DOI: 10.5152/turkjnephrol.2026.251306

Turkish Journal of Nephrology

Clinical Profile and Predictors of Kidney and Patient Outcomes in Anti–Glomerular Basement Membrane Disease: A 10-Year Single-Center Experience from South India

Jeeja Jaya Janardhanan, Namesh Kamat, Thotanolla Surya Prakash, Ram Rapur

Department of Nephrology, Sri Venkateswara Institute of Medical Sciences, Tirupati, India

ABSTRACT

Background: Anti–glomerular basement membrane (anti-GBM) disease is a rare, life-threatening cause of rapidly progressive glomerulonephritis, often presenting with pulmonary kidney syndrome and frequently progressing to end-stage kidney disease (ESKD) if diagnosis and treatment are delayed.

Methods: The authors conducted a retrospective observational study including all patients ≥ 10 years with serological and/or biopsy-proven anti-GBM disease treated at a tertiary care center in South India between January 2016 and January 2025. Demographic, clinical, serological, histopathological, and treatment data were collected, and predictors of dialysis independence, ESKD, and mortality were analyzed.

Results: Thirty-two patients were identified; mean age was 39.8 years, and 53% were male. At presentation, 81.3% required dialysis, mean serum creatinine was 9.15 mg/dL, and 25% had pulmonary hemorrhage; anti–neutrophil cytoplasmic antibody co-positivity was seen in 34.4%. All patients received plasma exchange plus corticosteroids and cyclophosphamide. At the last follow-up, 25% recovered kidney function, 62.5% were dialysis dependent, and 12.5% had died. Multivariable logistic regression analysis demonstrated that dialysis requirement at presentation and interstitial fibrosis on kidney biopsy were independent predictors of failure to achieve kidney recovery, while older age and pulmonary involvement predicted mortality.

Conclusions: In this single-center Indian cohort, anti-GBM disease was characterized by delayed presentation with advanced kidney failure, leading to low kidney recovery and high ESKD burden despite standard therapy. Early recognition, rapid referral, and timely initiation of plasma exchange–based immunosuppression are crucial to improve outcomes in resource-limited settings.

Keywords: Anti-GBM disease, crescentic glomerulonephritis, immunosuppression, plasma exchange, rapidly progressive glomerulonephritis

Corresponding author: Jeeja Jaya Janardhanan ✉ jeeja87here@gmail.com **Received:** December 31, 2025 **Revision Requested:** January 14, 2026 **Last Revision Received:** April 9, 2026 **Accepted:** May 2, 2026 **Publication Date:** October 9, 2026

Cite this article as: Janardhanan JJ, Kamat N, Prakash TS, Rapur R. Clinical profile and predictors of kidney and patient outcomes in anti–glomerular basement membrane disease: a 10-year single-center experience from South India. *Turk J Nephrol.* 2026;35(4):292-303.

INTRODUCTION

Anti–glomerular basement membrane (anti-GBM) disease is a rare but life-threatening autoimmune disorder caused by circulating antibodies against the α3 chain of type IV collagen in the glomerular and alveolar basement membranes.[1] It typically presents as rapidly progressive glomerulonephritis, with or without pulmonary hemorrhage (Goodpasture syndrome), and has an estimated incidence of 0.5-1.8 cases per million population per year.[2,3]

The disease results from antibody binding to the non-collagenous domain of the α3 chain, leading to complement activation and inflammatory injury. Kidney biopsy characteristically shows crescentic glomerulonephritis with linear immunoglobulin (IgG) deposition along the

This work is licensed under a Creative Commons Attribution 4.0 International License.

glomerular basement membrane on immunofluorescence, which is diagnostic of anti-GBM disease.[4,5]

Anti-GBM disease usually presents acutely with rapidly worsening kidney function, hematuria, and proteinuria, and pulmonary involvement occurs in about 40-60% of patients, ranging from mild hemoptysis to life-threatening diffuse alveolar hemorrhage.[6,7] Disease severity at presentation, especially the need for dialysis, is a key determinant of outcome and is associated with poor kidney prognosis.[8] Other adverse factors include older age, severe reduction in urine output, high serum creatinine at diagnosis, and extensive crescentic or chronic changes on kidney biopsy. A subset of patients with anti-GBM disease (10-40%) also have concurrent anti-neutrophil cytoplasmic antibody (ANCA) positivity, most commonly p-ANCA (perinuclear) or MPO-ANCA (myeloperoxidase).[9]

Standard therapy includes high-dose corticosteroids, cyclophosphamide, and daily plasma exchange until anti-GBM antibodies become undetectable.[10] Despite this approach, kidney outcomes remain poor in patients with advanced kidney failure at presentation, with dialysis-independence rates varying widely (8-95%) depending on disease severity and timing of treatment initiation.[11]

Long-term relapse rates in anti-GBM disease are low (<5%), but patients who fail to recover kidney function often develop end-stage kidney disease (ESKD) with substantial morbidity.[12] Although patient survival has improved with advances in supportive care and kidney replacement therapy, mortality remains higher than in the general population, especially among those with pulmonary-kidney syndrome.[13]

Data on anti-GBM disease from Indian tertiary care centers remain limited.[14]

This retrospective study describes a decade of experience with anti-GBM disease at a tertiary care center in South India. The authors analyzed patient demographics, clinical presentation, treatment, and kidney and patient outcomes and examined factors associated with dialysis independence and progression to ESKD. The authors also assessed pulmonary involvement, ANCA co-positivity, and histopathological findings.

MAIN POINTS

- Anti-GBM disease is an uncommon but serious cause of rapidly progressive kidney failure in South India.
- Most patients reached the hospital with severe kidney dysfunction, and the need for dialysis at presentation strongly predicted poor kidney outcomes.
- Better baseline kidney function and absence of chronic changes on biopsy were associated with recovery and dialysis independence.
- Lung involvement significantly increased the risk of death despite standard treatment.
- Early recognition and timely initiation of plasma exchange and immunosuppression are essential to improve patient outcomes.

MATERIAL AND METHODS

Study Design and Setting

This was a retrospective observational cohort study conducted at Sri Venkateswara Institute of Medical Sciences, Tirupathi, Andhra Pradesh, India. The Department of Nephrology provides comprehensive kidney care, including inpatient and outpatient services, hemodialysis, kidney biopsy with immunofluorescence, and plasma exchange therapy. All patients diagnosed with anti-GBM disease who presented to or were treated between January 2016 and January 2025 were included. This period allowed adequate case accrual and follow-up to evaluate long-term kidney and patient outcomes. Patients' demographic, laboratory values, physical findings and biopsy evaluations as well as treatment documents were retracted from their hospital recordings.

