Anti-Glomerular Basement Membrane Disease Combined with IgA Nephropathy: A Case Report and Literature Review

Limin Wang,[1,2#] Meichun Huang,[2#] Jun Liu,[3] Xiuxiu Li,[2] Liping Zhao[2*]

[1]Department of Nephrology, Tongxiang First People's Hospital, Tongxiang, 314500, Zhejiang, China
[2]Renal Department, Tongde Hospital of Zhejiang Province, Hangzhou, 310012, China
[3]Department of Pathology, Tongde Hospital of Zhejiang Province, Hangzhou, 310012, China

[#]Both authors contributed equally to this work and should be considered as equal first coauthors.

Keywords. Anti-glomerular basement membrane disease; IgA nephropathy; Anti-glomerular basement membrane antibody

Anti-glomerular basement membrane (GBM) disease is a rare autoimmune disease characterized by injury to small blood vessels in the kidneys and/or lungs. IgA nephropathy is the most common primary glomerular disease diagnosed in China. However, the co-occurrence of anti-GBM illness and IgA nephropathy has been infrequently documented in the literature. At present, its mechanism is unknown, the treatment approach is unclear, and the prognosis remains poor. Herein, we report a case of anti-GBM disease complicated with IgA nephropathy, in which renal function returned to normal state following treatment with a combination of prednisone and cyclophosphamide. This case suggests that in patients with anti-GBM disease and IgA nephropathy, the combination of glucocorticoids and cyclophosphamide may serve as an optimum treatment choice.

IJKD 2025;19:361-8
www.ijkd.org

Case Report

INTRODUCTION

Anti-glomerular basement membrane (GBM) disease is a rare autoimmune disease, with reported incidence rate of 1.64 cases per million people/year.[1] It is characterized by the presence of pathogenic antibodies targeting type IV collagen in the GBM as well as in the alveolar basement membrane.[2-3] Clinically, 80%-90% of patients present with rapidly progressive glomerulonephritis (RPGN), whereas 40%-60% of patients present with Goodpasture's syndrome,[2] both of which are associated with poor prognosis and high mortality. The pathogenic autoantibodies in anti-GBM disease are generally comprised of IgG, though rare cases involving other isoforms such as IgA, IgM, *etc.* have been reported.[4-6] The clinical manifestations of anti-GBM disease mediated by IgA or IgM are often atypical, and serum GBM antibody can be negative. Anti-GBM disease can occur alone or in combination with other renal diseases. For instance, it has been reported that 5% of anti-neutrophil cytoplasm antibody (ANCA)-associated-vasculitis patients can exhibit GBM antibody positivity, whereas up to 1/3 of anti-GBM nephropathy patients have anti-neutrophil cytoplasm antibody (ANCA) positivity.[7] Our center has previously reported a case of anti-GBM nephropathy concomitant with ANCA-associated vasculitis (AAV), that was treated with plasma exchange, corticosteroids and cyclophosphamide. After few years of follow-up, patient's serum creatinine level is approximately1.24mg/dL.[8] Anti-GBM disease has also been reported in patients with membranous nephropathy,[9] though its co-occurrence with other primary glomerular diseases is rare. Nevertheless, a few cases of concurrent IgA nephropathy have been reported. Currently, the pathological mechanism remains unclear, and the efficacy and prognosis are uncertain. Herein, we report a case of anti-GBM nephropathy combined with IgA nephropathy. After treatment and follow-up for one year, the renal function has now returned to the normal. Finally, a literature review was

also conducted, summarizing and comparing the clinical manifestations, treatment and prognosis of an additional 10 cases of combined GBM disease and IgA nephropathy.

CASE PRESENTATION

The patient was a 38-year-old female who had been working in a vehicle management office for five years and had a history of occupational exposure to gasoline. She was admitted to the hospital on July 5th, 2022, due to bilateral lower limb swelling, proteinuria, and hematuria for the past month. She had no history of hemoptysis, rash, fever, arthralgia, etc., and nor any history of hypertension and diabetes mellitus. Routine urine test had urine red blood cells 2+/ high power field (HPF), urinary protein excretion was 3163.84 mg/24h. Renal function tests revealed blood urea nitrogen (BUN) of 5.7mmol/L, serum creatinine (Cr) 0.93mg/dL. The anti- GBM antibody titer was 42.24 RU/mL. Other laboratory tests, including antinuclear antibodies (ANA), ANCA, complement levels, tumor markers, thyroid function test, hepatitis panel and human immunodeficiency virus were normal. A Pulmonary CT scan showed no abnormality (Figure 1). The main laboratory results are shown in Table 1.

