Proteinase 3-antineutrophil cytoplasmic antibody-positive necrotizing crescentic glomerulonephritis complicated by infectious endocarditis: a case report

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Abstract

Background: Proteinase 3-antineutrophil cytoplasmic antibody has been reported to be positive in 5–10% of cases of renal injury complicated by infective endocarditis; however, histological findings have rarely been reported for these cases.

Case presentation: A 71-year-old Japanese man with a history of aortic valve replacement developed rapidly progressive renal dysfunction with gross hematuria and proteinuria. Blood analysis showed a high proteinase 3-antineutrophil cytoplasmic antibody (163 IU/ml) titer. *Streptococcus* species was detected from two separate blood culture bottles. Transesophageal echocardiography detected mitral valve vegetation. Histological evaluation of renal biopsy specimens showed necrosis and cellular crescents in glomeruli without immune complex deposition. The patient met the modified Duke criteria for definitive infective endocarditis. On the basis of these findings, the patient was diagnosed with proteinase 3-antineutrophil cytoplasmic antibody-positive necrotizing crescentic glomerulonephritis complicated by *Streptococcus* infective endocarditis. His renal disease improved, and his proteinase 3-antineutrophil cytoplasmic antibody titer normalized with antibiotic monotherapy.

Conclusion: Few case reports have described histological findings of proteinase 3-antineutrophil cytoplasmic antibody-positive renal injury complicated with infective endocarditis. We believe that an accumulation of histological findings and treatments is mandatory for establishment of optimal management for proteinase 3-antineutrophil cytoplasmic antibody-positive renal injury complicated with infective endocarditis.

Keywords: Necrotizing crescentic glomerulonephritis, Infective endocarditis, Proteinase 3-antineutrophil cytoplasmic antibody

Background

Proteinase 3-antineutrophil cytoplasmic antibody (PR3-ANCA) has been reported to be positive in 5–10% of cases of renal injury complicated by infective endocarditis [1]; however, histological findings have rarely been reported for these cases. In addition, the clinical course and optimal treatment have not been fully clarified.

We report a case of a patient with rapidly progressive PR3-ANCA-positive necrotizing crescentic glomerulonephritis complicated by *Streptococcus* infective endocarditis. The patient's renal disease improved with antibiotic therapy without any immunosuppressive agents, and his PR3-ANCA titer normalized in accordance with improving infective endocarditis.
Case presentation

Our patient was a 71-year-old Japanese man who had undergone the Bentall procedure and biological aortic valve replacement for the treatment of descending aortic aneurysm and aortic regurgitation at 70 years of age. Thereafter, his renal function had been normal (serum creatinine level, 0.93 mg/dl) without hematuria and proteinuria. Two months before admission, he had appetite loss, malaise, and gross hematuria. One month before admission, he noticed purpura on his lower extremities. A laboratory examination conducted by his primary care physician showed anemia (hemoglobin, 9.2 g/dl), thrombocytopenia (platelet count, 10 × 10⁴/μl), hematuria, and proteinuria. Therefore, he was referred to our hospital for further management.

Upon admission, his body temperature was 36.9 °C, and his blood pressure was 120/60 mmHg. Anemia, edema, and symmetrically distributed palpable purpura of the lower extremities were observed. He had no characteristic physical findings of infective endocarditis, such as Osler nodes, Roth spots, and Janeway lesions. Cardiac auscultation revealed 2/6 systolic reflux murmur at the cardiac apex. Blood analysis showed that the patient’s serum creatinine level was elevated at 2.34 mg/dl, and his serum hemoglobin level was reduced at 7.6 g/dl. Urinalysis showed proteinuria at 0.74 g/g Cr and microscopic hematuria. PR3-ANCA level was elevated at 163 IU/ml (normal range, < 10 IU/ml). The patient had negative test results for hepatitis B antigen, hepatitis C antibody, cryoglobulin, antistreptolysin O, antineutrophil cytoplasmic antibody, and myeloperoxidase-antineutrophil cytoplasmic antibody, RBC red blood cells, RNP ribonucleoprotein, Sm Smith, WBC white blood cells.

