of retinitis detection, and the immuno-suppression was tapering and graft nephrectomy was planned.

In conclusion, we suggest the retinitis may be due to severe antigen–antibody complex vasculitis, which showed in the graft biopsy, but CMV reactivation after strong immunosuppression such as OKT3 monoclonal antibody therapy cannot be excluded.

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The role of surgery and itraconazole in Aspergillus peritonitis in CAPD

Sir,

In their Case Report and review of literature, Tanis et al. [1] found only 13 cases of peritonitis which may be attributed to Aspergillus. In only five of these there were more than one positive culture and in only one were the classical hyphae seen. Two also recommend catheter removal and antifungal agents. Nearly all patients had amphotericin B and only one had itraconazole as well. Recently we had an experience to suggest that catheter removal and amphotericin may result in inadequate treatment in a case of microscopy- and culture-confirmed Aspergillus peritonitis, and that surgical intervention in combination with prolonged use of itraconazole may be required.

The patient, a 37-year-old male presented with fever and generalized abdominal tenderness in June 1995. Before this visit, he had had a cough which was treated with antibiotics. He first presented in end-stage renal failure of unknown cause and was commenced on CAPD in January 1994. He was apyrexial with diffusely tender abdomen with rebound tenderness. Exit site and tunnel were normal. PD fluid was turbid with 920 w.b.c./µl (90% neutrophils). Gram-positive cocci were seen and the patient was treated with cephalizin and gentamicin i.p., which was changed on day 7 to vancomycin and amikacin i.p. because of lack of improvement. Previous dialysate cultures were negative. On day 13, the CAPD catheter was removed because of persisting symptoms and cloudy dialysate. Postoperatively the patient developed septicaemic shock, requiring the addition of dopamine, cefazidime and clexacin intravenously. On day 18, PD fluid culture sent on day 13 showed Aspergillus niger, although the catheter tip sent for routine culture was negative. As the patient was well, no treatment was given.

By day 22, the patient was noted to have increasing ascites. Aspiration revealed a cloudy fluid with 2000 polymorphs/µl, staining revealed typical Aspergillus hyphae, which was confirmed on three subsequent samples. Amphotericin B (30 mg/day) was given intravenously. A repeat aspiration of the abdomen on day 50, revealed a persistently cloudy fluid with neutrophils, although fungal stains and cultures were negative. Abdominal ultrasound revealed multiple loculated collections. A surgical referral for open drainage was made. At laparotomy, the bowel was seen to be coated with fibrous capsule, and a large collection containing fibrin and turbid fluid was drained.

By day 65, after completing a cumulative dose of 1.5 g of amphotericin, the patient was well. As a small collection was seen on ultrasound, he was started on itraconazole 300 mg twice daily and discharged. The collection persisted and was noted to have increased in size to 10 × 10 cm. On day 120, straw-coloured fluid was aspirated, revealing 80 lymphocytes/µl, protein 51 g/l on examination, and was negative on culture. The patient was well on haemodialysis with no abdominal symptoms and an ESR of 38 mm/h. Itraconazole dose was reduced to 200 mg twice daily and a follow-up ultrasound arranged for 1 month.

It is reasonable to assume that the diagnosis is correct as hyphae were seen in several samples and Aspergillus confirmed on culture. Repeated bacterial and mycobacterial cultures were negative. The patient was not immuno-suppressed and the only risk factor appeared to be antibiotic usage. It is not certain whether Aspergillus is the primary cause of peritonitis or whether this developed after weeks on antibiotics. The source of the infection was also not clear, though this was not investigated thoroughly. After catheter removal, despite treatment with adequate dose and duration of amphotericin, the patient still required open drainage. The infection appears to induce a strong fibrous reaction, leading to formation of loculated collection which resulted in both poor drug accessibility and failure to return to CAPD. In retrospect, it cannot be certain whether surgery could have been avoided by earlier catheter removal and initiation of antifungal therapy.

As smaller collections persisted, long-term antifungal therapy was necessary. The role of itraconazole in treatment of fungal peritonitis is at present unclear. Certainly it is an effective oral agent against systemic aspergillosis [2] and has relatively few side-effects [3]. It is, however, poorly soluble in water and is not detected in the peritoneal fluid following oral administration [4]. On the other hand it is effective in cryptococcal meningitis despite low measurable concentrations in the CSF, an effect explained by high meningeal concentration of the drug [3]. Although high levels of itraconazole have been measured in the omentum [5], there is scant data at present to support its use in fungal peritonitis. In this patient, after 60 days of treatment, he was well and fluid aspirated was clear, suggesting a possible role for itraconazole in the treatment of aspergillus peritonitis. Further studies may be useful to define its role in peritonitis due to fluconazole-resistant Candida species and Aspergillus.

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Influence of methodological differences on microbiological diagnosis of CAPD peritonitis

Sir,

Peritonitis is still the most important complication of continuous ambulatory peritoneal dialysis (CAPD) [1]. Different