Inclusion Criteria

Patients were included in the study if they met all of the following criteria:

1. Age ≥ 10 years at presentation.
2. Clinical diagnosis of anti-GBM disease defined by at least one of the following:

- Positive circulating anti-GBM antibody detected by enzyme-linked immunosorbent assay or other immunoassay, OR
- Kidney biopsy demonstrating linear IgG staining along the glomerular basement membrane on immunofluorescence microscopy consistent with anti-GBM disease.

3. Presented to and/or received treatment during the study period.
4. Written informed consent, including parent or guardian consent for underage patients, was waived by the Institutional Ethics Committee because this was a retrospective study using anonymized clinical data.

Exclusion Criteria

Patients were excluded if they met any of the following criteria:

1. Insufficient medical records to extract key demographic, clinical, treatment, and outcome data.
2. Alternative primary diagnosis that could explain linear IgG deposition on kidney biopsy.
3. Patients referred only for a single consultation opinion with no treatment or follow-up data available.

Treatment Protocol

Treatment followed standard protocols based on KDIGO 2021 guidelines for anti-GBM disease.[15] All patients received standard

therapy consisting of high-dose corticosteroids, cyclophosphamide, and therapeutic plasma exchange. Intravenous methylprednisolone (500-1000 mg/day for 3 days) was administered, followed by oral prednisolone (0.5 mg/kg/day). Cyclophosphamide was administered intravenously in a dose of 500 mg/m^2 monthly for 6 doses. Plasma exchange was performed on alternate days, exchanging approximately 60 mL/kg plasma per session. Treatment was continued for 10-14 sessions or until anti-GBM antibodies became undetectable.

Anti-GBM antibody levels were reassessed following completion of 7 sessions of plasma exchange therapy. If positive at the end of 7 sessions, then titer was repeated at the end of the 10th and 14th sessions of plasma exchange.

Definitions

The following standardized definitions were used throughout the study:

Dialysis Independence at Discharge :

Patient not requiring kidney replacement therapy at the time of hospital discharge and not restarted on dialysis within 2 weeks of discharge.

Kidney Recovery Categorized as:

- Complete recovery: Return to baseline kidney function or eGFR > 60 mL/min/1.73 m^2 with serum creatinine within 20% of baseline.
- Partial recovery: Improvement in serum creatinine but persistent chronic kidney disease (eGFR < 60 mL/min/1.73 m^2) without dialysis dependence.

End-Stage Kidney Disease

Requirement for chronic dialysis (≥3 months) or kidney transplantation.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics version 26.0 (IBM SPSS Corp.; Armonk, NY, USA). Continuous variables were tested for normal distribution using the Shapiro–Wilk test and are presented as mean ± standard deviation or median with interquartile range as appropriate. Categorical variables are expressed as frequencies and percentages.

Age categories used in categorical analysis (<30, 31-40, 41-50, >50 years) were defined based on clinically relevant age strata commonly used in previous anti-GBM cohort studies.

Outcome analysis was performed using 2 approaches: comparison among 3 outcome groups (recovery, dialysis dependence, death) and binary comparison between survivors and non-survivors.

Comparisons between 2 groups were performed using Student's *t*-test or Mann–Whitney *U*-test for continuous variables and chi-square or Fisher's exact test for categorical variables. Comparisons among the 3 outcome groups (recovery, dialysis dependence, and death) were performed using one-way ANOVA or Welch's ANOVA when variance inequality was present.

Multivariable logistic regression analysis was performed to identify independent predictors of kidney recovery and mortality. Variables with $P < .10$ in univariate analysis or those considered clinically relevant were included in the multivariable model. Adjusted odds ratios (aOR) with 95% confidence intervals were calculated.

Kaplan–Meier survival analysis was performed to estimate patient survival and dialysis-free kidney survival.

Ethical Considerations

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. The Institutional Ethics Committee, Sri Venkateswara Institute of Medical Sciences, Tirupathi, Andhra Pradesh, India reviewed the study. Due to the retrospective observational design using anonymized clinical data, the committee granted a waiver of formal ethical approval according to institutional policy. Therefore, no ethics approval number was assigned.

RESULTS

Patient Characteristics and Demographics

A total of 38 patients with suspected anti-GBM disease were identified during the study period. Six patients were excluded due to incomplete records or alternative diagnoses, leaving 32 patients included in the final analysis (Figure 1). The demographic and baseline clinical characteristics of the cohort are summarized in Table 1. Seventeen patients (53.1%) were male and 15 (46.9%) were female.

Twenty-six patients (81.3%) required dialysis at presentation, while 6 patients (18.8%) did not require dialysis initially. All 32 patients (100%) received plasma exchange therapy as part of their treatment regimen.

Post-Treatment Serological Response

After completion of plasma exchange therapy, anti-GBM antibody status was assessed. Nine patients (28.1%) achieved negative anti-GBM antibody status, 21 patients (65.6%) remained positive for anti-GBM antibodies, and 2 patients (6.3%) had no post-treatment testing documented (these were patients who died). Among patients tested for p-ANCA after plasma exchange, 27 patients (84.4%) were negative and 5 patients (15.6%) remained positive.

