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CASE REPORT

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C3 glomerulopathy associated with mycoplasma pneumoniae infection and positive IgA staining

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Abstract

Background Patients with C3 glomerulopathy (C3G) often have a history of infection, which implies that infection may lead to abnormal activation of the complement alternative pathway (CAP) and induce the development of C3G. However, patients with postinfectious glomerulonephritis (PIGN) often have a low serum C3 concentration and positive glomerular C3 staining, consistent with the activation of the CAP. PIGN, especially if it involves simultaneous IgA deposition, is often difficult to differentiate from C3G.

Case Presentation In this study, we report the consequences of *Mycoplasma pneumoniae* (MP) infection in a 66-year-old male Chinese patient, who developed persistent hypocomplementemia, gross hematuria, and rapidly progressive glomerulonephritis. The findings of the histologic examination of an initial renal biopsy were consistent with a diagnosis of IgA-dominant postinfectious glomerulonephritis. The sample was negative for Gd-IgA1 staining. After treatment with antibiotics, glucocorticoids, and mycophenolate mofetil, the patient's serum creatinine decreased from a peak of 387 μmol/L to 195 μmol/L prior to discharge, and there was a partial response in his urinary protein concentration. After 2 months, his serum C3 concentration had returned to normal. However, owing to reinfection with MP the patient's serum creatinine rapidly increased again to 475.07 μmol/L, and this was accompanied by a decrease in serum C3 concentration (> 8 months) and positivity for C3 nephritis factor. Examination of both renal biopsies showed stronger immunostaining for C3 than for IgA in the glomeruli.

Conclusion Thus, MP infection can cause sustained activation of the CAP, leading to C3G. For patients with MP infection, if there is an ongoing decrease in complement C3 levels and a progressive increase in serum creatinine, it is crucial to be vigilant for possible C3G and to consider the use of immunosuppressive therapy in conjunction with anti-infective treatment to prevent the ongoing activation of the CAP.

Keywords C3 glomerulopathy, *Mycoplasma pneumoniae*, IgA-dominant postinfectious glomerulonephritis, Acute kidney injury, Renal biopsy

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Background

C3 glomerulopathy (C3G) is a rare renal disease that pathologically manifests as C3 accumulation in the glomeruli, but with absent or minimal immunoglobulin deposition [1]. Many glomerular diseases involve abnormal regulation of complement activation, deposition, or degradation, leading to the deposition of C3 fragments in glomerulus [2]. Histologic analysis of the kidneys of patients with C3G reveals glomerular immunostaining for C3 (twice as much staining as for any other immune deposit), but this is often accompanied by the deposition of immunoglobulins. A study of a C3G cohort at the Mayo Clinic showed that 45% of the patients had immunoglobulin deposition (trace to 1+) [3]. The light microscopic changes associated with C3G can be diverse and be mesangial proliferative, membranoproliferative, and endocapillary proliferative; crescents may also be present [2].

Nearly 30% of patients with C3G have a history of infection [3], indicating that this may cause abnormal activation of the complement alternative pathway (CAP) and lead to the development of C3G. However, patients with postinfectious glomerulonephritis (PIGN) often have low serum C3 concentrations and glomerular C3 immunostaining, which is associated with the activation of the CAP. In clinical practice, some cases of PIGN are principally characterized by C3 deposition, often associated with the simultaneous deposition of IgA (codominant), and such patients are often difficult to differentiate from those with C3G [4]. Moreover, C3G and PIGN may overlap and some patients with PIGN may progress to develop C3G [5]. Owing to the difficulty in distinguishing the two using renal histology or clinical and laboratory indices, the possibility of C3G should be considered for patients with atypical PIGN [2].

Mycoplasma pneumoniae (MP) is a common cause of upper and lower respiratory tract disease. Indeed, MP accounts for approximately 2%-12% of cases of adult community-acquired pneumonia [6–8]. There have been reports of C3G caused by MP infection, but immunofluorescence studies of such cases typically reveal C3 deposition alone, with no transformation process [9]. Here, we report the case of an older male patient with persistent gross hematuria, nephrotic syndrome, and rapidly progressive nephritis syndrome that developed following infection with MP. The initial pathological diagnosis was IgA-dominant postinfectious glomerulonephritis. Subsequently, the patient' experienced repeated infections when his C3 concentration decreased again. On the basis of this and the findings of both renal biopsies, a diagnosis of C3G was made. We discuss in detail the diagnostic process and the differential diagnosis, as well as the possible mechanisms involved, in order to provide assistance with the diagnosis and treatment of similar patients in the future.

