immediately after prostatic biopsy in case an unusual organism gets into the blood stream. By the time illness is apparent sensitivities might be available. Also the urine should be cultured about one week after the procedure to exclude persistent urinary tract infection.

We are engaged in a further trial using the same antibiotic cover but with preliminary cleansing of the bowel with a phosphate enema followed by an aqueous solution of providone-iodine applied through a proctoscope. To date none of the blood samples taken five minutes after biopsy have been positive (six patients), and time may tell whether it is sufficient to use just this local prophylaxis.

We thank Professor C H Lack for helpful advice, Dr B Subagio for help in the laboratory, Mr R P M Miles and Mr W F P Gammie for allowing us to study their patients, and the Chichester Ladies’ Circle for financial support.

References

(Accepted 29 September 1978)

## SHORT REPORTS

### Unusual chlamydial infection in a human renal allograft recipient

Immunocompromised patients commonly experience repeated infections due to a wide spectrum of opportunist organisms. We report here an infection with a chlamydial agent that produced a near-fatal illness in a renal transplant recipient.

#### Case report

A 38-year-old housewife received a cadaveric renal transplant in October 1975. Graft function remained excellent and no rejection episodes occurred. Her only problems since transplant had been tertiary hyperparathyroidism, recurrent bacteriuria, and a chronic ringworm infection caused by *Microsporum canis* contracted from her family of cats. She was admitted on 11 November 1977 with a 10-day history of malaise and lethargy. She was pyrexial and the spleen tip was palpable, but examination otherwise showed nothing abnormal. During the next two weeks her condition deteriorated steadily. She became hyperpyrexial and developed hepatosplenomegaly and an unproductive cough. Liver enzyme concentrations rose shortly after admission. The serum aspartate transaminase (serum AST; SGOT) concentration reached a peak of 103 IU/l on 17 November, and the creatinine clearance fell from a preadmission 70 ml/min to 21 ml/min on 21 November. Azathioprine was discontinued on 11 November because of leukopenia. All bacteriological investigations gave negative results. Acid-fast bacilli were not found in gastric washings and chest radiographs remained normal. Serial titres of complement-fixing antibodies against multiple microbiological agents were stable. The Paul-Bunnell and hepatitis B surface antigen tests gave negative results. Bone marrow trephine and liver biopsy sections both contained foci of necrosis with inflammatory cell infiltration. A provisional diagnosis of disseminated tuberculous infection was made. On 16 November she was started on rifampicin 600 mg and isoniazid 300 mg daily, corticosteroids were stopped, and normal human immunoglobulin was given. By 21 November she was semicomatose and profoundly ill. Because of rising liver enzyme concentrations rifampicin was discontinued and streptomycin substituted. After her condition had further deteriorated corticosteroids were reintroduced on 24 November. The next day she began gradually to recover.

The results of chlamydial serology are shown in the table. Serum taken on 16 November was fractionated by Sephadex G-200, and 52% of the complement-fixing activity resided in the IgM fraction. Subsequently the titre of complement-fixing antibodies rose while that of specific IgM antibodies declined. Clearly the patient had had a primary infection with a chlamydial agent, but we were reluctant to diagnose psittacosis without evidence of lung infection. Furthermore, her only contact with birds had been at her local pet shop, and these birds showed no evidence of psittacosis. Serum samples were therefore examined by the microimmunofluorescence technique for antibodies against a variety of subgroup A (*C trachomatis*) and subgroup B (*C psittaci*) chlamydial agents. Among the significant rises in antibody titres the most striking was against isolate 457-F, a feline keratoconjunctivitis agent of subgroup B that can cause feline pneumonitis. This was the only *Chlamydia* against which high-titre specific IgM was detected in the patient’s sera. Her three cats were vencesected in February 1978, but none had antibodies against the feline keratoconjunctivitis agent. Serum from one (‘*Sooty’*), however, showed an immunofluorescence titre of 128 against a subgroup A serotype. This cat had been ill with a cough at the end of October 1977.

#### Comment

The patient’s most striking antibody rise was against the feline keratoconjunctivitis agent, but this *Chlamydia* has been associated only with conjunctivitis in humans. Nevertheless, possibly the patient’s

