A case report of PR-3-ANCA-positive glomerulonephritis with histological features of GPA associated with infectious endocarditis

Momoko Hirata, MDa, Haruhisa Miyazawa, MD, PhD, Junki Morino, MD, Shohei Kaneko, MD, Saori Minato, MD, Yana Katsunori, MD, Hiroki Ishii, MD, PhD, Taisuke Kitano, MD, Kiyonori Ito, MD, PhD, Keiji Hirai, MD, PhD, Takashi Oda, MD, PhD, Akira Shimizu, MD, PhD, Yoshihiko Ueda, MD, PhD, Yoshiyuki Morishita, MD, PhD

Abstract

Rationale: Several renal diseases are associated with infectious endocarditis. However, there are few reports on patients with granulomatosis with polyangiitis (GPA) associated with infectious endocarditis, and there is no consensus for appropriate treatment.

Patients concerns: A 35-year-old man with congenital ventricular septal defect presented severe anemia, hematuria and proteinuria. The blood and urine examinations showed elevated white blood cells (12,900 cells/μL), C-reactive protein level (13.1 mg/dL) and proteinase 3-anti-neutrophil cytoplasmic antibody (PR3-ANCA) level (11.0 IU/mL), severe anemia (hemoglobin: 6.1 g/dL) and renal dysfunction [estimated glomerular filtration rate (eGFR): 12.7 ml/min.1.78 m2 with hematuria and proteinuria].

Diagnoses: The patient was diagnosed with crescentic glomerulonephritis with histological features of GPA associated with infectious endocarditis by renal biopsy and transthoracic echocardiography.

Interventions: Antibacterial drugs (ampicillin-sulbactam) were administrated. No immunomodulating agents were used because immunosuppressive drugs may worsen infectious endocarditis. Subsequently, renal function and urinary findings improved. However, infectious endocarditis was not improved. Therefore, valve replacements and ventricular septal closure surgery were conducted.

Outcomes: Thereafter, his postoperative course was uneventful, renal function improved (eGFR: 64.3 ml/min.1.78 m2), and PR3-ANCA level normalized.

Lessons: We reported a case report of PR3-ANCA positive glomerulonephritis with histological features of GPA associated with infectious endocarditis by renal biopsy and transthoracic echocardiography.

Abbreviations: GPA = granulomatosis with polyangiitis, PR3-ANCA = proteinase 3-anti-neutrophil cytoplasmic antibody.

Keywords: granulomatosis with polyangiitis, infectious endocarditis, proteinase 3-anti-neutrophil cytoplasmic antibody positive glomerulonephritis

1. Introduction

Several forms of renal disease are accompanied with infectious endocarditis.[1–3] Patients with infectious endocarditis can also develop multiple serological abnormalities including antineutrophil cytoplasmic antibody (ANCA) production, notably proteinase-3-ANCA (PR3-ANCA). Additionally, granulomatosis with polyangiitis (GPA) (previously known as Wegener’s granulomatosis) is a small vessel vasculitis associated with
ANCAs, especially PR3-ANCA. However, few reports have described patients with GPA associated with infectious endocarditis.

Herein, we describe a 35-year-old man who presented rapidly progressive crescentic glomerulonephritis with histological features of GPA associated with infectious endocarditis. The patient’s renal biopsy revealed histopathological features of GPA. He was treated successfully with antibacterial monotherapy, and the titer of PR3-ANCA normalized with improvement of renal disease.

2. Case presentation

A 35-year-old man presented with congenital ventricular septal defect. Periodic medical examinations showed normal heart function that required no intervention. Additionally, to this point, he had no history of renal dysfunction such as proteinuria or hematuria. Although he was asymptomatic, renal dysfunction and severe anemia were identified upon routine medical checkup, and he was advised to be admitted to hospital. On admission, he had normal temperature at 36.3°C, he had elevated white blood cells [12,900 cells/µL (normal range: 3900~9800 cells/µL)] and C-reactive protein level [13.1 mg/dL (normal range: 0.00~0.14 mg/dL)], severe anemia [hemoglobin: 6.1 g/dL (normal range: 12.0~17.6 g/dL)], and renal dysfunction [estimated glomerular filtration rate (eGFR): 12.7 mL/min/1.73 m² with hematuria and proteinuria]. The blood culture was not conducted. He was treated with an antibacterial agent for suspected infection and transfusions for the anemia. However, his condition and laboratory parameters, including renal dysfunction, did not improve. Therefore, 3 days after admission, he was referred to our department for further management.