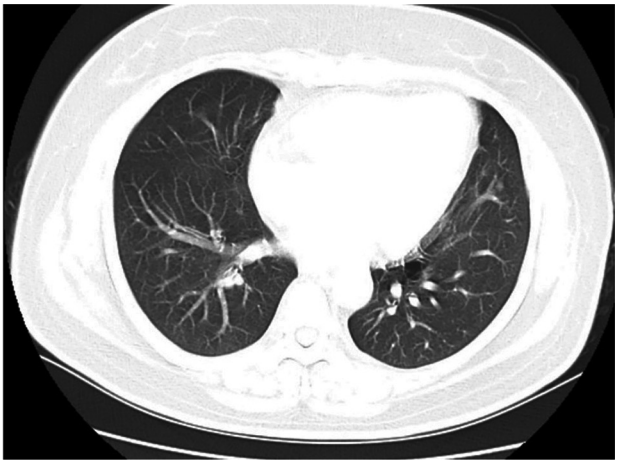


Figure 1. Lung CT scan of the patient revealing no signs of alveolar hemorrhage.

Table 1. Laboratory characteristics at time of kidney biopsy

Component	Value	Reference range	Interpretation
Urinalysis			
Urinary protein	2+	Negative	High
Red blood Cells (n/HPF)	2+	0-3	High
White blood Cells (n/HPF)	1+	0-5	High
Urinary protein excretion (g/24 h)	1.920	0-0.2	High
Hematology			
White blood cells (×10^9 /L)	9.3	3.5-9.5	Normal
Red blood cells (× 10^{12}/L)	4.19	3.8-5.1	Normal
Platelets (×10^9 /L)	298	125-350	Normal
Hemoglobin (g/L)	124	115-150	Normal
Blood chemistry			
Blood urea nitrogen (mmol/L)	5.7	2.6-7.5	Normal
Serum creatinine (mg/dL)	0.93	0.45-0.94	Normal
Serum albumin (g/L)	34.2	40-55	Normal
Uric acid (mg/dL)	8.32	2.35-5.85	High
Immunology			
C3 (g/L)	0.86	0.70-1.40	Normal
C4 (g/L)	0.16	0.10-0.40	Normal
IgG (g/L)	14.6	8.60-17.40	Normal
IgA (g/L)	3.18	1.00-4.20	Normal
IgM (g/L)	2.10	0.50-2.80	Normal
SIFE	Negative	Negative	Normal
Anti-GBM antibody (RU/mL)	42.24	< 20 RU/mL	Positive
ANCA	Negative	Negative	Normal
ANA	Negative	Negative	Normal
Blood Kappa Light Chains (g/L)	12	6.29-13.50	Normal
Blood Lambda Light Chains (g/dL)	6.16	3.13-7.23	Normal

SIFE, serum immunofixation electrophoresis; ANCA, antineutrophil cytoplasmic antibody; Anti-GBM, anti-glomerular basement membrane; ANA, antinuclear antibodies; HPF, high power field; C3, complement 3; C4, complement 4.

A kidney biopsy was performed for this patient, which revealed 21 glomeruli on light microscopy (LM), four of which were globally sclerosed and one showed segmental sclerosis. A total of eight crescents were found, including four cellular (Figure 2A), and four fibro-cellular crescents (Figure 2B). The remaining glomeruli showed slightly increased segmental mesangial matrix, with mild proliferation of mesangial cells. Renal tubular epithelial cells showed swelling and degeneration, with hyaline casts. Approximately 30% of tubules demonstrated focal atrophy, accompanied by interstitial fibrosis and infiltration with inflammatory cells involving roughly 10% of the interstitial area. Some small renal vessels showed focal hyaline degeneration. Congo red staining was also negative.

Immunofluorescence study revealed linear positivity for IgG (1+) along the glomerular basement membrane. (Figure 2C), together with mesangial staining for (2+) IgA (Figure 2D) and (2+) IgM. Kappa, lambda and C3 were also detected in the mesangial and glomerular basement membrane area, with relatively a weak deposition of C1q.

Electron microscopy (EM) revealed deposition of electron-dense substances along the subendothelial aspect of the basal membrane of segmental capillary loop (Figure 3A), and in the glomerular mesangial area (Figure 3B).

