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CASE REPORT

Immune-Complex Mediated Crescentic Membranoproliferative Glomerulonephritis Presenting as Rapidly Progressive Renal Failure: A Case Report

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Summary

Rapidly progressive glomerulonephritis represents a spectrum of diseases characterized by crescent formation in glomeruli and rapid decline in renal function. Membranoproliferative glomerulonephritis (MPGN) with crescentic transformation is rare and often presents with severe renal impairment. We report a case of a 40-years hypertensive woman who initially presented with acute kidney injury (AKI) and was treated as acute pyelonephritis. Due to rapid deterioration of renal function requiring hemodialysis, she was evaluated for RPGN. Renal biopsy revealed immune-complex mediated crescentic MPGN. Early aggressive immunosuppressive therapy resulted in significant renal recovery and dialysis independence.

Keywords: Crescentic MPGN; Rapidly progressive glomerulonephritis; Immune-complex mediated glomerulonephritis; Cyclophosphamide; Renal biopsy

Background

Rapidly progressive glomerulonephritis (RPGN) is a clinical term that is defined by the sudden and accelerated development of renal insufficiency ("rapidly progressive") due to cellular proliferation within and inflammation of glomeruli ("glomerulonephritis"). Common diagnostic criteria for RPGN include a 50% loss of renal function over 3 months and evidence of glomerular injury, in the form of an active urinary sediment including hematuria[1]. RPGN is classified into 3 main types based on immune deposit distribution and visualization through immuno-fluorescence and electron microscopy: anti-glomerular basement membrane disease, immune-complex glomerulonephritis, and pauci-immune glomerulo-nephritis[2]. MPGN can present as RPGN. MPGN is now classified based on immune-pathogenesis into immune-complex mediated, complement-mediated and immuno-fluorescence-negative subgroup[3,4]. Crescentic MPGN is uncommon and may clinically mimic ANCA-associated vasculitis, often leading to empirical treatment prior to histological diagnosis.

Case Presentation

Mrs. X, a 40-years woman with newly diagnosed hypertension, presented with fever, abdominal pain, oliguria, anasarca, and progressive deterioration of renal function. She was initially admitted to the department of medicine and treated as a case of acute pyelonephritis with AKI. Despite standard therapy, her renal function deteriorated rapidly with rising serum creatinine, reduced urine output, and generalized edema. She required three sessions of hemodialysis due to worsening renal function and volume overload. In view of rapidly progressive renal failure, RPGN was suspected and she was transferred to the department of nephrology for further evaluation. Urine R/M/E showed protein: 3+, RBC: 30-35/HPF (15% dysmorphic), urine C/S revealed no growth of bacteria. 24 hours UTP was 8 gm in 1.2 L urine, serum albumin was 28 gm/L. Serum creatinine was 2 mg/dl initially then rapidly raised to 10.8mg/dl over 10 days, after three session HD serum creatinine was 4.35 mg/dl. A thorough clinical and

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serological workup was performed, including ANA, anti-dsDNA, ANCA, and viral markers, all of which were negative. Serum C3 was low but serum C4 was normal. S.TSH was 4.89 µIU/ml, SGPT was 24 U/L, FBS was 5 mmol/L and 2HABS was 5.6 mmol/L. USG of whole abdomen showed right kidney was 11.89 cm, left kidney was 10.79 cm with well- maintained CMD, cortical thickness and echogenicity of both kidneys, moderate ascites with normal hepato-biliary and pancreatic system. Echocardiography revealed no RWMA, good left ventricular systolic function (LVEF: 60%).

Initial Management

After exclusion of active infection, the patient was empirically treated as ANCA-associated vasculitis due to RPGN. She received intravenous pulse methyl-prednisolone for three consecutive days followed by oral prednisolone, following the reduced-dose steroid strategy of the PEXIVAS trial[5]. Intravenous cyclophosphamide was administered at a dose of 12.5 mg/kg.

Renal Biopsy Findings

Renal biopsy showed features of immune-complex mediated crescentic membranoproliferative glomerulonephritis. Light microscopy revealed diffuse endocapillary hypercellularity with cellular crescent formation in 10 out of 14 glomeruli. Glomerular basement membrane was focally thickened with double contour formation. No glomerulosclerosis found. Intestinal fibrosis and tubular atrophy (IFTA) occupied 20% of cortical area. Severe acute tubular injury was seen. Blood vessels were unremarkable. Immunofluorescence demonstrated granular deposition of immunoglobulins and complement in the mesangium and along the glomerular basement membrane (Figure 1 and 2).

