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Case Report

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# "My Legs Are Swollen" Rapidly Progressive IgA-Dominant MPGN in a Young Woman: A Case Report.

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A 26-year-old women with a history of recurrent upper respiratory tract viral infections and psychiatric disorder had been followed in primary care for the past three years due to glomerulonephritis, which progressed to end-stage renal disease (ESRD stage 5) requiring hemodialysis, scheduled for September 2025.

## 1. Patient Data

Three years prior, the patient presented with bilateral lower-limb edema, decreased urine output, asthenia, and sleep disturbances.

## 2. Medical History

2002–2009: Multiple episodes of streptococcal tonsillitis and otitis treated with penicillin.
Childhood: Asthma.
2018: Anxiety, agoraphobia, bulimia, and depression.
2020: Abdominal pain and suspected urinary tract infection.
2022: Onset of renal and worsening psychiatric symptoms.

## 3. Clinical Course

July 2022: Fatigue, abdominal pain, and oliguria.
Laboratory findings (July 2022):

- Estimated glomerular filtration rate (eGFR): 90 mL/min/1.73 m²
- Urinalysis revealed persistent proteinuria

Laboratory findings (2023):

- eGFR: 90 mL/min/1.73 m²
- Proteinuria: +++ (15 g/day)

Laboratory findings (2024):

- eGFR decline from 22 to 9 mL/min/1.73 m²

Progressive renal impairment:

- Blood urea nitrogen increased from 26 to 100 mg/dL
- Serum albumin: 27.8 g/L
- Urinalysis: >100 red blood cells/high-power field

February 2024:

- Proteinuria: 2–3 g/day
- Creatinine: >5 mg/dL
- Anuria
- Initiation of hemodialysis

## 4. Kidney Biopsy Findings

Kidney biopsy revealed a renal cortex containing 22 glomeruli, including one globally sclerosed glomeruli. The remaining glomeruli displayed a lobular membranoproliferative pattern characterized by diffuse mesangial expansion and hypercellularity, together with focal endocapillary hypercellularity due to monocyte infiltration occluding capillary lumina. Marked extracapillary proliferation was observed, including three cellular crescents and six fibrocellular crescents, involving approximately 43% of glomeruli. Two fibrous crescents associated with Bowman capsule disruption were also identified. Silver methenamine staining demonstrated focal duplication of the glomerular basement membrane ("double contours") with cellular interposition. Protein reabsorption droplets were observed within podocytes.

Moderate tubulointerstitial inflammatory infiltrates and acute tubular injury with granular casts and regenerative changes (mitotic activity) were present. Tubular epithelial cells showed prominent cytoplasmic vacuolization compatible with lipid reabsorption. Mild interstitial fibrosis and tubular atrophy (IFTA) involved approximately 25% of the sampled renal cortex. One artery demonstrated mild intimal thickening, while arterioles showed no significant histopathological abnormalities.

Immunostaining for DNAJB9 was negative, and Congo red staining was also negative. Direct immunofluorescence microscopy (5 glomeruli examined, none globally sclerosed) demonstrated granular mesangial and subendothelial deposits positive for IgA (+++), kappa (++), lambda (++), C3 (++), IgG (+), and IgM (+/-), with negative staining for C1q and fibrinogen.

Overall findings were consistent with IgA-dominant membranoproliferative glomerulonephritis with extracapillary proliferative features (43% cellular and fibrocellular crescents), associated moderate tubulointerstitial nephritis, acute tubular necrosis with regenerative changes, and mild interstitial fibrosis/tubular atrophy.