### Results of serological tests for Chlamydia antigens

<table>
<thead>
<tr>
<th>Tests and chlamydial antigens used</th>
<th>Antibody titres (%, IgM) of sera on dates shown</th>
<th>Patient’s sera</th>
<th>Cats’ sera (1/2/78)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8/11/77</td>
<td>16/11/77</td>
<td>1/12/77</td>
</tr>
<tr>
<td>Complement fixation:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Pioltosisis/LGV</em>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microimmuno fluorescence:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup A strains</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A *</td>
<td>&lt;8</td>
<td>32 (52)</td>
<td>1024 (17)</td>
</tr>
<tr>
<td>B *</td>
<td>&lt;8</td>
<td>128 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>G *</td>
<td>&lt;8</td>
<td>128 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>LGV L, H, and III *</td>
<td>&lt;8</td>
<td>512 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>Subgroup B strains</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3L **</td>
<td>&lt;8</td>
<td>128 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>3L-90 **</td>
<td>&lt;8</td>
<td>128 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>3L-13 **</td>
<td>&lt;8</td>
<td>128 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>457-F **</td>
<td>&lt;8</td>
<td>128 (0)</td>
<td>ND</td>
</tr>
<tr>
<td>457-F **</td>
<td>&lt;8</td>
<td>64 (25)</td>
<td>4096 (0)</td>
</tr>
</tbody>
</table>

ND = Not done.
* IgM fraction anticomplementary.
* = Human *C psittaci* strain. ** = Human *C psittaci* strain. *** = Pigeon *C psittaci* strain. **** = Enzooric abortion agent of ewes. ****** = Cat keratoconjunctivitis agent (feline pneumonitis).
immunosuppressive treatment allowed such an infection to become disseminated. From the start of her illness the patient was thought to have contracted an infection from one of her cats. She admitted to close contact with them and her previous ringworm was further evidence of this. But the puzzling fact remains that Sooty’s antibody profile was suggestive of a subgroup A infection while the patient’s antibody response suggested infection with a subgroup B Chlamydia.

Because of the multiple treatment we cannot attribute the patient’s recovery to any one drug. Rifampicin, however, is active against C trachomatis infection of the eye and so may have been beneficial.

1 Spencer, E S, Danish Medical Bulletin, 1975, 22, 234.

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Antenatal diagnosis of fetal duodenal atresia by ultrasonic scan

Fetal malformations of the central nervous system can be detected early by ultrasonic examination. Gastrointestinal lesions, however, many of which may require immediate surgical correction after delivery, are more difficult to find.

Case history

A healthy 26-year-old woman whose first pregnancy had been uncomplicated booked at 14 weeks and attended the antenatal clinic regularly. The uterine size was consistently two weeks too large for her dates. Ultrasound scan at 19 weeks and six days verified the given dates. Other findings were normal. At 30 weeks there was apparent polyhydramnios: the uterus was tense and four weeks too large for dates. X-ray examination at 33 weeks showed no fetal abnormality. At 34 weeks the patient was admitted in early premature labour. Intrauterine roridae was given to decrease uterine activity and betamethasone to reduce the possibility of neonatal respiratory distress.

A further ultrasonic scan four days after admission showed a biparietal measurement of 93.0 mm—again consistent with the patient’s dates. The fetal head and spine were normal. On taking cross-sections of the upper fetal abdomen, however, double fluid-filled structures were clearly visible (see figure). Measurement of the total internal uterine volume (6942 cm³) confirmed the apparent polyhydramnios. Slight uterine activity persisted over the next two weeks. It increased at 36 weeks. The cervix was found to be 5-cm dilated. Labour was allowed to proceed, amniotomy was performed, and six hours later a 2150 g female infant was delivered normally. A nasogastric tube was passed immediately and a large amount of bile-stained fluid aspirated. A clinical diagnosis of Down’s syndrome was subsequently confirmed by chromosomal analysis. Erect x-ray examination of the infant at 12 hours showed the “double-bubble” shadow in her abdomen, confirming duodenal atresia. The parents refused surgical treatment. The baby died at 8 days. Permission for necropsy was not given.

Comment

Polyhydramnios, in the absence of a maternal cause, may be associated with fetal abnormality in as many as half the cases. Lloyd and Clatworthy found 43%, abnormal fetuses in their series of 76 pregnancies complicated by polyhydramnios. High small-bowel obstruction, proximal to the ligament of Treitz, was associated with polyhydramnios in 47%, of 49 pregnancies. Obstruction distal to the ligament of Treitz did not appear to give rise to excess liquor. Fonkalsrud et al in a review of 503 infants with congenital duodenal atresia or stenosis found additional congenital malformations in 48%. Polyhydramnios was present in 45% of these cases and prematurity or dysmaturity (defined as a birth weight below 2500 g) in 51%. Down’s syndrome was present in 30%.


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Serum 2-hydroxybutyrate dehydrogenase activity and ineffective erythropoiesis

High levels of plasma lactate dehydrogenase activity are commonly found in patients with megaloblastic anaemia. Imperfect erythroblast maturation in this disorder results in the destruction of vast numbers of developing red cells rich in the anionic lactate dehydrogenase (LDH) isoenzymes LDH1 and LDH2. This is believed to be the cause of the