On admission, his body temperature was 36.7°C, and his blood pressure was 150/106 mm Hg. A systolic murmur was auscultated in the intercostal space at the left sternal border, and bilateral edema of the lower extremities was observed. There were no other physical findings of infectious endocarditis such as Osler’s nodes, Roth spots, or Janeway lesions. Blood testing indicated renal dysfunction [estimated glomerular filtration rate (eGFR): 13.8 mL/min/1.73 m², blood urea nitrogen: 54 mg/dL], inflammation [white blood cells: 14,960 cells/µL, C-reactive protein: 11.09 mg/dL, procalcitonin: 1.16 ng/mL (normal range: <0.5 ng/mL)], and anemia (hemoglobin: 8.8 g/dL). Urinalysis showed microscopic hematuria, and proteinuria (1.49 g/Cr). PR3-ANCA level was elevated at 11.0 IU/mL (normal range: <2.0 IU/mL), and antistreptolysin O was elevated at 367 IU/mL (normal range: 0~240 IU/mL). Hepatitis B antigen, hepatitis C antibody, antinuclear antibody, myeloperoxidase-ANCA, and antilongomer basement membrane antibody were negative. Serum complement C3 [39 mg/dL (normal range: 86~160 mg/dL)] and CH50 [< 10 U/mL (normal range: 30~46 U/mL)] levels were decreased, while C4 [26 mg/dL (normal range: 17~45 mg/dL)] level was normal. Two blood cultures were negative. Detailed laboratory data on admission are shown in Table 1. Transthoracic echocardiography showed a vegetation on the pulmonary valve, and computed tomography showed multiple nodular shadows in bilateral lung fields, and bilateral kidney enlargement. The patient met the modified Duke criteria for definitive infectious endocarditis.

On the second hospital day, we performed renal biopsy to evaluate his renal disease. Histological analysis revealed that 57% (8/14) of glomeruli showed cellular crescents (Fig. 1B, star) with intraglomerular neutrophil infiltration (Fig. 1A, arrowhead). Necrotizing granuloma formation was observed in the tubulointerstitial area (Fig. 1C, arrowhead). Figure 1D shows a high magnified image of Figure 1C. Granuloma formation around an artery was observed. Necrotizing granulomatous arteritis of arterioles was also observed (Fig. 1E). Arterioles demonstrating necrotizing vasculitis with fibrin exudation (Fig. 1F and 1G, arrowheads) and those with granulomatous arteritis without fibrin exudation were observed (Fig. 1F and 1G). Immunofluorescence microscopy showed granular C3 deposition along capillary walls in glomeruli without deposition of immunoglobulins or other complement factors (Fig. 1H and 1I). Nephritis-associated plasmin receptor (NAPr) staining and plasmin activity on glomeruli were negative (data not shown). Electron microscopy showed small electron-dense deposits in subendothelial and paramesangial areas (Fig. 1J). On the basis of these findings, the patient was diagnosed with crescentic glomerulonephritis with histological features of GPA associated with infectious endocarditis. Although he did not have upper respiratory lesions, he met the diagnostic criteria for GPA (abnormal urinalysis, abnormal findings on chest imaging, granuloma on biopsy, and PR3-ANCA positivity). We began therapy with antibacterial drugs (ampicillin-sulbactam) only. No immunomodulating agents were used because immunosuppressive drugs may worsen infectious endocarditis. Subsequently, the inflammatory response decreased steadily, and renal function, the urinary findings and the levels of PR3-ANCA improved [CRP: 2.09 mg/dL, eGFR: 40.4 mL/min/1.78 m², proteinuria 2.32 g/Cr, PR3-ANCA: 9.71 IU/mL] on hospital day 30 with antibiotic therapy for management of the endocarditis (Fig. 2). He did not require dialysis.

