**Bartonella Endocarditis and Pauci-Immune Glomerulonephritis**

A Case Report and Review of the Literature

Jillian E. Raybould, MD,* Alison L. Raybould, MD,† Megan K. Morales, MD,* Misbah Zaheer, MD,‡ Michael S. Lipkowitz, MD,‡ Joseph G. Timpone, MD,* and Princy N. Kumar, MD*

### 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

(Infec Dis Clin Pract 2016;24: 254–260)

Culture-negative endocarditis comprises 8.1% of cases of all infectious endocarditis. *Bartonella* is the most common cause of culture-negative endocarditis in the United States. 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. One study found that 45% of patients with *Bartonella* endocarditis have kidney failure. 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. 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. 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. 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. There are many reports with *Bartonella* endocarditis being associated with positive ANCA and glomerulonephritis.

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 × 10^9/mm^3 with a normal differential, hemoglobin of 8.6 g/dL, and platelet count of 121 × 10^9/L. 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...
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 m²). 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 burnetii 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 doxycycline 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 doxycycline. 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 doxycycline 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, and since then, multiple case series have been reported in the literature. In 1 study, 28% of 348 cases of culture-negative endocarditis were caused by *Bartonella* species. Other *Bartonella* species have been reported to cause 3% of all infectious endocarditis. Ninety-five percent of *Bartonella* endocarditis is caused by either *B. quintana* or *B. henselae*. However, other *Bartonella* species including *B. koehlerae*, *B. alantica*, *B. elizabethae*, and *B. vinsonii* have also been reported as causing culture-negative endocarditis in humans. 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 house infections are known risk factors for *B. quintana* endocarditis. Seventy to 84.2% of cases of endocarditis had positive blood cultures. Tissue cultures also have a relatively low yield. As both blood and tissue cultures are unreliable, serum antibodies are useful to confirm the diagnosis of *Bartonella* endocarditis. In particular, the presence of pericarditis and coronary arteritis occurs in 6% to 44% of cases, with the most commonly encountered disorders being pericarditis and coronary arteritis. Valvular involvement has been reported and usually manifests as aortic regurgitation or thickening, stenosis, prolapse, or regurgitation of the mitral valve. 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. Although valvular involvement in patients with ANCA vasculitis has been reported, vegetations are rare.

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. However, c-ANCA directed against PR-3 positivity has also been well documented in infective endocarditis, and more specifically in *Bartonella* endocarditis. 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.

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. 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. 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
| 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 azthromycin 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 ofloxacin 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, ciclophosphamide | Gentamicin, ceftriaxone 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, ciclophosphamide | 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)
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. 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. 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. Of note, 44% of patients from the previously mentioned cohort met the criteria for pauci-immune staining.

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. 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 immunocompetent and immunocompromised patients with post-streptococcal glomerulonephritis.82

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.11 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. In addition, only aminoglycosides are bactericidal. 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. 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. However, given the association between Bartonella endocarditis and glomerulonephritis, use of nephrotoxic agents is cautioned. Among such patients, rifampin along with doxycycline can be used instead of aminoglycoside for at least 6 weeks.

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. Monitoring serologies may be useful in defining antibiotic duration. A decline and stabilization in antibody titers suggest a lower risk of recurrence. Continued stabilization of titers after discontinuation of therapy further supports effective clearance of the infection. Relapsing disease is seen in both immunocompetent and immunocompromised patients.
hosts, especially when therapy is prematurely discontinued. Relapsing disease can be treated with chronic suppressive therapy of doxycycline or erythromycin.

When treating bacterial endocarditis with secondary glomerulonephritis, treatment of the underlying infection usually leads to recovered renal function. 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. 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 associated with 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.

REFERENCES

1. Fowler VG, Miro JM, Hoen B, et al. Staphylococcus aureus endocarditis: a consequence of medical progress. JAMA. 2005;294(8):900.

2. Houptkan P, Raoult D. Blood culture–negative endocarditis in a reference center: etiologic diagnosis of 348 cases. Medicine (Baltimore). 2005;84(3):162–173.

3. Khalighi M, Nguyen S, Wideman J, et al. Bartonella endocarditis–associated glomerulonephritis: a case report and review of the literature. Am J Kidney Dis. 2014;63(6):1060–1065.

4. Majumdar A, Chowdhary S, Ferreira MA, et al. Renal pathological features of Bartonella endocarditis. Am J Kidney Dis. 2015;65(1):172–178.

5. Teoh LS, Hart H, Soh C, et al. Bartonella henselae aortic valve endocarditis mimicking systemic vasculitis. BMJ Case Rep. 2010 Oct 21, doi:10.1136/bcr.04.2010.2945, 2015 Sep 21.

6. Harper L, Savage CO. Pathogenesis of ANCA-associated systemic vasculitis. J Pathol. 2000;190:349–359.

7. Cunha BA. Diagnostic implications of ANCA-associated diseases. Infect Dis Clin Pract. 2002;10:158–159.

8. Mege JL, Escallier JC, Capo C, et al. Anti-neutrophil cytoplasmic antibodies (ANCA) and infection. Adv Exp Med Biol. 1993;336:353–356.

