Recurrence of Idiopathic Membranous Nephropathy in HLA-Identical Allograft

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Dear Sir,

We report a case of recurrent membranous nephropathy (rMGN) in an allograft, out of 200 live-related renal transplant (LRT) we have operated so far.

S.S., a 49-year-old male first presented with idiopathic nephrotic syndrome (NS) in 1984: blood pressure was normal. He was empirically treated with prednisolone (1 mg/kg/day) for an adequate period without any response. Renal biopsy done later in November, 1985, revealed MGN. Thereafter, he received only symptomatic treatment. He developed ESRD by the end of 1987 and underwent renal transplantation in March, 1988, the donor being his HLA-identical brother sharing A2, A10, B21 and B40 antigens. On the 17th postoperative day, he was discharged from the hospital with normal renal function and urinary protein 1.3 g/day. He remained asymptomatic till the 8th month, when he developed NS with a proteinuria of 8.5 g/day and creatinine clearance of 78 ml/min. Percutaneous graft biopsy done showed MGN.

Since the first case report in 1975 [1], 28 cases of rMGN have been reported so far, including the present case. Details of 23 cases could be found in the literature. Of the remaining 5 cases, in 1981 Pirson et al. [2] and Blanc-Brunat et al. [3] reported 2 cases each. In the same year, Cosyn et al. [4] also reported a de novo MGN which recurred in the 2nd and 3rd transplant. Cases of rMGN have been published mostly as case reports. In three major series, First et al. [5] reported 1 of 14 cases, Montagnino [6] 3 of 9 cases and Mirzyeka [7] 4 of 7 cases of rMGN. These cases had usually an aggressive primary disease; mean duration between diagnosis and ESRD being 4.0 years. Recurrent MGN is common in males and in LRT. Patients with LRT develop rMGN earlier than patients with cadaver renal transplants (CRT). Of 23 cases, 11 allografts were from LRT, 8 being 100% HLA-matched. In cases of rMGN from haploidentical donors, A2 antigen is common in all 4 cases where HLA of patients are known. Montagnino [6] reported 3 of 6 cases of rMGN while on ciclosporin and steroid for the first time in 1989. rMGN presents as early posttransplant variable proteinuria. Hypertension has been frequently found. Renal function at onset was normal. Prognosis of rMGN is relatively poor in LRT as compared to CRT. At 3-year follow-up, our case had heavy proteinuria (6.0-8.0 g/day) with abnormal renal function (serum creatinine 4.5 mg%). It appears that a group of idiopathic MGN patients have a rapid progressive course leading to ESRD in the short period. It may be reasonable to

consider delaying transplantation in such patients to avoid early recurrence of disease. If it is done, a cadaver source should be preferred to a living donor. But in countries where facilities for cadaver kidneys do not exist, a haplo-nonidentical match with ciclosporin as immunosuppressor should be used.

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0028-2766/92/

0603-0366\$2.75/0