Dear Sir,

I would like to present an unusual case of the nephrotic syndrome in a 44-year-old Jamaican with known mild sickle cell disease, who presented with a 1-month history of cough, weight loss, night sweats and breathlessness. On examination, he had left middle and lower zone consolidation in the chest, with dense shadowing on the chest X-ray in this area. Sputum showed large numbers of Mycobacterium tuberculosis, later found to be fully sensitive to anti-tuberculous drugs. He was started on oral steroids, rifampicin, isoniazid, ethambutol and pyrazinamide. On admission his albumin was 26 g/l (30–46 g/l), with proteinuria on dipstick testing. One week later, he developed ankle oedema, with a urinary protein loss of 5.6 g/24 h with an EDTA-GFR of 39 ml/min. After initial rehydration his serum creatinine remained within normal limits, with a normal or slightly raised urea. Ultrasound examination showed diffusely bright normal-sized kidneys. After a stormy course over the next few months, he gradually improved, with resolution of his ankle oedema. His serum albumin increased to 36 g/l, his EDTA-GFR to 52 ml/min, although still with 4.2 g/24 h proteinuria. Ethambutol was stopped and his steroid dose reduced. Chest X-ray showed contraction of the destroyed section of the left lung. Renal biopsy showed segmentally accentuated mesangiocapillary glomerulonephritis, with granular capillary wall localisation of IgA, IgM, C3 and C19.

These renal glomerular abnormalities are similar to previous reports of sickle cell disease and the nephrotic syndrome, with membranoproliferative/mesangiocapillary glomerulonephritis [1, 2], although IgG and IgM rather than IgA and IgM deposition was reported. Membranoproliferative glomerulonephritis has also been reported in a patient with tuberculosis [3], and a focal proliferative glomerulonephritis with granular staining for IgA and IgM, and C3 in the mesangium and capillary loops has been described in another patient with tuberculosis and moderate proteinuria [4]. No patient with both conditions has previously been described, and whether the IgA in this case was directed against a mycobacterial antigen is unknown: certainly the development of pulmonary tuberculosis heralded the onset of the nephrotic syndrome and a worsening in renal function, and whether this was mediated via an immune complex nephritis due to mycobacterial antigen, or simply the effects of a severe illness on top of sickle nephropathy is a matter for speculation. As far as his management was concerned, the use of anti-tuberculous drugs in the presence of renal abnormalities required careful monitoring, especially the ethambutol which is renally excreted; a low dose of 10 mg/kg was chosen and the serum level monitored.

References