9. Sugiyama H, Sahara M, Imai Y, et al. Infective endocarditis by Bartonella quintana masquerading as antineutrophil cytoplasmic antibody-associated small vessel vasculitis. Cardiology. 2009;114:208–211.

10. Salvado C, McKinian A, Rouvier P, et al. Rapidly progressive crescentic glomerulonephritis and aneurism with antineutrophil cytoplasmic antibody: Bartonella henselae endocarditis. Presse Med. 2013;42(6 pt 1):1060–1061.

11. Satake K, Ohsawa I, Kobayashi N, et al. Three cases of PR3-ANCA–positive subacute endocarditis caused by attenuated bacteria (Propionibacterium, Gemella, and Bartonella) complicated with kidney injury. Mod Rheumatol. 2011;21:536–541.

12. Spack DH, Callis KP, Pauw DS, et al. Endocarditis caused by Rochalimaea quintana in a patient infected with human immunodeficiency virus. J Clin Microbiol. 1993;31:692–694.

13. Drancourt M, Mainardi JL, Bouquie P, et al. Bartonella (Rochalimaea) quintana endocarditis in three homeless men. N Engl J Med. 1995;332(7):419–423.

14. Raoult D, Fournier PE, Drancourt M, et al. Diagnosis of 22 new cases of Bartonella endocarditis. Ann Intern Med. 1996;125(8):646–652.

15. Raoult D, Fournier PE, Vandenesch F, et al. Outcome and treatment of Bartonella endocarditis. Arch Intern Med. 2003;163(2):226–230.

16. Fournier PE, Mainardi JL, Raoult D. Value of micrommunoﬂuorescence for diagnosis and follow-up of Bartonella endocarditis. Clin Diagn Lab Immunol. 2002;9:795–801.

17. Fournier PE, Lelièvre H, Eykyn SJ, et al. Epidemiologic and clinical characteristics of Bartonella quintana and Bartonella henselae endocarditis: a study of 48 patients. Medicine (Baltimore). 2001;80(4):245–251.

18. Avidor B, Graidy M, Efrat G, et al. Bartonella koehleae, a new cat-associated agent of culture-negative human endocarditis. J Clin Microbiol. 2004;42:3462–3568.

19. Raoult D, Roblot F, Rolain JM, et al. First isolation of Bartonella altstata from a valve of a patient with endocarditis. J Clin Microbiol. 2006;44:278–279.

20. Jeanlaude D, Godmer P, Leverdier D, et al. Bartonella altstata endocarditis in a French patient in close contact with rabbits. Clin Microbiol Infect. 2009;15:110–111.

21. Daly JS, Worthington MG, Brenner DJ, et al. Rochalimaea elizabethae sp isolated from a patient with endocarditis. J Clin Microbiol. 1993;31:872–881.

22. Fenolla F, Sire S, Raoult D. Bartonella vinsonii subsp arupensis as an agent of blood culture–negative endocarditis in a human. J Clin Microbiol. 2005;43:945–947.

23. Roux V, Eykyn SJ, Wylie S, et al. Bartonella vinsonii subsp berkoffii as an agent of afebrile blood culture–negative endocarditis in a human. J Clin Microbiol. 2000;38:1698–1700.

24. Oltarte L, Ampofo K, Thorrel EA, et al. Bartonella vinsonii endocarditis in an adolescent with congenital heart disease. Pediatr Infect Dis J. 2012;5:531–534.

25. del Valle Mendoza J, Silva Caso W, Tinco Valdez C, et al. Diagnosis of Carrión's disease by direct blood PCR in thin blood smear–negative samples. PLoS One. 2014;9:e92283.

26. Vikram HR, Bacani K, DeValeria PA, et al. Bivalvar Bartonella henselae prosthetic valve endocarditis. J Clin Microbiol. 2007;45(12):4081–4084.

27. Yamada Y, Ohkusu K, Yanagihara M, et al. Prosthetic valve endocarditis caused by Bartonella quintana in a patient during immunosuppressive therapies for collagen vascular diseases. Diagn Microbiol Infect Dis. 2011;70:395–398.
32. Vermeulen MJ, Herremans M, Verbakel H, et al. Serological testing for Bartonella henselae infections in The Netherlands: clinical evaluation of immunofluorescence assay and ELISA. Clin Microbiol Infect. 2007;13(6):627–634.

33. Giladi M, Kletter Y, Avidor B, et al. Enzyme immunoassay for the diagnosis of cat-scratch disease defined by polymerase chain reaction. Clin Infect Dis. 2001;33:1852–1858.

34. Lesprit P, Noel V, Chazouilleres P, et al. Cure of Bartonella endocarditis of a prosthetic aortic valve without surgery: value of serologic follow up. Clin Microbiol Infect. 2003;9:239–241.

