Clinical and Histological Sequence of Recurrent Focal Segmental Glomerulosclerosis

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Dear Sir,

The recurrence of focal segmental glomerulosclerosis (FSGS) in renal transplants is clearly established [1], Epithelial cell proliferation and detachment and the presence of foam cells are the hallmarks of the early lesions [2]. We have had the opportunity to study sequentially the clinical and histological evolution of a patient with FSGS in whom the disease recurred in the renal allograft. In this case the early lesion was a mesangial proliferative focal segmental lesion that preceded the apparition of the typical hyalinosis.

A 20-year-old women was started on haemodialysis in 1983 because of FSGS. In May, 1985, a cadaveric renal transplant was performed under prednisone and azathioprine treatment. After surgery the patient presented with acute tubular necrosis and anuria. A first renal biopsy (RB) performed the 15th day unfortunately did not show glomeruli at light microscopy (LM), but electron microscopy (EM) revealed signs of acute rejection and tubular necrosis without evidence of glomerular alterations. She was treated with methylprednisolone pulses of 0.5 g during three days. On 40th day a second RB (fig. 1a) demonstrated at LM oedema and an interstitial cellular infiltrate without glomerular lesions. Immuno-fluorescence (IF) was negative. Again she was treated with steroids, and 7 days later the diuresis increased and renal function improved. Proteinuria appeared when the glomerular filtration rate was 10 ml/ min, increasing into a nephrotic range following renal function recovery. A third RB was made in the fourth month posttransplant (fig. 1b). At LM a focal and segmental proliferative lesion without hyalinosis foam cells or epithelial cell proliferation was seen. IF showed IgM, C3, C1q and C4 deposition focal and segmental. In the EM there were segmental dense deposits in some mesangial areas and fusion of epithelial foot processes. There was no evidence of rejection. In April 1986, 11 months posttransplant, a fourth RB (fig.

Fig. 1. Recurrent FSGS. a Second RB on 40th day without glomerular alterations, b Third RB showing proliferative segmental lesions, c Fourth RB with FSGS and tubulo-interstitial affection, d Fourth RB with segmental hyalinotic lesions, a, b, d HE. × 400. c HE. × 100.

lc, d) was made because of deterioration of renal function and persistent nephrotic syndrome. The histological study demonstrated clearly the presence of focal segmental hyalinosis, with IgM and C3 deposition in IF.

Our patient developed nephrotic proteinuria following renal function recovery after a very prolonged period of acute tubular necrosis with evidence of acute rejection. The histological sequence (see fig. 1) showed that initially there were no suggestive signs of FSGS: we

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didn't see proliferation nor detachment of epithelial cells or mesangial foam cells. Later, when proteinuria started, a proliferative focal segmental lesion appeared, followed by the demonstration of the typical histological findings of FSGS. In this case we demonstrated that FSGS was superimposed on mesangial proliferative lesions as described occasionally in the original disease [1]. On the other hand, it can be speculated that FSGS could be the consequence rather than the cause of proteinuria, in a similar fashion to the puromycin aminonucleoside model [3]. The normal glomerular findings in the early biopsies, when the patient was anuric, point to this possibility.

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