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Bartonella Endocarditis and Pauci-Immune Glomerulonephritis
A Case Report and Review of the Literature

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Abstract: Among culture-negative endocarditis in the United States, *Bartonella* species are the most common cause, with *Bartonella henselae* and *Bartonella quintana* comprising the majority of cases. Kidney manifestations, particularly glomerulonephritis, are common sequelae of infectious endocarditis, with nearly half of all *Bartonella* patients demonstrating renal involvement. Although a pauci-immune pattern is a frequent finding in infectious endocarditis–associated glomerulonephritis, it is rarely reported in *Bartonella* endocarditis. Anti–neutrophil cytoplasmic antibody (ANCA) positivity can be seen with many pathogens causing endocarditis and has been previously reported with *Bartonella* species. In addition, ANCA-associated vasculitis can also present with renal and cardiac involvement, including noninfectious valvular vegetations and pauci-immune glomerulonephritis. Given the overlap in their clinical presentation, it is difficult to differentiate between *Bartonella* endocarditis and ANCA-associated vasculitis but imperative to do so to guide management decisions. We present a case of ANCA-positive *Bartonella* endocarditis with associated pauci-immune glomerulonephritis that was successfully treated with medical management alone.

Key Words: *Bartonella* endocarditis, endocarditis-associated glomerulonephritis, culture-negative endocarditis, pauci-immune glomerulonephritis

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Culture-negative endocarditis comprises 8.1% of cases of all infectious endocarditis.[1] *Bartonella* is the most common cause of culture-negative endocarditis in the United States.[2] Despite advanced diagnostic testing, culture-negative endocarditis remains a diagnostic challenge because it is associated with a variety of systemic manifestations.

Kidney disease is a common manifestation of infectious endocarditis, with nearly 40% to 50% of patients demonstrating parenchymal infarction, hematuria, or glomerulonephritis, with glomerulonephritis being the most common.[3] One study found that 45% of patients with *Bartonella* endocarditis have kidney failure.[2] Endocarditis-associated glomerulonephritis can show significant variability in histopathologic appearance including the more well-known immune complex–mediated glomerulonephritis but pauci-immune glomerulonephritis may also be seen.[4] Because of this variability, a patient's renal disease can be misdiagnosed as a vasculitis rather than infectious endocarditis–related glomerulonephritis.

Further contributing to this diagnostic challenge is that noninfectious endocardial involvement is a known part of the spectrum of manifestations of anti–neutrophil cytoplasmic antibody (ANCA)–associated vasculitis, occurring in 6% of cases.[5] Specifically, valvular involvement can be seen in ANCA-associated vasculitis, such as granulomatosis with polyangiitis (GPA), microscopic polyangiitis, and Churg-Strauss syndrome. Diagnosis of GPA relies heavily on positive serum ANCA.[6] Although a positive ANCA is thought to strongly correlate to certain vasculitic diseases, ANCA positivity can be seen in a variety of infectious diseases as well, including bacterial endocarditis, invasive amebiasis, Legionnaire disease, leptospirosis, invasive aspergillosis, and human immunodeficiency virus (HIV) infection.[7,8] There are many reports with *Bartonella* endocarditis being associated with positive ANCA and glomerulonephritis.[3,5,9–11]

There remains significant overlap between ANCA-positive culture-negative endocarditis with associated pauci-immune glomerulonephritis and glomerulonephritis from ANCA-associated vasculitis with associated endocardial involvement. Differentiating these 2 diseases can be difficult but is crucial because treatment of an active infection with immunosuppressive agents can be life threatening. Here, we report the second case of c-ANCA–positive *Bartonella* endocarditis with pauci-immune glomerulonephritis.

CASE REPORT

A 55-year-old African American man with a history of alcohol abuse and homelessness presented to the hospital with a 1-week history of lower-extremity swelling and dyspnea on exertion. He also complained of fatigue, lumbar back pain, and a 10-lb unintentional weight loss during the past month. He denied fevers, chills, and night sweats. The patient's medical history was notable only for depression with a prior hospitalization for a suicide attempt and heavy alcohol use. Although homeless, he occasionally lived with his sister and her cat. He denied recent travel but reported having a louse infection a few months before presentation.

