


Conflict of interest statement. None declared.

**Table 1.** Laboratory findings on 20 HD patients after 6 months of high-dose (25 mg) Fol supplementation

<table>
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<th>Basal</th>
<th>6 months</th>
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<tr>
<td>Fol (ng/ml)</td>
<td>4 ± 3</td>
<td>24 ± 0*</td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>11.5 ± 1.67</td>
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<tr>
<td>rHu-EPO (i.v. IU/week)</td>
<td>8950 ± 6645</td>
<td>12 550 ± 14 687</td>
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*P < 0.01 vs Basal.

Advance Access publication 27 February 2006

High-dose folic acid supplements and responsiveness to rHu-EPO in HD patients

Sir,

Schiffl and Lang [1] demonstrated that high-dose supplements of folic acid in elderly maintenance haemodialysis (HD) patients with normocytic anaemia have no effect on rHu-EPO (recombinant human erythropoietin) responsiveness. We can show data on 20 HD patients without macrocytic anaemia (F = 5, M = 15; age 74 ± 13 years; dialysis age 93 ± 95 months) supplemented with high-dose calcium levofolinate (Fol). Fol (25 mg) was administrated orally to all 20 HD patients at the end of each HD session for 6 months. All patients received weekly thrice HD using synthetic high-flux membranes, always reaching a Kt/v > 1.2. Active bleeding, haemolysis or myeloproliferative disease was never observed during the follow-up. Data on Fol, haemoglobin (Hb) plasma levels and weekly i.v. rHu-EPO dosage are summarized in Table 1 as mean ± SD. Our results confirm the data published by Schiffl and Lang [1], where high-dose Fol supplements do not influence the response to rHu-EPO in normocytic HD patients.

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Advance Access publication 13 February 2006

**Fatal Candida famata peritonitis complicating sclerosing peritonitis in a peritoneal dialysis patient**

Sir,

Fungi are rare causes of secondary peritonitis [1]. Most of these are caused by Candida species although other yeasts and dimorphic fungi may be isolated in some cases. We recently came across one such case of sclerosing peritonitis with superimposed Candida famata infection.

A 35-year-old male with a failed renal transplant, on continuous ambulatory peritoneal dialysis (CAPD) since 1997, developed Staphylococcal peritonitis while resident in South Africa. This was successfully treated with a course of vancomycin. However, 6 weeks later, he again manifested with signs of CAPD peritonitis. *Candida famata* was isolated from the peritoneal fluid. A CT scan revealed a loculated fluid collection lying anteriorly within the abdomen, containing several pockets of gas as well as a moderately thick capsule suggestive of infected sclerosing peritonitis. The above findings were confirmed on laparotomy for removal of the tenckhoff catheter. The patient was started on intravenous fluconazole with intraperitoneal amphotericin, which was later converted to intravenous voriconazole. A relaparotomy was done to free the encased bowel. Further laparotomies were done to evacuate blood clots and lavage. The patient also received intraperitoneal tauroline washouts during this period. However, he failed to respond to therapy and subsequently died.

Sclerosing peritonitis is an unusual form of peritonitis. This disease was first described in 1974 following oral use of beta blockers, especially practolol [2,3]. In 1983 sclerosing peritonitis was first described in a CAPD patient [4]. Other causes include luteinized thecoma, chlorhexidine washout, keratoconjunctivitis sicca and peritoneal sarcoidosis. Chronic intestinal obstruction with profound weight loss or abdominal mass is the most common presentation. Other manifestations include haemoperitoneum and peritonitis. Peritonitis has been reported to occur in 38% of cases, with fungal peritonitis in 7% [5]. The development of bacterial or fungal peritonitis may bring the disease to light earlier, as in our case. Most cases of fungal peritonitis are caused by *Candida* (50–85%) with the majority being caused by *Candida albicans*. Other yeasts implicated include Cryptococcus, Trichosporon and Rhodotorula species. Dimorphic fungi causing peritonitis include Aspergillus, Penicillium and Paecilomyces. Management strategies include prompt diagnosis and removal of the dialysis catheter with administration of systemic antifungals.
Candida famata is an uncommon yeast. Previously called Torulopsis famata and Debaryomyces hansenii, the yeast is found in many dairy products like cheese. It is an opportunistic pathogen that is commensal in the oral cavity. The fungus has been implicated in sporadic case reports as causing onychomycosis, systemic blastomycosis, extrinsic allergic alveolitis, systemic fungaemia and endophthalmitis. Candida famata has been very rarely isolated in the culture of peritoneal fluid in peritonitis. The first and only documented case report in existing literature was reported in 1994. The yeast is increasingly isolated from patients and was found in 1.45% of urinary tract infections and in about 1–2% of patients with fungaemia [6]. Recently we reported a case of mediastinitis with Candida famata [7].

Rigby and Hawley [5], while reporting the Australian experience, noted that in most patients in whom sclerosing peritonitis was complicated by peritonitis, bowel function did not recover and the patient usually died of ongoing sepsis. This was exactly our experience, in that all efforts at treatment failed and the patient eventually succumbed to his illness.

To conclude, sclerosing peritonitis complicated by fungal peritonitis is a serious complication. Newer strains of candida are being implicated. Candida famata is currently emerging as a significant pathogen in humans.

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Renal/liver transplant Unit  
Freeman Hospital  
Newcastle upon Tyne  
United Kingdom  
Email: Ajay.Gupta@ncl.ac.uk


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Resumption of peritoneal dialysis after transcutaneous treatment of a peritoneal leakage using fibrin glue

Sir,

A common cause of technique failure of peritoneal dialysis (PD) are defects in the integrity of the peritoneal membrane [1,2]. Evidence-based guidelines for the management of PD-associated leakages are not available. Here, we report a case of dialysate leakage into the abdominal wall successfully managed with fibrin glue.

A 39-year-old woman with end-stage renal disease (ESRD) and a history of primary phospholipid antibody syndrome was admitted for initiation of continuous aubulatory peritoneal dialysis (CAPD). Surgical re-replacement of mitral valve prosthesis was performed in February 2005 due to endocarditis. She developed ESRD post-operatively. The PD-catheter was inserted by laparoscopy. Four weeks after hospital discharge she was readmitted with a painful swelling in the inferior right abdominal part. Her body weight had increased during the previous 4 days, accompanied by reduced ultrafiltration.

The swelling of the abdominal wall persisted after dialysate removal. Ultrasound examination revealed a massive abdominal wall oedema and a defect in the parietal peritoneal membrane (Figure 1). Loss of integrity occurred in the region of the surgical scar. The clinical presentation and ultrasonographic findings led to the diagnosis of a peritoneal leakage in the abdominal wall. PD was stopped and haemodialysis was initiated. The dialysis solution was completely absorbed within 1 week, but the size of the peritoneal defect remained unchanged (Figure 2A).