Case report

A case of *Bartonella henselae* native valve endocarditis presenting with crescentic glomerulonephritis

Roshni Patel *, Kansas Koran, Mark Call, Amanda Schnee

Prisma Health Infectious Disease Specialists, 890 West Faris Road, Suite 520, Greenville, SC 29605, USA

**ARTICLE INFO**

**ABSTRACT**

*Bartonella* endocarditis is often an elusive diagnosis, usually derived from evaluating multiple laboratory tests and assessment of presenting symptoms. Herein we describe a case of *Bartonella henselae* native mitral valve endocarditis with an initial presentation of volume overload and renal failure. The *Bartonella* organism is tedious to isolate from culture medium, causing most diagnoses to be delayed. Due to the destructive nature of *B. henselae* endocarditis, the need for rapid identification remains prudent. This therefore creates an opportunity for Next Generation Sequencing (NGS) to be used. We further summarize the varied presentations that may be associated with *B. henselae* endocarditis, and hope that this will heighten the clinicians' awareness of this entity when presented with acute onset renal failure and culture negative vegetations.

© 2021 The Authors. Published by Elsevier Ltd.

**A R T I C L E  I N F O**

Article history:
Received 8 June 2021
Received in revised form 29 November 2021
Accepted 15 December 2021
Available online xxxx

**Keywords:**
*Bartonella*
Culture negative endocarditis
Next Generation Sequencing
Crescentic glomerulonephritis

**Introduction**

From the flea’s stomach to the human heart, *Bartonella henselae* remains elusive in its abilities and characteristics, rising in the ranks as one of the most common causes of culture negative endocarditis. They are fastidious, facultatively intracellular and pleomorphic gram-negative bacilli with the ability to evade culture detection and produce biofilms. The two species that predominantly stand out for causing culture negative endocarditis are *B. henselae* and *B. quintana*. *Bartonella*’s slow growing behavior and requirement for heme create a difficult environment for culture detection. Herein, we will describe a case of *Bartonella* endocarditis complicated by crescentic glomerulonephritis. We will review the pathogenesis of these infections, alternate presentations, diagnostic modalities and the currently available therapies.

**Case report**

65-year-old Caucasian male with a medical history significant for bicuspid aortic valve, childhood aortic valve procedure (patient was unable to clarify, but no prosthetic material) and first-degree heart block, who presented with progressive shortness of breath over a two month period. He had acute worsening two to three days prior to admission with a twenty-minute episode of chest pain day of admission. He recorded a 100.1°F temperature at home with chills. Other past medical history was notable for hyperlipidemia, prior stroke with residual right-sided deficit, 48-pack-year tobacco history, and a social history significant for both indoor and outdoor pets consisting of cats, dogs and birds. Vital signs at admission temperature 100.1°F, blood pressure 136/67, pulse 87, and oxygen saturation 99% on room air. Physical exam with bibasilar crackles with faint expiratory wheezing. A II/VI systolic crescendo–decrescendo murmur with radiation to the carotids was noted on auscultation with minimal lower extremity edema. Labs were remarkable for a white blood cell count of 4600 /μL, hemoglobin 9 g/dL, platelets 117,000 /μL, creatinine 1.95 mg/dL, normal liver function tests. SARS-CoV-2 RNA and influenza screens were negative. He was started on empiric pneumonia treatment with vancomycin, ceftriaxone and azithromycin. On hospital day 2, a transthoracic echocardiogram revealed mild to moderate mitral regurgitation with a mobile dense mass adherent to the anterior leaflet concerning for a vegetation. The aortic valve was not visualized well. One set of blood cultures was collected prior to antimicrobial initiation, eventually returning negative. That evening he developed acute tachycardia, hypotension and hypoxia. CT of the chest was obtained revealing peripheral ground-glass infiltrates with “crazy paving.” He underwent a bronchoscopy with negative studies. Infectious disease was consulted on hospital day 3 for multifocal pneumonia as well as mitral valve findings. Blood cultures were obtained and were negative. Plasma Next Generation