However, the vegetation on the pulmonary valve grew gradually, and an aortic regurgitation appeared. Therefore, valve replacements and ventricular septal closure surgery were required.

We repeated the renal biopsy on hospital day 36 to re-evaluate his renal pathological condition before receiving heart operation. According to histological analysis, 31% (4/13) of glomeruli showed fibrous or cellular crescents, GPA was not observed, and neutrophil and lymphocyte infiltration had improved. His renal function and urinary findings showed further improvement (eGFR: 50.8 mL/min/1.78 m², proteinuria 1.25 g/Cr, CRP: 0.95 mg/dL) on hospital day 48. Then, we performed pulmonary and aortic valve replacement and ventricular septal closure on hospital day 53. Histopathological features of the replaced valves were consistent with infectious endocarditis. His postoperative course was uneventful, renal function further improved (eGFR: 64.3 mL/min/1.78 m²) on hospital day 63. The size of the multiple nodular shadows on chest computed tomography decreased, and he was discharged on hospital day 70. After that, his renal function maintained (eGFR: around 60 mL/min/1.73 m² and proteinuria: <0.5 g/Cr) and PR-3 ANCA levels showed normal range on periodical examination in outpatient department.

3. Discussion

We report a case of rapidly progressive crescentic glomerulonephritis with histological features of GPA associated with infectious endocarditis. Our patient met the diagnostic criteria for GPA.[4,5] Although several forms of renal disease have been reported to be associated with infectious endocarditis,[6–7] to our knowledge, patients with crescentic glomerulonephritis accom-
patients with infectious endocarditis-associated glomerulonephritis (regardless of ANCA positivity) revealed the most common histological change was necrotizing and crescentic glomerulonephritis (53%), followed by endocardial proliferative glomerulonephritis (37%). C3 deposition was prominent in all cases, whereas IgG deposition was observed in <30% of cases. In the present case, we observed C3 deposition along glomerular capillary walls without deposition of immunoglobulins or electron-dense deposits in subendothelial and parameangial areas. These findings suggest mechanisms associated with infectious endocarditis that are independent of PR3-ANCA contribution to development of renal disease in this patient. In contrast, other studies reported that immune complexes containing C3 can be deposited in glomerular capillary walls in patients with ANCA-associated glomerulonephritis without evidence of underlying infections. Further studies and case analyses are necessary to fully elucidate the mechanisms underlying the development of glomerulonephritis associated with infectious endocarditis, including the pathological roles of ANCA and C3.