On physical examination on admission, the patient's temperature was 38.8°C, blood pressure was 169/82 mm Hg, pulse rate was 81 beats/min, and respiratory rate was 18 breaths/min, with an oxygen saturation of 98% on room air. He was in no acute distress. He had poor dentition, jugular venous distension, a III/VI systolic murmur over the apex radiating into the axilla, as well as a faint diastolic murmur heard at the left upper sternal border. He had crackles at bilateral lung bases and had pitting edema of the bilateral lower extremities. No skin rashes were identified.

The basic laboratory data revealed a leukocyte count of $4.0 \times 10^3/mm^3$ with a normal differential, hemoglobin of 8.6 g/dL, and platelet count of 121×10^3 μL. Serum urea nitrogen was 42 mg/dL, and creatinine was 5.51 mg/dL, elevated from 0.71 mg/dL 6 months prior. Serum albumin was low at 1.9 g/dL, but all other

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liver function tests were normal. A serum B-type natriuretic peptide was markedly elevated to 11,474 pg/mL. The urinalysis showed proteinuria (protein excretion, 100 mg/dL), hematuria (>50 red blood cells/high-power field), and the presence of white blood cells (26 white blood cells/high-power field). Erythrocyte sedimentation rate and C-reactive protein were elevated to 141 mm/h and 81.4 mg/L, respectively. He tested negative for syphilis with a negative rapid plasma reagin, and his rapid HIV test was negative. A chest radiograph showed diffuse, coarse, interstitial markings bilaterally consistent with pulmonary edema.

As part of the workup for new-onset renal failure, he had a 24-hour urine protein collection showing nephrotic-range proteinuria with 3.564 g/24 h. A renal ultrasound revealed bilateral enlarged kidneys but no hydronephrosis. Serum protein electrophoresis and urine protein electrophoresis were both normal. The rheumatoid factor was elevated to 213 IU/mL. Anti–nuclear antibody and complement 4 level were normal. The serum complement 3 level was reduced at 27 mg/dL (reference range, 90-180). In addition, he had a positive c-ANCA at 1:1024; p-ANCA was negative. A repeat c-ANCA obtained 4 days later was positive at 1:128. Proteinase 3 antibody (PR-3) was more than 8.0 U with a myeloperoxidase antibody of less than 0.2 U.

The patient's renal function continued to worsen with a creatinine rising to 5.9 mg/dL (estimated glomerular filtration rate of 12 mL/min per 1.73 m^2). Further investigation of acute renal failure included a renal biopsy. Renal biopsy (Fig. 1A) under light microscopy showed focal proliferative glomerulonephritis with rare active crescent and no necrotizing lesion. Immunofluorescence revealed 2+ staining for C3, 1+ staining for immunoglobulin A (IgA), and trace staining for IgG, IgM, and C1q—most consistent with a pauci-immune pattern. On electron microscopy (Fig. 1B, C), no electron-dense deposition was seen. Overall, the pathologist concluded that this was consistent with ANCA-associated glomerulonephritis.

Simultaneously, an echocardiogram demonstrated a moderately dilated left ventricle with normal systolic function, severe aortic regurgitation, and a long mobile echodensity attached to the ventricular side of the aortic leaflets consistent with a 1.7 × 0.3 cm vegetation. In addition, there was mild to moderate thickening of the mitral leaflets with a small, mobile, echogenic mass attached to the anterior leaflets, consistent with a vegetation. Blood cultures obtained on admission when the patient was febrile were without growth at 28 days. Given concern for infectious endocarditis, broad-spectrum antibiotics were initiated with intravenous vancomycin and cefepime. Three sets of aerobic and anaerobic blood cultures were obtained before initiation of antibiotics, and all remained negative. Of note, the patient was febrile during only the first 24 hours of admission and defervesced after initiation of antibiotics, by day 2 of hospitalization.

Workup for culture-negative endocarditis included negative serum serologies (IgG, IgM) for *Brucella* and *Coxiella burnetti* and a negative *C. burnetti* serum polymerase chain reaction (PCR). Serum *Bartonella* PCR was also negative. Serologic testing revealed significantly high serum IgG titers of more than 1:1024 (expected value, <1:128) against both *B. henselae* and *B. quintana* and *B. quintana* IgM titers of more than 1:20; *B. henselae* IgM titers were less than 1:20. Thus, the antibiotic regimen was changed to gentamicin and doxycyline for treatment of *Bartonella* endocarditis. Gentamicin was discontinued after a baseline audiogram revealed moderate to severe sensorineural hearing loss and in light of his continued renal failure. Oral rifampin was added to doxycyline. In addition, cardiac valve surgery was recommended as a means of treating his infection and repairing his severe aortic regurgitation. However, the patient repeatedly refused surgery.

