Dear Sir,

The association of crescentic glomerulonephritis and membranous nephropathy is a rare condition. The natural history of the disease is characterized by nephrotic syndrome and rapidly progressive glomerulonephritis (RPGN) leading to end-stage renal failure (ESRF). We report a case of a young patient with crescentic membranous glomerulonephritis who normalized renal function after immunosuppressive treatment.

A 24-year-old man was admitted to our division for nephrotic syndrome with renal failure. One year earlier, he presented proteinuria, microhematuria and normal renal function. Six months prior to admission, he showed peripheral edema and hypertension and was treated with furosemide 25 mg/day and prednisone 25 mg/day. On admission, physical examination revealed generalized edema and severe hypertension (190/120). Laboratory data showed: hemoglobin 11.1 g/dl, hematocrit 32.3%, WBC 5,600/mm3, platelet count 337,000/mm3, ESR 66 mm at the lst h, BUN 32 mg/dl, serum creatinine 2.5 mg/dl. Urinalysis showed 8 RBC per high-power field (HPF) and fatty casts 3-6/HPF, 24-hour protein excretion was 30 g and throat culture was negative. Serum albumin was 1.6 g/l, serum C3 was normal, ANA, anti-DNA antibodies, cryoglobulins and circulating anti-GBM antibodies were absent. Echotomography showed enlarged kidneys, ECG and chest X-ray were normal. Five days after admission, serum creatinine rose from 2.5 to 4.3 mg/dl.

A percutaneous renal biopsy was performed: eleven glomeruli showing diffuse basement membrane thickening were present.

Fig. 1. Diffuse spiking of the epithelial side of glomerular basement membrane. On the left side of the picture a cellular crescent is present. Silver-Methanamine. × 500.

Fig. 2. Glomerulus with a circumferential cellular crescent. Thickening of capillary walls and mesangial sclerosis are also present. PAS. × 250.

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ent; at methanamine silver stain spiking formation on the epithelial side of the basement membrane was evident (fig. 1). Eight glomeruli showed circumferential cellular crescent formation (fig. 2). Diffuse interstitial edema, focal tubular atrophy and interstitial mononuclear cell infiltration was also present. On direct immunofluorescence, diffuse granular subepithelial staining for IgG and C3 was present.

Treatment with pulse methylprednisolone 15 mg/kg/day i.v. for 3 days followed by oral prednisone 1 mg/kg/day was instituted. Oral cyclophosphamide 1 mg/kg/day was associated and 3-liter plasma exchange every other day was performed.

Ten days later, serum creatinine decreased to 1.6 mg/dl, so that plasma exchange was interrupted after a total of 6 sessions; however, serum albumin was 2.4 g/l and proteinuria was 10 g/day. Corticosteroid therapy (1 mg/kg/day) was continued for 2 months and then tapered until suspension 3 months after the beginning. Oral cyclophosphamide was also interrupted after 3 months of therapy; at this time renal function was normal.

After 5 years from discharge, renal function remained normal (serum creatinine 1.4 mg/dl, creatinine clearance 82 ml/min/1.73 m2), while nephrotic syndrome was still present. Blood pressure was normal with diuretic, beta-blocker and calcium antagonist therapy.

However, at this time renal function began to decline so that serum creatinine was 4 mg/dl and creatinine clearance 26 ml/min/1.73 m2 6 years after diagnosis. Nephrotic syndrome and hypertension were still present. A second renal biopsy was performed: eight glomeruli showing mesangial sclerosis and diffuse basement membrane thickening with spiking formation were present, diffuse tubular atrophy and interstitial fibrosis were also present. At immunofluorescence diffuse subepithelial granular staining for IgG and C3 was present. No immunosuppressive therapy has been instituted at this time.

Idiopathic membranous nephropathy complicated by extensive crescent formation has been reported in only 10 cases [1,2] and was associated with circulating anti-GBM antibodies in 3 cases [3, 4]. Therapy with corticosteroid and cytotoxic drugs did not alter the course of the disease that led most patients to ESRF within 1 year, while it slightly improved renal function in only 1 patient [1].

Plasma exchange therapy associated with corticosteroids was performed only in 1 patient and did not improve renal function [2].

The case described here shows normalisation of renal function after plasma exchange and immunosuppressive therapy; moreover, renal function remained normal 5 years after the therapy was discontinued.

Plasma exchange, high-dose corticosteroid and cytotoxic treatment can be beneficial in patients with crescentic membranous glomerulonephritis presenting with rapidly progressive renal failure. Early diagnosis and treatment are mandatory for these patients.

References
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341