

# UNCOMMON PRESENTATION OF MULTIPLE MYELOMA (MM): ATYPICAL MANIFESTATION OF KIDNEY DISEASE

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## Introduction

Kidney involvement has been frequently observed in hematologic disorders. Proliferative GN with monoclonal immunoglobulin deposits (PGNMID) is the second most prevalent form of monoclonal gammopathy of renal significance (MGRS) after amyloid light chain amyloidosis.

The primary diagnosis based on IF commonly identifies IgG3 kappa as the predominant monoclonal Ig in glomerular deposits. However, subsequent studies have also reported cases of PGNMID with IgG4, IgA, and IgM heavy chain subtypes, as well as light chain (LC)-only deposition.

We present a case of PGNMID, multiple myeloma, type 2 diabetes mellitus, hepatitis C virus infection, and Type 1 cryoglobulinemia. The case includes a four-year follow-up with two kidney biopsies, supplemented by a literature review.

## Disease Progression and Second Biopsy:

In 2022, 33 months later after initial presentation, the patient experienced a recurrence of disease. A second kidney biopsy was performed, and the diagnosis of PGNMID by the monoclonal pattern of IgM kappa (Fig.1-5).

Subsequent IEP of blood and urine revealed monoclonal gammopathy of the kappa type. These findings, along with the clinical presentation, were indicative of MM. The bone marrow specimen was presented with plasmacytosis.

