Continuous Ambulatory Peritoneal Dialysis, Protective against Developing Dialysis-Associated Amyloid?

C.		Tielemans ^a
M.		Dratwa ^a
P.		Bergmann ^a
M.		Goldman ^b
B.		Flamion ^a
F.		Collart ^a
R.		Wens ^a

^aDepartment of Medicine, Hôpital Universitaire Brugmann and ^bDepartment of Nephrology, Dialysis, and Transplantation, Hôpital Erasme, Université Libre de Bruxelles, Brussels, Belgium

Dr. C. Tielemans, Hôpital Universitaire Brugmann, Place Van Gehuchten 4, B-1020 Bruxelles (Belgium)

Dear Sir,

We read with much interest the paper by Sethi et al. [1], reporting a significant decrease in serum β_2 -microglobulin ($s\beta_2M$) levels in 6 patients changed from hemodialysis (HD) on cuprophane membranes to continuous ambulatory peritoneal dialysis (CAPD). Since the pathogenesis of dialysis-associated amyloidosis is presumably related to the long-term persistence of raised β_2M 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 $s\beta_2M$ levels at a 1-year interval in 25 CAPD patients and 25 patients hemodialyzed with cuprophane 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 $s\beta_2M$ levels increase with declining renal function [4]. The mean duration ($\pm$ 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 $RCCr$ ($\pm$ 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, $s\beta_2M$ 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, $s\beta_2M$ levels remained lower throughout the study in the patients on CAPD. In both HD and CAPD groups $s\beta_2M$ 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 $s\beta_2M$ levels as a function of $RCCr$ (slope: $F = 4.77$; $p < 0.05$; height: $F = 8.82$; $p < 0.01$). In CAPD, the β_2M daily peritoneal output averaged 38 mg – ranging from 16 to 59 mg – and was directly correlated with $s\beta_2M$ levels ($p < 0.01$).

Table 1. Evolution with time of $s\beta_2M$ (mg/l) in HD and CAPD

Our study thus indicates that $\text{s}\beta_2\text{M}$ 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 $\beta_2\text{M}$ in CAPD patients, which progressively takes over from renal function, while there is no significant clearance of $\beta_2\text{M}$ by cuprophane 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 $\text{s}\beta_2\text{M}$ 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 $\beta_2\text{M}$ remains obviously insufficient, however, and $\text{s}\beta_2\text{M}$ levels increase with time on CAPD. Thus, if $\beta_2\text{M}$ amyloidosis is related to increased $\text{s}\beta_2\text{M}$ 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 $\beta_2\text{M}$ amyloidosis [6].

CAPD, Protective against Developing Dialysis-Associated Amyloid?

175

References

Sethi, D.; Brown, E.A.; Gower, P.E.: CAPD, protective against developing dialysis-associated amyloid? *Nephron* 50: 85–86 (1988).

Tielemans, C; Dratwa, M.; Bergman, P.; et al.: Continuous ambulatory peritoneal dialysis vs haemodialysis: a lesser risk of amyloidosis? *Nephrol. Dial. Transplant. J.* 291–294 (1988).

Spertini, F.; Wauters, J.-P.; Poulenas, I.: Carpal tunnel syndrome, a frequent invalidating long-term complication of chronic haemodialysis. *Clin. Nephrol.* 27:95–101 (1984).

Vincent, C; Revillard, J.-P.; Galland, M.; Traeger, J.: Serum beta-2-microglobulin in hemodialyzed patients. *Nephron* 21: 260–268 (1978).

Ballardie, F.W.; Kerr, D.N.S.; Tennent, G.; Pepys, M.B.: Hemo-dialysis versus CAPD: equal predisposition to amyloidosis? *Lancet* i: 795–796 (1986).

6 Dratwa

M.; Telerman

N.; Goffin

Y.; Van Hoof

I.; Demey

A.: Beta-2-microglobulin amyloidosis in a CAPD patient; in Khanna

Nolph

Prowant

Twardowski

Oreopoulos

Advances in continuous ambulatory peritoneal dialysis. *Perit. Dial. Bull.*

vol. 4

pp. 73–78 (Toronto