Based on the kidney biopsy pathological findings, the patient was diagnosed with anti-GBM disease (with crescents involving 38% of glomeruli) and IgA nephropathy (Oxford classification: M0E0S1T1. The main pathology in the renal biopsy was crescents responsible for hematuria, proteinuria, and elevated serum creatinine level. The crescents were attributed to anti-GBM disease based on the characteristic appearance. Therefore, the scoring of crescents was not added to the Oxford classification

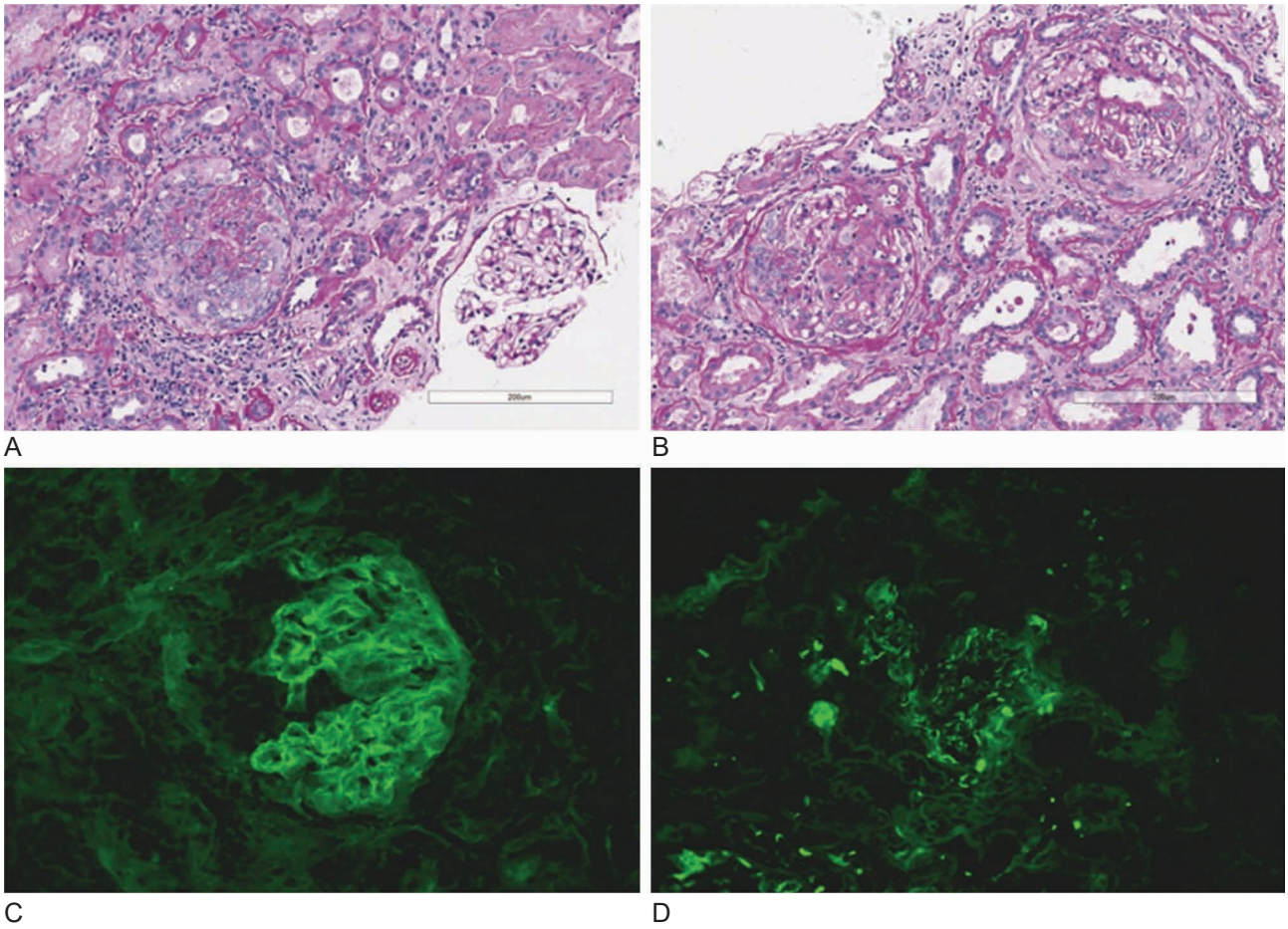
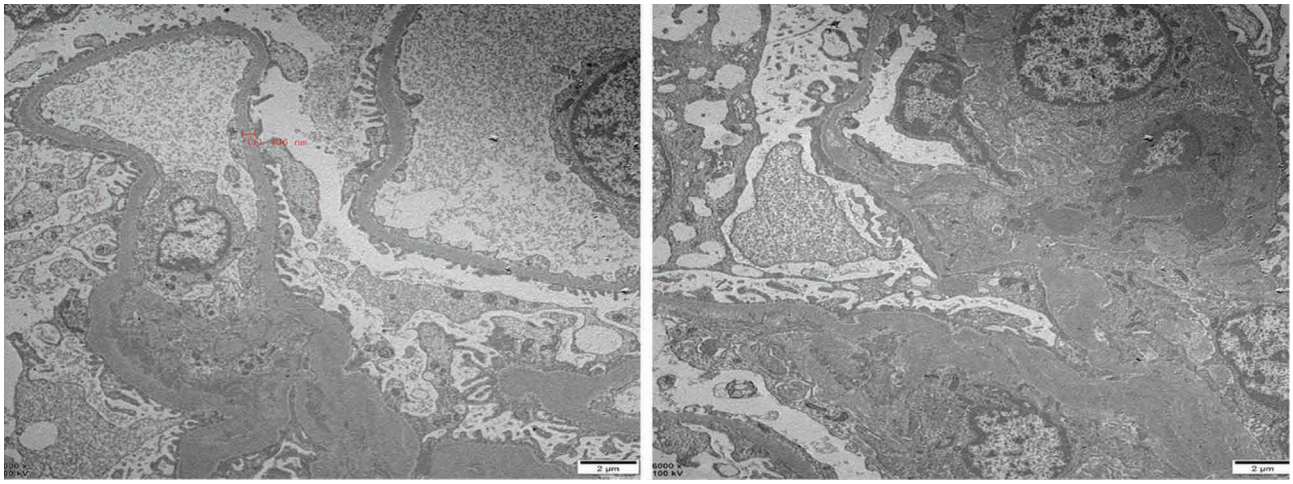