Differential Diagnosis

The differential diagnoses included:

- Pauci-immune crescentic glomerulonephritis
- Lupus nephritis
- Anti-glomerular basement membrane disease
- Complement-mediated MPGN (C3 glomerulopathy)

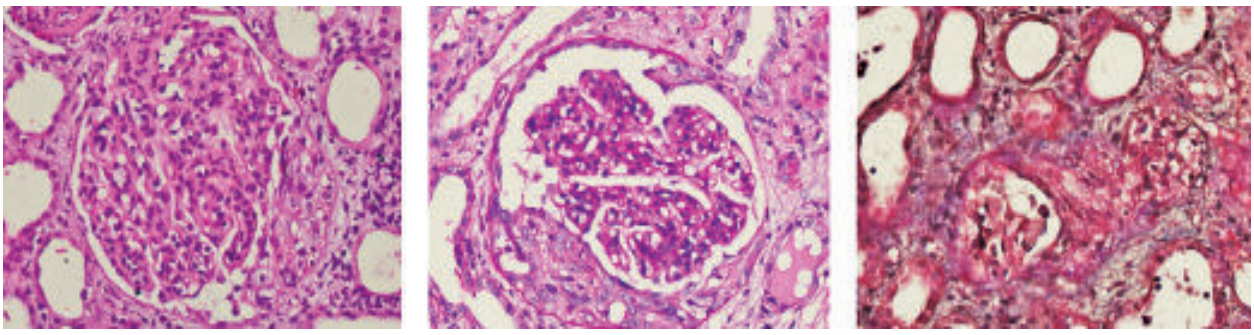
The presence of immune-complex deposition on immunofluorescence supported the diagnosis of immune-complex–mediated crescentic MPGN.

Definitive Treatment

As per KDIGO 2021 glomerular diseases recommendation, MPGN presented as RPGN should be treated as ANCA-associated vasculitis. Following histological confirmation, the patient completed six doses of intravenous cyclophosphamide with tapering oral corticosteroids. Supportive treatment included blood pressure control with renin–angiotensin system blockade and careful management of fluid and electrolyte balance. She is now on maintenance therapy with azathioprine.

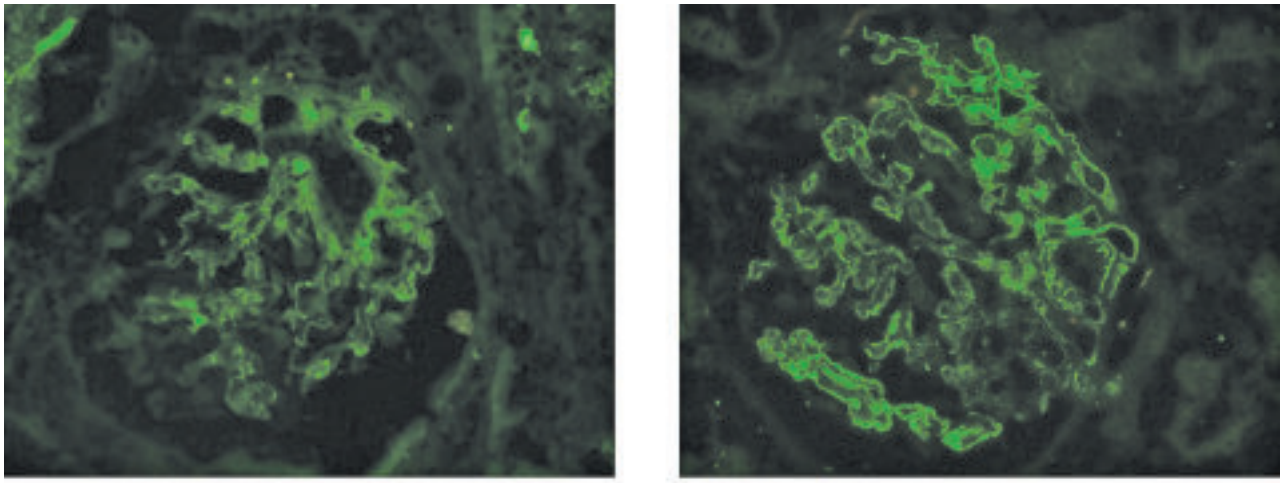
Outcome and Follow-up

Her renal function gradually improved, proteinuria decreased, and hemodialysis was discontinued. At follow-up, she remained dialysis-free with stable renal function and marked clinical improvement. In her last follow up serum creatinine was 1.06mg/dl, serum albumin was 44gm/L and 24 hours UTP was 0.53 gm in 1.8 L urine.