After 2 weeks of antibiotic therapy with doxycyline and rifampin, his creatinine remained unchanged. He then received 2 days of pulse methylprednisolone. His creatinine minimally improved after pulse steroids to 4.81 mg/dL. He refused further care in the hospital and was discharged on doxycycline, rifampin, and oral prednisone (60 mg/d).

He followed up in the clinic 10 days after discharge and admitting to stopping his medications 2 days before his follow-up appointment. He was instructed to resume his medication, and his prednisone was titrated to 40 mg daily. At that time, his creatinine was 3.5 mg/dL. *Bartonella henselae* and *B. quintana* IgM titers were less than 1:20, *B. henselae* IgG titers were more than 1:2056, and *B. quintana* IgG titers were more than 1:1024.

He had continued noncompliance with his medications, and 9 days later, he presented to a local hospital with shortness of breath secondary to worsening congestive heart failure. Repeat echocardiogram demonstrated a decreased systolic function (ejection fraction

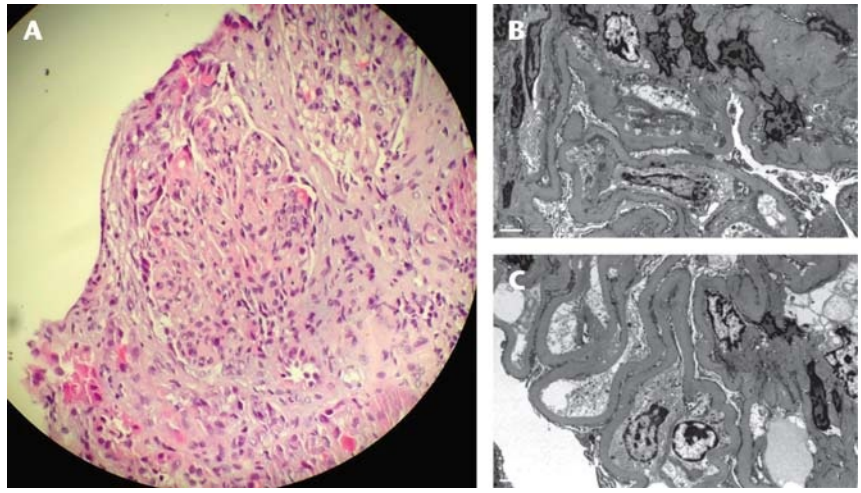

FIGURE 1. Histologic examination of renal biopsy. A, Light microscopy with hematoxylin and eosin stain showing focal proliferative glomerulonephritis with increased cellularity and small subepithelial crescent and no necrotizing lesion. Also noted moderate chronic tubulointerstitial disease with mild tubular atrophy and mixed interstitial infiltrate. B, Electron microscopy showing glomeruli with diffuse and nodular mesangial matrix expansion with occasional densities. C, Electron microscopy showing no subepithelial or subendothelial immune complexes and with visceral epithelium with diffuse foot process effusion.

of 35%–40%). The aortic and mitral vegetations seen on previous echo had resolved, although he had continued moderate aortic regurgitation and mild mitral regurgitation. He was restarted on rifampin and doxycycline. Oral prednisone was tapered to 15 mg/d.

Two weeks after discharge, he followed up in the clinic and reported compliance with his medications, although he had mistakenly only been taking 5 mg of prednisone daily. Symptomatically, he felt well. After 3 months of therapy, his repeat creatinine was 1.09 mg/dL and his *Bartonella* titers had started to decline, with IgG *B. henselae at* 1:1280 and *B. quintana* at 1:1280. His prednisone was tapered off, and he was continued on doxycycline and rifampin for a 6-month course.

