

[CASE REPORT]

Glomerulonephritis Associated with Infective Endocarditis Showing Serological Positivity for PR3-anti-neutrophil Cytoplasmic Antibody and Anti-glomerular Basement Membrane Antibody

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Abstract:

We herein report a case of crescentic glomerulonephritis (GN) associated with infective endocarditis (IE). A 61-year-old-woman presented with a fever and renal dysfunction and was diagnosed with IE. The patient was positive for proteinase 3-anti-neutrophil cytoplasmic antibody (PR3-ANCA) and anti-glomerular basement membrane (GBM) antibodies. Renal biopsy findings showed crescentic GN with isolated deposition of C3c, a serum conversion product of complement C3. Given these clinical findings, the patient was diagnosed with infective endocarditis (IE)-associated GN. Antibiotic therapy was continued without immunosuppressive agents. After the initiation of the antibiotics, the fever resolved, and the renal function gradually recovered. This case highlights the notion that laboratory findings should be carefully evaluated with reference to other findings.

Key words: anti-glomerular basement membrane antibody, anti-neutrophil cytoplasmic antibody-associated vasculitis, infective endocarditis, proteinase 3-anti-neutrophil cytoplasmic antibody

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Introduction

Infective endocarditis (IE) is an important disease in the differential diagnosis of a fever of unknown origin. For the diagnosis of IE, it is important to identify the pathogen by blood culture tests based on the Duke criteria (1). The most common symptom of IE is a fever; however, it can present with various signs and symptoms, such as cerebral complications and splenomegaly (1). IE also affects the kidneys, and the presenting symptoms are hematuria and acute kidney injury (AKI) (1, 2).

Hypovolemia, an impaired cardiac function, tubular intoxication, glomerular injury, and vascular obstruction are included among the various causes of AKI (3), which presents with a rapid decline in the renal function. The Kidney Disease Improving Global Outcomes has recommended a

staging system for the severity of the AKI according to the serum creatinine (Cr) and urine output (4). Even a slight increase in the Cr level has been shown to increase the risk of death; therefore, an early diagnosis and prompt treatment of AKI are important (5).

AKI due to anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) and anti-glomerular basement membrane (GBM) antibody disease accounts for approximately 10% of all AKI cases, with patients often presenting with urinary protein (UP) and urinary occult blood (UOB) (6). Typical histopathological findings of AAV and anti-GBM antibody disease are disrupted crescentic glomerulonephritis (GN). Immunohistochemistry (IHC) and immunofluorescence (IF) show no significant deposition of immunoglobulin or complement in AAV with renal involvement or linear IgG deposition in anti-GBM antibody disease. AAV and anti-GBM antibody disease mainly present with

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rapidly progressive GN; therefore, prompt treatment with immunosuppressive therapy is important (6).

ANCA and anti-GBM antibodies are measured as reference markers for the diagnosis and disease activity. ANCA is divided into perinuclear (P)-ANCA and cytoplasmic (C)-ANCA, as measured by immunoassays or indirect immunofluorescent techniques (7). Multiple ANCA-specific antigens have been identified, and proteinase 3 (PR3)-ANCA, the main specific antigen of C-ANCA, is strongly associated with AAV (8, 9). ANCAs are also positive under various conditions, such as infectious diseases, malignant diseases, collagen diseases, and drug-related adverse effects (10). It is likely that ANCA activates neutrophils in response to genetic and environmental factors that cause GN (10-12).

In contrast, anti-GBM antibodies are extremely sensitive and specific for anti-GBM antibody disease (13). Although anti-GBM antibody is highly specific, approximately 7.5-14% of patients with AAV are positive for anti-GBM antibodies (14). Clinicians often encounter difficulty in the diagnosis of patients with infection-associated GN with ANCA antibody and ANCA-associated GN because infection-associated GN and ANCA-associated GN present with similar clinical symptoms, such as a fever, malaise, and weight loss. It is therefore necessary to accurately determine which is the cause of renal dysfunction and to provide prompt treatment.

