Aortic Thrombosis Complicating the Nephrotic Syndrome

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Dear Sir,

A wide variety of complications can occur in patients with nephrotic syndrome. These include acute renal failure, thrombosis, infections and hyperlipidaemia [1]. Thrombosis occurs particularly in patients with membranous nephropathy, but is also seen in renal amyloidosis [2]. Whereas in adults the majority of thromboses are venous, arterial thromboses are more common in children, affecting almost every artery in the body including, in one reported case, the aorta [3, 4]. We present here a second case of thrombosis of the abdominal aorta which complicated amyloidosis-induced nephrotic syndrome in an 18-year-old girl with severe juvenile-onset rheumatoid arthritis.

Case Report

An 18-year-old university student presented with severe nephrotic syndrome. Nine years earlier juvenile-onset rheumatoid arthritis had been diagnosed which had been treated with corticosteroids. Her medications on admission comprised prednisone 5 mg per day, frusemide 40 mg on alternate days and piroxicam 10 mg twice daily. The patient was markedly cushingoid in appearance from past high-dose steroids. There was severe arthropathy involving the hands, wrists, neck, hips, knees and ankles. Anasarca was present with massive pitting oedema extending to the abdominal wall, bilateral pleural effusions and ascites. Serum sodium was 141 mmol/l (141 mEq/l), serum potassium 3.7 mmol/l (3.7 mEq/l), serum chloride 103 mmol/l (103 mEq/l), serum bicarbonate 26 mmol/l (26 mEq/l), blood urea 9.1 mmol/l (25.1 mg/dl) and serum creatinine 94 μ mol/l (1.06 mg/dl). Serum albumin was 25 g/l (2.5 g/dl) and globulin 28 g/l (2.8 g/dl). Plasma cholesterol was 21.4 mmol/l [823 mg/dl normal range: 3.5–6.5 mmol/l (135–250 mg/dl)]. Haemoglobin was 118 g/l (11.8 g/dl), haematocrit 37.5%, white cell count $77 \times 10^9/l$ and platelet count $386 \times 10^9/l$. The international normalized ratio (INR) for prothrombin time was normal at 1.0, and the activated partial thromboplastin time was 30 s (normal range: 25–35 s). Urinary protein excretion was 8.4 g/24 h. The ESR was markedly elevated at 121 mm in the first hour.

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Fig. 1. Translumbar aortogram showing complete occlusion of the infrarenal aorta.

One week following admission, a percutaneous renal biopsy was performed which showed extensive renal amyloidosis.

Twelve days after admission, diuretic therapy was intensified to frusemide 80 mg twice daily, chlorthalidone 50 mg twice daily and amiloride 10 mg daily, for resistant oedema. Eighteen days

after admission, the patient became severely ill, and developed pain and absent pulses in both lower limbs. The haematocrit at this time had

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increased to 43.3%. A translumbar aortogram revealed complete occlusion of the infrarenal abdominal aorta (fig. 1). Urgent surgical intervention was arranged. Extensive thrombotic occlusions of the abdominal aorta and both iliac arteries were found and cleared. Histological examination showed fresh, laminated, platelet thrombi without areas of organisation.

The patient made an uneventful post-operative recovery. She was commenced on long-term oral anticoagulation. Unfortunately, her previously normal renal function began to deteriorate steadily, and chronic haemodialysis was instituted 10 months later. Following an unsuccessful renal transplantation in October 1986, a second cadaveric renal transplantation was carried out in November 1987, 3 years after commencement of dialysis. She presently enjoys satisfactory renal transplant function (serum creatinine 118 $\mu\text{mol/l}$; 1.33 mg/dl). Immunosuppressive therapy comprises prednisone 9 mg and ciclosporin a 7 mg/kg daily. She continues on warfarin, maintaining an INR of 1.5–2.0 times control.

Discussion

Thrombosis in nephrotic syndrome is probably due to a variety of factors. It is believed that disturbances of platelet function may account for a major part of the thrombotic tendency [5].

Arterial thrombi, as in this case, are composed mainly of platelets.

As far as we are aware, this is only the second report in the literature of aortic thrombosis complicating nephrotic syndrome. In our patient, thrombosis coincided with the period of maximum diuretic therapy. Presumably haemoconcentration, as evidenced by the rise in haematocrit, was the primary inciting event for thrombosis.

Whereas it is generally accepted that the nephrotic patients most at risk for thrombosis are those with a serum albumin of less than 20 g/l (2.0 g/dl) [1], it is clear

that thrombosis may also occur at higher albumin concentrations, as in this patient. There may be an indication for the use of prophylactic anticoagulation in patients with severe proteinuria and oedema, despite albumin levels greater than 20 g/l (2.0 g/dl). In addition, judicious prescription of diuretics may help to avoid the occurrence of this potentially devastating complication in patients with nephrotic syndrome.

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Announcement

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