Figure 2. Histopathology of kidney biopsy. (A) Light microscopy revealed a cellular crescent. (B) Light microscopy revealed a fibro-cellular crescent, and a small cellular crescent. (C) Immunofluorescence showing linear deposition of IgG along the GBM. (D) Immunofluorescence showing mesangial staining with IgA. Ig = immunoglobulin; GBM = glomerular basement membrane.



A B

Figure 3. Electron microscopy of kidney biopsy revealed electron-dense deposits under the endothelium of the basal membrane of the segmental capillary loop (A). Electron microscopy revealed electron-dense deposits in the glomerular mesangial area (B).

of IgA nephropathy. The patient initially declined cyclophosphamide treatment due to the concerns about potential side effects. Hence, she was started with prednisone 60 mg/day orally for eight weeks. However, the serum creatinine level increased from 0.93mg/dL to 1.18mg/dL, the 24-hour urinary protein fluctuated between 2500.79 and 2584.83 mg, and the anti-GBM antibody titer was observed to be 37.36 RU/ml, indicating poor efficacy. Consequently, prednisone was gradually tapered and monthly intravenous infusion of 0.7g cyclophosphamide was added to the treatment. The 24-hour urinary protein excretion decreased from 3163.84 mg to 856.8 mg following the administration of cyclophosphamide. The serum creatinine level was0.94mg/dL, and anti-GBM antibody titer

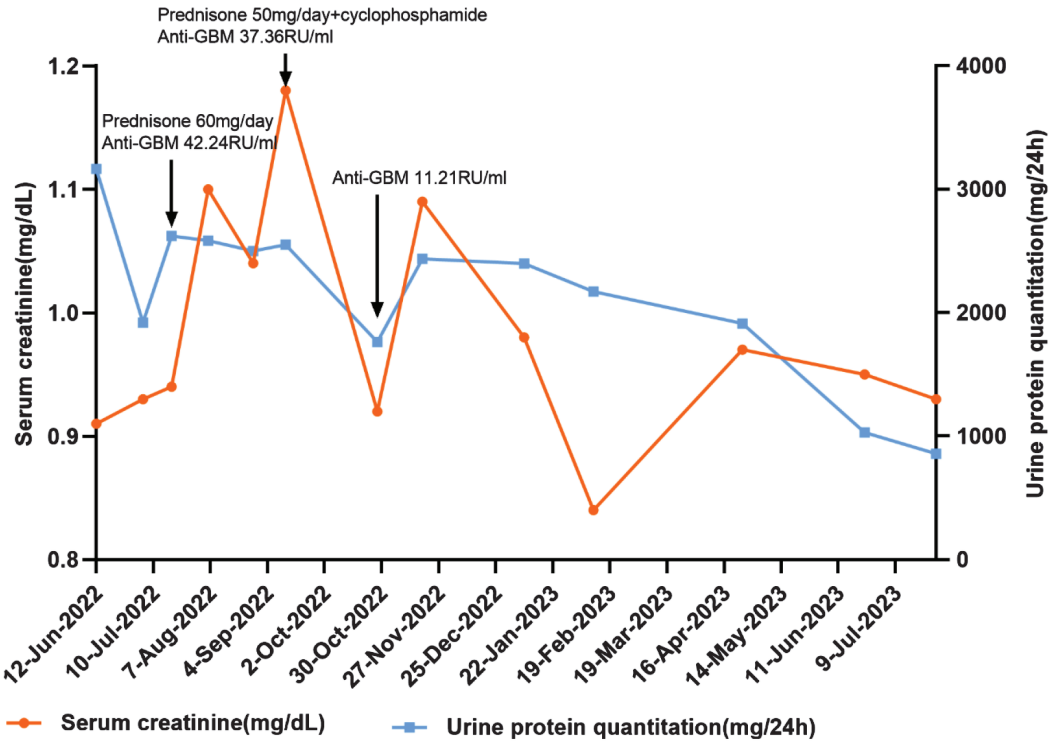


Figure 4. The trend of serum creatinine and proteinuria during treatment

normalized (11.21 RU/mL). The changes in the serum creatinine level during the treatment is depicted in Figure 4.

