Dear Sir,

Recent reports suggest that raised levels of \( \beta_2 \)-micro-globulin (\( \beta_2 \)M) in long-term haemodialysis patients lead to the deposition of dialysis-associated amyloid (DAA) [1]. Amyloid derived from this precursor molecule is of importance because of its implication in the development of dialysis arthropathy, recurrent carpal tunnel syndrome and bone amyloidosis [2, 3]. However, the question as to whether patients on continuous ambulatory peritoneal dialysis (CAPD) have an equal predisposition to developing DAA remains unanswered. Although there have been reports which suggest that elevated levels of \( \beta_2 \)M in CAPD patients may predispose them to developing DAA [4], histological confirmation of this has not yet been published. In this letter we present results which suggest that CAPD may be protective against this form of amyloidosis.

Between March 1986 and January 1987, 6 patients on maintenance haemodialysis using cuprophane membranes have changed to CAPD because of problems with vascular access. Their mean length of time on haemodialysis was 6 years (range 1.5–13). Samples for \( \beta_2 \)M were taken whilst they were on haemodialysis and then after they had been established on CAPD for a mean of 3 months. Samples were taken before dialysis whilst patients were undergoing haemodialysis. Laboratory measurement was with the Pharmacia \( \beta_2 \)M radioimmunoas-say kit.

There was a significant difference in \( \beta_2 \)M concentration between the two treatment modalities (\( p < 0.02 \), paired t test) and all 6 patients demonstrated a lowering in the concentration of \( \beta_2 \)M. The mean (± SD) \( \beta_2 \)M level in the patients whilst on haemodialysis was 59.4±17.2 mg/l, and after changing to CAPD, 35.5±4.5 mg/l (normal range 1–3 mg/l).

These results demonstrate a significant lowering in \( \beta_2 \)M concentration during treatment with CAPD in patients who have previously been on maintenance haemodialysis. This suggests that CAPD is less likely to result in elevation of serum \( \beta_2 \)M levels. It has been shown that the in vitro formation of amyloid fibrils from intact \( \beta_2 \)M is dependent upon increasing the concentration of \( \beta_2 \)M [5]. If a comparable process can occur in vivo then amyloidogenesis in dialysis patients would be dependent upon exceeding a threshold concentration of \( \beta_2 \)M. Should this be the case then CAPD patients would be less likely to develop DAA. This assertion is supported by the fact that there have been no reports in the literature of the development of DAA in CAPD patients [6].

2 of these 6 patients also had dialysis arthropathy at rheumatological assessment. On conversion to CAPD they both showed a significant improvement in severity of pain and range of movement of affected joints. The possible relationship between a fall in \( \beta_2 \)M level and relief of shoulder
pain has been previously reported after changing dialyser membrane from cuprophane to polysulfone [7]. In this report we demonstrate a similar relationship between clinical improvement and the lowering of β2M levels after changing to CAPD. However, other factors such as the bio-incompatibility-related inflammatory reactions which occur with cuprophane membranes [8, 9] but not with CAPD need to be taken into account.

References


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