



## Plasma Exchange is a Therapeutic Procedure used in the Management of Rapidly Progressive Glomerulonephritis (RPGN): A Case Report

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### Abstract

Rapidly Progressive Glomerulonephritis (RPGN) is a clinical syndrome characterized by a rapid decline in kidney function and the presence of crescents on kidney biopsy, requiring immediate treatment to prevent disease progression. A 34-year-old man was admitted to the hospital with generalized body swelling that began in the eyelids one week prior. He had a history of fever before admission, which subsided upon hospitalization. Additional complaints included shortness of breath, cough, and runny nose. Past medical history included hypertension and hiperuresemia. In recent days, he experienced oliguria. Physical examination revealed pitting edema in the lower extremities. Laboratory tests showed anemia, hypoalbuminemia, and rapid decline in kidney function. Urinalysis revealed protein +3, leukocytes sediment 80.1/μL, erythrocytes sediment 468/μL, and Esbach 0.5 g/L. Complement C3 53 mg/dL, C4 13.8 mg/dL, ANA, and anti-dsDNA were negative, with no available data for anti-GBM, ANCA and kidney biopsy Segmental Mesangial Hypercellular Glomerulus and focal segmental sclerosis. Chest X-ray showed cardiomegaly, bronchopneumonia, and left pleural effusion. Abdominal ultrasound revealed bilateral parenchymal renal disease. The patient was treated with high dose steroids, antibiotics, antihypertensives, symptomatic medications, two sessions of hemodialysis, and four sessions of plasma exchange (PLEX). The combination therapy of high dose steroids and followed by hemodialysis and plasma exchange, resulted in significant reduction of edema and improvement in kidney function, demonstrating the effectiveness of an aggressive immunomodulatory regimen. In pauci-immune or idiopathic RPGN, the combination of high-dose steroids and plasma exchange along with dialysis provides a rapid clinical response, increases the chance of remission, and preserves kidney function.

### Introduction

Rapidly Progressive Glomerulonephritis (RPGN) is one of the most severe and progressive forms of glomerulonephritis, characterized by a rapid decline in renal function within days to

weeks. Histopathologically, RPGN is marked by the formation of crescents in the glomeruli due to proliferation of parietal epithelial cells and infiltration of inflammatory cells in response to glomerular capillary injury (Mohamed et al., 2024; Meena et al., 2026; Kurts et al., 2025). Clinically, RPGN may present with hematuria, proteinuria, hypertension, and a rapid deterioration of renal function, which can progress to end-stage renal disease if not treated promptly (Graňák et al., 2025; Sahu et al., 2025; Berrocal et al., 2026). The prevalence is relatively significant among cases of severe glomerulonephritis. In developed countries, RPGN is estimated to account for approximately 10–15% of glomerulonephritis cases requiring kidney biopsy (Aghsae Fard & Zahedian, 2024; Palma et al., 2025; Covic et al., 2025). In Indonesia, prevalence data remain limited due to the lack of a national registry; however, local studies indicate that RPGN occurs at a relatively high rate among patients with acute nephritis and rapid loss of renal function (Jauhal et al., 2026; Hull et al., 2026; Floege et al., 2025). The reported one-year mortality rate ranges from 20–30%, depending on the underlying etiology, delay in diagnosis, and response to therapy (Grillo et al., 2025; Gettings et al., 2025; Kamalo et al., 2026). Moreover, approximately 30–50% of patients may progress to end-stage chronic kidney disease and require long-term renal replacement therapy such as hemodialysis or kidney transplantation (Cortazar et al., 2023; Turra et al., 2026; Zisman-Ilani et al., 2026). Identifying the specific type of RPGN is crucial, as it directly influences therapeutic decisions and prognosis. Given its high progression rate and the risk of irreversible renal failure, RPGN is considered a nephrological emergency requiring early detection, accurate diagnosis, and intensive management to reduce both short- and long-term mortality and morbidity (Aghsae Fard & Zahedian, 2024; Rovin et al., 2021; Jakubowska et al., 2026).