DISCUSSION

The diagnosis of anti-GBM disease relies primarily on the detection of anti-GBM antibodies in conjunction with glomerulonephritis.[2,10-11] The kidney immunopathology of anti-GBM disease is mainly characterized by linear deposition of immunoglobulin along the GBM, which is usually IgG, but rarely IgA or IgM may also be found.[4-6] Anti-GBM antibody can be detected in the serum of 87-90% of patients with anti-GBM disease.[12] However, approximately 10% of patients with anti-GBM disease possess negative serum anti-GBM antibodies. There are several possible explanations for this, including the presence of non-IgG anti-GBM antibodies (IgA or IgM etc.),[13] the higher affinity of the antibodies towards GBM,[14] lower sensitivity of detection by ELISA method,[15-16] or of cryptic and highly conformation-dependent epitopes on α3(IV)NC1.[17] In addition, these antibodies can be negative if the samples are obtained during the late course of disease when circulating antibodies have disappeared.[11] Although serum anti-GBM antibody positivity is highly specific for diagnosing anti-GBM disease, there have been reports of positive serum anti-GBM antibody in viral infections such as Hantavirus, HIV, *etc.*[18-19]

Currently, the standard treatment of Anti-GBM disease is based mainly on plasma exchange, to remove circulating pathogenic autoantibodies. Immunosuppression with corticosteroids combined with cyclophosphamide or even rituximab can be administered, to inhibit further autoantibody production and to suppress kidney inflammation.[11,20] It has been found that early and active treatment can significantly improve the prognosis of patients and reduce the need for dialysis. At present, there are reports describing few atypical anti-GBM disease, found in combination with ANCA-associated vasculitis, membranous nephropathy, and a few cases with IgA nephropathy. The pathological mechanisms and treatment options for these diseases are currently unclear, and hence further clinical research are needed. A literature search using PubMed Central (PMC) revealed 10 cases of concurrent anti-GBM disease and IgA nephropathy over the past15 years (Table 2).[21-30] It was found that one patient had negative serum anti-GBM antibody, whereas the others revealed positive results. The patients had serum creatinine levels ranging from 1.5 to 23 mg/dL, with a crescent percentage of 12.5% to 100%. No patients exhibited alveolar hemorrhage. The therapies and prognosis of these patients varied markedly. So far, anti-GBM disease combined with IgA nephropathy has not shown better prognosis than anti-GBM disease alone.

In our case, initially treatment with prednisone alone was used: however, its efficacy was proved inadequate. After adding cyclophosphamide to the treatment, the condition improved significantly, proteinuria decreased, hematuria resolved, serum creatinine turned normal, and anti-GBM antibody titers were within the normal range; indicating that the crescentic pathological damage was mainly driven by anti-GBM disease. Since the combination of corticosteroids and cyclophosphamide was effective, plasma exchange (PE) therapy was not indicated.

Elevated serum creatinine (> 5.66 mg/dL), crescentic nephritis (with crescent involving more than 50% of glomeruli), and high titers of serum anti-GBM antibodies are often associated with poor renal function and prognosis.[20,31-32] Among the 10 patients with concurrent anti-GBM disease and IgA nephropathy in the literature, three patients required hemodialysis, and their initial serum creatinine level ranged from 6.6 to 23 mg/dL, while two patients with renal function recovery had initial serum creatinine levels between 1.74 and 1.97 mg/dL. This suggests that early active treatment is accompanied with better prognosis. According to the results of review analysis, our patient did not receive PE treatment due to the insignificant increase in the serum creatinine level. However, the efficacy of corticosteroid alone was not adequate, and the combination therapy with cyclophosphamide showed significant clinical efficacy. Therefore, there is currently no unified treatment plan for concurrent anti-GBM disease and IgA nephropathy. Patients with milder clinical presentations can initially be treated with corticosteroids combined with cyclophosphamide, whereas those with more severe conditions should receive PE treatment.