Case Report

A 61-year-old woman was admitted to our hospital with a fever, cough, and progressive renal dysfunction within three months after cardiovascular surgery. A year before admission, the patient had undergone aortic valve replacement (AVR) for severe aortic stenosis (AS). Six months after AVR, replacement of the ascending aorta was performed due to an enlarging aneurysm of the ascending aorta. After surgery, the patient developed bacteremia (blood culture test demonstrated methicillin-resistant coagulase-negative *Staphylococci*) of unknown origin. After four weeks of daptomycin (DAP) therapy, the patient presented to our hospital with abnormal urinalysis findings and elevated levels of serum Cr (Table). Approximately three months after the onset of renal dysfunction, the patient presented to our hospital with a persistent fever, cough, and malaise; she was therefore hospitalized for a closer examination.

On the day of admission, the patient had general fatigue and a fever. On an examination, her height was 162.7 cm, weight was 64.5 kg (1.6 kg weight loss within 4 months), body mass index (BMI) was 24.4 kg/m², temperature 37.1°C, pulse 80 beats per minute, blood pressure 167/71 mmHg, and oxygen saturation 98% while breathing ambient air. A physical examination revealed conjunctival pallor and abdominal tenderness. No other abnormality was noted on the physical examination.

The results of laboratory tests showed white blood cells 15,000/μL (neutrophils: 87%), hemoglobin 8.7 g/dL, total

protein 7.0 g/dL, albumin 2.6 g/dL, blood urea nitrogen 29 mg/dL, Cr 1.98 mg/dL (0.65 mg/dL at base line 3 months ago), estimated glomerular filtration rate 21.0 mL/min/1.73 m², C-reactive protein (CRP) 11.25 mg/dL, immunoglobulin (Ig) G 2,521 mg/dL, IgA 339 mg/dL, IgM 81 mg/dL, and complement 3 70 mg/dL. Complement 4 and total hemolytic complement levels were within the normal range. It was notable that PR3-ANCA (63.9 IU/mL; reference range <3.5 IU/mL) and anti-GBM antibodies (29.0 U/mL; reference range <7.0 U/mL) were positive. A urinalysis showed UP level of 3+ (8.15 g/g Cr), UOB level of 3+ [30> high-power field (HPF)], and various casts (Table). The patient was taking the following medications, including drugs to relieve her respiratory symptoms: aspirin (100 mg), azilsartan (40 mg), carvedilol (5 mg), atorvastatin (10 mg), lansoprazole (15 mg), codeine phosphate (15 mg), carbocysteine (1,500 mg), and inhaled vilanterol trifenatate fluticasone furoate (200 μg).

We suspected that the observed symptoms and renal dysfunction were induced by bacterial infection, so empiric antibiotic therapy with ceftriaxone (CTRX) was initiated. A low blood pressure was not observed in this patient's medical history, and the left ventricular ejection fraction by transthoracic echocardiography was 69%. We ruled out an impaired cardiac function and renal vascular obstruction by echography as other causes of renal dysfunction.

After the blood culture test was positive for CTRX-resistant *Staphylococcus capitis*, we added DAP to CTRX. However, the patient had abdominal tenderness in the left upper quadrant on the second day of admission, so [18F]-fluorodeoxyglucose-positron emission tomography/computed tomography (FDG-PET/CT) was performed. While FDG-PET/CT showed an infected prosthetic aortic valve and septic spleen embolism suggestive of IE, there were no signs of interstitial pneumonia or alveolar hemorrhage (Fig. 1).

The patient was diagnosed with IE by transesophageal echocardiography (TEE), and antibiotic therapy was continued. After the initiation of antibiotics, the size of vegetation did not change according to follow-up TEE. However, the fever resolved, and splenic infarction did not worsen on follow-up CT. The patient was discharged as IE improved with eight weeks of continuous antibiotic therapy with CTRX and DAP.

After discharge, the patient was prescribed oral clindamycin. Throughout this period, the renal function gradually improved to baseline levels of Cr, and the nephrotic-range proteinuria disappeared within 100 days; however, proteinuria of roughly 1 g/g Cr and urinary casts were sustained. A renal biopsy was thus performed for a close examination of renal dysfunction. Light microscopy showed focal crescentic sclerosing GN, with 28% (7/25) fibrocellular-to-fibrous crescents and 32% (8/25) global sclerosis. In addition, IF showed isolated mesangial deposition of C3c (Fig. 2). Based on these findings, the patient was diagnosed with IE-associated crescentic GN with pseudo-positive anti-GBM antibody. At this time, there was no active extracapillary pro-

Table. Laboratory Findings of Presented Case.