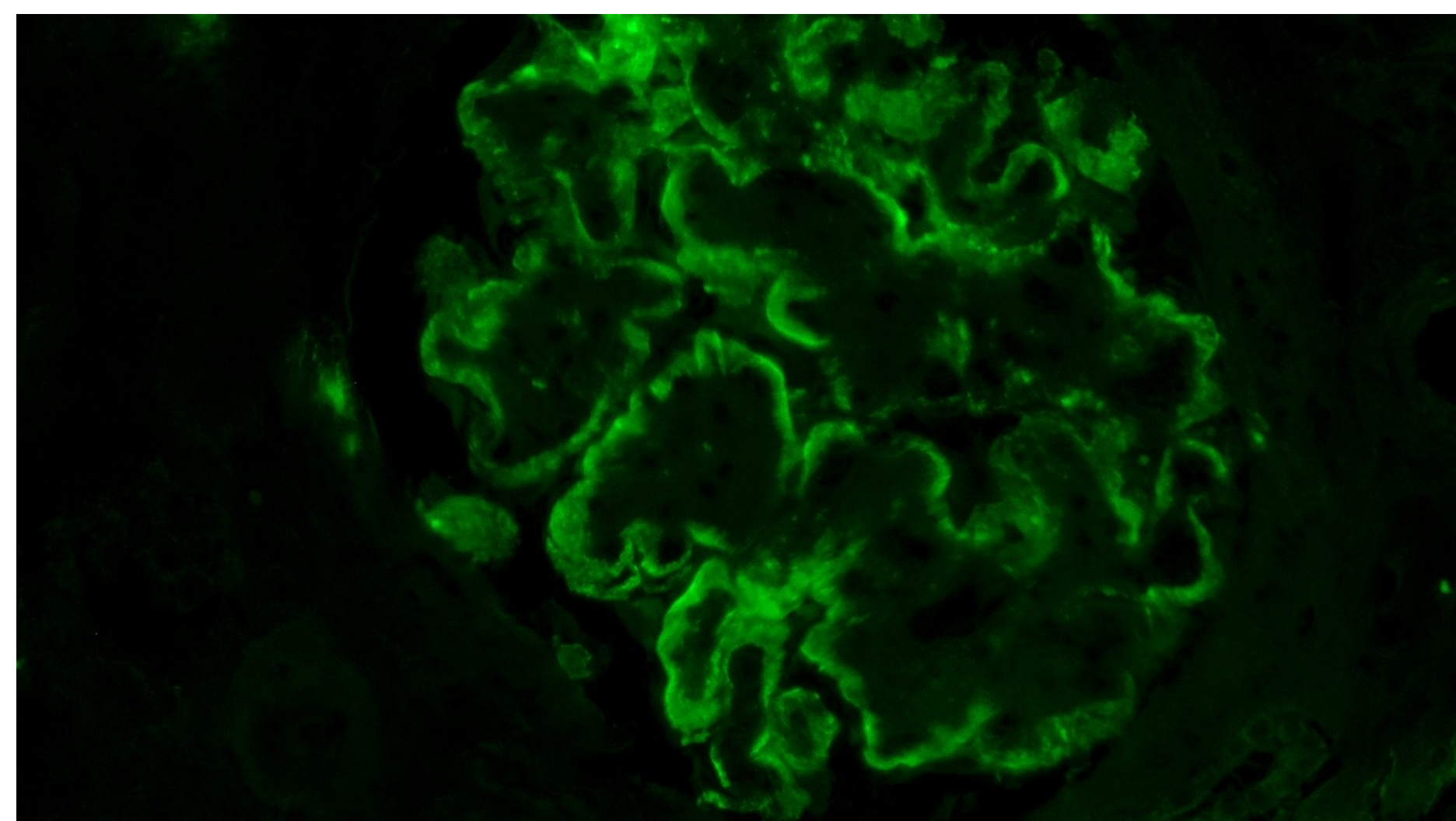


Fig.3 Deposits of IgM

## Further Treatment and Follow-Up:

Bortezomib, CYC, and dexamethasone were the starting chemotherapy agents. His condition improved and after a two-year follow-up, the patient remains stable with GFR 88mL/min, uPCR of 0.330 mg/mgCreat.

## Case presentation

A 65-year-old male was admitted to our hospital in 2020 with decreased kidney function (GFR 37 ml/min), proteinuria along with hematuria (uPCR 5.156 mg/mgCreat; Eryth 80/hpf, dysmorphic RBC 40%, acanthocytes >5% ), hypoalbuminemia (Alb 23.6 g/L), edema, palpable purpura, anemia, thrombocytopenia, and arterial hypertension. RF 168.6 U/mL. His past medical history was significant for T2DM since 2013, hepatitis C diagnosed nine months prior to admission (without treatment), self-resolved hepatitis B in 1990, and a history of TB cured in 2000. The First Kidney Biopsy and Initial Diagnosis: A kidney biopsy revealed acute tubular Injury with membranoproliferative glomerulonephritis with subendothelial deposition of monoclonal IgM along the GBM, Type I cryoglobulinemia. He subsequently underwent a standard bone marrow biopsy with no evidence of Mlg-s. Bone marrow aspiration was normal.