Table 1 Laboratory data

Variable	First renal biopsy	Second renal biopsy	Follow-up
Days	0	187	789
Albumin (g/L)	29.2	32	35.6
Serum creatinine (μmol/L)	363	475.07	100.64
Serum C3 (g/L)	0.53	0.379	0.852
Proteinuria (g/d)	5.54	3.48	0.16
Hematuria	Gross	Gross	Micro

Follow-up: last follow-up (October 2024); Gross: gross hematuria; Micro: microscopic hematuria

Case presentation

A 66-year-old male patient from the Han ethnic group in China presented with cough, yellow phlegm, and gross hematuria of 7 days' duration. In July 2022, physical examination and routine urinalysis revealed no abnormalities, and his serum creatinine (Scr) was 67.3 μmol/L. In early August, a cough, yellow phlegm, pharyngeal pain, and fever with a body temperature of 37.8 ℃ were present. Gross hematuria developed, with no substantial decrease in urine output. At the outpatient clinic, the following laboratory findings were made: albumin 40.9 g/L, Scr 117 μmol/L, urine protein 2+, urine blood 3+, urine white blood cells weakly positive, and 24-hour urine protein quantification 3.84 g/d. The urinary sediment included white blood cells (10–15 per high-power field) and red blood cells across the entire field (70% polymorphous). The patient had a 10-year history of hypertension, and this had been managed with nifedipine controlled-release and felodipine sustained-release tablets, resulting in good blood pressure control. He also had a 2-year history of Hashimoto's thyroiditis, and his thyroid function had been found to be low 1 year previously. He was taking 50 μg of levothyroxine per day, and an examination conducted in July 2022 showed normal thyroid function.

On August X, the patient was hospitalized. At admission, his blood pressure was 180/100 mmHg, he had mild edema in both lower limbs, and he had gained 4 kg during the preceding week. His white blood cell count was 4.9×10^9/L and the percentage of neutrophils was 52.9%. He also had a serum albumin concentration of 31.2 g/L, a highest Scr of 387 μmol/L, a C3 of 0.46 g/L, a high-sensitivity C-reactive protein of 16.5 mg/L, 24-hour urine protein loss of 5.54 g/24 h (urine volume 2.1 L), and a urinary N-acetyl-β-D-glucosaminidase of 10.3 U/L (Table 1). In addition, measurement of his IgG, IgA, IgM, C4, antistreptolysin O, rheumatoid factor, antinuclear antibody, antineutrophil cytoplasmic antibody, anti-glomerular basement membrane antibody, M-type

phospholipase A2 receptor antibody, and anti-extractable nuclear antigen antibody revealed no abnormalities. His serum MP IgM and IgG were both positive (1:80), but he had no serum IgM antibodies against *Legionella pneumophila*, *Chlamydia pneumoniae*, Q fever rickettsia, adenovirus, respiratory syncytial virus, influenza A virus, influenza B virus, and parainfluenza virus types 1, 2, or 3. Tests for hepatitis B, hepatitis C, AIDS, and syphilis were also negative. Chest CT revealed a bilateral lung infection with partial expansion, bilateral lower lobe involvement, and bilateral pleural effusion. Multiple bacterial cultures of the patient's sputum and throat swabs were negative, and no acid-fast bacilli were found on a sputum smear. The tuberculin skin test was negative, as were tests for nucleic acid, and IgM and IgG antibodies for SARS-Cov-2. COVID-19 vaccine had been administered 1 year previously, and he did not experience discomfort, rash, or gross hematuria at that time.

On August X, a renal biopsy was performed (Fig. 1). Of the 14 glomeruli visible on light microscopy of a histologic section, two were globally sclerotic; and there was mild-to-moderate diffuse proliferation of mesangial cells and matrix in the remaining glomeruli, segmental endothelial cell proliferation, and neutrophil infiltration. Two areas of focal segmental necrosis with crescent formation of glomeruli, three cellular crescents, and one small cellular fibrous crescent were visible. There was also diffuse acute tubular injury, erythrocyte casts, and moderate interstitial fibrosis. Immunofluorescence studies revealed IgA 2 + and C3 3+, but no staining for IgG, IgM, or C1q. κ 2 + and λ 1 + staining was also apparent, and these substances showed accumulation in the mesangial area as clumps around segmental capillary loops. Electron microscopy revealed blocky electronic dense deposits in the mesangial area, segmental subcutaneous tissue, and segmental basement membrane. Some of the deposits in the subepithelial had a "hump-like" shape, with shrunken