Table 1

| Laboratory findings on admission. | Unit | Normal range | Immunological testing | Unit | Normal range |
|----------------------------------|------|--------------|-----------------------|------|--------------|
| **Complete blood count and blood chemistry** | | | | | |
| WBC | 14960 | µL | 3900–9800 | ASO | 367 | IU/mL | 0–240 |
| Band | 1.0 | % | 0–19 | IgG | 3229 | mg/dL | 870–1700 |
| Segment | 90.0 | % | 25–72 | IgA | 479 | mg/dL | 110–410 |
| Eosinophil | 0 | % | 0–7.0 | IgM | 315 | mg/dL | 33–190 |
| Basophil | 0 | % | 0–2.0 | C3 | 39 | mg/dL | 86–160 |
| Lymphocyte | 4.0 | % | 19.0–49.0 | C4 | 26 | mg/dL | 17–45 |
| Monocyte | 5.0 | % | 3.4–9.0 | CH50 | <10 | IU/mL | 30–46 |
| RBC | 323 × 10^12/µL | | 427–570 | ANA | 11.0 | IU/mL | <2.0 |
| Hemoglobin | 8.8 | g/dL | 12.0–17.6 | PR3-ANCA | 11.0 | IU/mL | <2.0 |
| Hematocrit | 27.9 | % | 39.8–51.8 | MPO-ANCA | 1.0 | IU/mL | <3.5 |
| Platelet | 16.6 × 10^9/µL | | 13–36.9 | Anti-GBM-Ab | <1.0 | IU/mL | <7.0 |
| **Urine analysis** | | | | | |
| Gravity | 1.015 | | | pH | 5.0 | |
| RBC | 30–49 (dysmorphic) | | | WBC | 20–29 | | |
| Protein | 1.49 | g/dL | | NAG | 47.5 | IU/L | <7.0 |
| β2-MG | 273 | µg/L | | | | |
| BUN | 29–40 | | | | |
| Cr | 4.41 | mg/dL | | | |
| eGFR | 13.8 | ml/min/1.73m² | | | |
| **Immunological testing** | | | | | |
| ASO | 367 | IU/mL | 0–240 | | |
| IgG | 3229 | mg/dL | 870–1700 | | |
| IgA | 479 | mg/dL | 110–410 | | |
| IgM | 315 | mg/dL | 33–190 | | |
| C3 | 39 | mg/dL | 86–160 | | |
| C4 | 26 | mg/dL | 17–45 | | |
| CH50 | <10 | IU/mL | 30–46 | | |
| ANA | 11.0 | IU/mL | <2.0 | | |
| PR3-ANCA | 11.0 | IU/mL | <2.0 | | |
| MPO-ANCA | 1.0 | IU/mL | <3.5 | | |
| Anti-GBM-Ab | <1.0 | IU/mL | <7.0 | | |
| | | | | | |
| β2-MG = β2 microglobulin, ALT = alanine aminotransferase, ANA = anti-nuclear antibody, ASO = antistreptolysin O, AST = aspartate aminotransferase, BUN = blood urea nitrogen, C3 = complement component 3, C4 = complement component 4, Ca = calcium, CH50 = 50% hemolytic unit of complement, Cl = chloride, Cr = creatinine, CRP = C-reactive protein, eGFR = estimated glomerular filtration rate, GBM = antiglomerular basement membrane antibody, HbA1c = hemoglobin A1c, HPF = high-power field, Ig = immunoglobulin, IL = international unit, K = potassium, MPO-ANCA = myeloperoxidase antineutrophil cytoplasmic antibody, Na = sodium, NAG = N-acetyl-β-D-glucosaminidase, P = phosphate, PCT = procalcitonin, PR3-ANCA = proteinase-3 antineutrophil cytoplasmic antibody, RBC = red blood cell, TSAT = transferrin saturation, WBC = white blood cell, WF = whole field Anti-nuclear antibody.

