returning to the United Kingdom

During the past five years enterotoxigenic strains of *Escherichia coli* (ETEC) have been one of the most important causes of acute watery diarrhoea in areas of poor hygiene, notably in the tropics. Infants and young children are particularly at risk but adults may also be infected. Investigations in the United States of America and in Sweden suggest that ETEC are not important causes of sporadic diarrhoea in temperate areas of good hygiene. In a study of 61 children with acute diarrhoea in Boston, USA, no evidence was found of infection with ETEC,¹ and a study of 648 patients with diarrhoea in Sweden² concluded that ETEC were not important causes of diarrhoea. Nevertheless, ETEC are a significant cause of diarrhoea among visitors to tropical areas. Diarrhoea acquired after arrival in Mexico by students from the USA³ was due in 72% of cases to ETEC, and ETEC were found in 11% of travellers who acquired diarrhoea while abroad or shortly after returning to Sweden.² Because of air transport travellers may return home while still in the acute stage of an infectious diarrhoea acquired abroad. We sought to determine the importance of ETEC as a cause of travellers' diarrhoea in patients returning to the United Kingdom.

Methods, materials, and results

We studied 55 epidemiologically unrelated patients who developed diarrhoea during or shortly after travel abroad. Six were under 5 years old, two were between 5 and 14, and 47 were over 15. Fifteen patients had visited the Indian subcontinent, 11 had visited Spain or Portugal, and five had visited North Africa. In all, 21 different countries were represented. A further 50 patients with diarrhoea who had not recently been abroad were studied as a control group. Eleven were under 5 years old, six were between 5 and 14, and 33 were over 15. Sixty-two cultures were examined from each group of patients. All the cultures were identified as *E coli* by biochemical tests and were serotyped with antisera for O groups 1 to 164 and H antigens 1 to 56. The *E coli* strains were tested for the production of heat-stable enterotoxin (ST) by the infant mouse test, and for heat-labile enterotoxin (LT) by the CHO and Y1 tissue culture tests.⁴

ETEC were isolated from six (11%) patients who had recently travelled

abroad and from one (2%) patient with no history of recent travel. The seven ETEC strains belonged to seven different O groups. Three strains produced ST only, two produced LT only, and two produced ST and LT. Six of the patients with ETEC were adults and one was a 9-month-old baby (table).

Serotypes of ETEC in sporadic diarrhoea in 55 travellers and 50 non-travellers in England and Wales

Serotype	Enterotoxin produced	Country visited	Age of patient
O6.H16	ST + LT	Sierra Leone	Adult
O7.H18	LT	India	Adult
O20.H11	ST	Not abroad	Adult
O89.H-	ST	Middle East	Adult
O128.H18	ST	India	9 months
O148.H28	ST + LT	Yugoslavia	Adult
O ? H-	LT	France	Adult

Comment

Although ETEC have caused hospital outbreaks of infantile diarrhoea in Scotland and England,⁵ there is little information about their importance as a cause of sporadic diarrhoea in the community in the UK. Our findings suggest that ETEC are an uncommon cause of indigenous sporadic diarrhoea in the UK but confirm that they may be a significant cause of diarrhoea among travellers returning from abroad. These results closely agree with those of similar studies in Sweden. Laboratory workers investigating travellers' diarrhoea should be aware of the importance of ETEC. If salmonellae and shigellae are not isolated strains of *E coli* should be sent to the reference laboratory for serotyping and enterotoxin testing.

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Possible antigenic similarity between pulmonary carcinoma and cysts of *Echinococcus granulosus*

Antigenic similarity between parasites and some human carcinomas has been shown serologically.¹ The cystic intermediate stage of the cestode *Echinococcus granulosus* results in a chronic infection, known as hydatid disease. The test of choice for the immunodiagnosis of this disease has been the immunoelectrophoresis (IEP) test using as antigen concentrated *E granulosus* cyst fluid from sheep.² A test is considered positive when the patient's serum causes the precipitation of a band called arc 5, which is easily identified. In addition to this specific band, however, other bands may be precipitated by the sera of hydatidosis patients as well as by the sera of patients with other diseases.³ When the serum from a patient suffering from pulmonary carcinoma was tested using *E granulosus* cyst fluid a broad and intense precipitin band resulted.

Case report

A 42-year-old man, a farm worker all his life, was admitted to hospital in January 1978 complaining of tiredness, loss of appetite, persistent cough with occasional haemoptysis, and frequent night sweats. He had smoked 20-30 cigarettes a day for 25 years and had had an episode of renal colic in 1974. The results of clinical and laboratory investigations were normal except for a white cell count of $24.3 \times 10^9/l$ ($24\ 300/mm^3$) and an erythrocyte sedimentation rate of 113 mm in 1st h. The peripheral blood contained 86 %

neutrophils, 5% lymphocytes, 6% monocytes, and 3% eosinophils. Chest radiography showed a mass $6 \times 5 \times 5$ cm occupying the apical segment of the left dorsal lobe of the lung. Liver scan showed a space-occupying mass 5-6 mm in diameter in the posterior-dorsal region of the right lobe near the upper pole of the right kidney. This combination of masses in lung and liver in a farm worker suggested hydatid disease. Within 24 h of incubating a serum sample a broad and intense precipitin band with *E. granulosus* cyst fluid antigen developed in the IEP test where the diagnostic hydatidosis arc 5 is located. After a further 48 h of incubation, however, the edges of this precipitin band failed to join with those of the arc 5 given by the reference positive serum placed in the opposite trough of the same IEP plate (figure).