## Methods

### Case Illustration

A 34 year old man was admitted to the hospital with generalized body swelling, which began in the eyelids one week prior and progressively worsened to involve the entire body. He reported having a fever for one week, which improved after taking medication. Upon hospital admission, he was afebrile. Additional complaints included shortness of breath, cough, and nasal congestion. The patient reported cloudy yellow urine. His medical history was significant for hypertension and gout.

On physical examination, blood pressure was 180/90 mmHg, with other vital signs within normal limits. Conjunctivae were pale, and pitting edema was noted in both lower extremities. Examination of the head, chest, and abdomen was otherwise unremarkable. Laboratory results showed hemoglobin 7.2 g/dL, leukocyte count  $11.1 \times 10^3/\mu\text{L}$ , and platelet count  $131 \times 10^3/\mu\text{L}$ . Renal function tests revealed a blood urea nitrogen of 171 mg/dL and serum creatinine of 7.6 mg/dL, which increased rapidly over two days to 262 mg/dL and 10.3 mg/dL, respectively. Serum albumin was 2.1 g/dL, calcium 2.0 mmol/L, and other electrolytes were within normal range. Urinalysis demonstrated proteinuria (+3), leukocytes sediment  $80.1/\mu\text{L}$ , erythrocytes sediment 468  $1/\mu\text{L}$ , and Esbach 0.5 g/L. Antistreptolysin O was negative; Complement C3 53 mg/dL, C4 13.8 mg/dL, ANA 0.58 IU/mL, and anti-dsDNA 0.5 IU/mL were all within normal limits. Chest X-ray revealed cardiomegaly, bronchopneumonia, and left sided pleural effusion. Abdominal ultrasound demonstrated bilateral parenchymal renal disease. Kidney biopsy confirmed crescent formation in the glomeruli, Segmental Mesangial Hypercellular Glomerulus and focal segmental sclerosis, ANCA-Related RPGN.

The patient was diagnosed with acute glomerulonephritis, pauci immune type, with RPGN. Treatment included high dose intravenous methylprednisolone 500 mg every 12 hours for three days, followed by tapering to oral administration at 20 mg every 12 hours. Intravenous ampicillin sulbactam 1.5 g every eight hours was given for bronchopneumonia. Antihypertensive therapy consisted of amlodipine 10 mg once daily and imidapril 10 mg once

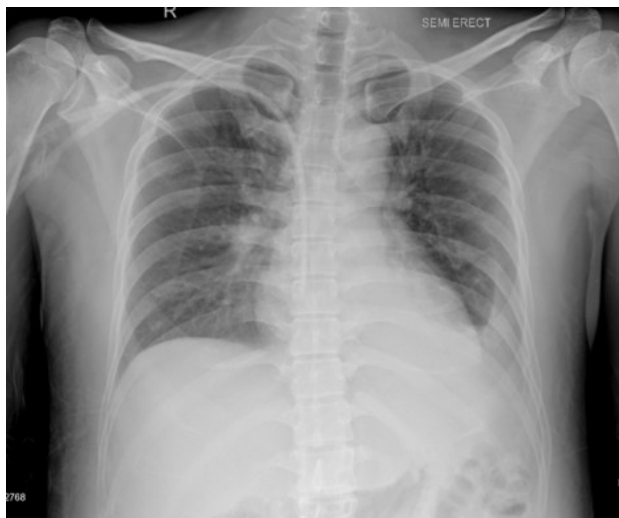
daily. Additional medications included sodium bicarbonate 500 mg every eight hours, folic acid 1 mg once daily, and packed red cell transfusion. Hemodialysis was performed twice, and Plasma exchange (PLEX) was carried out four times. After 30 days of hospitalization, renal function improved significantly, with blood urea nitrogen decreasing to 60 mg/dL and serum creatinine to 1.0 mg/dL, accompanied by marked clinical improvement.

## Result and Discussion

This 34 year old male presented with diffuse edema, hypertension, anemia, proteinuria, microscopic hematuria, and a rapid decline in renal function from an initial blood urea nitrogen of 171 mg/dL and serum creatinine of 7.6 mg/dL to 262 mg/dL and 10.3 mg/dL, respectively, within two days which is highly consistent with RPGN. Aggressive therapy comprising high dose steroids, PLEX, and dialysis represents the standard approach to prevent progressive glomerular injury (Rovin et al., 2021)(Odler et al., 2025).