---

*Corresponding author.
E-mail addresses: Roshni.patel2@prismahealth.org (R. Patel), Kansas.koran2@prismahealth.org (K. Koran), mark.call@prismahealth.org (M. Call), Amanda.schnee@prismahealth.org (A. Schnee).
Sequencing testing available on day 8 with 9653 DNA molecules per microliter (MPM) of *Bartonella henselae* detected. Given concern for *Bartonella* endocarditis, on hospital day 8 antimicrobials were changed to doxycycline and rifampin. *Bartonella quintana* and *Bartonella henselae* IgG testing positive with reflex titers of 1:512 and > / = 1:1024, respectively, and Brucella IgG positive at 3.02, all three available on day 9 (Table 1). Nephrology was consulted on day 5 secondary to progression of renal failure with creatinine rising to 2.13 mg/dL (prior normal creatinine). Given his pulmonary symptoms, autoimmune testing was completed on day 5 for anti-GMA, ANCA, and ANA negative, C3 and C4 borderline low at 80 mg/dL and 17 mg/dL, respectively. On day 9, renal biopsy was performed which showed focal necrotizing and crescentic glomerulonephritis. His renal function progressively worsened, but he remained non-oliguric. Renal dosed gentamicin was added to his therapy in place of rifampin on hospital day 17 after discussions with nephrology. That same day, he underwent a mitral valve replacement with intraoperative TEE noting stenotic mitral valve with central regurgitation, anterior and posterior leaflet thickening, and a bicuspid aortic valve with mild to moderate stenosis. Both mitral leaflets were excised, with the valve sent for pathology noting chronic inflammatory, negative for fungal and bacterial organisms, as well as culture which was negative. Unfortunately, 16s rRNA sequencing from the valve was requested but not sent. Postoperative course was complicated by worsening renal failure requiring hemodialysis and hemorrhage requiring sternal exploration and washout. Gentamicin was discontinued after 14 days of therapy to assist with renal recovery. The patient remained on doxycycline 100 mg BID planned for 6 weeks of treatment post valve replacement. Unfortunately, he suffered a sudden PEA arrest, and expired on hospital day 34.

**Discussion**

*Bartonella* species are fastidious, intracellular, gram-negative organisms that reside within erythrocytes. Due to this unique predilection, *Bartonella* is known to inhabit two specific sites: the gut of bloodsucking arthropods and bloodstream of mammalian hosts [1]. Cats have been identified as hosts when they become infected with *B. henselae* following the bite of infected fleas. *Bartonella* is transmitted to humans from a scratch (salivary contamination through grooming) or a bite from an infected cat. We reviewed 17 case reports from 2010 and 2020, 14 of those cases had contact with cats (Table 2).

*Bartonella* is a problematic organism to isolate from culture media. Blood cultures have as low as a 20% sensitivity [2]. Direct plating of blood or tissue is preferred with specialized agars (heart infusion, Trypticase soy, brucella agar, and Columbia agar supplemented with 5% hemoglobin) and incubated for up to 21 days. Special staining such as Warthin-Starry can be used but these are not specific for *Bartonella*. Serology seems to be the most informative. *B. henselae* IgG titers > 1:800 are considered to have the greatest sensitivity for endocarditis with positive predictive value of 0.955 [3]. Low level titers could indicate prior exposure to the bacteria or cross reactivity. IgM assays have lower sensitivity rates since assays react early in the disease process and wane after 10 weeks, but higher specificity than IgG [3]. False positive results may occur with *Coxiella burnetii* and *Chlamydia pneumoniae*. PCR testing has been utilized on both serum and tissue with serum sensitivity is 33% and tissue sensitivity is 92.5% [4]. 16s rRNA from tissue is considered the gold standard, but is invasive. 16s rRNA requires the use of primers that are limited to a certain portion of the genome and thus do not always provide accurate results [3]. Usually, the diagnosis of *bartonellosis* involves several methods to corroborate the results.