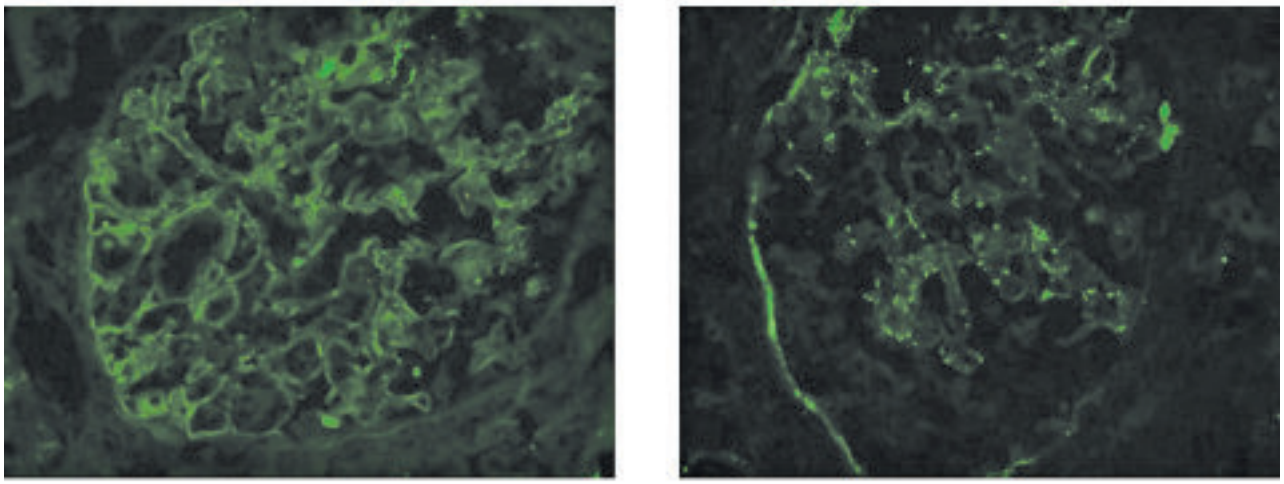


H&E PAS MT

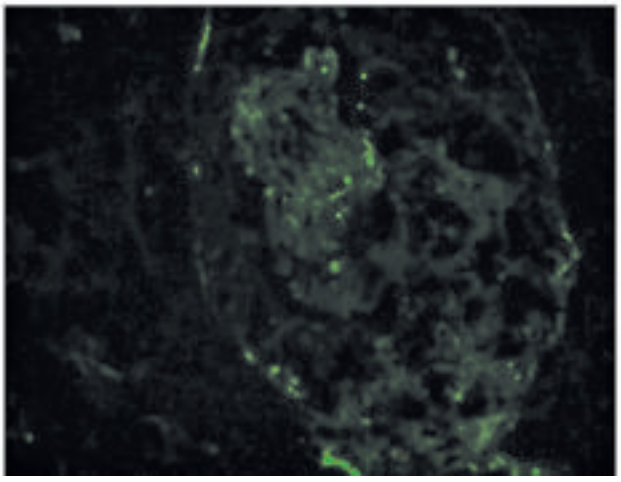
Figure 1. *Light Microscopic Images*



IgG C3



C1q Kappa



Lambda

Figure 2. *Direct immunofluorescence (DIF) Images*

Timeline of Events

Time	Clinical Events
Initial presentation	Fever, AKI, treated as acute pyelonephritis
Hospital course	Rapid rise in creatinine, anasarca, oliguria
Early admission	Three sessions of hemodialysis
RPGN suspected	Empirical steroids + cyclophosphamide
Renal biopsy	Immune-complex mediated crescenticMPGN
Follow-up	Improved renal function, dialysis-free

Discussion

This case highlights the diagnostic challenge of crescentic MPGN presenting as RPGN. Clinical presentation may closely resemble pauci-immune vasculitis, necessitating early empirical therapy. Renal biopsy remains essential for definitive diagnosis and prognostication. Aggressive immunosuppression can lead to favorable renal outcomes even in dialysis-dependent presentations.

Learning Points

- Membranoproliferative glomerulonephritis is a pattern of injury that requires immune-pathological classification.
- Crescent formation in immune-complex–mediated MPGN indicates severe disease and warrants urgent treatment.
- Renal biopsy with immunofluorescence is essential to differentiate immune-complex–mediated MPGN from other causes of rapidly progressive glomerulonephritis.

Conclusion

Immune-complex mediated crescentic MPGN should be considered in patients presenting with RPGN. Timely renal biopsy and early initiation of immunosuppressive therapy are crucial for renal recovery.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report.

Ethics Statement

Ethical approval was not required for this case report in accordance with institutional policy.

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Grindley MF: Manual of histologic and special staining Nephrologic, Elammarion, Paris, 1965.

ABBREVIATIONS

Angstrom	A
body surface area	BSA
body weight	body wt.
centimeter	cm
celius	C
complement components	C1,C2,C3
Correlation coefficient	r
creatinine clearance	Ccr.
curie (s)	Ci
Equivalents	Eq
Fahrenheit	F
Glomerular fitration rate	GFR
gram (s)	g
Grams per cent	g/100mi
half-time	tf1/2
hour (s)	hr
inch	inch