Table 1. Solutes' reduction rate during the three treatments

Variable	RR iHDF *vs* OL-HDF (%)	*P*	RR iHDF *vs* HFR (%)	*P*
Urea	79.5 ± 1.1 *vs* 80.4 ± 0.1	NS	81.3 ± 1.2 *vs* 80.5 ± 0.2	NS
Kt/V	1.59 ± 0.06 *vs* 1.63 ± 0.01	NS	1.68 ± 0.01 *vs* 1.64 ± 0.06	NS
Phosphate	59.9 ± 14.5 *vs* 58.7 ± 7.3	NS	72.1 ± 5.8 *vs* 62.9 ± 5.3	NS
Beta-2 MG	75.3 ± 1.3 *vs* 79.1 ± 0.5	<0.05	73.3 ± 1.9 *vs* 38.2 ± 1.1	<0.01
Hcy	47.1 ± 2.8 *vs* 39.4 ± 2.3	<0.05	53.1 ± 3.7 *vs* 47.8 ± 12.2	NS

Hcy reduction rate was high and about 50% both during iHDF and HFR, while it was lower in post-dilutional OL-HDF (~40%). Table 1 shows the most remarkable results.

This trial shows for the first time that iHDF is able to remove higher doses of total plasma hcy than OL-HDF with polyamide membrane. Moreover, this high hcy lowering capacity is equal to HFR which represents, together with superflux membranes, the dialysis technique with larger hcy removal. All types of solutes are well-removed by this new high-flux polysulfone membrane. Thus, we think that iHDF is not only a cost-effective alternative to other mixed techniques, but it could increase the rate of diffusive-convective treatments in dialysis units. Naturally, it will be necessary to perform prospective studies about its long-term efficacy.

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Antioxidant treatment in dialysis patients—importance of ferritin

Sir,

Cardiovascular disease remains one of the leading causes of mortality in haemodialysis (HD) patients. Abnormal oxidative stress and impaired antioxidant defence may contribute to accelerated atherogenesis associated with uraemia. I read with interest the article by Fumeron *et al*. [1], reporting that short-term oral vitamin C supplementation did not modify well-defined oxidative/antioxidative stress and inflammation markers in HD patients. As stated, they did not evaluate the effect of long-term intravenous vitamin C supplementation with lower or higher doses on oxidative stress, nor the efficacy of co-administration of vitamin E.

Iron is a powerful oxidizing substance. Ferritin may have other functions in addition to its well-described role in storing intracellular iron. Elevated ferritin levels are associated with an increased risk of atherosclerotic coronary artery disease and myocardial infarction. Recent proteomics and molecular biology studies have shown that ferritin levels in arteries are increased in diseased tissues [2]. Ferritin may be unregulated under particular physiological conditions and may act as a pro-oxidant. In the mammalian cell, iron stored in ferritin can participate in initiating lipid peroxidation [3].

High levels of serum ferritin may indicate iron overload. However, in many patients results may be elevated due to inflammation, even if iron stores are not increased. Limited evidence shows that elevated iron stores and high-dose intravenous iron therapy may increase morbidity and mortality in HD patients [4], and exacerbate increased oxidative stress in uraemic patients [5].

The upper limit of serum ferritin that has a potential for oxidative tissue damage related to increased iron storage or to intravenous iron treatment itself is unclear [6]. Reddi *et al*. [7] did not find a difference either in antioxidant enzymes, antioxidants or lipid peroxidation between dialysis patients with normal (<325 ng/ml) or higher than normal (>325 ng/ml) serum ferritin levels. On the contrary, in another study [8], malondialdehyde levels of dialysis patients with the highest ferritin levels (657–1251 μg/l) were significantly greater than those of the other two groups with lower ferritin levels (296–556 μg/l and 559–804 μg/l). In addition, this study showed that elevated serum ferritin levels may affect the levels of these lipophilic antioxidants. However, there is no study comparing malnutrition, inflammation, oxidative stress markers and atherosclerosis status in HD patients with high or low ferritin levels in detail.

Perhaps, Fumeron *et al*. [1] could divide the subjects into two groups with low and high ferritin levels by determining a cutoff ferritin value. Whether baseline oxidative/antioxidative stress parameters differ in these groups and change with oral 250 mg vitamin C supplementation in the vitamin C group could be evaluated. Thus, this study may provide an approach to the strategies of antioxidant treatment in the dialysis population.

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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.

Conflict of interest statement. None declared.

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

	Basal	6 months
Fol (ng/ml)	4 ± 3	24 ± 0*
Hb (g/dl)	11.5 ± 1.67	11.3 ± 1.38*
rHu-EPO (i.v. IU/week)	8950 ± 6645	12 550 ± 14 687

*$P < 0.01$ *vs* Basal.

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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 vericonazole. 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, keratinoconjunctivitis 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 *Paecelomyces*. Management strategies include prompt diagnosis and removal of the dialysis catheter with administration of systemic antifungals.