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separate blood culture bottles. On the third hospital day, renal biopsy was performed. Histological analysis revealed that 54% (6 of 11) of glomeruli showed partial fibrinoid necrosis with fragmentation of glomerular tufts (Fig. 1a), and 27% (3 of 11) of glomeruli showed cellular crescents (Fig. 1b). No fibrocellular or fibrous crescents and no endocapillary proliferation were found. The mesangium showed no increase in cells or matrix. The tubulointerstitium partially showed neutrophilic and lymphocytic infiltration in the peritubular capillary and atrophy (Fig. 1c). Fibrinoid necrosis was not observed in vessel walls. Immunofluorescence microscopy showed no deposition of immunoglobulins and complement factors. Electron microscopy showed small amounts of nonspecific electron-dense deposits in subendothelial areas and the paramesangial area. At this point, the patient met the modified Duke criteria for definitive infective endocarditis [2] (mitral valve vegetation on echocardiography, two positive blood cultures of Streptococcus species drawn 3 days apart, glomerulonephritis). On the eighth hospital day, transesophageal echocardiography revealed mitral valve vegetation. On the 12th hospital day, spinal magnetic resonance imaging showed pyogenic spondylitis at T7/T8 and L4/L5. On the basis of these findings, the patient was diagnosed with rapidly progressive PR3-ANCA-positive necrotizing crescentic glomerulonephritis complicated by Streptococcus infective endocarditis. Antibiotic therapy including cefazolin and penicillin G followed by oral administration of ampicillin was provided without immunosuppressive agents. Thereafter, his renal disease, endocarditis, and pyogenic spondylitis improved. He was discharged from our center on the 73rd hospital day. He has since received regular outpatient treatment in our department. At 7 months after discharge, his serum creatinine level had decreased to 1.43 mg/dl, his proteinuria had decreased to 0.15 g/g Cr, and his hematuria had decreased to 1.1 red blood cells per high-power field. His PR3-ANCA level had decreased to within the normal range (Fig. 2).

**Discussion and conclusions**

We report a case of rapidly progressive PR3-ANCA-positive necrotizing crescentic glomerulonephritis complicated by *Streptococcus* infective endocarditis. The patient’s renal disease improved with antibiotic monotherapy, which led to normalization of PR3-ANCA titer in accordance with improving infective endocarditis.

Renal disease associated with infective endocarditis shows various pathological changes including crescent formation, fibrinoid necrosis, mesangial cell proliferation,