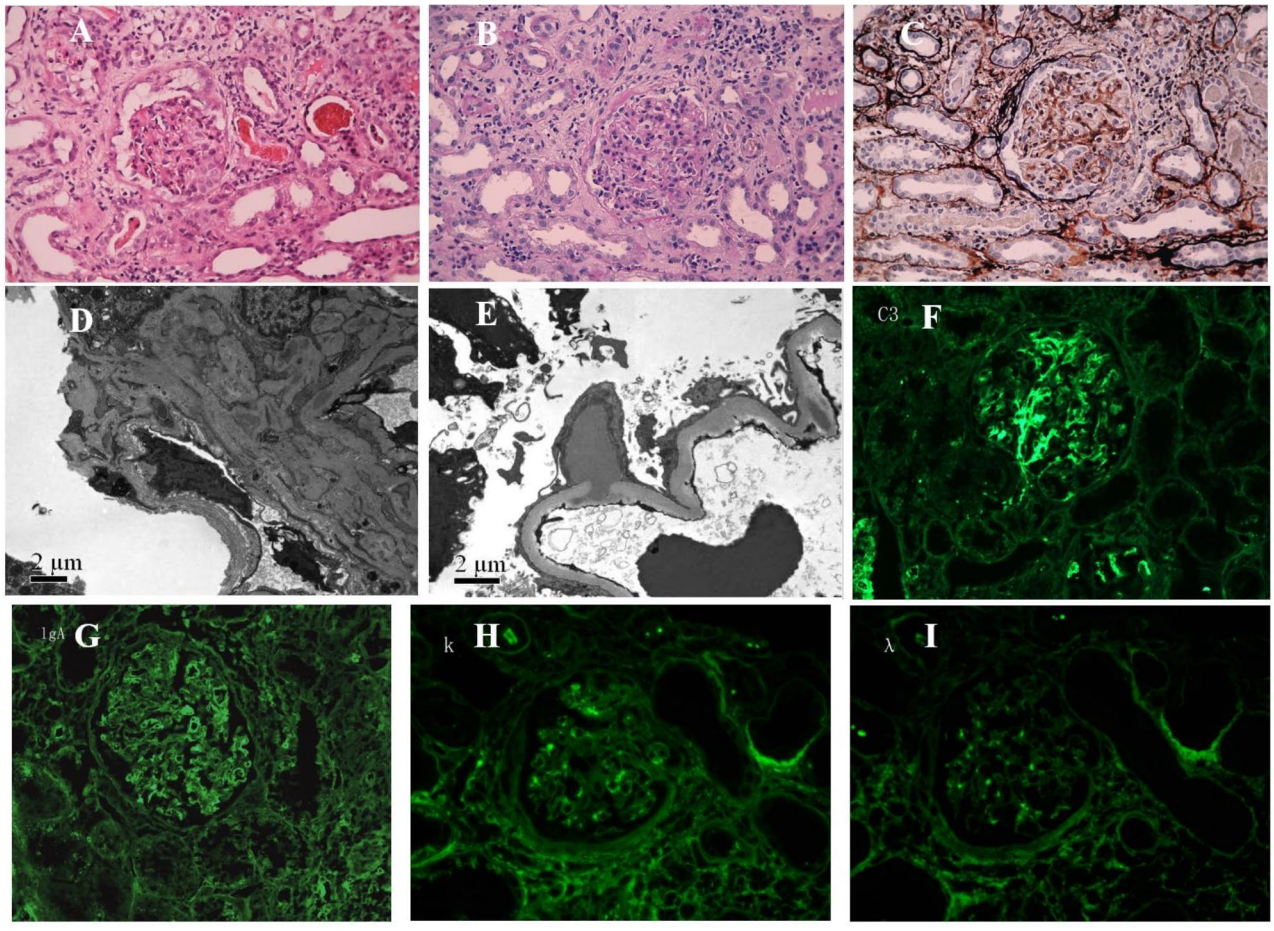

Fig. 1 Histological findings for the first renal biopsy. **(A)** Hematoxylin and eosin (H&E) staining and light microscopic examination (400×) revealed mesangial proliferative glomerulonephritis with crescent formation and red-cell casts in the renal tubules. **(B)** Periodic acid-Schiff staining (400×). **(C)** Periodic acid-Schiff-methenamine silver staining (400×). **(D)** Electron microscopy revealed blocky electron dense deposits in the mesangial area, segmental subcutaneous tissue, and segmental basement membrane (8,000×). **(E)** Electron microscopy revealed "hump-like" subepithelial immune-complex deposition (10,000×). **(F-I)** The glomeruli were positive for C3, IgA, kappa, and lambda (400×)