Outcomes

Figure 2 shows the outcome of patients with anti-GBM disease. Of the 26 patients who required dialysis at presentation, only 8 patients (25% of total cohort) achieved dialysis independence

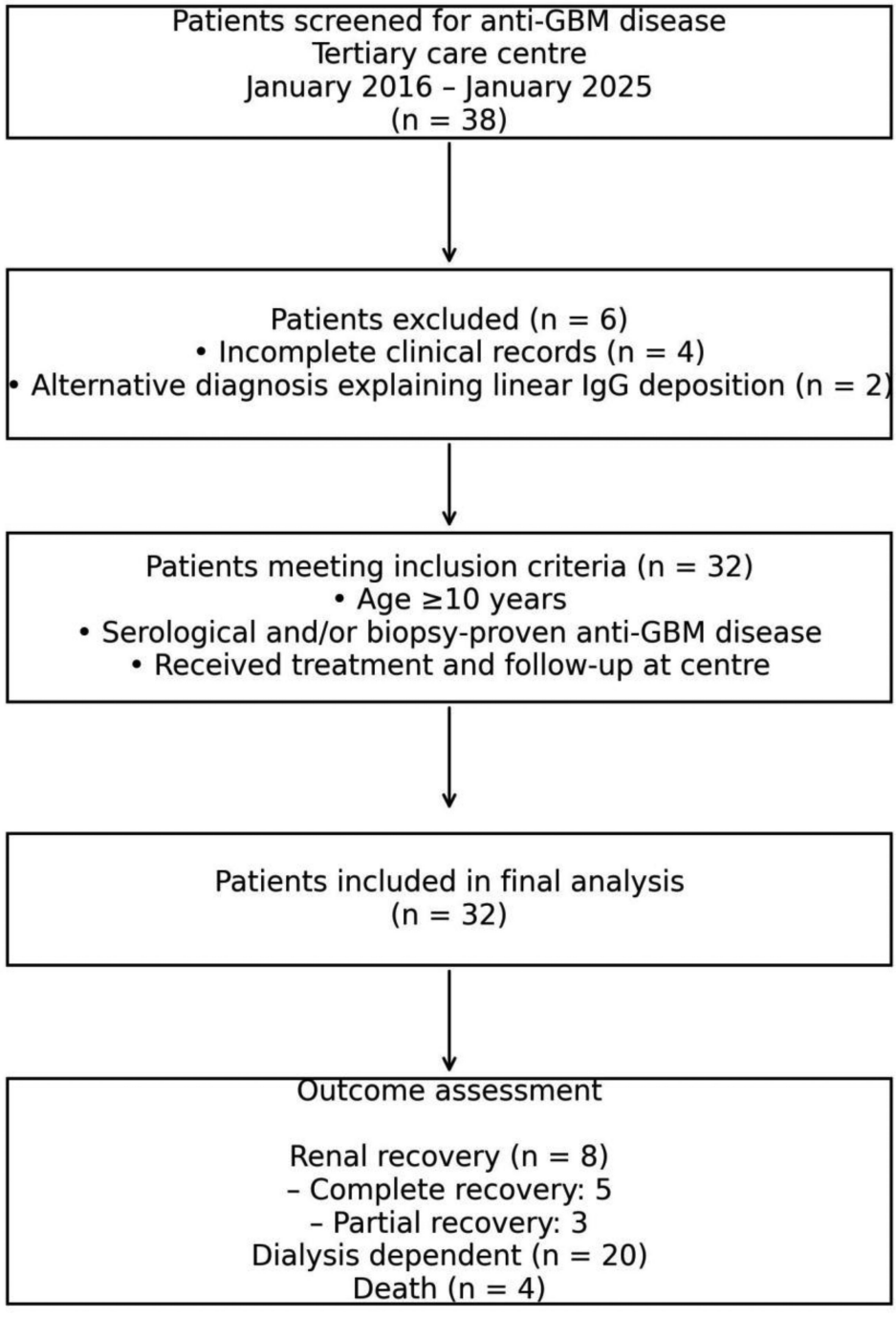

Figure 1. Flow diagram showing patient screening, exclusion, inclusion, and outcomes in the anti–glomerular basement membrane disease cohort (2016-2025).

and were off dialysis at discharge or during follow-up. Twenty patients (62.5%) remained dialysis dependent.

Overall Outcomes

At the end of follow-up, The outcomes at the end of the follow-up are presented in in Figure 1.

Among the 8 patients who recovered kidney function, 5 achieved complete recovery (Figure 1).

Predictors of Outcomes

Tables 2-5 show the predictors of outcomes.

Multivariable logistic regression analysis, as shown in Table 6, demonstrated that dialysis requirement at presentation (aOR 0.06, 95% CI 0.01-0.58, P = .015) and presence of interstitial fibrosis on kidney biopsy (aOR 0.08, 95% CI 0.01-0.72, P = .024) were independent predictors of failure to achieve kidney recovery. Pulmonary hemorrhage, baseline serum creatinine, and

Table 1. Baseline Characteristics

	Mean ± SD	Median (IQR)	Range
Age (years)	39.78 ± 14.94	41 (27-50.25)	10-71
Systolic blood pressure (mm of Hg)	154.69 ± 24.23	160 (142.5-170)	100-200
Diastolic blood pressure (mm of Hg)	93.13 ± 12.03	100 (90-100)	70-120
Hemoglobin (g/dL)	7.64 ± 1.4	7.7 (7-8.78)	4.4-10
Serum creatinine at presentation (mg/dL)	9.15 ± 4.38	10 (5.1-12.8)	1.7-15.9
Total leucocyte count (cells/mm^3)	11159.38 ± 3966.22	11000 (8925-12 875)	1200-21 000
eGFR at presentation (mL/min/1.73 m^2)	10.06 ± 10.96	6 (4-11.5)	3-55
Serum albumin (g/dL)	3.31 ± 0.39	3.25 (3-3.5)	2.7-4.1
Serum C3 (mg/dL)	189.1 ± 440.26	108 (100-124.43)	40.46-2590
Anti-GBM titer at presentation (Units/mL)	166.88 ± 151.52	100 (100-400)	20-400
24-hour urine protein (mg/day)	1519.25 ± 1137.79	1135 (985-1637.5)	451-5770
Cellular crescents (%)	29.09 ± 38.39	0 (0-50)	0-100
Fibrocellular crescents (%)	47.44 ± 40.76	50 (0-100)	0-100
Fibrous crescents (%)	3.2 ± 17.38	0 (0-0)	0-100

eGFR, estimated glomerular filtration rate; GBM, glomerular basement membrane.

age were not independently associated with kidney recovery after adjustment for other variables.

Because there were only 4 deaths, only 2 predictors couldsafely be included in the model (rule of 10 events per variable).

Multivariable logistic regression, as shown in Supplementary Table 1, identified older age (aOR 1.08 per year, 95% CI 1.01-1.16, P = .031) and pulmonary hemorrhage (aOR 4.52, 95% CI 1.12-18.2, P = .034) as independent predictors of mortality.

Kaplan–Meier survival analysis demonstrated that patient survival, as shown in Figure 3, remained relatively high during follow-up, with survival probabilities of 94% at 1 month and 89% at 3 months. All deaths occurred within the first 2 months after diagnosis. Kidney survival analysis showed a dialysis-free survival of 25% at 1 year, reflecting the severe kidney dysfunction at presentation in most patients.

