Membranous Nephropathy in a Patient with Ankylosing Spondylitis and Rheumatoid Arthritis

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Dear Sir,

The association of rheumatoid arthritis (RA) and ankylosing spondylitis (AS) is extremely rare. According to some authors [1, 2], taking into account the prevalence of each disease individually, the association rate of both entities could vary between 1:50,000 and 1:1,000,000 cases. Further, the presence of membranous nephropathy (MN) in patients with any of both rheumatic conditions is only exceptionally mentioned. Therefore we believe the present short report of a patient suffering from RA and AS who developed signs of renal function impairment due to histologically proven MN to be of interest. The patient was a 35-years-old man diagnosed having RA at the age of 23 by clinical and serological criteria [3, 4] with a positive latex reaction at a titer of 1:260. A total dose of 0.5 g gold salt was administered as an initial therapy followed in the ensuing years by variable dose schedules of steroid and/or nonsteroidal anti-inflammatory drugs. 12 years after the initial diagnosis, the patient was admitted to our department because of marked muscle stiffness and aching pain on the back. There was unequivocal x-ray evidence of advanced sacroileitis. The latex test was found to be positive at a titer of 1:2,560 but other tests including antinuclear antibodies were negative. CH50, C3 and C4 serum levels were well within the normal limits. The HLA phenotype was found to be A2/A3/Bw44, B27/Cw1, Cw5/4A, DR1. A skin biopsy failed to show immunoglobulin deposits. Significant proteinuria (4 g/24 h) was present, serum creatinine being 1 mg/dl (88 µmol/l) and serum albumin 30 g/l, but no gross abnormalities were detected on fresh urine dark field examination. A renal biopsy was performed that revealed a histological pattern of MN by LM and EM (fig. 1) as well as by immunofluorescence. 3 years later

Fig. 1. Electron micrograph showing electron-dense subepithelial deposits. no significant deterioration in renal function was apparent.

As alluded to earlier, the rare cases of nephropathy associated with AS are occasionally interpreted as a consequence of secondary amyloidosis and/or as an expression of analgesic nephropathy. However, some patients with mesangioproliferative and/or focal glomerulonephritis are known [4, 5], although only very recently, a patient with AS and MN has been reported. Despite the renal involvement in RA, most cases are either due to amyloidosis or to analgesic nephropathy. MN is known to be produced by penicillamine and gold therapy,

although some authors claim that chrysotherapy might only act by precipitating a previously existing MN [6]. Otherwise MN, believed to be connected with gold therapy, appears early in most cases with some rare exceptions. Thus, it is of further interest that in our patient clinical features and subsequent histological proof of

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renal involvement were discovered only 12 years after diagnosis. Incidentally, the usual DR2/DR3 HLA association with gold therapy induced nephropathy was not present in our case. As a final comment, it is worth stressing that to our knowledge this is the first report of concomitant RA and AS with histologically proven MN.

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