basal membrane segments and fusion of most of the epithelial foot processes. A pathologic diagnosis of IgA-dominant postinfectious glomerulonephritis with acute tubular injury caused by red blood cell tubular obstruction was made. Moreover, we performed Gd-IgA1 immunofluorescence staining of the renal biopsy specimen obtained from the present patient and another with primary IgA nephropathy (IgAN). The present patient was negative for Gd-IgA1, but the patients with IgAN was strongly positive (3+) (Fig. 2). Therefore, methylprednisolone was administered at a dose of 250 mg/day for 3 consecutive days, which was followed by a daily intravenous infusion of 40 mg and treatment with mycophenolate mofetil (MMF) 1.5 g/day, along with azithromycin and moxifloxacin as anti-infective treatment. As a consequence, the patient's Scr began to decrease, his urine output increased to between 3,500 and 4,500 ml/day, his body mass gradually decreased to its value 1 month previously, and the swelling in both of his lower limbs disappeared. Before discharge, his Scr had decreased to 195 μmol/L.

After discharge, the patient gradually discontinued his MMF and gradually reduced the dose of prednisone administered. His Scr decreased to 171 μmol/L, his serum C3 increased to normal, and his urinary protein quantification decreased to 1.53 g/24 h. His MP antibody (IgG type, 1:320) and macroscopic hematuria disappeared. However, on November X 2022, the patient developed another cough with yellow phlegm, his Scr had increased to 207 μmol/L, and his C3 had decreased to 0.67 g/L. Moxifloxacin was provided as a treatment, and after 2 weeks, his Scr had decreased to 179 μmol/L, but his serum C3 remained low, at 0.63 g/L. On December X, the patient was diagnosed with COVID-19, but his Scr was 165 μmol/L and his serum C3 was normal. On February X 2023, he experienced nasal congestion, runny nose, sore throat, and the gradual development of a cough with yellow phlegm. His gross hematuria reappeared, his Scr

