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

Sir,

Schiff and Lang [1] demonstrated that high-dose supplements of folic acid in elderly maintenance haemodialysis patients without macrocytic 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 thrice weekly haemodialysis 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 Schiff and Lang [1], where high-dose Fol supplements do not influence the response to rHu-EPO in normocytic HD patients.

Conflict of interest statement. None declared.

<table>
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<tr>
<th>Table 1. Laboratory findings on 20 HD patients after 6 months of high-dose (25mg) Fol supplementation</th>
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<td><strong>Basal</strong></td>
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<tr>
<td>Fol (ng/ml)</td>
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<td>Hb (g/dl)</td>
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<td>rHu-EPO (i.v. IU/week)</td>
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*P < 0.01 vs Basal.
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.

Conflict of interest statement. None declared.

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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 ambulatory peritoneal dialysis (CAPD). Surgical re-replacement of mitral valveprosthesis was performed in February 2005 due to endocarditis. She developed ESRD post-operatively.

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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).

Fig. 1. Ultrasonographic investigation of the abdominal wall in the region of the abdominal swelling showed a diffuse oedema starting from a defect in the peritoneum parietale. The discontinuation of the peritoneal membrane was directly located under one scar of the peritoneal catheter insertion. The central vertical hypodensity corresponded to the laparoscopic recess channel.