DISCUSSION

Bartonella species are an important cause of culture-negative endocarditis. The first definitive case was published in 1993,[12] and since then, multiple case series have been reported in the literature.[13–17] In 1 study, 28% of 348 cases of culture-negative endocarditis were caused by *Bartonella* species.[2] *Bartonella* species have been reported to cause 3% of all infectious endocarditis.[14] Ninety-five percent of *Bartonella* endocarditis is caused by either *B. quintana* or *B. henselae*.[2,14] However, other *Bartonella* species including *B. koehlerae*,[18] *B. alsatica*,[19,20] *B. elizabethae*,[21] and *B. vinsonii*[22–24] have also been reported as causing culture-negative endocarditis in humans.[25] Epidemiology studies have identified exposure to cats and preexisting valvular disease as risk factors for development of *B. henselae* endocarditis; homelessness, alcohol abuse, and prior louse infections are known risk factors for *B. quintana* endocarditis.[17] Seventy to 84.2% of cases of *Bartonella* endocarditis occur in men[17]; perhaps this is because men are more likely to be alcohol abusers and homeless. Furthermore, *B. quintana* endocarditis is less likely to cause infection in patients with underlying valvular disease when compared with other types of endocarditis.[17] The characteristics of the patient described here suggest *B. quintana* as the causative agent.

The aortic valve is the most commonly affected valve with aortic valve endocarditis occurring in 75% of cases of *Bartonella* endocarditis.[17] However, bivalvular involvement with the aortic and mitral valves—as was seen in our patient—is well documented, occurring in 13% of patients in one 48-patient case series.[17] Most cases of *Bartonella* endocarditis involve native valves, but those that involve prosthetic valves are associated with aggressive disease marked by valvular perforation and rapid progression to heart failure.[26–28]

Diagnosis of *Bartonella* endocarditis can be difficult and relies on a combination of multiple diagnostic tools. Routine blood cultures are often negative and require at least 21 days of incubation. In 1 multicenter study, only 25% of patients with *Bartonella* endocarditis had positive blood cultures.[14] Tissue cultures also have a relatively low yield. As both blood and tissue cultures are unreliable, serology is the most widely used technique for diagnosis. It is important to note that cross reactivity among the *Bartonella* species occurs,[13,14] as was seen in this patient who had an elevated IgG titer for both *B. quintana* and *B. henselae*. Cross reactivity can also occur with other organisms known to cause culture-negative endocarditis, specifically with *Coxiella burnetti* and *Chlamydia* species.[13,14,29]

This patient's epidemiologic characteristics and serologies confirmed *B. quintana* as the causative pathogen. An IgM titer of 1:16 or greater is considered evidence for early or recent *Bartonella* infection.[30] An IgG titer of greater than or equal to 1:256 strongly suggests acute or active infection,[30] and a 4-fold rise in IgG titers from acute to convalescent specimen is considered definitive.[31] Enzyme-linked immunosorbent assay IgM and IgG have a specificity of 91%, sensitivity for IgM is 43% to 65%, and sensitivity for enzyme-linked immunosorbent assay IgG is 53%.[32] The combined sensitivity is 85%, and specificity is 98.2%.[33] A high serologic titer strongly supports the diagnosis of *Bartonella* endocarditis, particularly in the right clinical context, and has a high positive predictive value.[14] An immunofluorescence assay titer of greater than or equal to 1:800 for IgG antibodies to either *B. henselae* or *B. quintana* has a positive predictive value of 0.955 for the detection of bartonellosis among patients with endocarditis.[16] Patients with delayed decreases in antibody titers may be at risk for endocarditis relapse. When clinical suspicion is high, titers should be repeated after 10 to 14 days for comparison and serial serologic testing performed every 1 to 2 months to help guide antibiotic duration.[34]

Polymerase chain reaction has played an important role in the diagnosis of *Bartonella* endocarditis. Although the serum *Bartonella* PCR was negative in the patient presented here, its sensitivity is generally poor at only 58%.[9] A positive serum PCR strongly correlates to an active infection given its near 100% specificity. One study reported PCR results that were positive for *Bartonella* on cardiac valve tissue in more than 95% of patients with *Bartonella* endocarditis, despite the fact that more than 60% of these patients underwent valve analysis after antibiotic administration.[35]