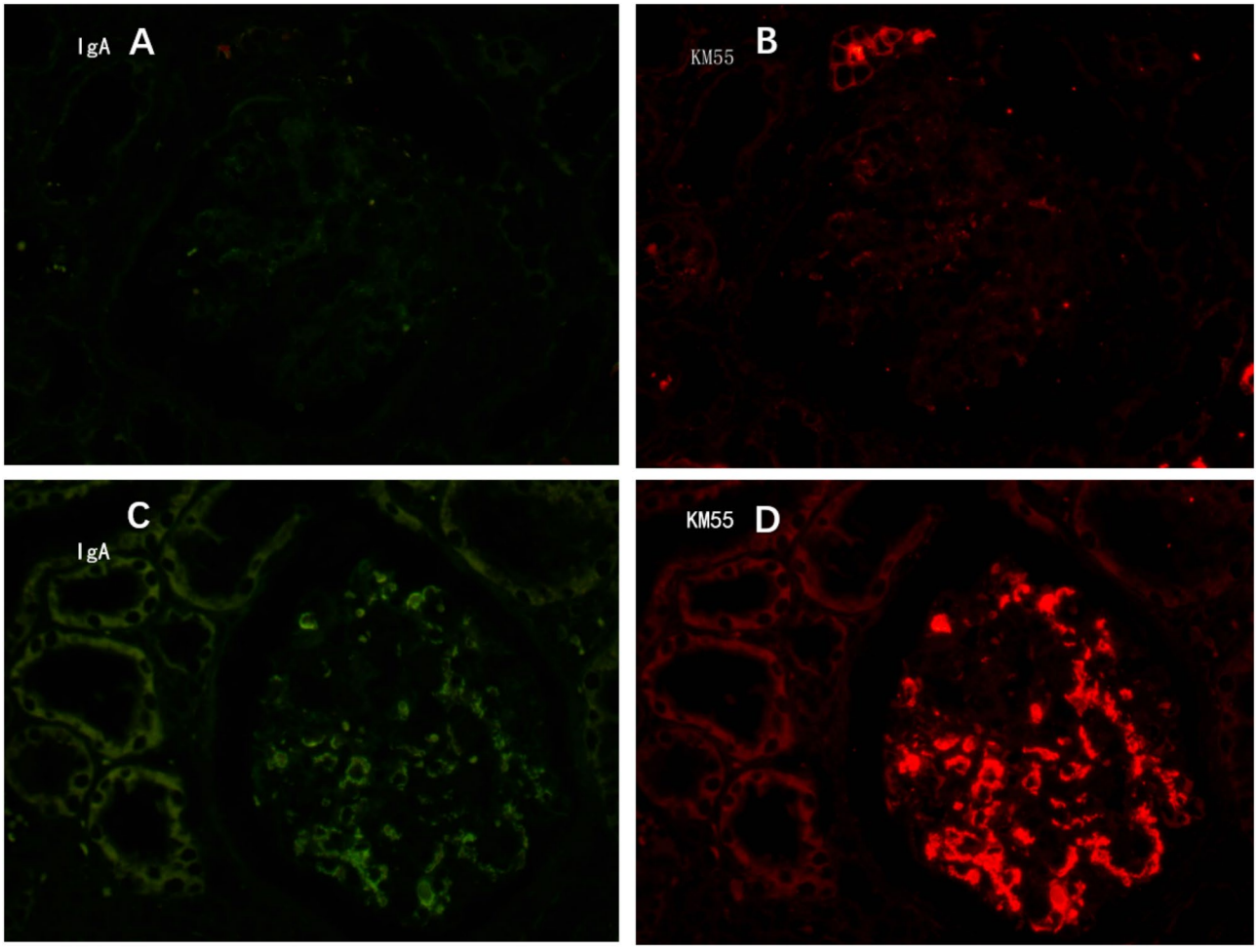

Fig. 2 Immunostaining for Gd-IgA1 in renal biopsy specimen. **(A)** Staining for IgA in a glomerulus from the present patient (400×). **(B)** Staining for Gd-IgA1 in the present patient (400×). **(C)** Staining for IgA in a glomerulus from a patient with primary IgAN (400×). **(D)** Staining for Gd-IgA1 in a renal biopsy from a patient with primary IgAN (400×)

rapidly increased to 475.07 μmol/L, his serum C3 was 0.379 g/L, his serum C4 was 0.132 g/L, and his 24-hour urine protein quantification had increased to 3.48 g/24 h. He was also C3 nephritis factor (C3NeF) positive.

On February X, a second renal biopsy was performed (Fig. 3), and the histologic section contained 30 glomeruli, of which three showed glomerular sclerosis; and there were four fibrous crescents with sclerosis, two cellular crescents, and 10 cellular fibrous crescents. There was also acute tubular injury with diffuse infiltration of monocytes and lymphocytes and fibrosis in the interstitium. Immunofluorescence studies revealed C3 3+, IgA 1+, IgM 1+, FRA 1+, Alb 1+, κ 1+, and λ 1+, in the form of granular deposition in the segmental mesangial area and segmental capillary wall. Immunohistochemistry reveals granular deposits of C4d (2+) in a segmental pattern within the capillary walls, segmental tubular basement membrane, and segmental small arterial walls. Electron microscopy revealed moderate-to-severe proliferation of glomerular mesangial cells and matrix, insertion of segmental mesangium, an increase in the number of cells in the segmental capillary luminas, the deposition of blocky electron dense substances in the mesangial and paramesangial areas, and extensive fusion of epithelial foot processes. The second biopsy was also negative for Gd-IgA1. Blood and urine immunofixation electrophoresis showed no abnormalities. In addition, no abnormalities were identified in the ratio of free light chains in blood and urine samples.

Methylprednisolone was administered at 300 mg/day for 3 consecutive days, followed by an intravenous infusion of 40 mg of methylprednisolone, four plasma exchanges, the intermittent infusion of plasma, and the administration of anti-infective agents (moxifloxacin and azithromycin). After discharge, the mediation was changed to oral prednisolone 50 mg/day and cyclophosphamide (CTX) 50 mg/day. On March X, the patient's Scr had decreased to 198.8 μmol/L. On September X, he underwent a follow-up examination, at which his Scr was 149 μmol/L, C3 0.652 g/L, C4 0.174 g/L, 24-hour urine protein quantification 0.57 g/day, and urine sediment red blood cells 5–10 per high-power field. Owing to the continued decrease in serum C3 concentration, the CTX was discontinued and replaced with MMF 1 g/day, in combination with ARB and compound sulfamethoxazole. On October X 2024, the patient's Scr had decreased to 100.64 μmol/L, his C3 was 0.852 g/L, his 24-hour urine protein loss was 0.16 g/day, and his urine sediment red blood cells count was 10–15 per high-power field (Fig. 4). His MMF dose was reduced to 0.5 g/day and was administered in combination with prednisone 5 mg/day.

