Sir,

In patients with nephrotic syndrome associated with systemic lupus erythematosus (SLE), the most common histological findings on renal biopsy are diffuse proliferative lupus nephritis (WHO class IV) and membranous lupus nephritis (WHO class V). We report a case of SLE with nephrotic proteinuria due to minimal change nephrotic syndrome (MCNS).

In 1995, SLE was diagnosed in a 29-year-old Japanese woman because of photosensitivity, arthralgia, positive antinuclear antibodies and lymphopenia. The nephrotic syndrome had occurred three times, in 1986, and 1995, with complete remissions following a short-term treatment with 30–40 mg of prednisolone daily. Renal biopsy was not performed. Neither non-steroids nor immunosuppressive agents were used for SLE. She was followed in the out-patient clinic and was admitted to our hospital in March 1998 because of oedema and severe proteinuria. Her blood pressure was 116/62 mmHg, white blood cells were 5000/mm³ and lymphocytes 1600/mm³ (885/mm³ in March 1998). Her platelet count was 388 000/mm³. Urinalysis revealed proteinuria (3.5 g/day). Total serum protein, albumin and total cholesterol were 4.7 g/dl, 2.2 g/dl and 409 mg/dl, respectively, and blood urea nitrogen and creatinine were 11.2 mg/dl and 0.5 mg/dl, respectively. C3, C4 and CH50 were within the normal range. Anti-double-stranded DNA antibody titre was 3 U/ml (normal range: <20). Antinuclear antibody was 1280 (≈40×). IgG, IgA and IgM were 574, 239 and 387 mg/dl, respectively. Creatinine clearance was 111.2 ml/min, and the selectivity index was 0.105. A renal biopsy was performed, which revealed normal glomeruli with only granular traces of mesangial IgM in immunofluorescence staining, with mild effacement of foot processes in electron microscopy, compatible with MCNS.

The patient was treated with 60 mg of prednisolone daily, followed by improvement of proteinuria and oedema. To date, there has been no relapse of the nephrotic syndrome.

Although lupus nephritis is usually preceded by an active immune disorder, detected by a high titre of anti-DNA antibody and low titres of complement [1], immunological exacerbation was not observed in the present case, despite the onset of proteinuria. MCNS was diagnosed because of the acute onset of nephrotic syndrome, its frequent relapses without impairment of renal function, complete remission following steroid therapy and the finding of normal glomeruli in the renal biopsy [2]. The renal biopsy disclosed in the present case only granular traces of mesangial IgM by immunofluorescence staining, which was compatible with the diagnosis of MCNS [3]. Furthermore, the low titre of IgG and the hyperselectivity of urinary protein excretion demonstrated by the selectivity index supported the diagnosis [4]. As only 21 cases of MCNS in SLE have been previously reported in the literature [5,6], the occurrence of a non-lupus nephritis in patients with SLE is a rare event. Although the pathogenesis of lupus nephritis and MCNS still remains unclear, altered T-lymphocyte function has been postulated to play a pathogenetic role in both disorders [7,8], thus representing a possible clue as to their association.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfl979
transplantation. His brother, aged 50 years and completely asymptomatic, came forward as a potential donor. Intravenous urography (IVU), done during the donor evaluation, was suggestive of retrocaval ureter on the right side. Inferior vena cavography (Figure 1) confirmed the diagnosis. The glomerular filtration rates (GFR) of the left and right kidney were 49.2 and 41.2 ml/min, respectively. Open right donor nephrectomy was done. On release of the clamps after vascular anastomosis in the recipient, there was progressive dilatation of the proximal ureter (Figure 2). The non-dilated segment of the ureter was trimmed until free peristaltic flow of urine was confirmed and only then implanted into the bladder over a Double–J stent. Post-operatively, the urine output was sluggish, associated with a marked increase in the dilatation of the transplanted ureter, which however responded to administration of Frusemide for 7 days. The need to administer repeated aliquots of Frusemide in the immediate post transplant period was possibly due to impaired peristalsis in the dilated segment of the ureter, which gradually recovered normal function, after the obstruction was relieved. A literature search revealed only one similar case in which Costea et al. [2] implanted the non-dilated portion of the retrocaval ureter into the bladder. In their report, the recipient developed obstruction and required revision of the ureteroneocystostomy.

On follow up at 6 weeks, the GFR of the transplanted kidney was 72.7 ml/min. We hypothesize that this increase of 76.4% in renal function, in addition to the compensatory increase in filtration, was also due to relief of the obstruction [3].

In conclusion, we feel that retrocaval ureters can be successfully used for transplantation. We recommend that aperistaltic non-dilated segment should be excised and the dilated portion of the ureter should be used for ureteroneocystostomy, only after peristaltic urinary flow has been demonstrated during the surgery.

Conflict of interest statement. None declared.

Fig. 1. Inferior vena cavaogram showing the S shaped course of the ureter up to the point of obstruction.

Fig. 2. Distal non dilated and aperistaltic segment of ureter (arrow). On excision of this segment there is free flow of urine.

doi:10.1093/ndt/gfi106

Advance Access publication 23 August 2005

Supplementation with anti-oxidants Vitamin C and E decreases cyclosporine A trough-levels in renal transplant recipients

Sir,

Blackhall et al. [1] recently reported that anti-oxidant supplementation with vitamin C, vitamin E and β-carotene resulted in a 24% decrease in cyclosporine A (CsA) trough-levels in renal transplant recipients. We agree with the authors that this is an important finding requiring confirmation. Supplementation of anti-oxidant vitamins is not uncommon in renal transplant recipients, because there is strong belief that these supplements are of special benefit to subjects with increased oxidative stress [2,3]. However, injudicious use of these vitamins might evoke rejection, if they indeed decrease blood CsA concentrations.

We recently finished a 3 month double-blind placebo-controlled trial of combination therapy of vitamin C (1000 mg/day) and vitamin E (300 mg/day) in 56 renal transplant recipients. All patients were on a CsA-based immunosuppressive regimen. Our Institutional Review Board had approved the study and all participants signed informed consent. Serum creatinine and trough-levels of CsA were measured at baseline and at the end of the study by the Jaffe’s method (MEGA AU 510; Merck Diagnostics, Darmstadt, Germany) and a fluorescence polarization immunoassay (Abbott Diagnostics; Abbott Park, IL, USA), respectively. Two subjects in the treatment group and one in the placebo group did not complete the study because of hospitalization for cardiovascular events, and were therefore not eligible for analyses.