Letters to the Editor

Listeria monocytogenes peritonitis during continuous ambulatory peritoneal dialysis (CAPD)

Sir,

Listeria monocytogenes has become increasingly recognized as an important cause of sepsis during the perinatal period, and also of opportunistic infections in adults. According to Seeliger¹ the usual site of entry in man is the intestine via the oral route. We report here a case of *Listeria* peritonitis in a CAPD patient. Although this species is a recognized cause of sepsis in renal transplant and haemodialysis patients,^{2,3} this is only the second documented case of CAPD peritonitis.

The patient was a 53 year old man with Wegener's granulomatosis and chronic renal failure, receiving treatment with cyclophosphamide (25 mg/day). He had been on CAPD for 6 months when he came to the Renal Dialysis Unit complaining of lower abdominal pain and cloudy bags. His peritoneal dialysis (PD) fluid showed 800 white cells/mm³ with 40% polymorphonuclear leucocytes, 45% mononuclear cells and 15% degenerated cells. Microscopical examination of the dialysis sediment showed no organisms. However, culture of the dialysate fluid in Bactec bottles (Bactec NR 660 system) for 24 hours yielded Gram-positive rods subsequently identified as *Listeria monocytogenes*.

Initially the patient was treated empirically with vancomycin and aztreonam for 48 hours. Although the organism was sensitive to vancomycin the PD fluid remained cloudy and *L. monocytogenes* was again isolated 48 hours after the start of treatment. Therefore, the antibiotics were stopped and treatment with ampicillin 50 mg/l in every bag and oral pivampicillin 500 mg b.d. was started. The infection responded within 72 hours, and therapy was continued for 3 weeks as is our policy.

Because of the unusual nature of the infecting organism, an environmental investigation was done. The patient admitted dropping the connector site of his line on the carpet, which constituted a violation in aseptic technique. Under these circumstances the patient should have contacted the Renal Dialysis Unit immediately, as is the policy of that unit, where the external catheter would be changed and a single prophylactic dose of antibiotic given. Swabs were taken from the patient's mouth, throat, nose, PD catheter insertion site and the carpet. A stool specimen was also taken. He also mentioned that he had eaten cottage cheese the day before the onset of his peritonitis, although he said that he had washed and disinfected his hands prior to the exchange. In view of the association of *L. monocytogenes* and soft cheeses, the remains of the cheese (which had been stored at 4°C) was also sent to our microbiology department. *L. monocytogenes* was not isolated from any of the above specimens.

This is the second documented case of *L. monocytogenes* peritonitis in a CAPD patient⁴. In contrast to the previous reported case the organism was not isolated from the blood.

This patient was immunocompromised due to his renal condition and treatment with cyclophosphamide, and

this may have made him more susceptible to opportunistic infection by *L. monocytogenes*.

In several previous cases of infections of the PD fluid by unusual organisms we have been able to trace the source of infection^{5,6} but here it was not possible.

L. monocytogenes may sometimes be confused with diphtheroids, which should not be regarded as 'contaminants' if isolated:⁴ we have found *Corynebacterium* spp. (including a strain belonging to the JK group) causing CAPD peritonitis in 3 patients over the past 14 months.

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Pericarditis due to *Salmonella dublin*

Sir,

I would like to report a case of bacterial pericarditis caused by a combination of *Salmonella dublin* and *Morganella morganii*. This rare complication of salmonella food poisoning assumes greater importance because of the increasing frequency of this infection in the community and the large population of immunosuppressed patients who may be susceptible.

A 52 year old woman with multiple myeloma was treated with three courses of chemotherapy including adriamycin, BCNU, melphalan and cyclophosphamide. Response was initially good; however, 5 months after diagnosis she developed renal failure. Haemodialysis was performed through a subclavian line whilst a surgical fistula matured. During this period she presented with clinical features suggestive of pericarditis which was

ascribed to her uraemia. Treatment was commenced with sulindac.