	Reference range, adults	3 months before admission	On admission, this hospital
Blood cell counts			
WBC (/μL)	3,300-8,600	6,600	15,000
Neutrophils (/μL)	1,620-6,540	4,640	12,990
Basophils (/μL)	0-150	50	450
Monocytes (/μL)	110-600	320	5,250
Lymphocytes (/μL)	960-3,100	1,330	1,440
RBC (×10 ⁴ /μL)	386-492	345	358
Hb (g/dL)	11.6-14.8	8.9	8.7
PLT (×10 ⁴ /μL)	15.8-34.8	24.6	23.3
Coagulation			
PT (%)	80.0-127.0	84.6	94.9
APTT (s)	26.9-38.1	33.7	39.3
Fibrinogen (mg/dL)	200-400	NA	414
D-dimer (μg/mL)	0.0-1.0	2.1	2.9
Biochemistry			
AST (IU/L)	13-30	18	39
ALT (IU/L)	7-23	11	26
ALP (IU/L)	106-322	271	250
LDH (IU/L)	124-222	160	304
Na (mEq/L)	138-145	141	134
K (mEq/L)	3.6-4.8	3.9	3.5
Cl (mEq/L)	101-108	107	101
Ca (mg/dL)	8.8-10.1	9.4	9.5
P (mg/dL)	3.7	3.7	3.0
BUN (mg/dL)	8-20	12	29
Cr (mg/dL)	0.46-0.79	0.72	1.98
eGFR (mL/min/1.73m ²)	NA	63	21.0
UA (mg/dL)	2.6-5.5	5.9	5.9
CRP (mg/dL)	0.00-0.14	1.76	11.25
TP (g/dL)	6.6-8.1	7.3	7.0
Alb (g/dL)	4.1-5.1	3.2	2.6
IgG (mg/dL)	861-1,747	2,260	2,521
IgA (mg/dL)	93-393	343	339
IgM (mg/dL)	50-269	57	81
C3 (mg/dL)	73-138	99	70
C4 (mg/dL)	11-31	19.9	15.9
CH50 (U/mL)	31.6-57.6	62.4	56.1
PR3-ANCA (IU/mL)	0.0-3.4	3.4	63.9
MPO-ANCA (U/mL)	0.0-3.4	<1.0	<1.0
Anti-GBM antibody (U/mL)	0.0-6.9	5.9	29.0
Urine analysis			
Dipstick			
Protein	Negative	Negative	3+
Blood	Negative	2+	3+
Urinary protein (g/g Cr)	NA	0.27	8.15
NAG (U/g Cr)	≤5.6	10.0	NA
Sediment			
RBC (/HPF)	<4	>30	>30
WBC (/HPF)	<4	<4	5-9
Cast			
Hyaline (/WF)	NA	1-9	30-99
Granular (/WF)	NA	ND	10-29
RBC (/WF)	NA	ND	30-99
WBC (/WF)	NA	ND	10-29
Fibrin (/WF)	NA	ND	1-9

Alb: albumin, ALP: alkaline phosphatase, ALT: alanine aminotransferase, APTT: activated partial thromboplastin time, AST: aspartate aminotransferase, BUN: blood urea nitrogen, Ca: calcium (corrected), CH50: 50% hemolytic complement unit, Cl: chlorine, Cr: creatinine, CRP: C-reactive protein, C3: complement component 3, C4: complement component 4, eGFR: estimated glomerular filtration rate, GBM: glomerular basement membrane, Hb: hemoglobin, HPF: high power field, IgA: immunoglobulin A, IgG: immunoglobulin G, IgM: immunoglobulin M, K: potassium, LDH: lactate dehydrogenase, MPO-ANCA: myeloperoxidase anti-neutrophil cytoplasmic antibody, NA: not applicable, Na: sodium, NAG: N-acetyl-β-D-glucosaminidase, ND: not detected, P: phosphorus, PLT: platelet, PR3-ANCA: proteinase 3-anti-neutrophil cytoplasmic antibody, PT: prothrombin time, RBC: red blood cell, TP: total protein, UA: uric acid, WBC: white blood cell, WF: whole field