Although definitive diagnosis with valve PCR is ideal, this is not always possible based on certain real-world limitations. Without definitive diagnosis, differentiating infectious endocarditis with associated glomerulonephritis from a vasculitis with cardiac involvement can be challenging. Cardiac involvement in GPA occurs in 6% to 44% of cases, with the most commonly encountered disorders being pericarditis and coronary arteritis.[36,37] Valvular involvement has been reported and usually manifests as aortic regurgitation or thickening, stenosis, prolapse, or regurgitation of the mitral valve.[38] One case series describing echocardiographic findings in patients with GPA found that the aortic valve thickening occurred in 8 of 9 patients, 7 of whom had aortic insufficiency. However, there were no discrete vegetations identified.[39] Although valvular involvement in patients with ANCA vasculitis has been reported, vegetations are rare.[40]

Echocardiographic findings are not definitive for distinguishing infectious versus autoimmune etiology for vegetations. Serum autoantibodies also are generally nondiagnostic because a positive RF, ANA, and ANCA may be seen in both vasculitis and infectious endocarditis. In particular, the presence of c-ANCA is generally thought to be highly specific for GPA.[41] However, c-ANCA directed against PR-3 positivity has also been well documented in infectious endocarditis[42–46] and more specifically in *Bartonella* endocarditis.[3,5,9–11,26,27,47] The presence of c-ANCA in infectious endocarditis is unclear: it may represent a false-positive or related to the infection or the production of c-ANCA may be induced through B-cell activation after release of PR-3 from neutrophils.[43,48]

The association between positive c-ANCA and infectious endocarditis is reported with enough frequency that it is generally regarded as prudent to rule out endocarditis in patients with a positive c-ANCA and suspected vasculitis before initiating treatment with immunosuppressives. There may be some benefit in monitoring ANCA titers while treating *Bartonella* endocarditis because the ANCA titer normalizes with resolution of infection.[42] Although interpretation of a positive c-ANCA may be difficult, it is likely that hypocomplementemia and the presence of at least one other autoantibody such as RF, ANA, or cryoglobulin are more suggestive of an ANCA-positive bacterial endocarditis rather than an ANCA vasculitis.[46] The patient presented here had hypocomplementemia, a positive RF, and positive c-ANCA titer.

Further confounding the diagnosis of c-ANCA vasculitis and infectious endocarditis is the presence of renal involvement. Although renal disease is a hallmark feature of c-ANCA vasculitis, it

TABLE 1. Characteristics of Patients With *Bartonella* Endocarditis and Biopsy-Proven Glomerulonephritis

Case	Age/Sex	Organism	Light Microscopy	Immunofluorescence	Electron Microscopy	ANCA	Immunosuppression	Treatment*	Outcome
Bookman et al, case 1	53 y/F	*B. henselae* by serology	Diffuse/segmental necrosis/crescents, No endocapillary hypercellularity	IgM/C3, strong capillary loop and mesangial; IgG, moderate capillary loop and mesangial	EDD, mesangial; subepithelial (occasional)	Negative	Intravenous methylprednisolone, oral prednisone	Doxycycline and ceftriaxone for 6 wk	Readmitted at 4 mo with renal failure. Deceased
Bookman et al, case 2	35 y/M	*B. henselae* by serology	Focal/segmental necrosis/crescents, No endocapillary hypercellularity	IgM/C3, strong capillary loop and & mesangial; IgG, moderate capillary loop and mesangial	EDD, mesangial; subendothelial	Negative	Oral prednisone	Doxycycline and tobramycin for 6 wk*	Discharged postoperatively with stable renal function
Bookman et al, case 3	46 y/M	*B. henselae* by serology	Focal/segmental necrosis/crescents, Mild endocapillary hypercellularity	IgM/C3, strong capillary loop and mesangial; IgG, moderate/segmental capillary loop and mesangial	EDD, mesangial; subendothelial	Negative	None	Ceftriaxone and azithromycin for 6 wk*	Discharged postoperatively with stable renal function
van Tooren et al, case 1	53 y/M	*Bartonella* species by serology	Diffuse proliferative glomerulonephritis, Focal crescents	Immunoglobulins/C3/C1q, diffuse granular capillary loop*	Not reported	Negative	None	Ceftazidime and oxfloxacine preop Doxycycline postop for 6 wk*	Improved renal function; *B. henselae* antibody titers normalized
Salvado et al	78 y/F	*B. henselae* by serology	Diffuse endocapillary hypercellularity and fibrinoid necrosis, Diffuse crescents	IgM/C3/C1q, strong capillary loop	Not reported	c-ANCA + anti-PR3 +	None	Doxycycline for 8 wk	Normal renal function; negative ANCA and PR3 at 2 mo
Sugiyama et al	64 y/M	*B. quintana* by serology	Focal glomerular sclerosis	Complement deposition	Not reported	c-ANCA +	None	Ceftriaxone and doxycycline for 6 wk*	Normal renal function, negative c-ANCA at 8 mo
Vikram et al	43 y/M	*B. henselae* by PCR	Focal segmental necrosis/crescents	Pauci-immune	Not reported	c-ANCA + anti-PR3 +	Oral prednisone, cyclophosphamide	Gentamicin, ceftriax one, and vancomycin for 6 wk, doxycycline for 1 y*	Stable condition 18 mo postoperatively
Turner et al	58 y/M	*B. henselae* by PCR	Focal crescents	IgA, mesangial immune complexes	Not reported	c-ANCA + anti-PR3 +	Oral prednisone, cyclophosphamide	Doxycycline and gentamicin for 2 wk followed by 5 wk doxycycline*	Normal renal function; negative c-ANCA and anti-PR3 10 mo postoperatively