Fig. 1 Renal biopsy findings. a Glomerulus with partial fibrinoid necrosis with fragmentation of glomerular tufts (arrows) (periodic acid-methenamine silver stain; original magnification, 400×). b Glomerulus with cellular crescentic formation (arrows) (periodic acid-Schiff stain; magnification, original magnification, 400×). c Tubulointerstitium with sporadic neutrophil infiltration in the peritubular capillary (arrows) and atrophy (broken line) (periodic acid-Schiff stain; original magnification, 100×)
and endothelial cell thickening in the glomerulus and tubulointerstitial damage with infiltration of immune cells [3–7]. PR3-ANCA has been reported to be positive in 5–10% of cases of renal disease complicated with infective endocarditis [1]. It is considered that PR3-ANCA may be produced as a result of an immune response against infection by sharing epitopes with cytoplasmic antigens of neutrophils in cases of infective endocarditis [8]. The produced PR3-ANCA is then speculated to contribute to fibrinoid necrosis, crescent formation, and granulomas in the kidney [9]. However, the lack of sufficient histological findings of PR3-ANCA-positive renal diseases complicated by infective endocarditis prevents clarification of detailed pathological changes in the kidney. Although many cases of PR3-ANCA-positive renal disease complicated by infective endocarditis have been reported, including crescentic glomerulonephritis, endocapillary proliferative glomerulonephritis, mesangial proliferative glomerulonephritis, and focal segmental glomerulosclerosis, only three cases showed necrotizing crescentic glomerulonephritis complicated by infective endocarditis [10–34] (Table 2). Regarding treatment for PR3-ANCA-positive renal disease complicated by infective endocarditis, previous studies suggested antibiotic monotherapy for patients with low PR3-ANCA titers (< 25 IU/ml) and combination therapy with immunosuppressive agents, including steroids for patients with high PR3-ANCA titers (> 50 IU/ml), when the patients’ condition does not improve with antibiotic monotherapy [22, 35]. The three previous cases of PR3-ANCA-positive necrotizing crescentic glomerulonephritis showed various PR3-ANCA titers (2.96, > 8.0, and 85 IU/ml) and were treated with immunosuppressive agents such as corticosteroids in addition to antibiotics. Among those three cases, the renal disease resolved completely in two patients but progressed to end-stage renal disease in the other (Table 2). The other types of PR3-ANCA-positive renal disease complicated by infective endocarditis also showed various PR3-ANCA titers (3–359 IU/ml) and were treated with antibiotics with or without immunosuppressive agents (Table 2). Regarding treatment outcomes, most renal diseases recovered, except for one patient with crescentic glomerulonephritis with high PR3-
| Age (years)/sex | Renal biopsy histology (IF/EM) | PR3-ANCA (IU/mL) | Microbe detected | Past medical history | Treatments | Outcome | Reference |
|-----------------|-------------------------------|------------------|-----------------|---------------------|------------|---------|-----------|
| 54/M 59/M 67/M 6/M 12/F 14/M 18/F 24/M 26/F 36/M 43/M 46/M 47/M 50/M 54/M 55/M 67/M | Focal necrotizing crescent GN (negative/no deposits) Focal necrotizing crescent GN (negative/no deposits) Focal necrotizing crescent GN (IgA⁺, IgM⁺, IgG⁺, C3⁺, C1q+/mesangial and subendothelial dense deposits) Crescentic GN (no mention) Crescentic GN (C3⁺/subendothelial dense deposits) Crescentic GN (no mention) Crescentic GN (IgG[1+], IgM[3+], C3[2+], C1q[2+] /subendothelial dense deposits) Crescentic GN (C3⁺/mesangial and subendothelial dense deposits) Crescentic GN (IgM⁺, C3⁺/no mention) Crescentic GN (negative/no mention) Crescentic GN (negative/no mention) Crescentic GN (C3⁺ C1q⁺/no mention) Crescentic GN (IgM⁺, C3⁺, C1q⁺/mesangial and subendothelial dense deposits) Crescentic GN (negative/not performed) Crescentic GN (IgM⁺, C3⁺/no deposits) Crescentic GN (C3[2+], IgA⁺/no deposits) Crescentic GN (IgM⁺, C3⁺, C1q⁺/no mention) | 2.96 > 8.0 85 14 160 359 14 25 160 247 3 8.0 41 | Streptococcus mutans Enterococcus faecalis Gemella sanguinis Bartonella henselae CHD VSD ASD, recent dental Tx Bartonella henselae Bartonella quintana Bartonella henselae, Bartonella quintana | No mention No mention Bartonella henselae CHD Chronic HCM, depression VSD No mention Infective endocarditis (blood culture was negative) No mention Toxoplasmosis, depression Thoracic aortic aneurysm repair | Piperacillin, tazobactam, cyclophosphamide, corticosteroids Pulse methylprednisolone ⇒ prednisolone Ceftriaxone, gentamicin, methylprednisolone Doxycycline, rifampicin Prednisolone, intravenous cyclophosphamide ⇒ azathioprine, MMF, prednisolone, CV surgery Antibiotic therapy, methylprednisolone Doxycycline, rifampicin Amoxicillin, gentamicin, penicillin Amoxicillin, gentamicin, penicillin CV surgery | Complete recovery Hemodialysis Temporary hemodialysis ⇒ complete recovery Complete recovery Complete recovery Complete recovery Complete recovery Temporary hemodialysis ⇒ complete recovery Death Recovery Recovery Recovery Recovery Recovery Recovery Recovery | [10] [11] [12] [13] [14] [15] [16] [17] [18] [19] [20] [21] [22] [23] [24] [25] |
ANCA titers (143 IU/ml), both of whom died (Table 2). In our patient, necrotizing crescentic glomerulonephritis improved with antibiotic monotherapy, and PR3-ANCA titer normalized in accordance with improving infective endocarditis; however, PR3-ANCA titer was highly elevated at 163 IU/ml. The results of our patient’s case suggest that antibiotic monotherapy can be effective even if the PR3-ANCA titer is considerably high in PR3-ANCA-positive necrotizing crescentic glomerulonephritis complicated by infective endocarditis. However, caution is needed with the use of immunosuppressive agents because they may exacerbate bacteremia and infective endocarditis. Furthermore, a greater accumulation of cases with histological evidence is needed to investigate optimal treatments for PR3-ANCA-positive renal disease complicated with infective endocarditis.

In conclusion, we describe a case of a patient with PR3-ANCA-positive necrotizing crescentic glomerulonephritis complicated by infective endocarditis. His renal disease was improved with antibiotic agents, and his PR3-ANCA titer normalized in accordance with improving infective endocarditis.

**Abbreviations**

ABPC: Ampicillin; ALT: Alanine aminotransferase; ASD: Atrial septal defect; ASO: Antistreptolysin O; AST: Aspartate aminotransferase; AVR: Aortic valve replacement; β2-MG: β2-Microglobulin; BUN: Blood urea nitrogen; C3: Complement component 3; C4: Complement component 4; CEZ: Cefazolin; CH50: 50% Homolytic unit of complement; CHD: Chronic heart disease; Cr: Creatinine; CRP: C-reactive protein; CV: Cardiovascular; DM: Diabetes mellitus; eGFR: Estimated glomerular filtration rate; EM: Electron microscopy; FSGS: Focal segmental glomerulosclerosis; IF: Immunofluorescence; IHD: Ischemic heart disease; Ig: Immunoglobulin; LM: Light microscopy; M: Male; MMF: Mycophenolate mofetil; MVP: Mitral valve prolapse; PR3-ANCA: Proteinase 3 antineutrophil cytoplasmic antibody; SSS: Sick sinus syndrome; Tx: Treatment; VSD: Ventricular septal defect