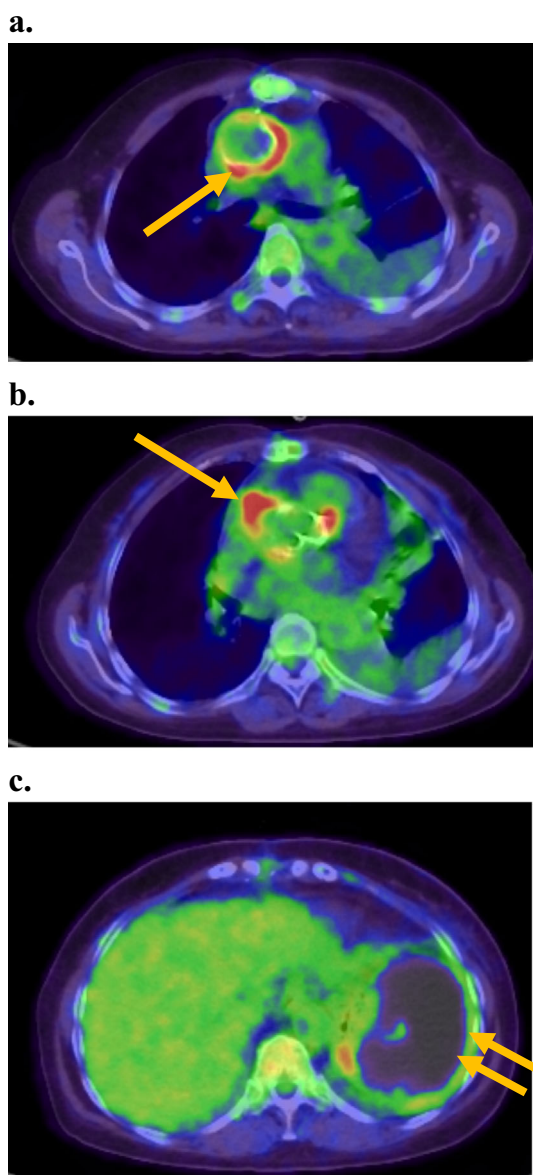


Figure 1. FDG-PET/CT. a-b: FDG-PET/CT showing a high uptake of FDG around the ascending aorta and replaced aortic valve (arrows). c: FDG-PET/CT showing a wide defect in the FDG uptake at the spleen (arrows). FDG-PET/CT: fluorodeoxyglucose-positron emission tomography/computed tomography

liferative lesion; we therefore did not add immunosuppressive therapy and continued antibiotic therapy alone for IE. The renal function considerably improved after long-term antibiotic therapy, and the PR3-ANCA and anti-GBM antibody titers decreased to within the normal range (Fig. 3).

Discussion

We encountered a case of GN induced by IE. Renal dysfunction associated with infections is common and shows various renal histopathological findings (15). Treatment, including immunosuppressive therapy, antibiotics, or surgery, should be performed based on pathophysiological conditions. Adequate and prompt treatment can improve the renal

function, as in this case. However, if infections cannot be controlled, renal dysfunction might progress and eventually lead to end-stage kidney disease (ESKD).

IE is an infection of the endocardial surface of the heart. The major risk factors include prosthetic valves, intracardiac devices, chronic rheumatic heart disease, intravenous catheter use, and diabetes mellitus (DM) (1). IE is a common disease; however, it is difficult to diagnose unless suspected and followed by a detailed examination. In this case, we suspected IE due to abdominal pain associated with splenic infarction, thus arriving at a conclusive diagnosis. Extracardiac complications of IE occur due to embolism and immunological phenomena (1, 16). Splenic infarction is an important extracardiac complication and is often caused by heart disease, including IE, atrial fibrillation, and hematopoietic disorders (1, 16). Splenic infarction occurs in approximately 40% of cases of left-sided IE, and the most common symptom of splenic infarction is sudden left-sided abdominal pain, as seen in this case (17). A low-attenuation area on CT is helpful for a conclusive diagnosis. There is no specific treatment; however, treatment is recommended according to the underlying disease.