Next Generation Sequencing (NGS) of microbial cell free DNA (mcDNA) is a new and emerging diagnostic test. This method is noninvasive with a short turnaround time (28 h), detecting the normal products of bacterial turnover within the body. When we compare the turnaround time to serology, NGS and IFa are almost equal, adding in two to four days for NGS specimen shipping. As the test becomes more readily available, this should improve. The NGS test relies on sequencing mcDNA in circulating plasma to identify over 1000 pathogens (including bacteria, fungi, viruses) and reports a quantitative amount as molecules per microliter (MPM). In one study, NGS reported a 95% sensitivity in culture positive endocarditis even with antimicrobial pre-treatment [3]. NGS was ordered for our patient given the expected low yield from routine blood cultures and to capture other fastidious organisms. From reflection of our case, the differential diagnoses were initially broad, therefore using NGS and IFA together helped secure a diagnosis. We used both tests to advocate for prompt surgical intervention. The downfalls to NGS are that it may pick up on low yield microbe/viroma, requiring clinical interpretation [6] and average NGS costs are roughly twenty times more expensive than IFA. During literature review, it was noted that in some cases patients were left on doxycycline until IFA titers decreased. Serial NGS testing could be considered to evaluate pathogen levels and monitor responses, as down-trending MPM levels correlate directly with decreasing amounts of viable pathogen [7]. This topic however, needs further investigation and would prove to be useful especially since serial IgG levels overtime have been inconsistent [8].

*Bartonella* is the leading cause of culture negative endocarditis in the US [9] and second most common CNE pathogen worldwide [4]. The species typically infects native valves with the aortic valve being the most commonly reported [4]. Prosthetic valve infections have been described, with clinical presentations that are usually severe with rapid progression to heart failure. Rarely, cases of myocarditis associated with *B. henselae* and *B.quintana* infections have been reported. Some reports resulting in sudden unexpected cardiac death in previously healthy individuals [2]. About 70% of patients require valve replacement secondary to severity of valve damage [10]. Mortality remains low at 7% [10]. Various reviews of pathology found histologic findings such as fibrosis, endothelial proliferation and neovascular formation that were distinctive to *Bartonella* [11–13]. The suggestion is for higher calcification and less extensive vegetation material, indicating a more chronic inflammatory process [12]. These findings are supportive of the need for surgical intervention in addition to antimicrobials for clearance rather than medical management alone.

Renal failure is a common manifestation of *Bartonella* endocarditis, with one study estimating greater than 40% of patients present with such findings [14]. Specifically, rapidly progressive renal failure is noted to be a clinical feature in infection-associated glomerulonephritis (GN). Serology testing is typically done to rule out an autoimmune diagnosis in these instances; however, renal biopsy to assess for histological patterns appears to be of vital diagnostic value. Severe damage to the glomerular capillary walls results in crescent formation, findings that can be seen with immune

---

**Table 1**

| Blood cultures x3 | Negative, only first set collected prior to antimicrobial administration |
|--------------------|--------------------------------------------------------------------------------|
| *B. quintana*, IgG with reflex titer | Positive, 1:512 |
| *B. quintana/B. henselae* IgM | Negative |
| *Bartonella henselae* IgG with reflex titer | Positive, > / = 1:1024 |
| Coxiella burnetti DNA Karius (Ref < 10) | Not detected |
| 9653 DNA molecules per microliter | |
| Brucella IgG 3.02, detected. IgM not detected | |
| Mitral Valve tissue pathology Negative CMS stain for fungal organisms, Negative bacterial organisms | |
| 16s rDNA on valve tissue Not done | |
| Renal biopsy Focal necrotizing and crescentic glomerulonephritis, immune complex type | |