Although the underlying mechanism is not fully understood, it has been demonstrated that

Table 2. Patient's characteristics based on the literature review

Authors	Age/ gender	Anti-GBM antibody	Lung hemorrhage (Y/N)	Serum creatinine (mg/dl)	Proteinuria	Hematuria	Renal biopsy (Immunofluorescence)	Crescent ratio (%)	Treatment	Prognosis
Shaojie et al. 2022.[21]	26/F	142RU/ ml	N	1.97	10.2g/24h	1258RBCs /ul	Linear deposition of IgG and C3 along the GCW, Clumps of IgA deposition in the mesangial area.	48	Corticosteroid, CTX, PE	Anti-GBM antibody negative, Normal renal function
Guo. et al. 2022.[22]	49/M	68RU/ml	N	1.5	5.16g/24h	45.09 RBCs / HPF	Linear deposition of IgG along the GCW, Granular and bolus-type deposition of IgA and C3 in the mesangial area.	70	Corticosteroid, CTX	Anti-GBM antibody negative. Serum creatinine 105μmol/ L~123μmol/L
Ge. et al. 2015.[23]	24/M	Positive	N	15.7	7.04g/24h	25-30 r RBCs /HPF	Linear deposition of IgG and C3 along GBM, Granular deposition of IgA in the mesangial area.	58	Corticosteroid, CTX, PE, Hemodialysis	Anti-GBM antibody negative, Hemodialysis
Qu. et al. 2022.[24]	48/F	551 U/ml	N	1.74	1g/24h	222.62 RBCs /HPF	Weak linear deposition of IgG along GCW, Lumpy deposition of IgA and C3 in the mesangial area.	13	Corticosteroid, CTX, PE, RTX	Anti-GBM antibody negative, Normal renal function
Xu et al.2016.[25]	50/F	258.3 EU/ml	N	1.59	0.41g/24h	10-15 RBCs / HPF	Linear deposition of IgG along the GBM, Lumpy deposition of IgA in the mesangium.	89	Corticosteroid, MMF	Anti-GBM antibody negative, Normal renal function
Suh et al. 2019.[26]	38/F	187.2 U/ mL	N	5.45	Urine protein/ creatinine 1.4 g/g	Many urine spot erythrocytes	Linear deposition of IgG along the GBM, Granular deposition of IgA in mesangial spaces .	69	Corticosteroid, CTX	Anti-GBM titer 15.6U/ mL, Serum creatinine 2.08 mg/dL
Yamaguchi et al.2013.[27]	46/M	214 EU	N	6.6	2.98 g/gCr	> 100 RBCs / HPF	Linear deposition of IgG along the GCW, Granular depositon of IgA and C3 in mesangial spaces.	94	Corticosteroid, Haemodialysis	The anti-GBM antibody decreased, Hemodialysis
Shenoy et al.2022.[28]	57/M	107 RU/L	N	23	NR	NR	Linear positivity along capillary walls with IgG, Diffuse and granular deposition in mesangium with IgA and C3.	100	Corticosteroid, Haemodialysis	Haemodialysis
Gupta et al. 2019.[29]	30/M	Positive	N	13.13	Urine protein/ creatinine 2.5 g/g	10-12 RBCs / HPF	Linear GBM staining with IgG and mesangial granular staining with IgA and C3.	100	Steroid, CTX, PE	Anti-GBM antibody negative. Serum creatinine 3.0 mg/dl
Bajaj et al. 2021.[30]	13/F	Negative	N	3.4	Urine protein/ creatinine 8.2 mg/mg	80–90 RBCs / HPF	Coarse mesangial deposits of IgA and C3 along with capillary wall linear positivity for IgG.	75	Corticosteroid, Haemodialysis, PE, CTX	Serum creatinine 1.26 mg/dl

Y, yes; N, no; CTX, cyclophosphamide; PE, Plasma exchange; NR, no report; GCW, glomerular capillary walls; HPF, high power field; GBM, glomerular basement membrane. RBCs, red blood cells.

the pathogenesis of anti-GBM disease includes both genetic and environmental factors; genetic susceptibility is closely related to the HLA-DRB1*1501, HLA-DRB1*0401, and HLA-DRB1*03 alleles.[33-34] In addition, environmental factors are mainly linked to exposure to hydrocarbons such as gasoline, diesel and paint. The pathogenesis of anti-GBM disease combined with IgA nephropathy remains unknown, and there is currently no evidence to suggest a causal relationship between the two diseases. Our patient had several years of exposure to car exhaust. Inhalation of vehicle exhaust may disrupt the quaternary structure of the alveolar basement membrane, inducing the formation of anti-basement membrane antibodies and leading to nephritis.

CONCLUSION

In summary, anti-GBM disease is associated with significant heterogeneity, such as a few atypical presentations and rare combination with other primary glomerulonephritis. Indeed, the pathological mechanism remains unclear, and its clinical manifestations vary significantly. Although the pathological manifestations in this patient were not mild, the impairment of renal function was relatively mild, and the therapeutic effect of prednisone combined with cyclophosphamide was significant.

ETHICAL STATEMENT

The study was approved by the Ethics Committee of Tongde Hospital of Zhejiang Province, China.

ACKNOWLEDGEMENTS

Not applicable.

