

## MEMBRANOPROLIFERATIVA GLOMERULONEPHRITIS SECONDARY TO MONOCLONAL GAMOPATHY – CASE REPORT

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## CASE PRESENTATION

A.S.M., 38 years old, man, reports GNRP with proteinuria and hematuria in 2020, with pulse therapy with Cyclophosphamide 1g for 6 months being indicated, but he remained symptomatic, with uncontrolled hypertension and increased nitrogen bases, when he underwent a kidney biopsy. He was hospitalized in June with hypertensive conditions, worsening renal function and nephritic/nephrotic syndrome, underwent renal replacement therapy for 45 days and was discharged from hospital on 07/15/2022.

In August, he returned to Nephrology with the results of the kidney biopsy, excluding SLE and cryoglobulinemia, and tests were requested to investigate the biopsy results: membranoproliferative glomerulopathy type 1/3, IgM and kappa +3. Fibrosis and atrophy in 40-50%. He was hospitalized days later for vomiting and watery diarrhea with hyponatremia, hypokalemia, and respiratory alkalosis.

At the beginning of 2023, he returns with test results: rheumatological profile and non-reactive serology. The myelogram was returned to the hematologist, who ruled out hematological disease after sending material again for analysis, concluding that it was mild interstitial plasmacytosis. Free light chains Kappa/Lambda ratio 1.84, Kappa 101 mg/dL and Lambda 55 mg/dL. In the 24-hour protein immunofixation, elimination of Kappa/Lambda light chains without

a paraprotein appearance was observed. Myelogram indicated mild polyclonal interstitial plasmacytosis. He was referred to the second hematologist for another diagnostic impression.

Therefore, Kappa Monoclonal Gammopathy was diagnosed and treatment was started. On 04/10/2023, the patient denies complaints and is in the third cycle of chemotherapy of Cyclophosphamide 395mg, Bortezomib 2.3 mg and Dexamethasone 40 mg, with improvement in the initial condition, with urea 127, creatinine 4.85, uric acid 6, 8, pH 7.23, 24h proteinuria of 10.2 g/24h.

## DISCUSSION

MPGN secondary to Monoclonal Gammopathy is caused by abnormal plasma cells that produce monoclonal proteins. The protein can deposit in the glomeruli, affecting patients. Treatment involves therapies that reduce protein production and kidney inflammation, including corticosteroids, chemotherapy, immunotherapy and immunosuppressants. There is relative refractoriness to treatment and symptoms persist in this patient, which deviates from the cynical picture of the main literature.

## FINAL COMMENTS

The importance of this case can be seen due to the persistence of the patient's clinical symptoms, even in the absence of evidence in the tests performed.