35. Marin M, Munoz P, et al. Molecular diagnosis of infective endocarditis by real-time broad-range polymerase chain reaction (PCR) and sequencing directly from heart valve tissue. Medicine (Baltimore). 2007;86(4):195–202.

36. Hoffman GS, Kerr GS, Leavitt RY, et al. Wegener granulomatosis: an analysis of 158 patients. Ann Intern Med. 1992;116:488–498.

37. Pinching AJ, Lockwood CM, Pussell A, et al. Wegener's granulomatosis: observations on 18 patients with severe renal disease. QJM. 1983;208:435–60.

38. Korantzopoulos P, Papaioannides D, Siogas K. The heart in Wegener's granulomatosis. Cardiology. 2004;102:7–10.

39. Morelli S, Gargo Di Castelmenardo AM, Conti F, et al. Cardiac involvement in patients with Wegener's granulomatosis. Rheumatol Int. 2000;19:209–212.

40. Gerbracht DD, Savage RW, Scharff N. Reversible valvulitis in Wegener's granulomatosis. Chest. 1987;92:182–183.

41. Lane SK, Gravel JW. Clinical utility of common serum rheumatologic tests. Am Fam Physician. 2002;65(6):1073–1080.

42. Chiurro J, Corrales-Medina VF, Garcia S, et al. Endocarditis associated with antineutrophil cytoplasmic antibodies: a case report and review of the literature. Clin Rheumatol. 2007;26:590–595.

43. Choi HK, Lamprecht P, Niles JL, et al. Subacute bacterial endocarditis with positive cytoplasmic antineutrophil cytoplasmic antibodies and anti-proteinase 3 antibodies. Arthritis Rheum. 2000;43(1):226–231.

44. 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(1):15–18.

45. Soto A, Jorgensen C, Oksman F, et al. Endocarditis associated with ANCA. Clin Exp Rheumatol. 1994;12(2):203–204.

46. Fukasawa H, Hayashi M, Kinoshita N, et al. Rapidly progressive glomerulonephritis associated with PR3-ANCA positive subacute bacterial endocarditis. Intern Med. 2012;51:2587–2590.

47. Turner JW, Pien BC, Ardoin SA, et al. A man with chest pain and glomerulonephritis. Lancet. 2005;365(9476):2062.

48. Boils CL, Naar SH, Walker PD, et al. Update on endocarditis-associated glomerulonephritis. Kidney Int. 2015;87:1241–1249.

49. Boils CL, Naar SH, Walker PD, et al. Infective endocarditis-associated glomerulonephritis: a report of 37 cases [Abstract]. Mod Pathol. 2012;25:396A.

50. Bookman I, Scholey J, Jassal S, et al. Necrotizing glomerulonephritis caused by Bartonella henselae endocarditis. Am J Kidney Dis. 2004;43(2):c25–c30.

51. Van Torren RM, van Leusen R, Bosch FH. Culture negative endocarditis combined with glomerulonephritis caused by Bartonella species in two immunocompetent adults. Neth J Med. 2001;59(5):218–224.

52. Sexton DJ, Spelman D. Current best practices and guidelines: assessment and management of complications in infective endocarditis. Cardiol Clin. 2003;21:273–282.

53. Rolain JM, Broqueni P, Koehler JE, et al. Recommendations for treatment of human infections caused by Bartonella species. Antimicrob Agents Chemother. 2004;48:1921–1933.

54. Baddour LM, Wilson WR, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications. Circulation. 2005;112:2373.

55. Foucault C, Raoul D, Broqueni P. Randomized open trial of gentamicin and doxycycline for eradication of Bartonella quintana from blood in patients with chronic bacteremia. Antimicrob Agents Chemother. 2002;47:2204–2207.

56. Lucey D, Dolan MJ, Moss CW, et al. Relapsing illness due to Rochalimaea henselae in normal hosts: implication for therapy. Clin Infect Dis. 1992;14:683–688.

57. Wong MT, Dolan MJ, Lattuada CP, et al. Neutrophetritis, aseptic meningitis, and lymphadenitis associated with Bartonella (Rochalimaea) henselae infection in immunocompetent patients and patients infected with human immunodeficiency virus type 1. Clin Infect Dis. 1995;21:352–360.

58. Gandhi T, Slater L, Welch D, et al. Bartonella including cat-scratch disease. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 2015;2(236):2649–2663.

59. Neugarten J, Baldwin DS. Glomerulonephritis in bacterial endocarditis. Am J Med. 1984;77:297–304.

60. Ghosh G, Sharma B, Katageri B, et al. ANCA positivity in a patient with infective endocarditis-associated glomerulonephritis: a diagnostic dilemma. Yale J Biol Med. 2014;87:373–377.

61. Koya D, Shibuya K, Kikkawa R, et al. Successful recovery of infective endocarditis with lymph node enlargement. Jpn J Infect Dis. 2007;60(3):1080.

62. Le Moing V, Lacassine F, Delahousse M, et al. Use of corticosteroids in endocarditis related to endocarditis: three cases and review. Clin Infect Dis. 1999;28(5):1057–1061.