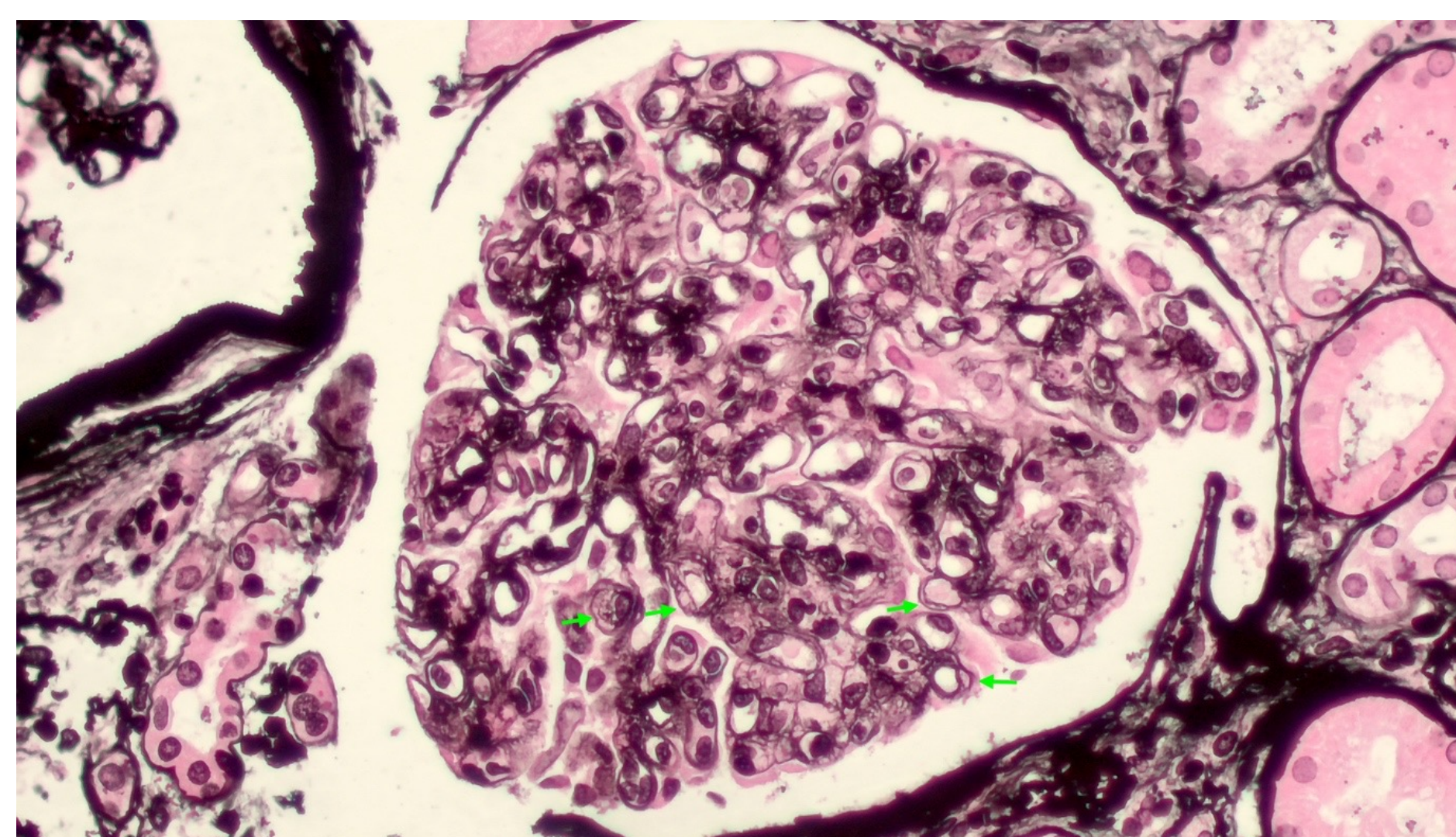


Fig. 1 Membranoproliferative pattern of glomerular injury

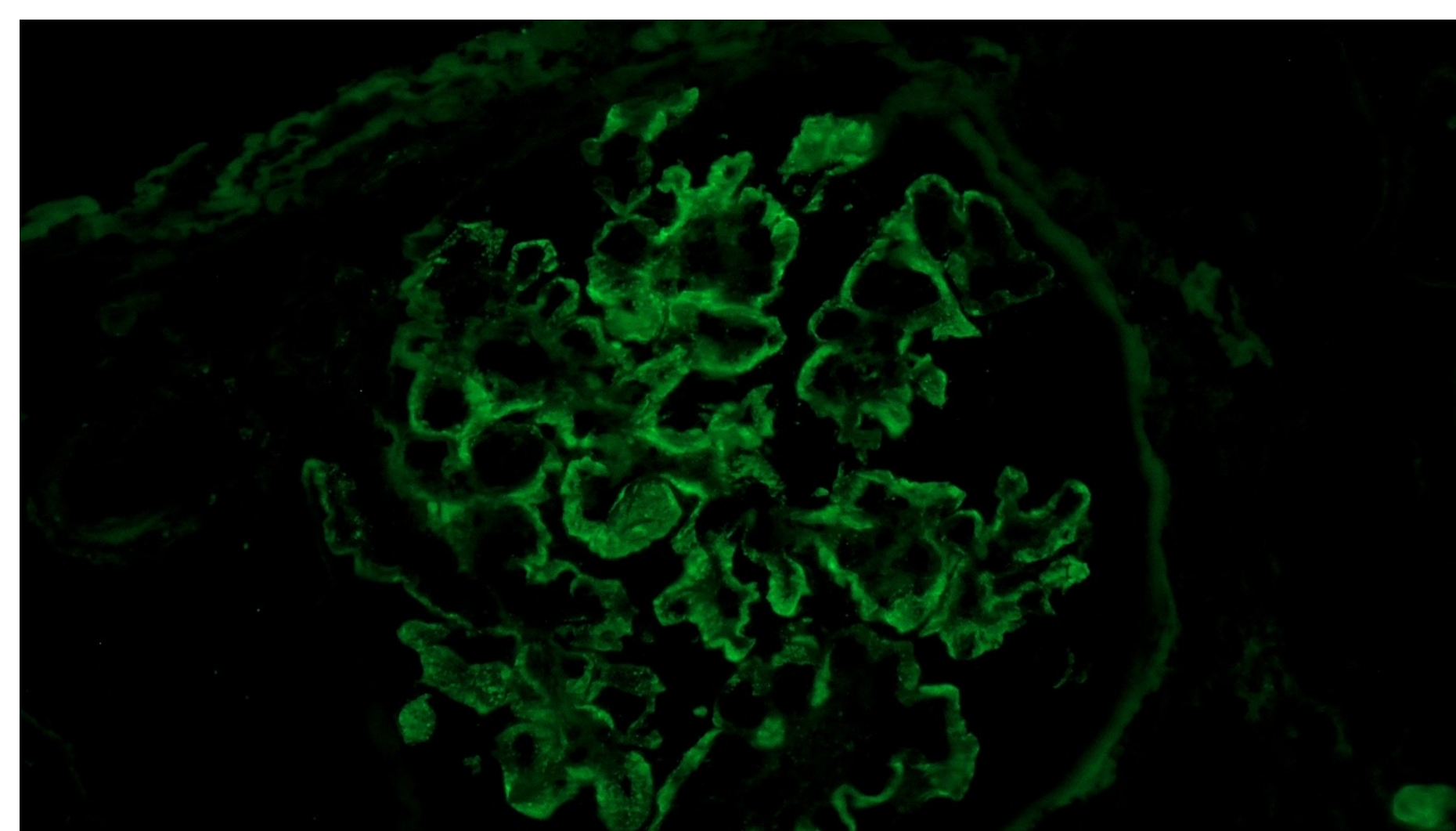


Fig 4. Deposits of kappa

## The First Kidney Biopsy and Initial Diagnosis:

A kidney biopsy revealed acute tubular Injury with membranoproliferative glomerulonephritis with subendothelial deposition of monoclonal IgM along the GBM, Type I cryoglobulinemia. He subsequently underwent a standard bone marrow biopsy with no evidence of Mlg-s. Bone marrow aspiration was normal.

## Initial Treatment and Outcome:

The patient was treated with Methylprednisolone, CYC, and PE (6 sessions). DDA - Sofosbuvir/Velpatasvir for HCV was started. CYC was subsequently replaced with MMF. After 12 months of treatment full clinical remission was achieved: GFR 56 ml/min), uPCR 0.56mg/mgCreat.