RPGN is a clinical syndrome characterized by a rapid decline in renal function usually defined as a >50% reduction in glomerular filtration rate (eGFR) within  $\leq 3$  months and histologically by the presence of crescents in glomeruli on kidney biopsy; This condition constitutes a nephrological emergency, requiring prompt intervention to prevent progression to chronic kidney failure (Arimura et al., 2016).

In this case, ANA 0.58 IU/mL and anti-dsDNA 0.5 IU/mL were both negative, making lupus nephritis unlikely, while no anti GBM or ANCA data were available. Based on these findings, the most probable diagnosis was pauci immune or idiopathic RPGN (Han et al., 2023)(Gluhovschi et al., 2023).



*Figure 1. Chest X-ray revealed cardiomegaly, bronchopneumonia, and left sided pleural effusion*

Generalized body edema pitting in the lower extremities was attributable to severe proteinuria and hypoalbuminemia serum albumin 2.1 g/dL. Urinalysis revealed proteinuria +3, leukocytes sediment 80.1/ $\mu$ L, erythrocytes sediment 468/ $\mu$ L, and Esbach 0.5 g/L, while abdominal ultrasound demonstrated bilateral parenchymal renal disease findings consistent with glomerulonephritis. Hypertension likely contributed to further glomerular damage. The patient had a history of hypertension and hiperuresemia, with previous use of antihypertensive agents amlodipine and imidapril that did not adequately control blood pressure. Chest radiography showed bronchopneumonia and pleural effusion, for which intravenous ampicillin sulbactam 1.5 g every eight hours was administered. Kidney biopsy confirmed crescent formation in the glomeruli, Segmental Mesangial Hypercellular Glomerulus and focal segmental sclerosis, ANCA-Related RPGN.

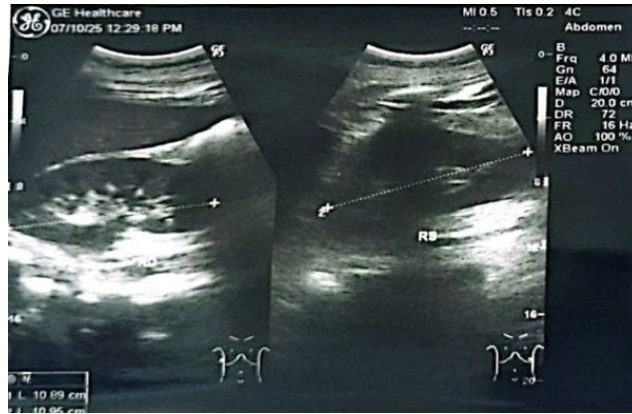


Figure 2. Abdominal ultrasound demonstrated bilateral parenchymal renal disease

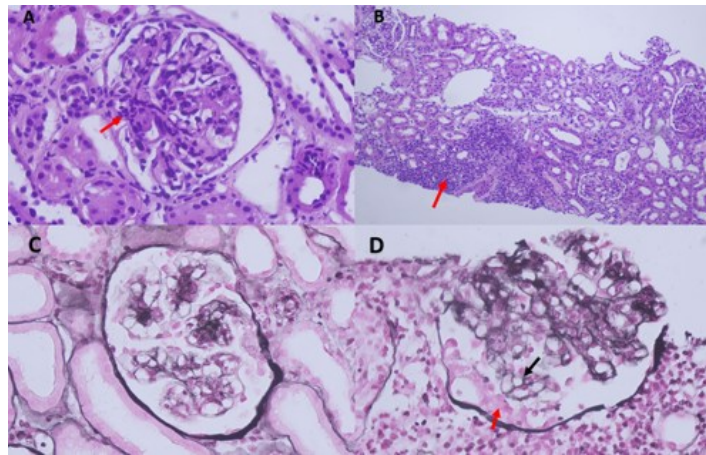


Figure 3. A. Focal hypercellularity glomerulus, neutrophil infiltration, B. Interstitial inflammation, no interstitial fibrosis, C. Segmental sclerosis glomeruli, no interstitial fibrosis, D. Cellular crescent, opened glomerular capillary lumina