In our patient, PR3-ANCA level was initially elevated, and it decreased with improvement of renal disease. PR3-ANCA is positive in 5% to 10% of patients with renal disease complicated with infectious endocarditis. Interestingly, Bele et al reported the success of combined treatment with antibiotics and immunosuppressants for systemic vasculitis, which may be caused by ANCA-associated infections. In contrast, others proposed treatment with antibiotics alone. Moreover, there are currently no guidelines for treatment of ANCA-positive glomerulonephritis associated with infectious endocarditis. Recently, A recent large cohort study that analyzed histological changes in kidney biopsies from 49 patients with infectious endocarditis-associated glomerulonephritis reported that Renal diseases associated with infectious endocarditis have been shown to result in various pathological changes including crescent formation, fibrinoid necrosis, and granuloma formation in the kidney. Renal diseases associated with infectious endocarditis have been shown to result in various pathological changes including crescent formation, fibrinoid necrosis, mesangial cell proliferation, endothelial cell thickening in glomeruli, and tubulointerstitial damage with infiltration of immune cells. A recent large cohort study that analyzed histological changes in kidney biopsies from 49 patients with infectious endocarditis-associated glomerulonephritis (regardless of ANCA positivity) revealed the most common histological change was necrotizing and crescentic glomerulonephritis (53%), followed by endocardial proliferative glomerulonephritis (37%). C3 deposition was prominent in all cases, whereas IgG deposition was observed in <30% of cases. In the present case, we observed C3 deposition along glomerular capillary walls without deposition of immunoglobulins or electron-dense deposits in subendothelial and parameangial areas. These findings suggest mechanisms associated with infectious endocarditis that are independent of PR3-ANCA contribution to development of renal disease in this patient. In contrast, other studies reported that immune complexes containing C3 can be deposited in glomerular capillary walls in patients with ANCA-associated glomerulonephritis without evidence of underlying infections. Further studies and case analyses are necessary to fully elucidate the mechanisms underlying the development of glomerulonephritis associated with infectious endocarditis, including the pathological roles of ANCA and C3.
In the present case, pathogenic bacteria were not identified by blood cultures or culture test of the replaced valves. Increased serum levels of antistreptolysin O suggest possible Streptococcus species infection. However, we did not observe NAPrlr staining or plasmin activity in kidney biopsies, which are histological markers for infection-related glomerulonephritis. We cannot exclude the involvement of infection, because NAPrlr staining can be negative depending on time from disease onset to biopsy.\textsuperscript{[16]} C3 deposition in glomeruli observed by immunofluorescence and electron-dense deposits observed by electron microscopy may indicate infection-related glomerulonephritis. We were unable to diagnose the pathogenic condition related to the multiple nodular

Figure 1. Renal biopsy findings. (A) Neutrophil infiltration within the glomerulus (hematoxylin-eosin stain; magnification, 600×). (B) Formation of cellular crescent (Periodic acid-methenamine-silver stain; magnification, 400×). (C) Necrotizing granuloma formation in the cortex area (Masson trichrome stain; magnification, 200×). (D) High magnified image of Fig. 1C (Masson trichrome stain; magnification, 400×). (E) Necrotizing granulomatous arteritis in arteriole (Masson trichrome stain; magnification, 600×). (F) Necrotizing and granulomatous arteritis in arteriole (Periodic acid-methenamine-silver stain; magnification, 800×). (G) Same area as in Fig. 1F (Masson trichrome stain; magnification, 800×). (H) No deposition of immunoglobulin G (immunofluorescence; magnification, 400×). (I) Granular complement component 3 staining on the mesangial areas and glomerular capillary walls (immunofluorescence; magnification, 400×). (J) Small electron-dense deposits in the subendothelial area (uranyl acetate lead citrate stain; magnification, 8000×).

Figure 2. The patient’s clinical course. CRP = C-reactive protein, eGFR = estimated glomerular filtration rate, PR3-ANCA = proteinase-3 antineutrophil cytoplasmic antibody, U-Pro = urinary protein.
shadows on bilateral lung computed tomographic images because the patient declined to undergo lung biopsy. These may have been septic emboli or granulomas.

Regarding treatment for PR3-ANCA-positive renal disease complicated with infectious endocarditis, previous studies suggested antibiotic monotherapy for patients with low PR3-ANCA titers (<50U/mL) and combination therapy with antibiotics and immunosuppressive agents for patients with high PR3-ANCA titers (>50U/mL) when the condition does not improve with antibiotic monotherapy. In our patient, PR3-ANCA titer was low (11.0IU/mL), and because of concern that immunosuppressive drugs would increase the risk of exacerbating infectious endocarditis, we initiated antibiotic monotherapy. The patient's PR3-ANCA titer normalized with improvement of renal disease. The results in our patient suggest antibiotic monotherapy can be effective for rapidly progressive crescentic glomerulonephritis with histological features of GPA associated with infectious endocarditis. However, histological evidence and more data regarding treatment outcomes are required for PR3-ANCA-positive renal disease associated with infectious endocarditis.

In conclusion, we report a case of PR3-ANCA positive rapidly progressive crescentic glomerulonephritis with histological features of GPA associated with infectious endocarditis. The monitoring of PR-3 ANCA level and repeated renal biopsy may be useful to evaluate the pathological condition of these cases.