GN, an extracardiac complication, is also known to be caused by the immunological phenomena associated with IE. AKI is the most common clinical syndrome associated with IE-associated GN (18). It has been reported that hypocomplementemia with ANCA antibody is associated with an increased incidence of IE-associated GN; the most common histopathological feature is necrotizing and crescentic GN (18).

We considered that the patient's main pathogenesis of crescentic GN was due to IE rather than AAV due to IE because of the clinical course after antibiotic alone and isolated deposition of C3c. Isolated deposition of C3 is the most common pathological features of IE-associated GN (18). In this case, PR3-ANCA and anti-GBM antibodies were within the normal range at three months before admission. In contrast, proteinuria and hematuria were positive, and C-reactive protein was mildly elevated three months before admission. Antibiotic therapy alone was effective in improving renal dysfunction and decreasing PR3-ANCA and anti-GBM antibody titers in conjunction with IE during the clinical course. It is possible that both ANCA and anti-GBM antibody were produced by the immune response to IE, and IE-associated GN beginning three months before admission became exacerbated. However, AAV after IE could not be ruled out because of the high levels of PR3-ANCA. Since IF showed no significant deposition of IgG, it is unlikely that the patient developed anti-GBM antibody disease. These results suggest that additional immunosuppressive therapy was not necessary in this case. However, anti-GBM antibody titers must be followed-up carefully (14).

Based on the findings of IF, C3 GN is also a differential diagnosis. However, the clinical evidence of infection and crescentic GN lacking an membranoproliferative GN pattern would favor IE-associated GN over C3GN (19). In addition,