DISCUSSION

This 10-year retrospective study describes a single-center Indian cohort of anti-GBM disease. The findings outline the clinical presentation, treatment, and outcomes of this rare but severe condition and highlight challenges specific to the Indian tertiary care setting, while also allowing comparison with international experience.

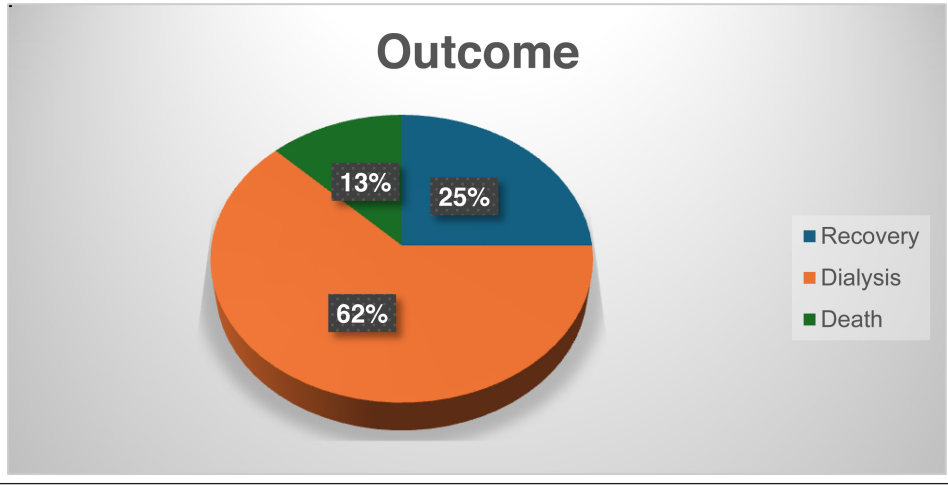

Figure 2. Outcome of patients with anti–glomerular basement membrane disease.

Table 2. Baseline Clinical and Pathological Characteristics According to Kidney Outcome Group (Recovery, Dialysis Dependence, Death)

			Outcome				
	Categories	N	Recovery (n (%))	Dialysis (n (%))	Death (n (%))	Chi-Square	*P*
Age categories, years	<30	12	4 (50)	8 (40)	0 (0)	5.307	.505
	31-40	2	1 (12.5)	1 (5)	0 (0)		
	41-50	10	1 (12.5)	7 (35)	2 (50)		
	>50	8	2 (25)	4 (20)	2 (50)		
Gender	Female	15	4 (50)	9 (45)	2 (50)	0.075	.963
	Male	17	4 (50)	11 (55)	2 (50)		
Smoking	No	26	8 (100)	16 (80)	2 (50)	4.431	.109
	Yes	6	0 (0)	4 (20)	2 (50)		
Anuria/oliguria	No	3	2 (25)	1 (5)	0 (0)	3.163	.206
	Yes	29	6 (75)	19 (95)	4 (100)		
ANCA	c-ANCA	1	0 (0)	1 (5)	0 (0)	6.343	.175
	Negative	21	3 (37.5)	14 (70)	4 (100)		
	p-ANCA+	10	5 (62.5)	5 (25)	0 (0)		
Pulmonary hemorrhage	No	24	7 (87.5)	16 (80)	1 (25)	6.267	**.044**
	Yes	8	1 (12.5)	4 (20)	3 (75)		
Dialysis	No	6	5 (62.5)	1 (5)	0 (0)	13.456	**.001**
	Yes	26	3 (37.5)	19 (95)	4 (100)		
PLEX	Yes	32	8 (100)	20 (100)	4 (100)	.	.
Immunosuppression	Yes	32	8 (100)	20 (100)	4 (100)	.	.
Interstitial fibrosis	No	13	8 (100)	5 (25)	0 (0)	16.453	**<.001**
	Yes	19	0 (0)	15 (75)	4 (100)		
Tubular atrophy	No	12	7 (87.5)	5 (25)	0 (0)	12.267	**.002**
	Yes	20	1 (12.5)	15 (75)	4 (100)		
Dialysis dependency at 1 year	No	8	8 (100)	0 (0)	0 (0)	64	**<.001**
	Not applicable	4	0 (0)	0 (0)	4 (100)		
	Yes	20	0 (0)	20 (100)	0 (0)		
Associations	ANCA vasculitis	11	5 (62.5)	6 (30)	0 (0)	5.07	.079
	None	21	3 (37.5)	14 (70)	4 (100)		
ANA	Equivocal	1	1 (12.5)	0 (0)	0 (0)	9.904	**.042**
	Negative	27	4 (50)	19 (95)	4 (100)		
	Positive	4	3 (37.5)	1 (5)	0 (0)		
ANA profile	Negative	30	7 (87.5)	19 (95)	4 (100)	3.653	.455
	Smd+	1	1 (12.5)	0 (0)	0 (0)		
	SS-A/SS-B +	1	0 (0)	1 (5)	0 (0)		
Anti-Ds DNA	Negative	32	8 (100)	20 (100)	4 (100)	.	.
Anti-GBM after PLEX	Negative	9	8 (100)	1 (5)	0 (0)	41.651	**<.001**
	None	2	0 (0)	0 (0)	2 (50)		

(*Continued*)

Table 2. Baseline Clinical and Pathological Characteristics According to Kidney Outcome Group (Recovery, Dialysis Dependence, Death) (*Continued*)

			Outcome				
	Categories	N	Recovery (n (%))	Dialysis (n (%))	Death (n (%))	Chi-Square	*P*
	Positive	21	0 (0)	19 (95)	2 (50)		
p-ANCA after PLEX	Negative	27	8 (100)	15 (75)	4 (100)	3.556	.169
	Positive	5	0 (0)	5 (25)	0 (0)		
Comorbidity	DM/HTN	2	0 (0)	1 (5)	1 (25)	6.48	.372
	HTN	5	1 (12.5)	3 (15)	1 (25)		
	No	24	6 (75)	16 (80)	2 (50)		
	HIV	1	1 (12.5)	0 (0)	0 (0)		

ANA, antinuclear antibody; ANCA, anti-neutrophil cytoplasmic antibody; DM, diabetes mellitus; GFR, estimated glomerular filtration rate; GBM, glomerular basement membrane; HIV, human immunodeficiency virus; HTN, systemic hypertension; PLEX, plasma exchange.
Bold values indicate statistical significance ($P < .05$).