Discussion and conclusions

C3G is relatively rare and caused by excessive activation of the complement pathway, leading to C3 deposition in glomeruli. The Mayo Clinic cohort study showed that nearly 30% of patients with C3G have a history of infection, with upper respiratory tract infections being

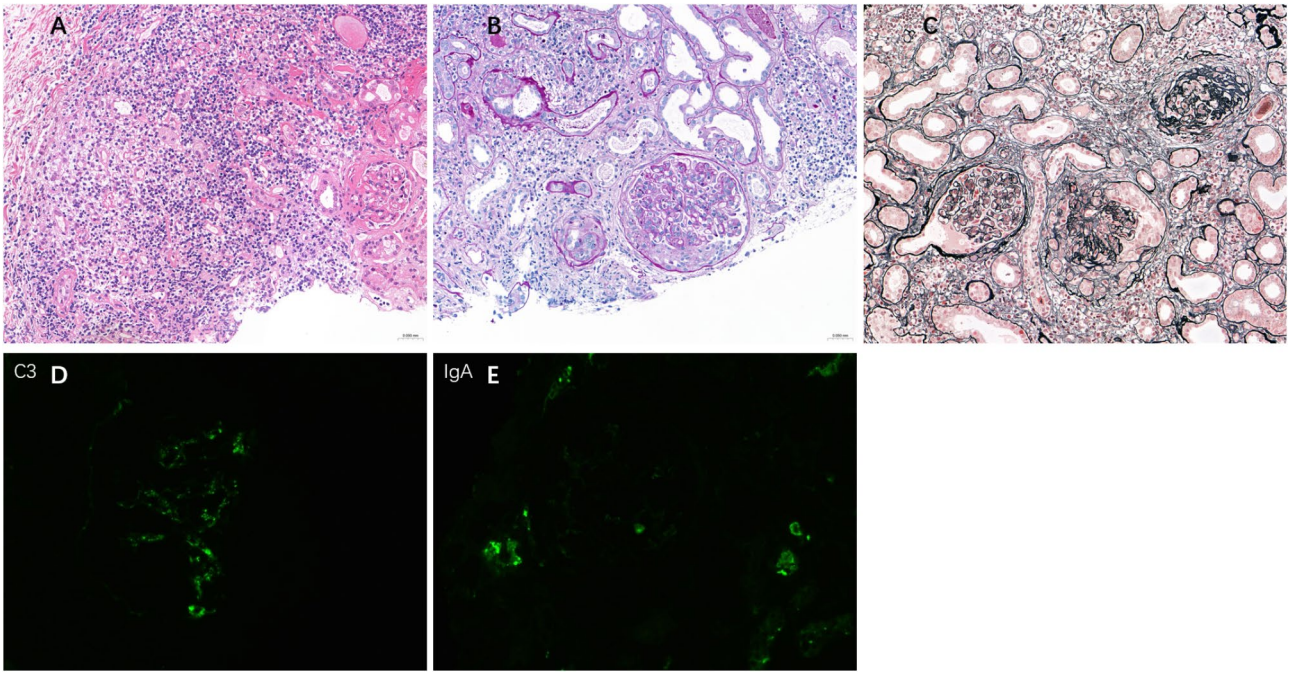

Fig. 3 Histological findings for the second renal biopsy. **(A)** Hematoxylin and eosin (H&E) staining (200×). **(B)** Periodic acid-Schiff staining (200×). **(C)** Periodic acid-Schiff-methenamine silver staining (200×). **(D)** Staining for C3 of a glomerulus from the present patient (400×). **(E)** Staining for IgA of a glomerulus from the present patient (400×)