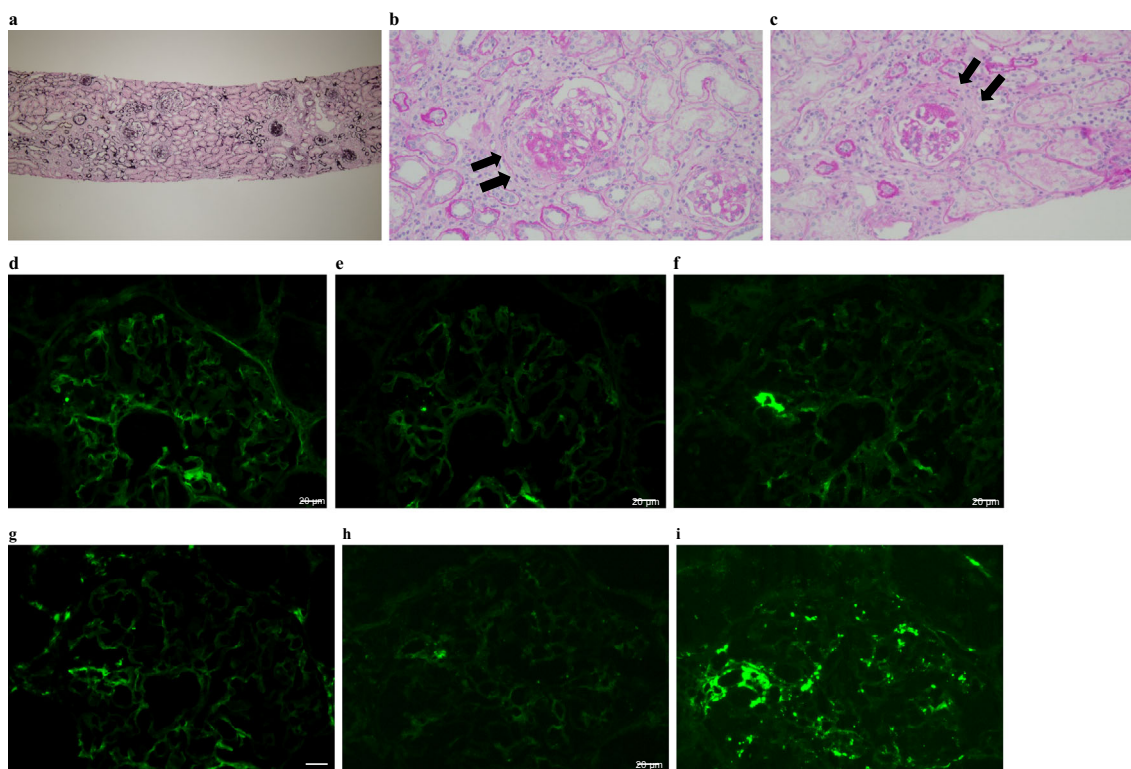


Figure 2. Renal biopsy findings of the present case. a: PAM stain showing focal crescentic sclerosing GN and global sclerosis of some glomeruli. Focal tubular atrophy with interstitial fibrosis and inflammatory infiltration was also noted. Tubular shedding cells were not observed. Original magnification $\times 40$. b-c: PAS stain showing a partly sclerotic glomerular tuft overlaid by a fibrocellular crescent (black arrows). Original magnification $\times 200$. d: Immunofluorescence showing no significant deposition of IgG. Original magnification $\times 400$. e: Immunofluorescence showing no significant deposition of IgA. Original magnification $\times 400$. f: Immunofluorescence showing no significant deposition of IgM. Original magnification $\times 400$. g: Immunofluorescence showing no significant deposition of Fib. Original magnification $\times 400$. h: Immunofluorescence showing no significant deposition of C1q. Original magnification $\times 400$. i: Immunofluorescence showing mesangial deposition of C3c. Original magnification $\times 400$. C3c: complement 3c, Fib: fibrinogen, IgA: Immunoglobulin A, IgG: Immunoglobulin G, IgM: immunoglobulin M, PAM: periodic acid-methenamine silver, PAS: periodic acid-Schiff

the patient was treated with several drugs, including antibiotics, before admission, so tubular intoxication is also a differential diagnosis. However, tubular shedding cells suggestive of tubular intoxication were not observed, and urinary N-acetyl- β -D-glucosaminidase levels were only mildly elevated three months before admission. These findings suggested that tubular intoxication with drugs was unlikely.

It has been reported that immunosuppressive therapy with glucocorticoids is effective in treating ANCA-positive renal dysfunction induced by IE (20). In a review of ANCA-positive renal dysfunction induced by IE, immunosuppressive therapy with glucocorticoids, azathioprine, rituximab, and cyclophosphamide was effective in a patient with crescentic GN on a renal biopsy. However, one-third of the cases improved with antibiotic therapy alone (20). Another review of IE-associated GN, including ANCA-negative patients, showed higher mortality rates in patients receiving combined immunosuppressive therapy. Mortality was also high in patients with a diffuse crescentic pattern of glomeru-

lar injury. The renal outcomes were better in the group of patients who underwent valve replacement or repair (18). These findings suggest that the efficacy of immunosuppressive therapy in patients with IE-associated GN is controversial.

Rapidly progressive GN associated with AAV or anti-GBM antibody requires adequate and prompt treatment with immunosuppressive therapy. It is reported that an early renal biopsy is important to differentiate AAV and IE-associated GN (21). This case suggested that blood cultures, the findings of IF or IHC, and PET-CT in addition to an early renal biopsy are useful clues for making a differential diagnosis of IE-associated GN and AAV/anti-GBM antibody disease. In particular, the findings of IF or IHC are useful, as the deposition pattern differs between IE-associated GN and AAV/anti-GBM antibody disease (18).

The clinical features of infection-related GN (IRGN) vary according to the underlying organisms (19). The two most frequently identified infectious organisms of IRGN are

