Amyloidosis in continuous ambulatory peritoneal dialysis

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Summary: We report a 53 year old man with chronic renal failure on continuous ambulatory peritoneal dialysis. Following eight episodes of severe peritonitis over a 2 year period, he died and was found to have widespread AA amyloid at post-mortem.

Introduction

Amyloidosis is an important factor in determining a patient's long-term prognosis. Systemic amyloidosis is frequently fatal and while an effective treatment for amyloidosis has not yet been developed, control of the predisposing condition in secondary amyloidosis may lead to remission or prevention. Amyloidosis has not previously been reported as a sequel of recurrent peritonitis in patients on continuous ambulatory peritoneal dialysis (CAPD). This observation has important long-term implications for patients with recurrent peritoneal infections.

Case report

A 53 year old man presented in July 1982 with a 7-month history of increasing dyspnoea. He was hypertensive, blood pressure 190/110 mm Hg and had cardiomegaly. Initial investigations were as follows: serum creatinine 840 μmol/l, serum urea 45.7 mmol/l, haemoglobin 7.7 g/dl. His urine was sterile and acellular with a trace of protein. Intravenous urography demonstrated small (8 cm) unobstructed kidneys. Despite control of his blood pressure his renal function deteriorated and he was commenced on CAPD in June 1983. Over the next two years he had eight episodes of peritonitis. In August 1985 following a loss of peritoneal membrane ultrafiltration, the peritoneal catheter was removed and he was started on haemodialysis. Two months later, he developed a further episode of peritonitis and died.

At autopsy, left ventricular hypertrophy, extensive coronary atherosclerosis and a recent small subendocardial infarct were found. The peritoneal cavity was obliterated by extensive fibrous adhesions and collections of pus were noted between the loops of small bowel. Both kidneys were small with multiple cysts of varying size. Microscopy demonstrated perivascular deposits of amyloid in the heart, liver, thyroid, adrenal, parathyroids, pancreas and kidneys. Global sclerosis of most of the glomeruli was present, with chronic tubular damage and interstitial fibrosis. Perivascular and interstitial amyloid was seen principally in the medulla (Figure 1). No amyloid was detected in any of the glomeruli. The potassium permanganate reaction of Wright was consistent with AA amyloid. Staining of the sections with specific sheep antiserum to human B2-microglobulin (IDRL, University of Birmingham) was negative.

Figure 1 A micrograph of post-mortem kidney showing profuse medullary amyloid deposition. Congo red stain (original magnification × 1000).

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Discussion

Amyloidosis is an infrequent but important complication of long term haemodialysis and seems to be related to the overall duration of therapy. It has recently been shown that the amyloid fibrils in these patients are homologous to β₂-microglobulin. This complication is more frequently encountered in those patients exposed to cuprophane rather than polyacrylonitrile membranes.

This is unlikely to be the mechanism in our patient as he was exposed to a cuprophane membrane during dialysis for only two months and the histological sections did not demonstrate β₂-microglobulin using specific peroxidase-conjugated antisera. In our patient the development of extensive systemic deposits of amyloid of the AA type was almost certainly a consequence of recurrent peritonitis. Amyloid of the AA type is a well recognized complication of chronic infections, but has hitherto not been described as a result of recurrent peritonitis in patients on CAPD.

The absence of glomerular amyloid deposits in association with small kidneys makes it unlikely that the original renal disease was due to amyloid.

Our observations suggest that some patients on CAPD who develop recurrent peritonitis may be at risk of developing secondary amyloidosis.

References