(Continued on next page)

TABLE 1. *(Continued)*

Case	Age/Sex	Organism	Light Microscopy	Immunofluorescence	Electron Microscopy	ANCA	Immunosuppression	Treatment*	Outcome
Khalighi et al	18 y/F	*B. henselae* by serology and *Bartonella* species by PCR	Diffuse proliferative glomerulonephritis, Focal crescents	IgM/C3, strong capillary loop and mesangium; C1q/κ/λ, moderate capillary loop and mesangium; IgG, weak capillary loop and mesangium	EDD, mesangial; subendothelial	c-ANCA + anti-PR3 +	Intravenous methylprednisolone, oral prednisone	Doxycycline and rifampin for 15 wk	*Bartonella* PCR undetectable; Renal function stable† No cardiac valvular destruction observed at 3 mo
Present report	55 y/M	*B. quintana* by serology	Focal proliferative glomerulonephritis, Rare active crescent	Pauci-immune	No EDD present	c-ANCA +	Intravenous methylprednisolone, oral prednisone	Doxycycline and rifampin for 24 wk	Renal function normalized, vegetations resolved and *B. quintana* titers declining

*Treatment included antibiotics along with valvular surgery.
†Repeat kidney biopsy revealed decreased endocapillary hypercellularity, segmental distribution, and rare fibrocellular crescents.
ANCA, anti–neutrophil cytoplasmic antibody; EDD, electron-dense deposits; IgM, immunoglobulin M; IgG, immunoglobulin G; PCR, polymerase chain reaction; PR3, proteinase 3.

is also a well-described complication of infectious endocarditis. There are 3 pathologic processes found in the kidney in patients with infectious endocarditis including abscess formation, infarction from septic emboli, and glomerulonephritis from immune-mediated mechanisms.[4] Glomerulonephritis in infectious endocarditis can be both "focal" and "diffuse." The largest biopsy-based cohort series on infectious endocarditis–associated glomerulonephritis reports that crescentic glomerulonephritis predominates as the most common histologic pattern, occurring in 53% of patients studied, followed by diffuse proliferative glomerulonephritis in 33%. Furthermore, ANCA positivity was seen in 28% of patients in this cohort.[48] Glomerulonephritis occurs by 2 main immunologic mechanisms: immune complex–mediated and ANCA-associated or pauci-immune. The commonest type of glomerulonephritis is vasculitic, with minimal to absent staining for immunoglobulins by immunofluorescence in 63% to 69% of patients with endocarditis-associated glomerulonephritis.[4,49] Of note, 44% of patients from the previously mentioned cohort met the criteria for pauci-immune staining.[48]

More specifically, glomerulonephritis in *Bartonella* endocarditis has also been observed to have a variable appearance on renal biopsy. Of the previously reported 9 cases of *Bartonella* endocarditis with associated glomerulonephritis who underwent a renal biopsy (Table 1), 8 patients demonstrated moderate to strong capillary loop and mesangial deposition of immune complexes (IgG, IgM, IgA, C3, or C1q) seen by immunofluorescence.[3,9,10,26,47,50,51] One case demonstrated pauci-immune staining.[26] The case reported here is now the second reported case of *Bartonella* endocarditis associated with pauci-immune glomerulonephritis. Of these cases, 60% were found to be c-ANCA positive (6 of 10 patients) including both patients with pauci-immune vasculitis.[3,9,10,26,47]

