Dear Sir,

We read with much interest the paper by Sethi et al. [1], reporting a significant decrease in serum ß2-microglobulin (sß2M) levels in 6 patients changed from hemodialysis (HD) on cuprophan membranes to continuous ambulatory peritoneal dialysis (CAPD). Since the pathogenesis of dialysis-associated amyloidosis is presumably related to the long-term persistence of raised ß2M levels in the extracellular fluids, the authors suggested that CAPD might be protective against this complication.

Using a quite different study design, we came to rather similar conclusions [2]. We compared sß2M levels at a 1-year interval in 25 CAPD patients and 25 patients hemodialyzed with cuprophan membranes. Patients were matched for duration on dialysis and residual clearance of creatinine (RCCr), since the incidence of dialysis-associated amyloidosis is known to increase with time on HD [3], and since sß2M levels increase with declining renal function [4]. The mean duration (± SEM) on dialysis was 40.2 ± 4.9 months (range: 8–95) in HD and 39.4 ± 4.9 months (range: 9–96) in CAPD; the RCCr (± SEM) was 1.2 ± 0.3 (range: 0–5.6) in HD and 1.3 ± 0.3 (0–7.0) in CAPD. As shown in table 1, sß2M levels increased with time, both in CAPD (p < 0.05) and in HD patients (p < 0.05), while RCCr decreased significantly (CAPD: p < 0.05; HD: p < 0.01). Nevertheless, sß2M levels remained lower throughout the study in the patients on CAPD. In both HD and CAPD groups sß2M levels correlated negatively with RCCr (HD: p < 0.05; CAPD: p < 0.01); however, the influence of residual renal function was much greater in HD patients, as demonstrated by comparison of the regression lines of sß2M levels as a function of RCCr (slope: F = 4.77; p < 0.05; height: F = 8.82; p < 0.01). In CAPD, the ß2M daily peritoneal output averaged 38 mg – ranging from 16 to 59 mg – and was directly correlated with sß2M levels (p < 0.01).

Table 1. Evolution with time of sß2M (mg/l) in HD and CAPD
Our study thus indicates that sβ2M levels increase when residual renal function decreases, both in HD and CAPD patients. These findings are in agreement with the other data in the literature [3]. Furthermore, the influence of RCCr was greater in HD than in CAPD patients; this is probably due to the significant peritoneal removal of β2M in CAPD patients, which progressively takes over from renal function, while there is no significant clearance of β2M by cuprophan membranes [5].

In contrast with the data of Ballardie et al. [5], our results are in agreement with those of Sethi et al. [1], showing that sβ2M levels are lower in CAPD than in HD patients. The discrepancies between these studies can be explained by the fact that our patients – but not those of Ballardie et al. – were matched for time on dialysis and RCCr. The peritoneal clearance of β2M remains obviously insufficient, however, and sβ2M levels increase with time on CAPD. Thus, if β2M amyloidosis is related to increased sβ2M levels, the apparition of this complication should simply be delayed in CAPD as compared to HD patients. Interestingly, we have recently observed 1 CAPD patient who developed a carpal tunnel syndrome with histologically demonstrated β2M amyloidosis [6].

CAPD, Protective against Developing Dialysis-Associated Amyloid?

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