Demographic and Clinical Characteristics

The mean age at presentation in the cohort was 39.78 years, which is consistent with the bimodal age distribution typically described for anti-GBM disease, with peaks in the third and sixth decades of life.[16] However, the cohort showed a relatively younger age profile compared to some Western series, which may reflect demographic differences in the Indian population or referral patterns to the tertiary center.[17]

The clinical presentation in the cohort was characterized by severe acute kidney injury, with 90.6% of patients presenting with oliguria or anuria and a mean serum creatinine of 9.15 mg/dL. The mean eGFR at presentation was only 10.06 mL/min/1.73 m^2, indicating that most patients had advanced kidney injury by the time of diagnosis. This delayed presentation may be attributed to several factors, including limited awareness of the disease among primary care physicians, lack of access to specialized diagnostic facilities in peripheral centers, and socioeconomic barriers to seeking timely medical care.[18]

Pulmonary Involvement

Pulmonary involvement, manifested as pulmonary hemorrhage, was documented in 25% of the patients. This frequency is lower than the 40-60% reported in many Western series.[19] The lower rate of pulmonary involvement may reflect true epidemiological differences, under-recognition of mild hemorrhage, or survival bias. In contrast, pulmonary hemorrhage was strongly associated with poor outcomes, with 75% of affected patients dying during follow-up ($P = .044$ for overall outcome; $P = .014$ for mortality). This finding underscores the grave prognosis associated with pulmonary-kidney syndrome and emphasizes the need for aggressive early intervention in such cases.[20]

Anti-Neutrophil Cytoplasmic Antibody Co-Positivity

Anti-neutrophil cytoplasmic antibody co-positivity was present in 34.4% of the patients (31.3% p-ANCA positive, 3.1% c-ANCA positive), which falls within the reported range of 10-40% in the literature.[21] The predominance of p-ANCA over c-ANCA is consistent with previous reports showing that MPO-ANCA is more commonly associated with anti-GBM disease than PR3-ANCA.[22] Although some studies suggest better kidney outcomes in ANCA-positive patients,[23] there was no statistically significant association between ANCA status and outcome in the cohort ($P = .175$). However, p-ANCA positivity was more frequent among patients who recovered than those who remained dialysis dependent, suggesting a possible trend limited by small sample size.

Histopathological Findings

The mean percentage of cellular crescents was 29.09%, while fibrocellular crescents averaged 47.44%. Notably, patients who achieved kidney recovery had significantly higher percentages of cellular crescents (75.75% vs. 14.6% in dialysis group, $P < .001$) and significantly lower percentages of fibrocellular crescents (6.25% vs. 60.9% in dialysis group, $P = .002$). This finding aligns with the established understanding that cellular crescents represent acute, potentially reversible injury, while fibrocellular and fibrous crescents indicate more advanced, irreversible damage.[24]

Interstitial fibrosis and tubular atrophy were strongly associated with poor outcomes, with all recovered patients showing no interstitial fibrosis ($P < .001$) and 87.5% having no tubular atrophy ($P = .002$). The high prevalence of interstitial fibrosis (59.4%) and tubular atrophy (62.5%) in the cohort suggests delayed presentation with irreversible nephron loss, a recognized predictor of poor prognosis in crescentic glomerulonephritis.[25]

Treatment and Outcomes

All patients received plasma exchange with corticosteroids and cyclophosphamide in line with current guidelines.[15] Despite this, only 25% achieved dialysis independence, while 62.5% progressed to ESKD and 12.5% died. These outcomes are poorer than reports from developed countries showing 40-50% kidney recovery[26] but are consistent with cohorts presenting

Table 3. Comparison of Categorical Variables Between Survivors and Non-survivors

	Categories	N	Death		Chi-Square	*P*
			Alive (n (%))	Death (n (%))		
Age categories	<30	12	12 (42.9)	0 (0)	3.657	.301
	31-40	2	2 (7.1)	0 (0)		
	41-50	10	8 (28.6)	2 (50)		
	>50	8	6 (21.4)	2 (50)		
Gender	Female	15	13 (46.4)	2 (50)	0.018	.893
	Male	17	15 (53.6)	2 (50)		
Smoking	No	26	24 (85.7)	2 (50)	2.93	.087
	Yes	6	4 (14.3)	2 (50)		
Anuria/oliguria	No	3	3 (10.7)	0 (0)	0.473	.492
	Yes	29	25 (89.3)	4 (100)		
ANCA	c-ANCA	1	1 (3.6)	0 (0)	2.395	.302
	Negative	21	17 (60.7)	4 (100)		
	p-ANCA+	10	10 (35.7)	0 (0)		
Pulmonary hemorrhage	No	24	23 (82.1)	1 (25)	6.095	**.014**
	Yes	8	5 (17.9)	3 (75)		
Dialysis	No	6	6 (21.4)	0 (0)	1.055	.304
	Yes	26	22 (78.6)	4 (100)		
PLEX	Yes	32	28 (100)	4 (100)	.	.
Immunosuppression	Yes	32	28 (100)	4 (100)	.	.
Interstitial fibrosis	No	13	13 (46.4)	0 (0)	3.128	.077
	Yes	19	15 (53.6)	4 (100)		
Tubular atrophy	No	12	12 (42.9)	0 (0)	2.743	.098
	Yes	20	16 (57.1)	4 (100)		
Dialysis dependency at 1 year	No	8	8 (28.6)	0 (0)	32	**<.001**
	Not applicable	4	0 (0)	4 (100)		
	Yes	20	20 (71.4)	0 (0)		
Outcome	Recovery	8	8 (28.6)	0 (0)	32	**<.001**
	Dialysis	20	20 (71.4)	0 (0)		
	Death	4	0 (0)	4 (100)		
Associations	ANCA vasculitis	11	11 (39.3)	0 (0)	2.395	.122
	None	21	17 (60.7)	4 (100)		
ANA	Equivocal	1	1 (3.6)	0 (0)	0.847	.655
	Negative	27	23 (82.1)	4 (100)		
	Positive	4	4 (14.3)	0 (0)		
ANA profile	Negative	30	26 (92.9)	4 (100)	0.305	.859
	Smd+	1	1 (3.6)	0 (0)		
	SS-A/SS-B +	1	1 (3.6)	0 (0)		
Anti-Ds DNA	Negative	32	28 (100)	4 (100)	.	.
Anti-GBM after PLEX	Negative	9	9 (32.1)	0 (0)	15.456	**<.001**
	None	2	0 (0)	2 (50)		
	Positive	21	19 (67.9)	2 (50)		
p-ANCA after PLEX	Negative	27	23 (82.1)	4 (100)	0.847	.358
	Positive	5	5 (17.9)	0 (0)		

ANA, antinuclear antibody; ANCA, anti-neutrophil cytoplasmic antibody; eGFR, estimated glomerular filtration rate; GBM, glomerular basement membrane; PLEX, plasma exchange.
Bold values indicate statistical significance ($P < .05$).
Bold values indicate statistical significance ($P < .05$).