---

**Table 2**

| Summary of diagnostic indices. |
|-------------------------------|
| Blood cultures x3 | Negative, only first set collected prior to antimicrobial administration |
| *B. quintana*, IgG with reflex titer | Positive, 1:512 |
| *B. quintana/B. henselae* IgM | Negative |
| *Bartonella henselae* IgG with reflex titer | Positive, > / = 1:1024 |
| Coxiella burnetti DNA Karius (Ref < 10) | Not detected |
| 9653 DNA molecules per microliter | |
| Brucella IgG 3.02, detected. IgM not detected | |
| Mitral Valve tissue pathology Negative CMS stain for fungal organisms, Negative bacterial organisms | |
| 16s rDNA on valve tissue Not done | |
| Renal biopsy Focal necrotizing and crescentic glomerulonephritis, immune complex type | |
Table 2
Summary of prior case reports of Bartonella endocarditis.

| Age/Gender | Contact with cats | Pre-existing heart valve disorder | Valve involved | Other manifestations | Blood cultures | Bartonella serology by IFA | Autoimmune workup | Other testing (Karius, etc) | 16S Ribosomal DNA amplification and sequencing | Treatment: surgical, antimicrobials | Outcome |
|------------|-------------------|-----------------------------------|----------------|---------------------|----------------|--------------------------|------------------|--------------------------|-----------------------------------------------|------------------------------------------|---------|
| [22] 19 yo Male | Yes | Yes, bioprosthetic pulmonic valve | Pulmonic | Crescentic glomerulonephritis (renal biopsy noting cellular crescent formation) | Negative after prolonged incubation | B. henselae 1:64,000 | ANA 1:160, anti dsDNA + 1:20, decreased C3 69 mg/dL and C4 7.7 mg/dL. CRP 149 mg/L. Coombs positive, elevated haptoglobin 238 mg/dL | Not applicable | PCR assay targeting B. henselae ribC gene, and DNA sequencing on valve. Valve tissue not submitted for pathology for special staining. | Empiric antimicrobials: Vancomycin and Ceftriaxone on admission. Doxycycline added when blood cultures returned negative. Pulse dose methylprednisolone and hydroxychloroquine added for presumptive SLE diagnosis, stopped once transesophageal echocardiogram noted destruction of bioprosthetic valve, positive bartonella titer. Changed to Doxycycline and Rifampin; aminoglycoside not used secondary to ongoing renal failure. Prosthetic pulmonic valve replacement. | Repeat serology undefined time reduced to 1:800, renal function deterioration requiring peritoneal dialysis. ANA and anti-dsDNA antibodies, complement titers remained normal off immunomodulatory therapy |
| [23] 65 yo Male | No, but homeless, alcoholism | Aortic and mitral regurgitation | Warts on AV and MV, moderate to severe AR | Cerebral aneurysm, vasculitis noted by skin biopsy of purpura, crescentic glomerulonephritis | Multiple negative | B. henselae 1:1024 B.q uintana 1:512 | Normal complement levels, elevated RF, proteinase 3-ANCA (PR3-ANCA) and myeloperoxidase ANCA | Not applicable | Not applicable | Amoxicillin-sulbactam IV 7 weeks and Gentamicin 2 weeks (culture negative endocarditis), followed by 2 weeks Ceftriaxone IV and Gentamicin IV and Doxycycline. Self discharged before more antimicrobials given. Declined valve surgery. | Creatinine, CRP and PR3-ANCA decreased, skin purpura disappeared. 2 months post DC echo with warts remarkably regressed |
| [10] 60 yo Male | Yes | None | Mitral | Subarachnoid hemorrhage/mycotic aneurysm | 4 set prior to antimicrobials Negative | B. henselae 1:1024 | None | Valve PCR testing positive B. henselae | Not applicable | Not reported | 2 weeks of Gentamicin plus Doxycycline, followed by 4 weeks of Doxycycline. Post-antimicrobial mitral valve replacement for continued deterioration and continued evidence of mycotic aneurysm. Retreated with 2 weeks of Gentamicin plus Doxycycline and 4 weeks of Doxycycline. | Not reported |
| [24] 66 yo female | Yes, bioprosthetic aortic valve | Negative TEE, but fulfilled all five conditions for modified Duke minor criteria | Crescentic glomerulonephritis, right central retinal artery occlusion, leukopenia & thrombocytopenia | Multiple sets negative | B. henselae 1:1600 | Multiple-ANA 1:320, Positive RF, c-ANCA positive | Bartonella PCR testing on kidney biopsy tissue negative | Not applicable | 2 weeks of Gentamicin and 4 months of Doxycycline, until titer decreased to 1:400. Almost started on Rituximab, in addition to steroids prior to return of positive B. henselae titer |
| [25] Male | Yes | None | None | None | None | None | None | Not applicable | Not applicable | | |

