Bilateral renal embolization as a therapy in proteinuria

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Received 27 January 1993; Revision received 24 March 1993; Accepted 27 May 1994

Introduction

The nephrotic syndrome is associated with protein loss from the kidneys, hypoalbuminaemia and hypercholes-
terinaemia. It can be the end-stage result of different types of diseases including all kinds of glomerulonephri-
tis. Metabolic diseases like diabetes mellitus or primary amyloidosis as well as so-called secondary amyloidosis caused by chronic inflammation can also result in proteinuria. For example, tuberculosis, osteomyelitis, ulcerative colitis and Crohn’s ileitis can cause secondary amyloidosis of the kidneys. In these cases secondary amyloidosis is an unspecific reaction of the kidneys to a chronic irritation, but the deposits of amyloid are not reversible. Infrequent causes for nephrotic syndrome are involvement of the kidneys in collagenosis, infection, drug reactions and multiple myeloma [1-3].

The first principle in treatment of the nephrotic syndrome is the attempt to cure or stop the underlying disease, but even if the underlying disease is stopped the renal amyloidosis remains. Therefore, the substitution of pathologically lost substances is evident to keep the oncotic pressure and immune resistance in balance. This is generally performed with oral and parenteral replacement of protein loss combined with salt restriction and specific drug therapy, which is usually a combination of steroids and immunosuppressives [2]. The concept is to make the basal membrane impervious to molecules weighing over 80,000 D. The physiological diuresis of protein is 150 mg/day and the blood albumin > 35 g/L. If the protein loss is below 5 g/day mild symptoms such as fever or orthostasis occur. If the protein loss is more than 5 g/day severe symptoms such as general oedema accompanied by lung or brain oedema as well as thrombosis caused by the loss of antithrombin III can occur. The loss of plasma proteins weakens the humoral infect barrier of the patient. A protein loss exceeding 20 g/day is extremely difficult to substitute with albumin perfusion for cardiovascular overload problems. Additionally, the patient has to bear a 24-h infusion daily. However, if this therapy fails and/or the underlying disease gets out of control an irreplaceable protein loss with a dramatic immune deficiency syndrome can result. In these cases a bilateral renal embolization via a percutaneous transfemoral approach can be performed as a last resort.

Patients

Case 1

R.S. was a 40-year-old man with previously diagnosed membrano-proliferative glomerulonephritis. He developed a nephrotic syndrome with daily loss of up to 40 g protein. The blood albumin was 11.2 g/L. The patient suffered weakness and showed general oedema combined with hypotension of 90/50 mmHg. His main problems were recurrent infections such as suppurative pneumonia associated with loss of immunoglobulins. In this bad
Case 2

H.S., a 53-year-old man, was referred to the hospital with end-stage renal failure and a tumor of the neck. Examination showed oedema of the legs and a soft 100 x 60-mm right submandibular tumour. Abnormal findings were a blood pressure of 80/60 mm Hg, a proteinuria of 19.3 g/L and a blood albumin of 12.8 g/L. The patient was treated with albumin perfusor (40 g/day) and the albumin pool could be improved to 18–22 g/L. Biopsies of the neck tumour and the kidneys showed lymph node tuberculosis and systemic amyloidosis, which produced progressive renal failure requiring haemodialysis. In this situation renal ablation could only improve the patient’s situation.

Case 3

A.A. was a 28-year-old man with a history of familial Mediterranean fever for 8 years, followed by secondary amyloidosis with splenomegaly and nephrotic syndrome. Upon examination he had extensive peripheral oedema and a blood pressure of 180/120 mm Hg, despite treatment with a combination of four antihypertensive drugs. Proteinuria of 12.3 g/L and a blood albumin of 16.9 g/L was present. Albumin infusion worsened the hypertension and did not replace the protein blood pool adequately. Therefore, renal embolization was performed.

In all cases angiography followed by bilateral renal embolization was performed in Seldinger technique via a femoral approach. After initial aortic angiography using a pigtail catheter a selective end-hole catheter (cobra configuration 5 F; Terumo, Frankfurt, Germany; Ödmann configuration 5 F; Cook, Mönchengladbach, Germany) with an inner diameter of 0.038" was inserted into the arteries and Gianturco coils (Cook, Mönchengladbach, Germany) were directed to the origin of the segmental branches of the renal arteries until blood stream stopped. Subsequent digital subtraction angiography (DSA) was obtained to confirm successful embolization. The number of coils used in the different arteries varied from two large and one small coil to three large, two medium and three small-sized coils.

Results

After embolectomy diuresis vanished in the first and third patient. The second patient was already on haemodialysis. Antibiotic therapy had to be performed in the first patient, whereas in the other two no clinical or laboratory signs of infection were present. Albumin levels were stable at 24 g/L in the second and 32 g/L in the third patient without substitution. The first patient was septic before procedure and died 14 days later in general sepsis. At autopsy multiple microabscseses were found in the lungs, liver and spleen. The kidney showed complete infarction, but no kidney abscesses were found.