Immunoelectrophoresis test for hydatidosis after 72 h incubation, using concentrated ovine *Echinococcus granulosus* cyst fluid antigen. Upper trough contained serum from patient with pulmonary carcinoma. Lower trough contained arc 5 positive serum from sheep-infected with *E. granulosus*.

The mass removed by radical left upper lobectomy was a squamous cell carcinoma with a necrotic core. On further laboratory tests, eight weeks after the lung operation and before exploratory surgery to investigate the mass in the liver area, the white blood cell count had dropped to $7.1 \times 10^9/l$ ($7100/mm^3$). The peripheral differential cell count had not altered. The erythrocyte sedimentation rate had also dropped to 26 mm in 1st h. A serum sample taken at this stage did not give any precipitin bands in the IEP test for hydatidosis. The mass was found to be a hard, calcified benign adrenal tumour with no histologically recognisable tissue.

Comment

The cyst fluid of *E. granulosus* contains a number of antigenic molecules which are apparently shared with a variety of parasites.³ Nevertheless, reports on cross-reactions between hydatid antigens and sera from patients with confirmed neoplastic growth are rare.¹ Recently a patient with multiple cysticercosis and myeloma was found to have circulating antibodies against the *E. granulosus* arc 5 antigen.⁴

The precipitin band that we observed did not dissolve in 5% trisodium acetate solution,³ indicating that it was the product of an antigen-antibody reaction. Because of its diffuse nature we could not definitely say that it did not join up with the arc 5 precipitin band in *E. granulosus* cyst fluid precipitated with hydatid-infected sheep serum. We have seen similar diffuse bands produced by serum from hydatid-infected sheep. Circumstantial evidence that the band was not stimulated by an *E. granulosus* infection was that the patient's serum eight weeks after the removal of the pulmonary carcinoma did not precipitate with *E. granulosus* cyst fluid. Since the patient had always worked on farms he had been exposed to hydatid organisms or other taeniid tapeworms, which are endemic in New Zealand. Nevertheless, careful palpation of the liver and lungs during surgery and further organ and bone scans revealed no other lesions. Therefore the antigens released by the pulmonary squamous cell carcinoma are apparently similar to antigens in hydatid cyst fluid.

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Penicillamine-induced neuromyotonia

Treatment of rheumatoid arthritis with penicillamine¹ has been associated with various unwanted effects,² including polymyositis and myasthenia gravis. We have recently encountered an additional neuromuscular complication of penicillamine treatment—neuromyotonia.

Case report

A 61-year-old woman developed seropositive, erosive rheumatoid arthritis in 1966. She was treated with analgesics and anti-inflammatory agents. In 1974 her arthritis became more active and penicillamine 750 mg daily was started. In 1975 her platelet count fell slightly and the penicillamine was reduced to 500 mg daily. Since then her rheumatoid arthritis has remained quiescent. For two months she had complained of feelings like "electric shocks" in the arms and legs with a "pulling" feeling in her muscles. She also noted some tingling round her mouth and had difficulty in opening her eyes. When attempting to move, her hands or toes would curl and she would momentarily be unable to grip objects. She was seen on one occasion to become rigid on standing.

On examination, she had difficulty in relaxing muscles after contraction. For example, her fingers after clenching remained flexed for up to 30 seconds before the forearm flexors suddenly relaxed. Relaxation could be induced by gently extending the fingers. She had similar prolonged contraction in her periorbital facial muscles with difficulty in opening her eyes after blinking. This was mistaken at first for ptosis. Extraocular movements were normal. There was no myasthenic weakness or percussion myotonia; sensory examination was normal; ankle jerks were diminished; joint deformities were only slight. Concentric needle electromyography (EMG) in the small hand muscles, biceps, and deltoids showed prominent spontaneous discharges (figure). These consisted of one or more motor units firing repetitively at

EMG: spontaneous firing of three motor units. Calibration: 100 μ V and 10 ms.

10 to 30/s. Electrode movement induced bursts of motor unit activity lasting for between 21 and 26 seconds. After brief voluntary muscular contraction similar activity was seen, continuing for the same time. Rare pseudomyotonic discharges were seen. Motor unit action potentials during voluntary activity were normal, and the interference pattern was full. Single fibre EMG showed no jitter or blocking, and the fibre density was normal. Nerve conduction velocity measurements were normal. The evoked response to repetitive stimulation of the ulnar nerve at the wrist, recording from the abductor digiti minimi, showed no decrement of the 4th potential at 2 Hz, but at 10 Hz and 20 Hz there was a 25% decrement. A needle biopsy of the right quadriceps muscle was normal; the blood creatine kinase concentration was also normal.

Neuromyotonia was diagnosed and phenytoin 300 mg daily was given. The patient noted improvement within three days. EMG studies six weeks later showed much reduction in the neuromyotonia. The penicillamine was stopped and the neuromyotonia disappeared. It did not recur when phenytoin was also discontinued.

Comment

The neuromyotonia in this patient was associated with penicillamine. There was no EMG evidence of neuropathy, although neuromyotonia has been described in patients with both congenital and acquired neuropathies.³ The pathogenesis of neuromyotonia is uncertain. It is probably due to spontaneous firing in the distal part of the axonal tree,