(continued on next page)
| Age/Gender | Contact with cats | Blood cultures | Bartonella serology by IFA | Autoimmune workup | Other testing (Karius, etc) | 16 S Ribosomal DNA amplification and sequencing | Treatment: surgical, antimicrobials | Outcome |
|------------|-------------------|----------------|---------------------------|-------------------|---------------------------|---------------------------------|---------------------------------|---------|
| [16] Male  | Unknown           | Bicuspid aortic valve | Aortic valve | Splenic infarct, immune complex glomerulonephritis | Negative | None | CRP 49.0, ESR 25.0, C4 23 mg/dL, ANCA negative, MPO-ANCA negative, PR3-ANCA positive | Valve path with necrosis, neutrophils, B. henselae on tissue culture and specialized stains PCR amplification of short fragment (151 bp) of the pap31 gene using specific primers (higher sensitivity than 16 S rDNA) | Not applicable | Levofoxacin, Azithromycin, doxycycline, and Gentamicin (no duration) | Gradual improvement |
| [26] Male  | Yes               | No              | Aortic valve | lymph nodes, lungs, bone, SQ, epididymis | Negative despite 3 weeks incubation | Not reported | None | C3: 81 mg/dL, C4: 4.2 mg/dL, RF: 102 IU/mL | Not applicable | Doxycycline + Rifampin + Gentamicin | Gradual improvement |
| [27] Male  | Yes               | Bioprosthetic pulmonic valve | Bioprosthetic pulmonic valve | Focal segmental proliferative glomerulonephritis and incomplete crescent formation | Negative | B. henselae IgG > 1:1024, IgM > 1:10 | Elevated ESR (CRP, C3 normal, C4 marginal low, ANCA positive | Prednisone and 6 weeks IV Ceftriaxone | Unknown | Unknown | Improved urinalysis and creatinine post treatment. ESR and CRP normalized |
| [28] Male  | Yes               | Unknown         | Aortic valve | Janeway lesion | Negative | B. henselae IgG 1:63,844, B. quintana 1:16,384 | None | Aortic valve tissue positive for B. quintana | Aortic Valve replacement Ceftriaxone + doxycycline + Azithromycin (added for salvage therapy). Gentamicin not used: CKD stage 3 | Not applicable | Doxycycline + Rifampin + Gentamicin | Deceased from massive transformation of brain infarct |
| [4] Male   | Yes               | Negative        | cANCA 1:256 | Not applicable | | | | | | | | (continued on next page) |
| Age/Gender | Contact with cats | Pre-existing heart valve disorder | Valve involved | Other manifestations | Blood cultures | Bartonella serology by IFA | Autoimmune workup | Other testing (Karius, etc) | 16S Ribosomal DNA amplification and sequencing | Treatment: surgical, antimicrobials | Outcome |
|-----------|------------------|---------------------------------|----------------|---------------------|---------------|------------------------|-----------------|--------------------------|--------------------------------|--------------------------------|---------|
| [29] | Male | No-Dogs | No | Aortic | Daily fever, splenomegaly | Negative | B. henselae > 1:64, B. quintana > 1:64 | Not done | Nested PCR targeting fstZ region and molecular testing targeting Bartonella spp DNA; both noting B. henselae | Not applicable | 2 weeks of PO Doxycycline, IV Ceftriaxone and IV Gentamicin, followed by additional 4 weeks of IV Ceftriaxone plus PO Doxycycline, then indefinite suppression with PO Doxycycline, Gentamicin+Unasyn, followed by Doxycycline 200 mg daily to complete 6 weeks | Followed 5 years later with no complications from endocardial vegetation |