Table 4. Comparison of Continuous Variables Between Survivors and Non-Survivors Using an Independent *t*-Test

Parameter	Alive		Death		*t*	*P*
	n	Mean ± SD	n	Mean ± SD		
Age (years)	28	37.29 ± 13.81	4	57.25 ± 11.3	−2.751	**.01**
SBP (mm of Hg)	28	153.93 ± 25.73	4	160 ± 8.17	−0.463	.647
DBP (mm of Hg)	28	92.86 ± 12.72	4	95 ± 5.77	−0.328	.745
Hemoglobin (g/dL)	28	7.66 ± 1.4	4	7.45 ± 1.54	0.283	.779
Serum creatinine at presentation (mg/dL)	28	9.07 ± 4.49	4	9.7 ± 4.09	−0.265	.793
Total count (cells/mm^3)	28	11 189.29 ± 3573.6	4	10 950 ± 6895.65	0.111	.912
eGFR at presentation (mL/min/1.73 m^2)	28	10.68 ± 11.6	4	5.75 ± 1.5	0.837	.409
Albumin (g/dL)	28	3.35 ± 0.39	4	3.03 ± 0.33	1.599	.12
Serum C3 (mg/dL)	28	201.73 ± 470.34	4	100.75 ± 6.18	0.423	.675
Anti-GBM titer at presentation (Units/mL)	28	165.71 ± 154.43	4	175 ± 150	−0.113	.911
24-hour urine protein (mg/day)	28	1609.07 ± 1189.65	4	890.5 ± 193.5	1.189	.244
Cellular crescents (%)	28	32.07 ± 39.86	4	8.25 ± 16.5	2.132	.061
Fibrocellular crescents (%)	28	45.29 ± 40.17	4	62.5 ± 47.87	−0.785	.438
Fibrous crescents (%)	28	3.65 ± 18.88	4	7.5 ± 15	0.374	.711

DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; GBM, glomerular basement membrane; SBP, systolic blood pressure.
Bold values indicate statistical significance ($P < .05$).

with severe disease,[27] reflecting advanced kidney failure at presentation, with 81.3% requiring dialysis and a mean serum creatinine > 9 mg/dL. The low kidney recovery observed in the cohort reflects the severity of disease at presentation, with the majority of patients requiring dialysis and demonstrating extensive chronic changes on kidney biopsy.

Table 5. Comparison of Continuous Variables Across Kidney Outcome Groups (Recovery, Dialysis Dependence, Death)

	Recovery (n = 8) Mean ± SD	Dialysis (n = 20) Mean ± SD	Death (n = 4) Mean ± SD	*F*/Welch Statistics*	*P*
Age (years)	36.25 ± 13.82	37.7 ± 14.14	57.25 ± 11.3	3.698	**.037**
SBP (mm of Hg)	137.5 ± 24.93	160.5 ± 23.5	160 ± 8.17	3.038	.063
DBP (mm of Hg)	87.5 ± 11.65	95 ± 12.77	95 ± 5.77	1.179	.322
Hemoglobin (g/dL)	7.35 ± 1.49	7.79 ± 1.39	7.45 ± 1.54	0.31	.736
Serum creatinine at presentation (mg/dL)	4.62 ± 3.46	10.85 ± 3.55	9.7 ± 4.09	8.657	**.001**
Total count (cells/mm^3)	12087.5 ± 4748.67	10830 ± 3060.12	10950 ± 6895.65	0.28	.758
eGFR at presentation(mL/min/1.73 m^2)	22.25 ± 16.52	6.05 ± 3.41	5.75 ± 1.5	3.709*	.058
Albumin (g/dL)	3.39 ± 0.43	3.34 ± 0.38	3.03 ± 0.33	1.293	.29
Serum C3 (mg/dL)	410.74 ± 880.81	118.12 ± 52.74	100.75 ± 6.18	1.439*	.27
Anti-GBM titer at presentation	145 ± 161.69	174 ± 154.93	175 ± 150	0.105	.901
24-hour urine protein (mg/day)	1945.38 ± 1433.53	1474.55 ± 1089.67	890.5 ± 193.5	1.203	.315
Cellular crescents (%)	75.75 ± 35.78	14.6 ± 25.71	8.25 ± 16.5	15.159	**<.001**
Fibrocellular crescents (%)	6.25 ± 17.68	60.9 ± 35.78	62.5 ± 47.87	14.29*	**.003**
Fibrous crescents (%)	0 ± 0	5.11 ± 22.33	7.5 ± 15	0.298	.74

DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; GBM, glomerular basement membrane; SBP, systolic blood pressure.
*Welch test.
Bold values indicate statistical significance ($P < .05$).

Table 6. Multivariable Logistic Regression Analysis for Predictors of Kidney Recovery in Anti-GBM Disease

Variable	Adjusted Odds Ratio	95% Confidence Interval	*P*
Dialysis requirement at presentation	0.06	0.01-0.58	.015
Interstitial fibrosis	0.08	0.01-0.72	.024
Pulmonary hemorrhage	0.34	0.05-2.19	.260
Baseline serum creatinine (per mg/dL increase)	0.90	0.69-1.17	.420
Age (per year increase)	1.01	0.95-1.07	.770

Predictors of Outcome

Our analysis identified several important predictors of outcome:

Dialysis requirement at presentation was the strongest predictor of poor kidney outcome ($P = .001$). consistent with previous studies identifying dialysis dependence at presentation as a key prognostic factor in anti-GBM disease.[19]

Baseline kidney function strongly influenced outcomes. Patients who recovered had significantly lower serum creatinine and higher eGFR compared with those who remained dialysis-dependent, underscoring the importance of early diagnosis and timely treatment to prevent irreversible kidney damage.