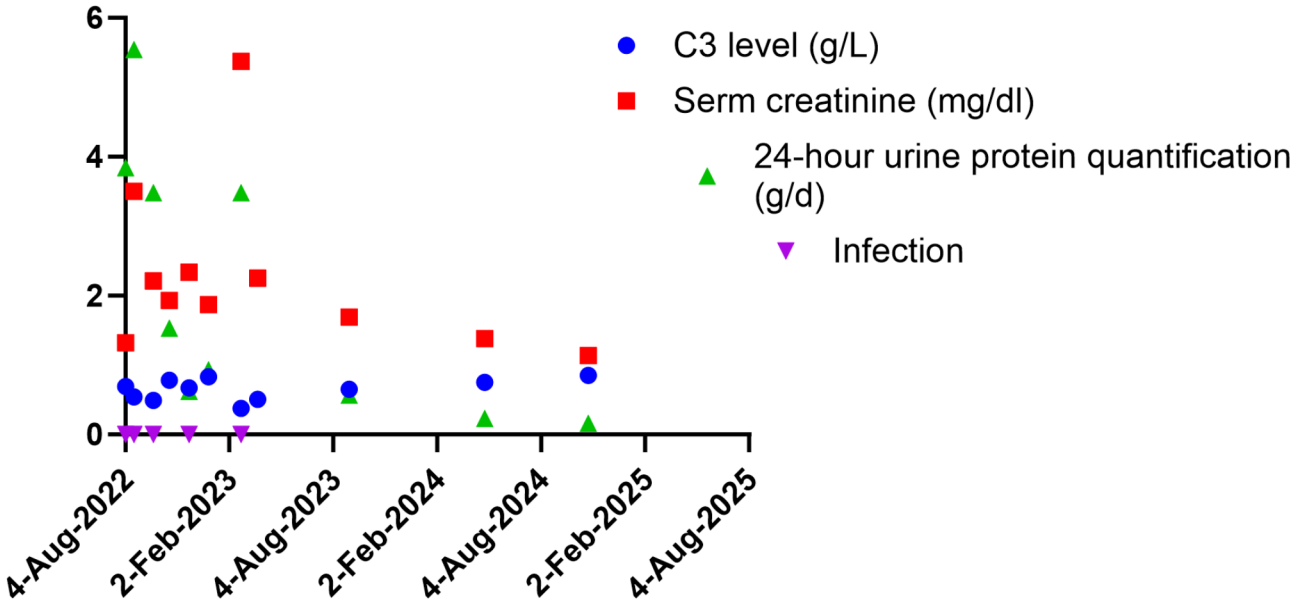

Fig. 4 The time series graph of the course of the disease

the most common, followed by urinary tract infections [3]. C3G and PIGN are difficult to differentiate, and their histologic manifestations and the associated serum concentrations of C3 and C4 are similar. Currently, a clinical diagnosis can only be made according to whether or not spontaneous remission occurs within 8–12 weeks [2].

Immunofluorescence studies have shown that only C3 deposition accounts for approximately 55-57% of patients, whereas others show a small amount of immunoglobulin deposition, with IgM being the most common form [3, 10]. However, only 4.4% of patients with C3G show IgA deposition [3].

The deposition of Gd-IgA1 in glomeruli is one of the pathological characteristics of IgAN. The KM55 kit contains a monoclonal antibody that specifically recognizes Gd-IgA1 and can be used for the identification of glomerular Gd-IgA1 [11]. Gd-IgA1 deposition is difficult to distinguish from primary IgAN, IgA vasculitis, IgAN secondary to IBD, psoriasis, and cirrhosis [11, 12]. However, it may be useful to distinguish primary IgAN from IgA-dominant postinfectious glomerulonephritis [12]. The Gd-IgA1 staining intensity in the kidneys of patients with IgA-dominant postinfectious glomerulonephritis was found to be significantly lower than that in patients with primary IgAN. Furthermore, using a threshold of 1+, the specificity and sensitivity for the diagnosis of primary IgAN were found to be 82% and 69%, respectively ($P=0.001$). Although there was no significant difference in the proportion of patients with IgA-dominant postinfectious glomerulonephritis (69%) or primary IgAN (100%) that were positive using a threshold of 0.5+, the negative predictive value of IgAN was 100% [12]. Gd-IgA1 staining in the present case was negative, and therefore primary IgAN could be ruled out.

The patient experienced multiple relapses of MP infection during the course of his disease, resulting in repeated increases in Scr, decreases in C3, and the expression of C3NeF. After treatment, his Scr gradually decreased, but his C3 continued to decrease. After 8 months, his C3 remained lower than normal, and the fluorescence intensity associated with C3 in the second of his renal biopsies was ≥2 orders of magnitude higher than that for IgA (3+vs. 1+). Therefore, it was possible to diagnose C3G. In our previous review [13] and in a multicenter cohort study conducted in France [14], the prevalence of a low serum C3 concentration of IgA-dominant postinfectious glomerulonephritis was 16-57%. A study of French multicenter C3G cohort revealed a higher prevalence of low serum C3 concentration of 40-60% [15], which was higher than the 16% revealed for the French multicenter IgA-dominant postinfectious glomerulonephritis cohort. Another cohort study of patients with C3G conducted in Italy yielded prevalences of low serum C3 concentration for patients with C3 glomerulonephritis (C3GN) and dense deposit disease (DDD) of 74% and 84%, respectively [16], which are significantly higher percentages than those reported for IgA-dominant postinfectious glomerulonephritis. Based on the above evidence, the prevalence of low C3 in patients with C3G is higher than that in patients with IgA-dominant postinfectious glomerulonephritis. Thus, for patients with IgA-dominant postinfectious glomerulonephritis with low C3 concentrations over 8–12 weeks or repeated episodes of low C3, the possibility of C3G should be considered.