**Author contributions**

Conceptualization: Yoshiyuki Morishita.

Data curation: Momoko Hirata, Haruhisa Miyazawa, Junki Morino, Shohei Kaneko, Saori Minato, Yanai Katsunori, Hiroki Ishii, Taisuke Kitano, Kiyonori Ito, Keiji Hirai, Takashi Oda, Akira Shimizu, Yoshishiko Ueda, Yoshiyuki Morishita.

Writing – original draft: Momoko Hirata, Haruhisa Miyazawa.

Writing – review & editing: Haruhisa Miyazawa, Yoshiyuki Morishita

**References**

[1] Nasr SH, Radhakrishnan J, D’Agati VD. Bacterial infection-related glomerulonephritis in adults. Kidney Int 2013;83:792–803.

[2] Kannan S, Matteo TK. Diffuse crescentic glomerulonephritis in bacterial endocarditis. Pediatr Nephrol 2001;16:423–8.

[3] Orfila C, Lepert JC, Modesto A, Goudable C, Suc JM. Rapidly progressive glomerulonephritis associated with bacterial endocarditis: efficacy of antibiotic therapy alone. Am J Nephrol 1993;13:218–22.

[4] Leavitt RY, Fauci AS, Bloch DA, et al. The American College of Rheumatology 1990 criteria for the classification of Wegener’s granulomatosis. Arthritis Rheum 1990;33:1101–7.

[5] Watts R, Lane S, Hanslik T, et al. Development and validation of a consensus methodology for the classification of the ANCA-associated vasculitides and polyarteritis nodosa for epidemiological studies. Ann Rheum Dis 2007;66:222–7.

[6] Bools CL, Nasr SH, Walker PD, Couser WG, Larsen CP. Update on endocarditis-associated glomerulonephritis. Kidney Int 2015;87:1241–9.

[7] Pierce D, Calkins BC, Thornton K. Infectious endocarditis: diagnosis and treatment. Am Fam Physician 2012;85:981–6.

[8] Bele D, Kojc N, Perše M, et al. Diagnostic and treatment challenge of unrecognized subacute bacterial endocarditis associated with ANCA-PR3 positive immunocomplex glomerulonephritis: a case report and literature review. BMC Nephrol 2020;21:40.

[9] Peng H, Chen WF, Wu C, et al. Culture-negative subacute bacterial endocarditis masquerades as granulomatosis with polyangiitis (Wege

[10] Subra JF, Michelet C, Laporte J, et al. The presence of cytoplasmic antineutrophil cytoplasmic antibodies (C-ANCA) in the course of subacute bacterial endocarditis with glomerular involvement, coincidence or association? Clin Nephrol 1998;49:15–8.

[11] Töth T. Crescentic involved glomerulonephritis in infective endocarditis. Int Urol Nephrol 1990;22:77–88.

[12] Neugarten J, Gallo GR, Baldwin DS. Glomerulonephritis in bacterial endocarditis. Am J Kidney Dis 1984;3:371–9.

[13] Langlois V, Lesourd A, Girszyn N, et al. Antineutrophil cytoplasmic antibodies associated with infective endocarditis. Medicine (Baltimore) 2016;95:e2564.

[14] Sumida K, Ubara Y, Nomura K, et al. ANCA-associated crescentic glomerulonephritis with immune complex deposits. Clin Nephrol 2012;77:454–60.

[15] Chen M, Xing GQ, Yu F, Liu G, Zhao MH. Complement deposition in bacterial infection-related glomerulonephritis. Kidney Int 2013;83:792–803.

[16] Pierce D, Calkins BC, Thornton K. Infectious endocarditis: diagnosis and treatment. Am Fam Physician 2012;85:981–6.

[17] Bele D, Kojc N, Perše M, et al. Diagnostic and treatment challenge of unrecognized subacute bacterial endocarditis associated with ANCA-PR3 positive immunocomplex glomerulonephritis: a case report and literature review. BMC Nephrol 2020;21:40.

