Letters to the Editor

Daily hemodialysis and nutritional status

To the Editor: A switch from conventional hemodialysis (CHD) to daily hemodialysis (DHD) has been found to improve nutritional status of hemodialysis patients [1]. This observation by Galland et al needed to be confirmed. We, too, evaluated the effect of a switch from CHD (3 × 4 hours/week) to DHD (6 × 2 hours/week), using bioelectrical impedance analysis (BIA); we also observed a dramatic improvement in patients’ lean body mass.

Fourteen volunteer patients were evaluated during 3 months on CHD and 12 months on DHD. The lean body mass was evaluated at −3, 0, +6 and +12 months, respectively. Five patients were excluded (renal transplantation in the first 6 months (N = 4), incomplete data (N = 1); data from the nine patients with 6 months DHD follow-up (six men, three women; 61 years old, range: 30 to 76 years), on CHD (five at home, three in a low-care unit, one in hospital) for 63 months (range: 8 to 137 months) was analysed by ANOVA for repeated measures with F or Friedman tests, as appropriate.

The lean body mass (mean ± SEM) increased (+1.3 ± 0.4 kg) during the first 6 months (P < 0.008) of DHD, while total body weight remained unchanged. After 6 months, two more patients were withdrawn (renal transplantation and aortic valve replacement, respectively). The lean body mass evolution of the remaining seven patients is presented (Fig. 1).

Based on these observations, a switch to DHD could be proposed to CHD patients with poor nutritional status. Further studies, including a larger number of patients, are required to confirm these encouraging results.

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Moderate hyperhomocysteinemia and oxidative stress

To the Editor: In uremic patients, hyperhomocysteinemia was found to be unrelated to measurements of oxidative stress [1]. The data imply that hyperhomocysteinemia is no primary risk factor for vascular lesions and thrombotic events in chronic renal failure.

Moderate hyperhomocysteinemia is considered to develop when dietary supplementation of folate is insufficient. Accordingly, folate supplementation can easily correct hyperhomocysteinemia, and an inverse relationship between homocysteine and folate concentrations usually exists, which was also observed in the study by Mezzano et al [1]. Hyperhomocysteinemia often coincides with elevated neopterin levels, which indicate immune activation and oxidative stress emerging from activated macrophages [2], and, in patients with uremia, increased neopterin concentrations are associated with protein oxidation products [3]. Also in the study of Mezzano et al, oxidation products correlated with markers of immune activation [1]. 5,6,7,8-Tetrahydrofolic acid, the biologically active cofactor, is very susceptible to oxidation, which may become relevant under oxidative stress conditions. Immune system-derived oxidative stress seems to be crucial for folate depletion, resulting in hyperhomocysteinemia even when dietary folate is within the recommended range [2]. In coronary artery disease, folate supplementation was found to improve endothelial function earlier than changes of homocysteine concentrations became apparent [4], and also the data of Mezzano et al support the view that hyperhomocysteinemia is an indirect consequence of hyperconsumption of antioxidant vitamins during prolonged states of immune activation. Then, hyperhomocysteinemia is not primary in the pathogenesis of vascular lesions and thrombotic events. Nevertheless, hyperhomocysteinemia still could contribute to accelerate the underlying pathogenic process.

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