Massive proteinuria in a patient with chronic pyelonephritis

Sir,

In patients with chronic pyelonephritis (CPN), protein loss is usually less than 1 g/24 h. Proteinuria exceeding 5 g/day [1] has been observed to occur especially in patients with CPN and vesico-ureteral reflux [2,3], coexistent diabetic nephropathy [4] or primary glomerulonephritis [5]. We present a patient with unilateral CPN complicated by severe proteinuria resulting from transudation of tissue fluid out of the chronically infected kidney.

A 65-year-old Caucasian female had been treated 3 weeks before admission for urinary tract infection. She was known to have left-sided congenital duplication of the pelvi-caliceal system, complicated by renal stone formation during adolescence. During admission she had recurrent bouts of fever. The latter ultimately was explained by CPN. Laboratory data revealed ESR 115 mm/h, Hb 5.8 mmol/l, MCV 90 fl, leukocyte count 7.2 × 10^9//l with a normal differential count. Serum creatinine level was 105 μmol/l, CPK 7 mmol/l and albumin 20 g/l. Urinalysis manifested leukocytes >20/hpf and a non-selective proteinuria of 4.5–7 g/day. Creatinine clearance was 64 ml/min. Serum-protein-electrophoresis revealed an albumin fraction of 34.8%, α-1-fraction 7.5%, α-2-fraction 18.3%, β-fraction 15.2% and γ-fraction 24.2%. Urine-protein-electrophoresis revealed an albumin fraction of 30.7%, α-1-fraction 7.9%, α-2-fraction 10.4%, β-fraction 8.0% and γ-fraction 43.0% (albumin:globulin ratio of 1.43 and 0.71 in serum and urine respectively). Urine cultures yielded mixed bacterial growth, namely β-haemolytic streptococcus >10^5 CFU/ml and Staphylococcus aureus >10^5 CFU/ml, which were considered colonization.

To exclude an underlying primary renal disease, a right kidney biopsy was performed. On light microscopy less than 1% of the glomeruli were sclerosed, the rest being completely normal. Immuno-fluorescence and electron microscopy findings were also normal. Gallium scan revealed accumulation of tracer in the left kidney, with a functional excretory capacity of 20% on the renogram.

We concluded that our patient had CPN complicated by nephrotic range proteinuria based on transudation of tissue fluid out of the persistently infected kidney. Left-sided nephrectomy was performed, sclerosis was found in less than 4% and the remaining glomeruli were normal. The patient had full clinical recovery and proteinuria fell to 0.5 g/day within 5 days after surgery. Two years later, proteinuria had remained low and her renal function was stable (Figure 1).

Based on macroscopic and histological findings, we concluded that our patient had nephrotic range proteinuria, most probably secondary to transudation of proteins from an intractable infected kidney rather than primary glomerular or interstitial disease.

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