Histopathological findings were strongly associated with outcomes. A higher proportion of cellular crescents was linked to better recovery, whereas fibrocellular crescents, interstitial fibrosis, and tubular atrophy were associated with poor outcomes, reflecting the balance between reversible acute injury and irreversible chronic damage in determining kidney recovery.

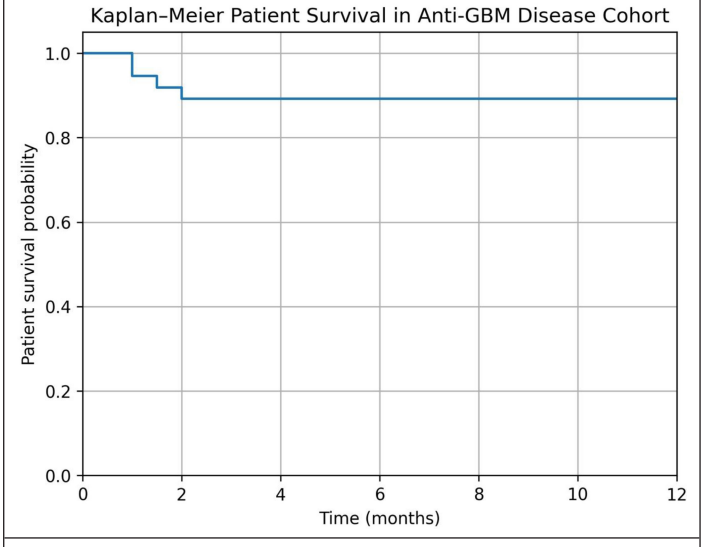

Figure 3. Kaplan–Meier curve showing patient survival in patients with anti–glomerular basement membrane disease.

Post-treatment anti-GBM antibody status was strongly associated with outcome ($P < .001$). All patients who recovered became antibody-negative after plasma exchange, whereas none with persistently positive antibodies achieved recovery, highlighting the importance of achieving serological remission and continuing therapy until antibodies are undetectable.

Older age was associated with higher mortality ($P = .01$), with patients who died being significantly older than survivors (57.25 vs. 37.29 years).

Pulmonary involvement was also linked to poor overall outcome ($P = .044$) and increased mortality ($P = .014$), with a 75% death rate among patients presenting with hemoptysis, highlighting the severity of pulmonary-kidney syndrome.

These findings remained significant in multivariable logistic regression analysis, confirming dialysis requirement at presentation and interstitial fibrosis as independent predictors of kidney recovery, while older age and pulmonary hemorrhage independently predicted mortality.

Data on anti-GBM disease from India are limited. Studies from PGIMER Chandigarh and CMC Vellore reported dialysis independence rates of 20% and 33%, respectively,[28,29] comparable to the findings and suggesting that delayed presentation with severe disease is a common challenge across Indian centers.

In contrast, recent international cohorts from the United Kingdom and France have reported higher kidney recovery rates of 40% and 47%, respectively.[30] These differences are likely related to earlier presentation and less severe disease at diagnosis rather than differences in treatment strategies.

At 1 year follow-up, 71.4% of survivors remained dialysis-dependent, representing progression to ESKD. This high rate of ESKD reflects the severity of initial presentation and the limited potential for kidney recovery once extensive irreversible damage has occurred. These patients will require long-term kidney replacement therapy with dialysis or kidney transplantation.

Implications for Clinical Practice

Our findings have important implications for clinical practice in resource-limited settings. Early recognition of rapidly progressive glomerulonephritis and prompt testing for anti-GBM antibodies are essential to avoid irreversible kidney damage. Delays in referral to centers capable of plasma exchange remain a major barrier to kidney recovery. The data also support early prognostic counseling, particularly for patients requiring dialysis at presentation or showing chronic changes on biopsy. Achieving post-treatment anti-GBM antibody negativity should be a key therapeutic goal. Future studies should focus on

multicenter data, earlier diagnostic pathways, and strategies to improve outcomes in patients with severe disease.

This study has several limitations. First, the retrospective design introduces potential selection bias. Second, the sample size is relatively small due to the rarity of anti-GBM disease. Third, this was a single-center study, which may limit generalizability. Fourth, the time interval between onset of symptoms and diagnosis was not consistently documented in the medical records and therefore could not be analyzed. Delayed diagnosis is likely to have contributed to the severe kidney dysfunction observed at presentation in many patients. Finally, although multivariable analyses were performed, the limited number of outcome events reduces statistical power.

This 10-year retrospective study from SVIMS, Tirupati, describes a single-center Indian cohort of anti-GBM disease and highlights the clinical presentation, treatment, and outcomes in a resource-limited setting. Despite standard therapy, delayed presentation was associated with poor kidney outcomes, underscoring the importance of early recognition, rapid referral, and timely initiation of plasma exchange–based immunosuppression. The findings provide useful outcome benchmarks and identify key prognostic factors that can aid clinical decision-making and patient counseling in similar settings.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that no Artificial Intelligence Tool was used in the preparation of the manuscript.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of The Institutional Ethics Committee of SVIMS, Tirupati (Approval No.: 1233; Date: March 14, 2025).

Informed Consent: N/A.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – J.J.J.; Design – J.J.J.; Supervision – R.R.; Resources – N.K.; Materials – N.K.; Data Collection and/or Processing – T.S.P.; Analysis and/or Interpretation – J.J.J., N.K.; Literature Search – N.K., J.J.J.; Writing Manuscript – J.J., N.K., T.S.P.; Critical Review – R.R.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: The authors declared that this study has received no financial support.