Administration of high dose steroids, such as intravenous methylprednisolone 500 mg every 12 hours for three days, rapidly suppresses inflammatory immune responses (Aghsae Fard & Zahedian, 2024; Leon-Astudillo et al., 2026; Nelson et al., 2026). PLEX performed four times in this case, facilitates the removal of pathogenic antibodies or immune complexes and is recommended in anti GBM disease as well as in RPGN with high ANCA titers (Zepeda et al., 2023; Godbole et al., 2026; Moorani et al., 2022). Two sessions of hemodialysis were carried out to address uremic toxin accumulation and maintain fluid balance (Cortazar et al., 2023; Han et al., 2023; Guo & O'Donnell, 2026). Supportive therapy included furosemide for edema, ampicillin sulbactam for pulmonary infection, antihypertensives, folic acid supplementation, and packed red cell transfusion for anemia (Wenhong et al., 2025; Rashed, 2022; Zhou, 2024).

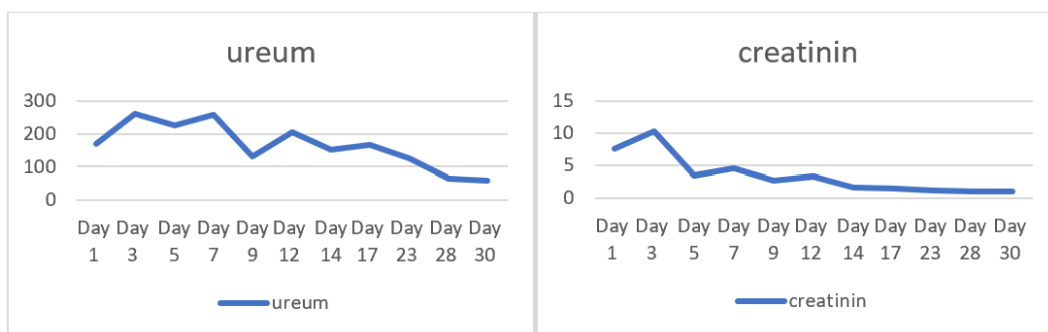


Figure 4. Kidney Function during Hospitalization

A rapid clinical response was observed within thirty days, with a marked reduction in edema and normalization kidney function. On the third day of hospitalization, hemodialysis was

initiated and subsequently followed by PLEX, resulting in clinical improvement. On the seventh day, a second hemodialysis session was performed in conjunction with PLEX. By the ninth day, a significant improvement in renal parameters was observed, with serum urea decreasing to 132 mg/dL and creatinine to 2.8 mg/dL. PLEX was continued with two additional sessions until day 30 of treatment, at which point renal function demonstrated marked recovery, with urea reduced to 60 mg/dL and creatinine to 1.0 mg/dL, demonstrating the effectiveness of the therapeutic regimen (Odler et al., 2025; Berti et al., 2019; Oberiukhtin et al., 2026). A favorable prognosis in such cases is supported by factors such as young age, early response therapy, and the absence of irreversible fibrosis on kidney biopsy. Studies have shown that combined therapy with steroids, hemodialysis, and PLEX achieves clinical remission in 60–70% of patients with anti GBM RPGN (Nakajima et al., 2025; Bell et al., 2021; Koirala et al., 2025).

Laboratory monitoring should include serum creatinine, estimated glomerular filtration rate (eGFR), electrolytes, and proteinuria every 1–2 weeks during the acute phase, then every 3–6 months thereafter. Serological testing, when available, should include ANCA and anti GBM antibodies to detect potential relapse (Aghsae Fard & Zahedian, 2024; Bell et al., 2021; Alberici et al., 2025).

## Conclusion

This case report describes a patient with RPGN. Negative serology for ANA and anti dsDNA excluded lupus, making pauci immune or idiopathic RPGN the most likely type. Aggressive therapy with high dose steroids, PLEX, and hemodialysis resulted in a rapid clinical response, increased the likelihood of remission, and preserved renal function. Routine laboratory monitoring, ANCA/anti GBM testing when available, and patient education on medication adherence and blood pressure control are essential to prevent relapse and long term complications.

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