Horseshoe Kidney, Focal and Sclerosing Glomerulonephritis and Primary Hypothyroidism

C.		Abson
M.		Jones
A.		Palmer
M.		Persey
R.		Gabriel

St. Mary's Hospital, London, UK

Dr. Roger Gabriel, St. Mary's Hospital, GB-London W2 1NY (UK)

Dear Sir,

We were interested to read the report of Chen and Wang-Shen [1] of a horseshoe kidney complicated by the development of a membranous glomerulonephropathy. There is a hypothyroid patient currently on long-term follow-up at this hospital who has focal and sclerosing glomerulonephritis in a horseshoe kidney. We believe these associations have not previously been reported.

A 52-year-old 80-kg man was referred in January 1987 with peripheral oedema for approximately 6 months. There was no past history referable to the urinary tract. In 1983 a diagnosis of primary hypothyroidism had been made [free T4 61 nM/l (4.7 µg/100 ml); T4 63 nM/l (4.9 µg/100 ml); TSH 28.5 MU/l, normal < 6.5, with positive thyroglobulin and microsomal antibodies]. Physical examination at the time of referral showed a standing diastolic blood pressure of 100 mm Hg together with moderate peripheral oedema. Urine microscopy showed multiple granular casts and erythrocytes. There was proteinuria of 7.7–14.4 g/day. Serum albumin was 24 g/l (2.4 g/100 ml), serum creatinine 129 µM/l (1.5 mg/100 ml), estimated clearance 70 ml/min/70 kg [2], serum cholesterol 13.5 mM/l (523 mg/100 ml) (normal 4.5–6.5; 174–252), serum triglyceride 4.35 mM/l (385 mg/100 ml) (normal 0.60–2.10; 53–186); LDL and VLDL evaluation indicated a type II B phenotype; the concentrations of serum immunoglobulins were normal; serum contained circulating immune complexes containing Clq, IgG and IgA; DNA binding was 10% (normal range < 30%); activities of haemolytic complement, C4 and C3, were both within the normal range. An intravenous urogram showed a horseshoe kidney. Intravenous digital subtraction arteri-ography demonstrated a single artery supplying the right moiety of the horseshoe kidney and two renal arteries to the left. A percutaneous renal biopsy of the right moiety was performed without difficulty under ultrasound control. By light and immunoperoxidase microscopy typical [3] focal and segmental glomerulosclerosis affecting 45% of glomeruli was diagnosed. The peripheral oedema was treated with frusemide. After 5 months, the serum creatinine had risen to 147 µM/l (1.7 mg/100 ml) and the estimated clearance had fallen to 53 ml/min/70 kg. Ciclosporin 100 mg at night was commenced and to date the patient has been followed 28 months during which time renal function has not deteriorated further, as judged by

either serum creatinine or clearance. After 11 months of ciclosporin treatment the nephrotic syndrome resolved and has not relapsed; a proteinuria of 1–2.5 g/day remains.

A horseshoe-shaped kidney is one of the more common renal malformations, occurring in about 0.25% of the population [4] while the incidence of focal and sclerosing glomerulonephritis presenting as nephrotic syndrome is unknown [5]. We think that the association of these two renal pathologies in our patient is chance and does not imply any causal relationship. Likewise we are not able to find any documentation between primary hypothyroidism and either of the renal conditions.

References

Chen A, Wang-Shen K: Horseshoe kidney and membranous nephropathy. *Nephron* 1990;54:283–284.

Berenack P, Gabriel R: Creatinine clearance without urine.

Abstract

s of the European Dialysis and Transplant Association.

Brussels 1985, p 62.

Chung J, Sobin LH: Focal/segmental lesions (including focal segmental glomerulosclerosis and focal glomerulonephritis): in *Renal Disease; Classification and Atlas of Glomerular Disease*. Tokyo, Igaku-Shoin, 1982.

Allen AC: Anomalies of the upper urinary tract; in *The Kidney: Medical and Surgical Diseases*, ed 1. New York, Grune & Stratton, 1951.

Sharpstone P, Ogg C, Cameron JS: Nephrotic syndrome due to primary renal disease in adults. I. Survey of incidence in South-East England. *Br Med J* 1969;2:533–535.