Treatment of *Bartonella* endocarditis is equally as difficult as its diagnosis. Surgical intervention in *Bartonella* endocarditis is a mainstay of therapy and can dramatically reduce mortality. *Bartonella* endocarditis patients undergo valvular surgery at higher rates than patients infected with other pathogens. In 1 case series, more than 90% of patients with *Bartonella* endocarditis underwent valvular surgery.[17] Without surgical intervention, infectious endocarditis with congestive heart failure mortality can be as high as 51%.[52]

The optimal antibiotic strategy for *Bartonella* endocarditis is largely unknown and relies heavily on retrospective data. In vitro susceptibilities do not correlate well with in vivo susceptibilities for a number of antibiotics. Although the minimal inhibitory concentration of most antibiotics against *Bartonella* species is low, minimal inhibitory concentration levels should not be relied on for the selection of antibiotics. [53] In addition, only aminoglycosides are bactericidal.[15] Per current Infectious Diseases Society of America guidelines, the preferred regimen for confirmed cases of *Bartonella* endocarditis includes doxycycline for a total of 6 weeks with concomitant gentamicin for the initial 2 weeks of treatment.[54] In 1 retrospective study of 101 patients with *Bartonella* endocarditis, aminoglycoside administered for more than 14 days was associated with a higher likelihood of recovery.[15] However, given the association between *Bartonella* endocarditis and glomerulonephritis, use of nephrotoxic agents is cautioned.[3] Among such patients, rifampin along with doxycycline can be used instead of an aminoglycoside for at least 6 weeks.[15,53,55,56]

An optimal duration of therapy is not well defined. Prolonged therapy may be indicated among HIV-infected patients, patients with persistent bacteremia despite appropriate antibiotic therapy, and patients who did not undergo valvular surgery.[57] Monitoring serologies may be useful in defining antibiotic duration. A decline and stabilization in antibody titers suggest a lower risk of recurrence.[34] Continued stabilization of titers after discontinuation of therapy furthermore supports effective clearance of the infection. Relapsing disease is seen in both immunocompetent and immunocompromised

hosts, especially when therapy is prematurely discontinued.[58] Relapsing disease can be treated with chronic suppressive therapy of doxycycline or erythromycin.[58]

When treating bacterial endocarditis with secondary glomerulonephritis, treatment of the underlying infection usually leads to recovered renal function.[59] However, when antibiotic therapy fails to return serum creatinine to baseline, corticosteroids may play a role. Multiple cases of ANCA-positive infectious endocarditis with associated glomerulonephritis have noted renal injury refractory to antibiotic therapy alone. In these instances, corticosteroids have resulted in rapid improvement of renal function.[60–62] Therefore, corticosteroids may be considered when treating infectious endocarditis with associated glomerulonephritis when antibiotic therapy fails to improve serum creatinine. In the patient presented here, 2 weeks of antibiotic therapy was completed before initiation of steroids. After steroid therapy, his serum creatinine demonstrated moderate improvement. However, we believe that the prolonged antibiotic treatment was primarily responsible for the resolution of kidney function, especially given that he was maintained on only low doses of prednisone with variable compliance and GPA seldom responds well to steroids alone.

The case presented here features a unique and complex presentation of an uncommon disease. The finding of ANCA-positive glomerulonephritis in a patient with culture-negative endocarditis can be difficult to distinguish from ANCA vasculitis with associated cardiac involvement. However, this distinction is critical because treatment of active infection with immunosuppressives may have severe consequences. In this case, the patient's clinical history of homelessness, alcohol abuse, and recent louse infection along with echocardiographic evidence of bivalvular vegetations supported the diagnosis of *Bartonella* endocarditis, and serum serology confirmed the diagnosis. Although a vast majority of *Bartonella* endocarditis patients require surgical intervention, this patient achieved clearance of the valvular vegetations as well as resolution of his kidney function with medical management alone—another remarkable feature of this case. Most notably, the case presented here marks the second report of *Bartonella* endocarditis with associated pauci-immune glomerulonephritis, highlighting the need for a high clinical suspicion of *Bartonella* endocarditis in patients with ANCA positivity and concurrent cardiac and renal disease.

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