CONFLICT OF INTEREST

The authors of this work have nothing to disclose.

FUNDING

No funding was obtained for this study.

REFERENCES

1. 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;11:1392-1399. doi: 10.2215/CJN.13591215. Epub 2016 Jul 11.
2. McAdoo SP, Pusey CD. Anti-Glomerular Basement Membrane Disease. Clin J Am Soc Nephrol. 2017;12:1162-1172. doi: 10.2215/CJN.01380217.
3. Asim M, Akhtar M. Epidemiology, Impact, and Management Strategies of Anti-Glomerular Basement Membrane Disease. Int J Nephrol Renovasc Dis. 2022;15:129-138. doi: 10.2147/IJNRD.S326427.
4. Bacalja J, Zibar L, Ljubanović DG. IgA-mediated anti-glomerular basement membrane disease. A case report. Nefrologia (Engl Ed). 2018;38:339-341. doi: 10.1016/j.nefro.2017.06.003.
5. Fervenza FC, Terreros D, Boutaud A, et al. Recurrent Goodpasture's disease due to a monoclonal IgA-kappa circulating antibody. Am J Kidney Dis. 1999;34:549-55. doi: 10.1016/s0272-6386(99)70084-3.
6. Nasr SH, Collins AB, Alexander MP, et al. The clinicopathologic characteristics and outcome of atypical anti-glomerular basement membrane nephritis. Kidney Int. 2016;89:897-908. doi: 10.1016/j.kint.2016.02.001.
7. Levy JB, Hammad T, Coulthart A, Dougan T, Pusey CD. Clinical features and outcome of patients with both ANCA and anti-GBM antibodies. Kidney Int. 2004;66:1535-40. doi: 10.1111/j.1523-1755.2004.00917.x.
8. Li X, Huang M, Liu J. ANCA-Associated Vasculitis With Anti-GBM Disease and Two Types of Tumors: A Case Report. Front Med (Lausanne). 2021;8:810680. doi: 10.3389/fmed.2021.810680.
9. Zhang S, Li C, Huang J, et al. Clinical and pathological features of anti-glomerular basement membrane disease associated with membranous nephropathy: an observational study. Ren Fail. 2022;44:1904-1914. doi: 10.1080/0886022X.2022.2141645.
10. Salama AD, Dougan T, Levy JB, et al. Goodpasture's disease in the absence of circulating anti-glomerular basement membrane antibodies as detected by standard techniques. Am J Kidney Dis. 2002;39:1162-7. doi: 10.1053/ajkd.2002.33385.
11. Hellmark T, Segelmark M. Diagnosis and classification of Goodpasture's disease (anti-GBM). J Autoimmun. 2014;48-49:108-12. doi: 10.1016/j.jaut.2014.01.024.
12. Jennette JC, Nickeleit V. Anti-glomerular basement membrane glomerulonephritis and Goodpasture syndrome. In: Jennette JC, Silva FG, Olson JL, et al. (eds). Heptinstall's Pathology of the Kidney. 7th edn Philadelphia, PA: Wolters Kluwer. 2015;657–684.
13. Wen YK, Wen KI. An unusual case of IgA-mediated anti-glomerular basement membrane disease. Int Urol Nephrol. 2013;45:1229-34. doi: 10.1007/s11255-012-0162-8.
14. Saxena R, Bygren P, Butkowski R, Wieslander J. Specificity of kidney-bound antibodies in Goodpasture's syndrome. Clin Exp Immunol. 1989;78:31-6.
15. Dougan T, Levy JB, Salama A, George AJ, Pusey CD. Characterization of autoantibodies from patients with Goodpasture's disease using a resonant mirror biosensor. Clin Exp Immunol. 2002;128:555-61. doi: 10.1046/j.1365-2249.2002.01867.x.
16. Levy JB, Turner AN, George AJ, Pusey CD. Epitope analysis of the Goodpasture antigen using a resonant mirror biosensor. Clin Exp Immunol. 1996;106:79-85. doi: 10.1046/j.1365-2249.1996.d01-815.x.
17. Jia XY, Qu Z, Cui Z, Yang R, Zhao J, Zhao MH. Circulating

anti-glomerular basement membrane autoantibodies against α3(IV)NC1 undetectable by commercially available enzyme-linked immunosorbent assays. Nephrology (Carlton). 2012;17:160-6. doi: 10.1111/j.1440-1797.2011.01511.x.

