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MABTHERA AS RESCUE THERAPY IN RAPIDLY PROGRESSIVE RENAL FAILURE DUE TO RARE IGA DOMINANT MPGN

Luigi Rossi1, Annalisa Teutono1, Piero Liisi1, Pasquale Libutti1, Francesco Casucci1, Carlo Lomonte1
1Nephrology, Ospedale Muli, Acquaviva delle Fonti, Italy

INTRODUCTION AND AIMS: Clinical and pathological overlap between IgA Nephropathy (IgAN) and membranoproliferative glomerulonephritis (MPGN) is well known since several years. IgA positive IgA dominant MPGN is uncommon disease described only as case reports. It affects most often children, patients with alcoholic cirrhosis and a recent report describes it as rapidly progressive renal failure and nephrotic range proteinuria in 35-years-old man needing hemodialysis. Usual treatment is based on long-term steroid therapy, with the possibility of association with other immunosuppressant. We describe the case of 81-year-old patient with IgA dominant MPGN who has been successfully treated with dual Rituximab administration.

METHODS: We describe the case of 81-years old man hospitalized for fever and acute renal failure. Anamnestically, diabetes mellitus and previous gastric resection for perforated ulcer were reported. At admission, serum Creatinine (sCr) was 4.4 mg/dl, serum Albumin (sAlb) 1.5 g/dl, proteinuria (uProt) 4.9 gr/24h. Other laboratory findings were: severe hypocomplementemia C3 (3 mg/dl), presence of circulating immune complexes, monoclonal gammopathy IgMk. We performed a Kidney biopsy that showed on light microscopy a membranoproliferative pattern with IgA and C3 deposits at immunofluorescence (IF). Considering rapidly progressive worsening of renal function and proteinuria, we decided to administer Rituximab 325 mg/m² (sCr was 6 mg/dl, uProt 8.6 gr/24h and sAlb 1.5 g/dl). Ten days later (sCr 3.9 mg/dl, uProt 3.6 gr/24h and sAlb 1.7), we performed second administration. One month after complete therapy, laboratoristic findings were: sCr 1.8 mg/dl, uProt 3.4 gr/24h, sAlb 1.8 g/dl. After another month, sCr 1.5 mg/dl, uProt 2 gr/24h, sAlb 2.3 g/dl.

RESULTS: In this case, the patient had a rapidly progressive renal failure. Laboratoristic and histological findings confirm activation of classical complement pathway, lead us towards diagnosis of IgA dominant form of MPGN. Considering worsen of renal function and patient comorbidities, we decided to administer Rituximab as rescue therapy, plus low dose of steroid (Prednisone 10 mg/die). Literature review shows that patients with MPGN due to a monoclonal gammopathy should undergo Rituximab treatment to attain remission of the hematologic dyscrasia. Moreover, recent evidences suggest that patients with MPGN associated with monoclonal immunoglobulin deposits and no overt hematologic cancer, had a good response to Rituximab, too. So we administered two 325 mg/m² doses of Rituximab at ten day-interval. Rapid response to therapy confirm the presence of unusual form of MPGN and highlights importance of Rituximab as rescue therapy.

CONCLUSIONS: To the best of our knowledge, this is the first report of Rituximab administration to treat IgA dominant MPGN. Our therapeutic approach was quickly effective and devoid of any side-effects.