**Table 2** Case reports of PR3-ANCA-positive renal injury complicated by infective endocarditis (Continued)

| Age (years)/sex | Renal biopsy histology (IF/EM) | PR3-ANCA (IU/mL) | Microbe detected | Past medical history | Treatments | Outcome | Reference |
|-----------------|-------------------------------|-----------------|------------------|----------------------|------------|---------|-----------|
| 72/F            | Crescentic GN (no mention)    | Positive        | Aggregatibacter| No mention           | Vancomycin, ceftriaxone | Temporary hemodialysis ⇒ recovery | [26]       |
| 42/M            | Diffuse endocapillary          | 21.3            | Staphylococcus aureus | Nothing | Cefazolin | Recovery | [27]       |
| 68/M            | Proliferative GN and crescentic GN (IgG[2+] | 102 | Negative | Schistosomiasis | Cefoperazone, tazobactam | Complete recovery | [28]       |
| 78/F            | Endocapillary proliferative GN (Ig+, C3+, C1q+/no mention) | 30 | Bartonella henselae | Hypertension | Doxycycline | Complete recovery | [29]       |
| 48/M            | Mesangial proliferative GN (C3+/no mention) | 12 | Negative | AlcOH, DM | Amoxicillin, gentamicin | Complete recovery | [20]       |
| 57/M            | Mesangial proliferative GN (IgG[2+], IgM, C3+/no mention) | 45 | Negative | Nothing | Corticosteroids ⇒ ampicillin, ceftriaxone, gentamicin, vancomycin | Recovery | [30]       |
| 74/M            | Mesangial proliferative GN (IgG[2+], C1q+/not performed) | > 100 | Bartonella henselae, Bartonella quintana | IHD, pacemaker, DM, pulmonary embolus | Antibiotic therapy | Recovery | [31]       |
| 78/M            | Mesangial proliferative GN (IgM, C3+, IgA, C1q+/subendothelial dense deposits) | 143 | Enterococcus faecalis | Coronary artery bypass surgery | Antibiotic therapy | Death | [32]       |
| 57/M            | FSGS (IgM, C3+/paramesangial dense deposits) | 40 | Negative | DM, AVR, aortic aneurysm | Pulse methylprednisolone ⇒ vancomycin, gentamicin, rifampicin | Plasmapheresis ⇒ recovery | [33]       |
| 64/M            | FSGS and mild interstitial infiltration (no mention) | 60 | Negative | Insidious mild renal dysfunction | Ceftriaxone, doxycycline | Complete recovery | [34]       |

**Abbreviations:** ASD atrial septal defect, AVR aortic valve replacement, CHD chronic heart disease, C3 complement component 3, CV cardiovascular, DM diabetes mellitus, EM electron microscopy, F female, FSGS focal segmental glomerulosclerosis, GN glomerular nephritis, IF immunofluorescence, IHD ischemic heart disease, Ig immunoglobulin, LM light microscopy, M male, MMF mycophenolate mofetil, MVP mitral valve prolapse, PR3-ANCA proteinase 3 antineutrophil cytoplasmic antibody, SSS sick sinus syndrome, Tx treatment, VSD ventricular septal defect
microscopy; ESR: Erythrocyte sedimentation rate; F: Female; FSGS: Focal segmental glomerulosclerosis; GBM: Glomerular basement membrane antibody; GN: Glomerulonephritis; HbA1c: Hemoglobin A1c; HPP: High-power field; IF: Immunofluorescence; Ig: Immunoglobulin; ID: Ischemic heart disease; LM: Light microscopy; M: Male; MIF: Mycoplasma fermentans; MPO-ANCA: Myeloperoxidase antineutrophil cytoplasmic antibody; MVP: Mitral valve prolapse; PCG: Penicillin G; PR3-ANCA: Proteinase 3-antineutrophil cytoplasmic antibody; RBC: Red blood cells; RNP: Ribonucleoprotein; Sm: Smith; SSS: Sick sinus syndrome; Tx: Treatment; VSD: Ventricular septal defect; WBC: White blood cells

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Authors’ contributions
KY and YK wrote the manuscript. KH and YM supervised the study. YU, MH, and YM undertook histological analysis. All authors participated in patient care. All authors read and approved the final manuscript.

Ethics approval and consent to participate
Approval of the institutional ethics committee was not required, because this is a case report without any experimental trial.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare that they have no competing interests.

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