[18] Peng H, Chen WF, Wu C, et al. Culture-negative subacute bacterial endocarditis masquerades as granulomatosis with polyangiitis (Wege

[19] Subra JF, Michelet C, Laporte J, et al. The presence of cytoplasmic antineutrophil cytoplasmic antibodies (C-ANCA) in the course of subacute bacterial endocarditis with glomerular involvement, coincidence or association? Clin Nephrol 1998;49:15–8.

[20] Töth T. Crescentic involved glomerulonephritis in infective endocarditis. Int Urol Nephrol 1990;22:77–88.

[21] Neugarten J, Gallo GR, Baldwin DS. Glomerulonephritis in bacterial endocarditis. Am J Kidney Dis 1984;3:371–9.

[22] Langlois V, Lesourd A, Girszyn N, et al. Antineutrophil cytoplasmic antibodies associated with infective endocarditis. Medicine (Baltimore) 2016;95:e2564.

[23] Sumida K, Ubara Y, Nomura K, et al. ANCA-associated crescentic glomerulonephritis with immune complex deposits. Clin Nephrol 2012;77:454–60.

[24] Chen M, Xing GQ, Yu F, Liu G, Zhao MH. Complement deposition in bacterial infection-related glomerulonephritis. Kidney Int 2013;83:792–803.

[25] Pierce D, Calkins BC, Thornton K. Infectious endocarditis: diagnosis and treatment. Am Fam Physician 2012;85:981–6.

[26] Bele D, Kojc N, Perše M, et al. Diagnostic and treatment challenge of unrecognized subacute bacterial endocarditis associated with ANCA-PR3 positive immunocomplex glomerulonephritis: a case report and literature review. BMC Nephrol 2020;21:40.

[27] Peng H, Chen WF, Wu C, et al. Culture-negative subacute bacterial endocarditis masquerades as granulomatosis with polyangiitis (Wege

[28] Subra JF, Michelet C, Laporte J, et al. The presence of cytoplasmic antineutrophil cytoplasmic antibodies (C-ANCA) in the course of subacute bacterial endocarditis with glomerular involvement, coincidence or association? Clin Nephrol 1998;49:15–8.

[29] Töth T. Crescentic involved glomerulonephritis in infective endocarditis. Int Urol Nephrol 1990;22:77–88.

[30] Neugarten J, Gallo GR, Baldwin DS. Glomerulonephritis in bacterial endocarditis. Am J Kidney Dis 1984;3:371–9.

[31] Langlois V, Lesourd A, Girszyn N, et al. Antineutrophil cytoplasmic antibodies associated with infective endocarditis. Medicine (Baltimore) 2016;95:e2564.

[32] Sumida K, Ubara Y, Nomura K, et al. ANCA-associated crescentic glomerulonephritis with immune complex deposits. Clin Nephrol 2012;77:454–60.

[33] Chen M, Xing GQ, Yu F, Liu G, Zhao MH. Complement deposition in renal histopathology of patients with ANCA-associated pauci-immune glomerulonephritis. Nephrol Dial Transplant 2009;24:1247–52.

[34] Yamakami K, Yoshizawa N, Wakabayashi K, Takeuchi A, Tadakuma T, Boyle MD. The potential role for nephritis-associated plasmn receptor in acute poststreptococcal glomerulonephritis. Methods 2000;21:185–97.

[35] Kishimoto N, Mori Y, Yamahara H, et al. Cytoplasmic antineutrophil cytoplasmic antibody positive pauci-immune glomerulonephritis associated with infectious endocarditis. Clin Nephrol 2006;66:447–54.

[36] Haseyama T, Imai H, Komatsuda A, et al. Proteinase-3-antineutrophil cytoplasmic antibody (PR3-ANCA) positive crescentic glomerulonephritis in a patient with Down’s syndrome and infectious endocarditis. Nephrol Dial Transplant 1998;13:2142–6.