C3NeF is detected in approximately 80% of patients with DDD and in 40-50% of those with C3GN [4]. Currently, it is believed that PIGN is actually a type of acute C3G [4] and is characterized by a transient abnormality in CAP and possibly transient C3NeF expression [17, 18]. C3NeF positivity was detected for the first time in patients suspected of IgA-dominant postinfectious glomerulonephritis, but it sometimes occurs in combination with glomerular immunostaining for C3 and a low serum C3 concentration, implying that IgA-dominant postinfectious glomerulonephritis can also be associated with a CAP disorder caused by infection and transient C3NeF positivity. Therefore, in future investigations of patients with IgA-dominant postinfectious glomerulonephritis, if a continuous decrease in complement C3 occurs or if there is strong positive glomerular C3 staining, the possibility of C3G should be considered and the C3NeF level should be evaluated. C4d is a product of activation of the classic and lectin pathways, and in theory, because C3G is caused by the abnormal activation of the CAP, there should be no deposition of C4d. However, a recent cohort study showed that only 17% of patients with C3G are C4d negative and 68.3% show strong C4d staining (2+-3+) [19]. Therefore, C4d cannot be used as a diagnostic marker of C3G.

A case of C3G caused by MP was reported [9]: a 58-year-old woman developed nephrotic syndrome, microscopic hematuria, and acute kidney injury (AKI) after being infected with MP. Renal biopsy showed diffuse endocapillary proliferative glomerulonephritis with crescents, and immunofluorescence revealed only C3 deposition in the mesangium. In addition, a 46-year-old male patient with postinfectious glomerulonephritis caused by MP infection was reported [20]. Similar to the present case, the patient experienced persistent gross hematuria in the absence of pain following infection with MP. He had a maximum urine protein content of 5.3 g/24 h, a C3 concentration of 0.647 g/L, AKI, and a maximum Scr of 601 μmol/L. Histologic investigation revealed MPGN, and immunofluorescence revealed IgG 1+, IgA 1+, and C3 3+, but electron microscopy was not performed. After undergoing anti-infection, glucocorticoid, and CTX treatment, the patient's renal function returned to normal and his proteinuria resolved. There is a possibility that the patient had C3G.

This case report had several limitations, including the failure to attempt to detect C3NeF during the early stage of management. Moreover, a case report study always has the potential for biased conclusions. However, the study also has some strengths. Because of comprehensive follow-up examinations, we can clearly see the relationship between low serum C3 concentration and recurrent infection. Gd-IgA1 staining in the present patient was negative, and therefore primary IgAN could be ruled out.

In conclusion, MP infection can cause sustained activation of the CAP, leading to C3G. For patients with MP infection, if there is an ongoing decrease in complement C3 level and a progressive increase in serum creatinine, it is crucial to be vigilant for possible C3G and to consider the use of immunosuppressive therapy in conjunction with anti-infective treatment to prevent the ongoing activation of the CAP.

Abbreviations

C3G	C3 glomerulopathy
CAP	Complement alternative pathway
PIGN	Postinfectious glomerulonephritis
MP	Mycoplasma pneumoniae
Scr	Serum creatinine
IgAN	IgA nephropathy
MMF	Mycophenolate mofetil
CTX	Cyclophosphamide
MPGN	Membranoproliferative glomerulonephritis
C3GN	C3 glomerulonephritis
DDD	Dense deposit disease
AKI	Acute kidney injury

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Author contributions

Z-YD: Writing– original draft, Writing– review & editing. J-JL: Writing– original draft, Writing– review & editing. G-YC: Writing–review & editing. S-XW: Writing–review & editing. X-LT: Visualization, Writing– review & editing. W-YH: Writing–review & editing. LJ: Data curation, Writing– review & editing. YS: Supervision, Writing– review & editing.

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Data availability

The original data collected during the study are included in the article. Further inquiries can be directed to the corresponding author.

Declarations

Ethics approval and consent to participate

Written informed consent to participate obtained from patient.

Consent for publication

Written informed consent for publication obtained from patient.

Competing interests

The authors declare no competing interests.

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