*The high percentage of crescents (43%) represented a marker of poor renal prognosis.*

*The coexistence of a membranoproliferative glomerulonephritis (MPGN) pattern with extracapillary proliferative features suggested an aggressive disease phenotype.*

*The presence of subendothelial immune deposits and C3 positivity supported an immune complex–mediated pathogenic mechanism. Tubulointerstitial fibrosis and tubular atrophy constituted important prognostic indicators associated with progressive renal dysfunction.*

*Histopathological findings, including negative DNAJB9 immunostaining and Congo red staining, allowed exclusion of fibrillary glomerulopathy and renal amyloidosis.*

## 5. Treatment

The patient received multiple immunosuppressive therapies, including:
Corticosteroids, Cyclophosphamide, Rituximab, Mycophenolate mofetil (MMF), Tacrolimus

Hemodialysis was initiated in February 2024.
The patient is currently undergoing chronic hemodialysis and remains on the waiting list for a kidney transplant scheduled for September 2025 and extended due to the desire for pregnancy.

## Comorbidities and Additional Treatments

- Hypertension: Angiotensin II receptor blocker (Losartan)
- Obesity / weight gain: Increase from 90 kg to 111 kg, treated with a GLP-1 receptor agonist (Semaglutide, Wegovy® 0.5–1 mg)
- Headache: Tramadol
- Bulimia and anxiety: Diazepam

## 6. Conclusions

The presence of extensive extracapillary proliferation, endocapillary hypercellularity, and early tubulointerstitial fibrosis likely contributed to the rapid progression toward end-stage renal disease(ESRD stage 5) despite aggressive immunosuppressive therapy. Regular monitoring of proteinuria and glomerular filtration rate (GFR) is essential for detecting treatment refractoriness and planning renal replacement therapy. No clear association between prior penicillin exposure and the glomerular disease was identified in this case. Psychiatric comorbidities may complicate the longitudinal management of patients with advanced chronic kidney disease. Primary care played a key role in the longitudinal follow-up of the patient, coordination with nephrology and psychiatry specialists, and management of sick leave, disability procedures, and social support documentation [1-9].

## Ethics Statement

This case report was conducted in accordance with the principles of the Declaration of Helsinki. Institutional ethics committee approval was not required for publication of a single anonymized case report according to local institutional policies.

## Informed Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical data.

## Conflict of Interest

The authors declare no conflicts of interest related to this work.


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## References


1. Endocapillary proliferative glomerulonephritis. Kidney Pathology.
2. Batsford, S. R., Mezzano, S., Mihatsch, M., Schiltz, E., & Rodreguez-Iturbe, B. (2005). Is the nephritogenic antigen in post-streptococcal glomerulonephritis pyrogenic exotoxin B (SPE B) or GAPDH?. *Kidney international, 68(*3), 1120-1129.
3. Ahn, S. Y., & Ingulli, E. (2008). Acute poststreptococcal glomerulonephritis: an update. *Current opinion in pediatrics, 20*(2), 157-162.
4. Rovin, B. H., Adler, S. G., Barratt, J., Bridoux, F., Burdge, K. A., Chan, T. M., ... & Floege, J. (2021). KDIGO 2021 clinical practice guideline for the management of glomerular diseases. *Kidney international, 100*(4), S1-S276.
5. Yu, L., Zhang, H., & Wu, Y. (2024). Association between different proportions of crescents and the progression of IgA nephropathy (IgAN): a systematic review and meta-analysis. *BMC nephrology, 25*(1), 396.
6. Filippone, E. J., & Farber, J. L. (2026). Primary membranoproliferative glomerulonephritis: natural history, pathogenesis, and treatment. *Frontiers in Nephrology, 6*, 1747678.
7. Roberts, I. S. (2014). Pathology of IgA nephropathy. *Nature Reviews Nephrology, 10*(8), 445-454.
8. Anguiano, L., Kain, R., & Anders, H. J. (2020). The glomerular crescent: triggers, evolution, resolution, and implications for therapy. *Current opinion in nephrology and hypertension, 29*(3), 302.
9. Rudnicki, M., Windpessl, M., Eller, K., Odler, B., Gauckler, P., Neumann, I., ... & Säemann, M. D. (2023). Diagnose und Therapie glomerulärer Erkrankungen mit einem membranoproliferativen Läsionsmuster (MPGN)–2023. *Wiener klinische Wochenschrift, 135*(Suppl 5), 688-695.