cultures of the cerebrospinal fluid and blood grew a β-haemolytic streptococcus not identifiable as a member of Lancefield group A, B, C, D, or G. Sensitivity plates subsequently showed the organism to be fully sensitive in vitro to both the drugs administered. Within 48 hours of this treatment the patient's fever had resolved, and after six days of gradual improvement he regained consciousness. As soon as he was well enough to concentrate, however, he complained of tinnitus, dizziness, and deafness in both ears. Intravenous antibiotics were stopped after 10 days and phenoxymethylpenicillin, 1·5 g four times daily, with probenecid, 500 mg three times daily, were given for a further five weeks. The patient was discharged after three weeks in hospital; since then he has suffered no recurrence of meningitis but remains disabled by dizziness, unsteadiness of gait, and deafness of his right ear.

The division of hospital infection (streptococcal reference) at the Public Health Service Laboratory, Colindale, identified his isolates from blood cultures and cerebrospinal fluid as *Streptococcus* group R (*Str suis* type II).

Comment

Group R streptococci were identified as a cause of septicaemic infections of pigs in 1959,[1] and nine years later three cases of human infection, two of meningitis and one of fatal septicaemia, were caused by this organism in Denmark.[2] In 1975 Zanen and Engel described ten cases of human meningitis due to group R streptococci which had occurred since 1968 in Holland[3]; of these, nine worked in the meat trade with pigs or pig carcasses (the other being a housewife), and of nine who recovered on antibiotic treatment five suffered permanent deafness and vertigo.

The first case of meningitis due to group R streptococci in England and Wales was recorded in a pork-pie factory worker in 1976,[4] and this was followed by a further case in a man with the same occupation[5]; both patients suffered residual deafness and vertigo. Wright (unpublished observations, Communicable Diseases Surveillance Centre) has described two more cases of infection occurring in pork handlers in Britain and has drawn attention to the almost invariable occupational association between humans handling pigs or pork products and group R streptococcal infections.

The case I have described highlights the occupational association with pigs in this rare disease, and in view of the disabling sequelae recorded in most cases, suggests that group R streptococcal infection in humans should be classed as an industrial disease; this could enable those who are incapacitated by the sequelae to benefit from industrial compensation.

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Strongyloides stercoralis infection in renal transplant recipients

Strongyloides stercoralis infection is endemic in tropical and subtropical regions. A host may remain infected but asymptomatic with this nematode for more than three decades.[1] Overwhelming infection with *S stercoralis* may occur in immunosuppressed hosts,[2] including renal transplant recipients.[3 4] We report two such infections in Guyanese recipients of transplants. At the time these patients developed symptoms of *S stercoralis* infection the immunosuppressive regimen for acute rejection in this unit was as follows: an increase in oral prednisolone to 200 mg daily, reducing by 50 mg steps every five days until the dose was 50 mg daily, after which it was reduced slowly to a minimum dose of 15 mg daily. Azathioprine was used at maximum tolerated dose. From our experience we now recommend prophylactic preoperative treatment against *S stercoralis* in transplant recipients who come from, or who have lived in, tropical or subtropical regions.

Case reports

Case 1—A 30-year-old man visited Guyana seven years before he received a cadaveric transplant. On the eighth postoperative day acute rejection occurred and he was treated successfully. Renal function deteriorated again, and on day 63 he received two single doses of 1 g methylprednisolone. On day 81 he developed dyspnoea, cyanosis, and haemoptysis, with widespread crepitations in the lungs. Chest radiography showed an alveolar infiltrate initially interpreted as pulmonary oedema. Larvae of *S stercoralis* were found in sputum and gastric aspirate. There was no eosinophilia. Thiabendazole 25 mg/kg body weight for 14 days cleared the larvae from his sputum and he made a good recovery. Creatinine clearance was 79 ml/minute. On day 157 he was readmitted with fever and apparent acute rejection. He was treated with 2 g methylprednisolone and prednisolone 100 mg daily for five days. Graft nephrectomy was performed on day 170 because of cortical necrosis. Immunosuppression was withdrawn. On day 175 he became severely dyspnoeic with massive haemoptysis. Chest radiography showed prominent alveolar infiltrate similar to the previous episode. *S stercoralis* larvae were again recovered from his sputum and gastric aspirate. Thiabendazole was started, but he died on day 179. Histological examination showed a single larva lying within an alveolus.

Case 2—A 37-year-old man with a history of pulmonary sarcoidosis had last visited Guyana 15 years before he received a kidney transplant. After operation he remained oliguric and on the fifth day was treated for presumed acute rejection. On day 30 he developed a cough with white frothy sputum, and a chest radiograph showed residual sarcoid changes. Sputum contained larvae of *S stercoralis*. There was no eosinophilia. He was treated with thiabendazole 25 mg/kg twice daily for 10 days and then prophylactically with three-day courses of thiabendazole monthly for six months. He had remained well four and a half years later.

Comment

Clinical infection with *S stercoralis* was closely related to high-dose prednisolone treatment in both patients. Respiratory distress mimicking pulmonary oedema is a common presentation.[2 3 4] Eosinophilia was not present in either patient before or after transplantation. Eosinophilia may be absent in infected individuals or as a result of therapeutic immunosuppression.[4] The first patient showed that conventional doses of thiabendazole in an immunosuppressed patient are insufficient to eradicate the nematode. The correct duration of treatment is arbitrary, but should be monitored by examination of concentrated stool samples for larvae.

S stercoralis infection in a transplant recipient is usually diagnosed when respiratory failure has developed and is fatal.[3 4] We now recommend prophylactic treatment of transplant recipients who come from endemic areas even if routine investigation does not show chronic infection. We adopted this policy in a third Guyanese patient, who developed no features suggestive of *S stercoralis* infection 12 months after transplantation, though larvae were never recovered from his stools.

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