18. Zijlstra HW, Mulder AHL, Geeraedts F, Visser F. Falsely positive anti-glomerular basement membrane antibodies in a patient with hantavirus induced acute kidney injury - a case report. BMC Nephrol. 2018;19:286. doi: 10.1186/s12882-018-1082-3.
19. Szczech LA, Anderson A, Ramers C, et al. The uncertain significance of anti-glomerular basement membrane antibody among HIV-infected persons with kidney disease. Am J Kidney Dis. 2006;48:e55-9. doi: 10.1053/j.ajkd.2006.06.007.
20. Heitz M, Carron PL, Clavarino G, et al. Use of rituximab as an induction therapy in anti-glomerular basement-membrane disease. BMC Nephrol. 2018;19:241. doi: 10.1186/s12882-018-1038-7.
21. Shaojie F, Sensen S, Jingda H, et al. Great prognosis of concurrent anti-GBM disease and IgA nephropathy in a young woman: A case report. Medicine (Baltimore). 2022;101:e30686. doi: 10.1097/MD.0000000000030686.
22. Guo C, Ye M, Li S, Zhu TT, Rao XR. Anti-glomerular basement membrane disease with IgA nephropathy: A case report. World J Clin Cases. 2022;10:3916-3922. doi: 10.12998/wjcc.v10.i12.3916.
23. Ge YT, Liao JL, Liang W, Xiong ZY. Anti-Glomerular Basement Membrane Disease Combined with IgA Nephropathy Complicated with Reversible Posterior Leukoencephalopathy Syndrome: An Unusual Case. Am J Case Rep. 2015;16:849-53. doi: 10.12659/ajcr.894619.
24. Qu W, Liu N, Xu T, et al. Case Report: Coexistence of Anti-Glomerular Basement Membrane Disease, Membranous Nephropathy, and IgA Nephropathy in a Female PatientWith Preserved Renal Function. Front Pharmacol. 2022;13:876512. doi: 10.3389/fphar.2022.876512.
25. Xu D, Wu J, Wu J, et al. Novel therapy for anti-glomerular basement membrane disease with IgA nephropathy: A case report. Exp Ther Med. 2016;11:1889-1892. doi: 10.3892/etm.2016.3149.
26. Suh KS, Choi SY, Bae GE, Choi DE, Yeo MK. Concurrent Anti-glomerular Basement Membrane Nephritis and IgA Nephropathy. J Pathol Transl Med. 2019;53:399-402. doi: 10.4132/jptm.2019.08.05.
27. Yamaguchi H, Takizawa H, Ogawa Y, Takada T, Yamaji I, Ura N. A case report of the anti-glomerular basement membrane glomerulonephritis with mesangial IgA deposition. CEN Case Rep. 2013;2:6-10. doi: 10.1007/s13730-012-0029-y.
28. Shenoy SV, Rao IR, Prabhu RA, et al. Crescentic glomerulonephritis due to coexistent IgA nephropathy and anti-glomerular basement membrane disease in a patient with COVID-19 disease: A case report. Nephrology (Carlton). 2022;27:727-728. doi: 10.1111/nep.14076.
29. Gupta Y, Swain M, Gowrishankar S. Antiglomerular Basement Membrane Disease Combined with IgA Nephropathy. Indian J Nephrol. 2019;29:375-377. doi: 10.4103/ijn.IJN_309_18.
30. Bajaj V, Thakur S, Barwad A, Sinha A, Bagga A, Singh G. IgA Nephropathy and Atypical Anti-GBM Disease: A Rare Dual Pathology in a Pediatric Rapidly Progressive Glomerulonephritis. Glomerular Dis. 2022;2:54-57. doi: 10.1159/000521582.
31. Zahir Z, Wani AS, Prasad N, Jain M. Clinicopathological characteristics and predictors of poor outcome in anti-glomerular basement membrane disease - a fifteen year single center experience. Ren Fail. 2021;43:79-89. doi: 10.1080/0886022X.2020.1854301.
32. Hellmark T, Segelmark M, Unger C, Burkhardt H, Saus J, Wieslander J. Identification of a clinically relevant immunodominant region of collagen IV in Goodpasture disease. Kidney Int. 1999;55:936-44. doi: 10.1046/j.1523-1755.1999.055003936.x.
33. Levy JB, Turner AN, Rees AJ, Pusey CD. Long-term outcome of anti-glomerular basement membrane antibody disease treated with plasma exchange and immunosuppression. Ann Intern Med. 2001;134:1033-42. doi: 10.7326/0003-4819-134-11-200106050-00009.
34. Fisher M, Pusey CD, Vaughan RW, Rees AJ. Susceptibility to anti-glomerular basement membrane disease is strongly associated with HLA-DRB1 genes. Kidney Int. 1997;51:222-9. doi: 10.1038/ki.1997.27.

*Correspondence to:
Liping Zhao, Renal Department, Tongde Hospital of Zhejiang Province, No. 234 Gucui Road, Hangzhou, Zhejiang, 310012, China
Tel: +86-0571-89972000
E-mail: xyw1123123@163.com

Received August 2024
Revised November 2024
Accepted October 2025