REFERENCES

1. Pedchenko V, Bondar O, Fogo AB, et al. Molecular architecture of the Goodpasture autoantigen in anti-GBM nephritis. *N Engl J Med*. 2010;363(4):343-354. [CrossRef]
2. Goodpasture EW. The significance of certain pulmonary lesions in relation to the etiology of influenza. *Am J Med Sci*. 1919;158(6):863-870. [CrossRef]
3. Savage CO, Pusey CD, Bowman C, et al. Antiglomerular basement membrane antibody mediated disease in the British Isles 1980-4. *Br Med J (Clin Res Ed)*. 1986;292(6516):301-304. [CrossRef]
4. Hudson BG, Tryggvason K, Sundaramoorthy M, et al. Alport's syndrome, Goodpasture's syndrome, and type IV collagen. *N Engl J Med*. 2003;348(25):2543-2556. [CrossRef]
5. Salama AD, Levy JB, Lightstone L, et al. Goodpasture's disease. *Lancet*. 2001;358(9285):917-920. [CrossRef]
6. Lazor R, Bigay-Gamé L, Cottin V, et al. Alveolar hemorrhage in antibasement membrane antibody disease: a series of 28 cases. *Med (Baltim)*. 2007;86(3):181-193. [CrossRef]
7. Hellmark T, Segelmark M. Diagnosis and classification of Goodpasture's disease (anti-GBM). *J Autoimmun*. 2014;48-49:108-112. [CrossRef]
8. Levy JB, Turner AN, Rees AJ, et al. Long-term outcome of anti-glomerular basement membrane antibody disease treated with plasma exchange and immunosuppression. *Ann Intern Med*. 2001;134(11):1033-1042. [CrossRef]
9. Rutgers A, Slot M, van Paassen P, et al. Coexistence of anti-glomerular basement membrane antibodies and myeloperoxidase-ANCAs in crescentic glomerulonephritis. *Am J Kidney Dis*. 2005;46(2):253-262. [CrossRef]
10. Johnson JP, Moore J Jr, Austin HA 3rd, et al. Therapy of anti-glomerular basement membrane antibody disease: analysis of prognostic significance of clinical, pathologic and treatment factors. *Med (Baltim)*. 1985;64(4):219-227. [CrossRef]
11. Alchi B, Griffiths M, Sivalingam M, et al. Predictors of renal and patient outcomes in anti-GBM disease: clinicopathologic analysis of a two-centre cohort. *Nephrol Dial Transplant*. 2015;30(5):814-821. [CrossRef]
12. Tang W, McDonald SP, Hawley CM, et al. Anti-glomerular basement membrane antibody disease is an uncommon cause of end-stage renal disease. *Kidney Int*. 2013;83(3):503-510. [CrossRef]
13. Canney M, O'Hara PV, McEvoy CM, et al. Spatial and temporal clustering of anti-glomerular basement membrane disease. *Clin J Am Soc Nephrol*. 2016;11(8):1392-1399. [CrossRef]
14. Datta SK, Kumar V, Pathak M, et al. Anti-glomerular basement membrane disease: a single center experience. *Indian J Nephrol*. 2015;25(3):162-167.
15. Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGO 2021 clinical practice guideline for the management of glomerular diseases. *Kidney Int*. 2021;100(4S):S1-S276. [CrossRef]
16. Donaghy M, Rees AJ. Cigarette smoking and lung haemorrhage in glomerulonephritis caused by autoantibodies to glomerular basement membrane. *Lancet*. 1983;2(8364):1390-1393. [CrossRef]
17. Williamson SR, Phillips CL, Andreoli SP, et al. A 25-year experience with pediatric anti-glomerular basement membrane disease. *Pediatr Nephrol*. 2011;26(1):85-91. [CrossRef]
18. Modi GK, Jha V. The incidence of end-stage renal disease in India: a population-based study. *Kidney Int*. 2006;70(12):2131-2133. [CrossRef]
19. Merkel F, Pullig O, Marx M, et al. Course and prognosis of anti-basement membrane antibody (anti-BM-Ab)-mediated disease: report of 35 cases. *Nephrol Dial Transplant*. 1994;9(4):372-376. [CrossRef]

20. Zamora MR, Warner ML, Tuder R, et al. Diffuse alveolar hemorrhage and systemic lupus erythematosus. Clinical presentation, histology, survival, and outcome. *Med (Baltim)*. 1997;76(3):192-202. [CrossRef]
21. Bosch X, Mirapeix E, Font J, et al. Prognostic implication of anti-neutrophil cytoplasmic autoantibodies with myeloperoxidase specificity in anti-glomerular basement membrane disease. *Clin Nephrol*. 1991;36(3):107-113.
22. Short AK, Esnault VL, Lockwood CM. Anti-neutrophil cytoplasm antibodies and anti-glomerular basement membrane antibodies: two coexisting distinct autoreactivities detectable in patients with rapidly progressive glomerulonephritis. *Am J Kidney Dis*. 1995; 26(3):439-445. [CrossRef]
23. Segelmark M, Hellmark T. Anti-glomerular basement membrane disease: an update on subgroups, pathogenesis and therapies. *Nephrol Dial Transplant*. 2019;34(11):1826-1832. [CrossRef]
24. Jennette JC. Rapidly progressive crescentic glomerulonephritis. *Kidney Int*. 2003;63(3):1164-1177. [CrossRef]
25. Berden AE, Ferrario F, Hagen EC, et al. Histopathologic classification of ANCA-associated glomerulonephritis. *J Am Soc Nephrol*. 2010;21(10):1628-1636. [CrossRef]
26. Touzot M, Poisson J, Faguer S, et al. Rituximab in anti-GBM disease: a retrospective study of 8 patients. *J Autoimmun*. 2015;60:74-79. [CrossRef]
27. Peto P, Salama AD. Update on antiglomerular basement membrane disease. *Curr Opin Rheumatol*. 2011;23(1):32-37. [CrossRef]
28. Prabhakar D, Rathi M, Nada R, et al. Anti-glomerular basement membrane disease: case series from a tertiary center in North India. *Indian J Nephrol*. 2017;27(2):108-112. [CrossRef]
29. Alexander S, Yusuf S, Rajan G, et al. Crescentic glomerulonephritis: what's different in South Asia? A single center observational cohort study. *Wellcome Open Res*. 2020;5:164. [CrossRef]
30. McAdoo SP, Pusey CD. Anti-glomerular basement membrane disease - treatment standard. *Nephrol Dial Transplant*. 2025;41(1):42-54. [CrossRef]

Supplementary Table 1. Multivariable Logistic Regression for Predictors of Mortality			
Variable	**Adjusted Odds Ratio**	**95% CI**	***P* Value**
Age (per year increase)	**1.08**	**1.01-1.16**	**.031**
Pulmonary hemorrhage	**4.52**	**1.12-18.2**	**.034**