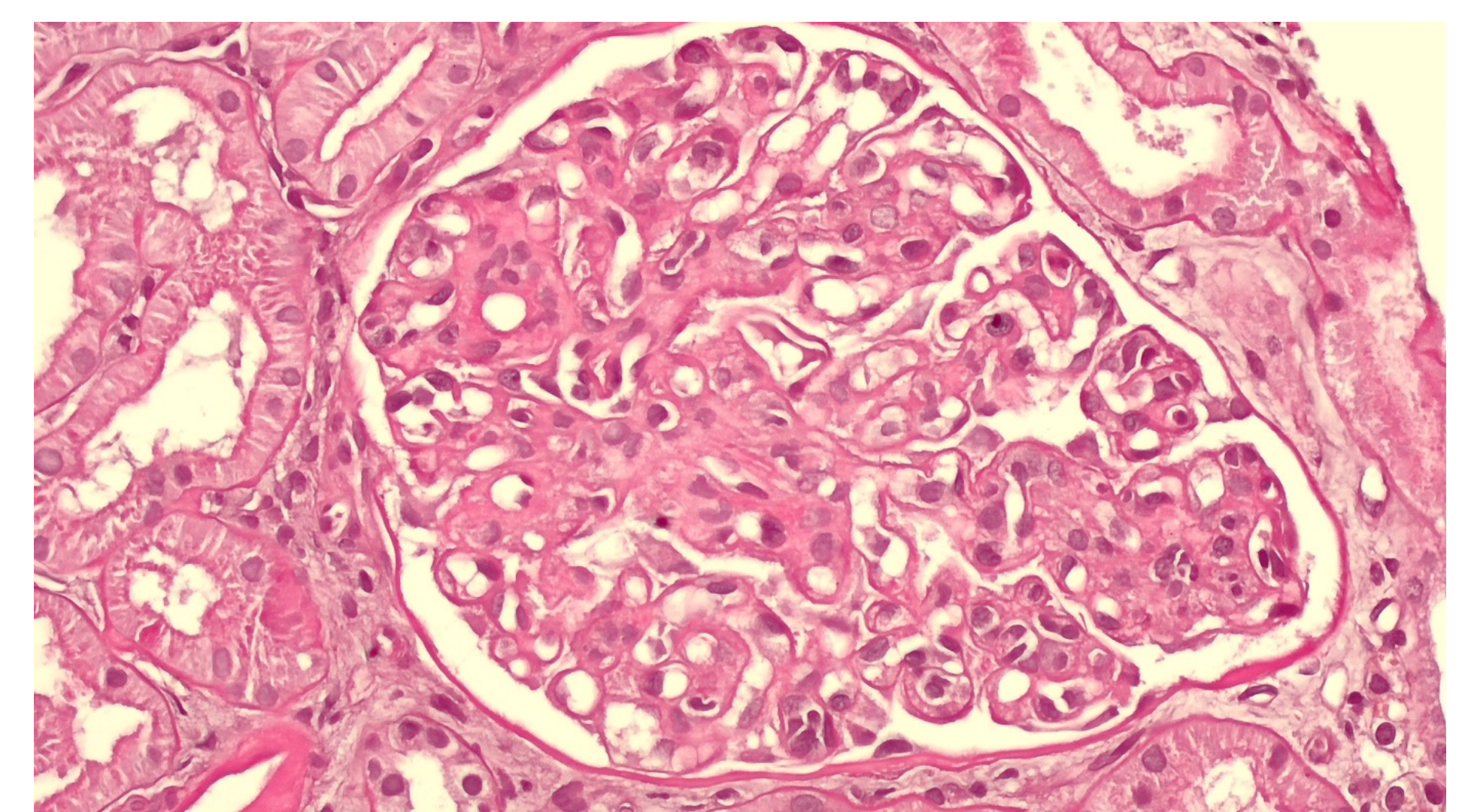


Fig.2 Membranoproliferative pattern of glomerular injury

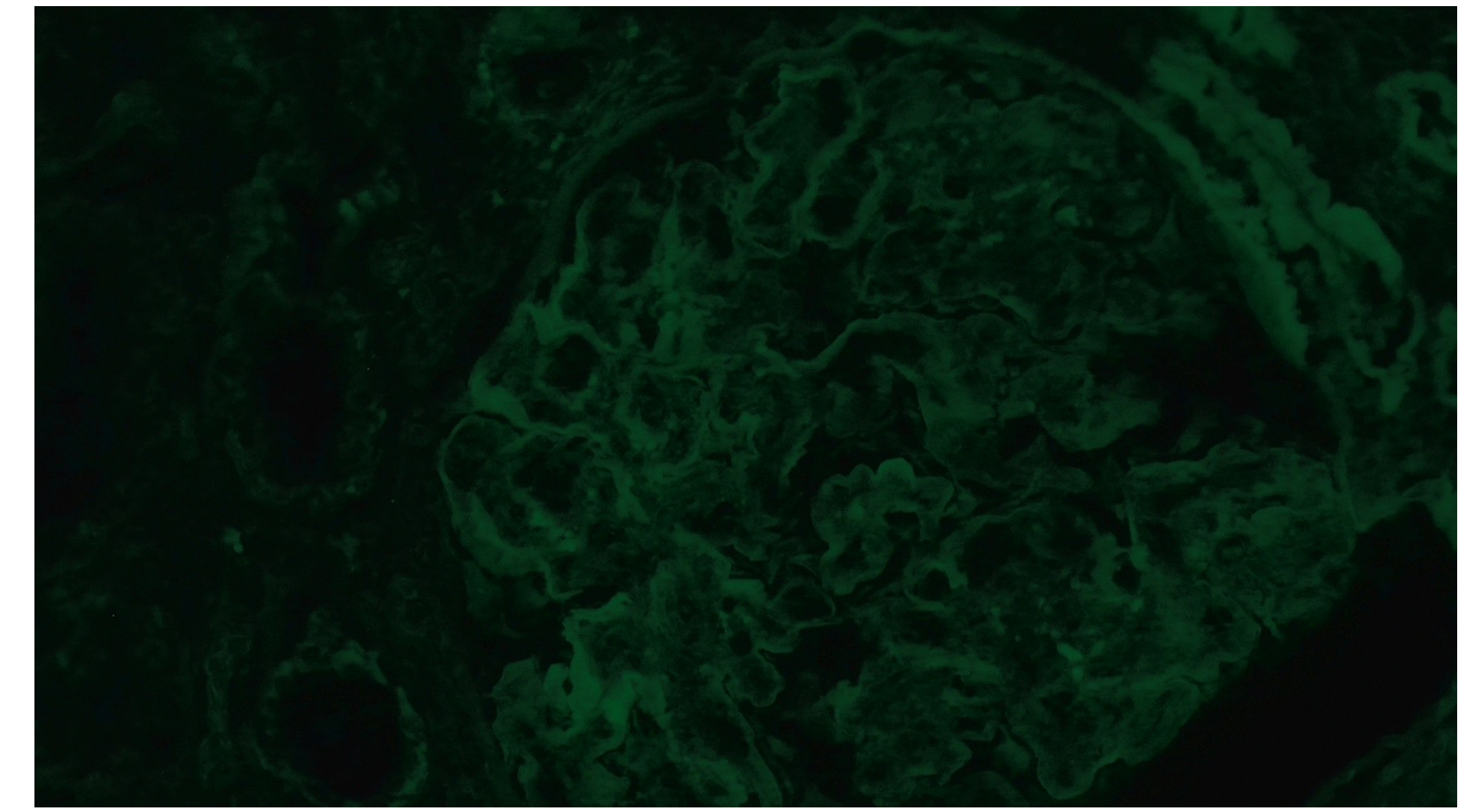


Fig.5 Deposits of lambda

## Discussion

Our case demonstrates unusual flow of the kidney damage in the time frame of 33 months: the first MPGN followed by full remission and no bone marrow damage, and 33 months later PGNMID with the full-blown MM. Given that the patient had both DM and hepatitis C induced cryoglobulinemia, there was no suspect to take the patient under closer observation until the relapse of the kidney damage.

It is important to note that PGNMID is associated not only with MGRS but also with malignancies, infections, connection with HCV infection has also been reported. Thereafter, the primary presentation of the kidney damage was addressed to the presence of the HCV and after achieving the full remission, no other complication was expected in the future.

Compared to other MGRS conditions, the detection rate of M protein in PGNMID is relatively low. Approximately 70% of cases show no detection of Mlg-s in either blood or bone marrow. It is important to note that a low serum M protein detection rate does not necessarily correlate with the severity of kidney damage.

## Conclusion

The presented case shows uncommon manifestation of a hidden monoclonal gammopathy. More significant, prospective, multicenter studies are necessary to compare the safety and efficacy of early treatment regimens, particularly in patients without detectable Mlg-s.

**Keywords:** Glomerulonephritis, MPGN, PGNMID, Multiple Myeloma, Onconeurology, Type I Cryoglobulinemia, Hematologic Malignancy

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