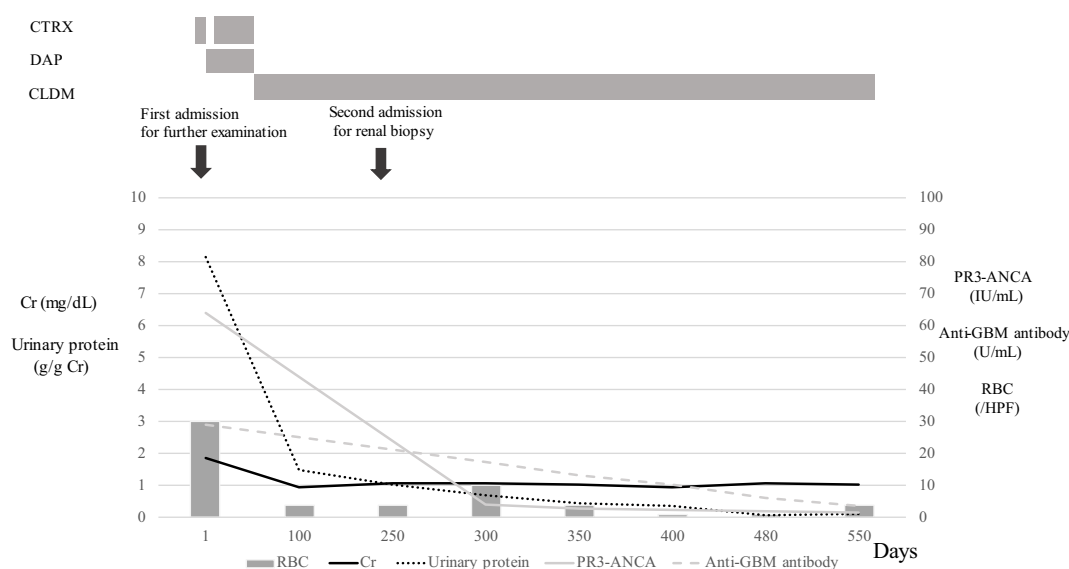


Figure 3. Clinical course of the present case. The levels of Cr, UP, and hematuria improved after the initiation of antibiotic therapy, and the renal function did not worsen. PR3-ANCA and anti-GBM antibody titers also decreased to within the normal range. The patient is under regular follow-up at our hospital and at another hospital. To date, the patient has not been treated with immunosuppressive agents, such as steroids. CLDM: clindamycin, Cr: creatinine, CTRX: ceftriaxone, DAP: daptomycin, GBM: glomerular basement membrane, HPF: high power field, MPO-ANCA: myeloperoxidase-anti-neutrophil cytoplasmic antibody, PR3-ANCA: proteinase 3-anti-neutrophil cytoplasmic antibody, RBC: red blood cell, UP: urinary protein

Streptococcus and *Staphylococcus*. *Staphylococcus* is more common in developed countries and elderly patients, and DM is a major risk factor for *Staphylococcus*-related GN. Despite treatment with immunosuppressive therapy, antibiotics, or surgery, 8-54% of patients with IRGN develop persistent renal dysfunction, and 4-33% progress to ESKD. If *Staphylococcus* is detected, renal dysfunction is typically more markedly progressed due to the advanced age of patients and the presence of underlying diseases, such as DM (19).

Several limitations associated with the present study warrant mention. First, a renal biopsy was not performed in the acute phase, so the serum Cr levels had improved from the peak by the time of the renal biopsy. The histological findings showed several global scleroses and were fully evaluated during the acute phase. As a result, endocapillary proliferative GN or thrombotic microangiopathy may not have been observed. Second, IF showed no significant deposition of IgG, but indirect IF for anti-GBM antibodies was not performed. Whether this was a true or false positive thus remains unclear.

In conclusion, ANCA and anti-GBM antibody testing are very useful for the diagnosis and monitoring of AAV and anti-GBM antibody disease. Infectious disease is also a trigger for AAV and anti-GBM antibody disease, while infectious disease is the reason for the serological positivity of these antibodies. Therefore, a renal biopsy is an essential tool for making decisions regarding additional immunosuppressive therapy. If a patient's symptoms indicate the probability of IE based on risk factors, the presence of vegeta-

tions should be aggressively investigated by TEE. PET-CT can not only assist in the diagnosis of IE but also help identify instances of a fever of unknown origin, as in this case. It is important to make a comprehensive assessment based on physical and imaging findings, rather than one based solely on a blood test.

Informed consent was obtained from the patient. All procedures were performed in accordance with the principles of the Declaration of Helsinki.

The authors state that they have no Conflict of Interest (COI).

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