Several days later she was admitted with fever and diarrhoea. Stool and blood cultures grew *Salmonella dublin* which was treated with a 3-week course of ampicillin. The source appeared to be precooked turkey bought in a local shop. A good clinical recovery was made. One month later, however, the patient re-presented with chest pain and dyspnoea. Pericardial tamponade was suspected and emergency pericardiocentesis was performed releasing 500 ml of pus. Culture of this revealed growth of *Salmonella dublin* and *Morganella morganii* which was treated with intravenous antibiotics. The blood film revealed a recurrence of her myeloma and despite therapy death occurred several days later.

Non-typhoidal salmonella pericarditis is very rare, with less than 25 cases reported in the literature. This is the first case reported due to *Salmonella dublin* which is endemic in cattle causing acute enteritis and spontaneous abortion. Human isolations are uncommon, but in one third of cases this organism was grown from the blood,¹ making this a relatively invasive organism with commensurate ability for causing serious extra-intestinal disease.

Our patient had a number of features predisposing to infection including renal failure, multiple myeloma and previous immunosuppressive therapy. Presumably the area of pericardial inflammation due to uraemia was a fertile nidus for infection during the bacteraemic phase. The failure of ampicillin to eradicate the organism in spite of *in vitro* sensitivity, may have been due to her immunosuppression although relapse after treatment is well-known, even in healthy individuals. A longer course of antibiotic treatment with the addition of a second agent such as co-trimoxazole or chloramphenicol may be desirable.

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Primary abdominal actinomycosis – an unusual presentation

Sir,
We report a case of primary actinomycosis that presented in a unique manner that suggested a rectus sheath haematoma.

A 61 year old man presented with a 3-week history of increasingly severe right sided abdominal pain. It began after an episode of heavy lifting, was worse on coughing and moving and was not relieved by a 7 day course of erythromycin. On examination he had a pyrexia of 37.5°C, with a tender fluctuant swelling overlying the right rectus abdominus muscle. A diagnosis of rectus sheath haematoma was made but incision of the anterior rectus sheath revealed a large cavity containing foul smelling pus. A small yellow granule was seen and this

was sent with the pus for immediate microbiological examination. The posterior rectus sheath and peritoneum were intact. The cavity was cleaned and a corrugated drain inserted. Microscopy of the 'sulphur granule' and pus revealed Gram-positive branching bacilli but no growth of actinomyces was obtained on culture. The patient was started on penicillin and was discharged from hospital after 5 days. A barium meal, follow-through and enema were all normal, confirming the diagnosis of primary actinomycosis. He remained well and was discharged from follow up after 3 months treatment.

Actinomycosis is an infectious disease, usually caused by *Actinomyces israelii*, a Gram-positive branching anaerobic bacterium. Abdominal actinomycosis has a predilection for the right iliac fossa and occurs following appendectomy or abscess drainage. When the intestinal mucosal barrier is destroyed by disease or trauma the actinomyces can penetrate into adjacent tissue and become pathogenic.¹ The resulting pus contains 'sulphur granules', which are hard, gritty, yellow granules 2 mm in diameter. Each represents a mycelial mass cemented together by host calcium phosphate in response to inflammation. Treatment is by surgical drainage followed by a prolonged course of high-dose penicillin. Primary actinomycosis of the abdominal wall is extremely rare.² Its occurrence in the absence of an associated lesion in the gastro-intestinal tract is unexplained.³ In the few reported cases it has presented as a chronic disease mimicking an intra-abdominal malignancy.⁴ Actinomyces was not cultured in this case, but any antibiotic treatment is likely to suppress growth sufficiently to render cultures negative.⁵

We can find no previous report of primary abdominal actinomycosis presenting with acute abdominal pain.

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Intestinal spirochaetosis in HIV infected homosexual men

Sir,
Digestive tract infection is frequent in human immunodeficiency virus (HIV) seropositive subjects and numerous microorganisms have been implicated. Intestinal spirochaetosis should probably be added to the list.

We recently diagnosed intestinal spirochaetosis in two