| [30] | Male | Yes | Yes, bicuspid aortic valve | Aortic valve | No | Negative | Not available in UK | None mentioned | Aortic valve | Not applicable | Aortic valve replaced. Amoxicillin/Gentamicin, followed by Doxycycline x 6 weeks and Gentamicin x 64 days | Full recovery, no long term follow up |
| [31] | Female | Yes | Bioprosthetic pulmonary valve and pacemaker | Aortic graft and Aortic grafting | Negative TEE | Acute diffuse proliferative glomerulonephritis | Negative | B. henselae IgG 1:1024, IgM 1:64, B. quintana negative | Decreased C3/C4, initial ANCA inconclusive, positive in speckles pattern, PR3 Ab positive | Not applicable | 15 weeks of Doxycycline and Rifampin | Bartonella PCR undetectable, Serum creatinine declined over 3 months |
| [32] | Male | Yes | Aortic graft, mechanical aortic valve and MV annuloplasty ring | Aortic valve | Splenomegaly, Elevated CRP, thrombocytopenia, fevers, and acute kidney injury | Negative | Not checked | None mentioned | Serum PCR +, confirmed with Western Blot | Not checked | No surgery. Ceftriaxone/ Doxycycline, followed by Ceftriaxone for 8 weeks and Gentamicin for 4 weeks then life-long doxycycline | Full recovery |
| [33] | Male | Yes | Bicuspid aortic valve | Aortic valve | Brain, kidney and spleen infarcts | Negative | B. henselae IgG > 2048, IgM 20 | Elevated ESR, CRP, RF, cANCA and anti-PR3 antibody. C3 low, C4 normal, Lupus anticoagulant detected | Not done | 16S rRNA positive, Histopath of valve with poorly staining coccobacilli, Aortic valve culture on special agar plates positive for B. henselae, Aortic valve grindings analyzed for rpoB gene + B. henselae | Initially treated with aspirin and unfractionated heparin; steroids were planned but not started. IV Ceftriaxone 6 weeks, IV Gentamicin 2 weeks and Doxycycline PO 3 months | Full recovery at 7 months, inflammatory marker normalized |
| Patel et al., 2021 – current case | 65 Male | Yes | Mitral | Bicuspid aortic valve, repair in childhood | Crescentic glomerulonephritis, respiratory failure | Negative | B. henselae > 1:1024, B. quintana > 1:512 | ANA/ANCA negative for B. henselae | Karius, positive for B. henselae | Not performed | Mitral valve replacement. Rifampin + doxycycline x 17 days then gentamicin + doxycycline x 14 days, plan for doxycycline x 6 weeks thereafter | Deceased on hospital day 34 following PEA arrest |
complex diseases such as postinfectious GN, lupus nephritis, IgA nephropathy and vasculitis, as well as anti-glomerular basement membrane disease and antineutrophil cytoplasmic antibody (ANCA) vasculitis [15]. Interestingly, there is evidence of ANCA positivity in serum for many Bartonella endocarditis patients, with one review demonstrating 77% of cases with these findings [16]. Furthermore, a 2014 case report and literature review of Bartonella endocarditis cases reported that 50% of the eight reviewed cases were c-ANCA positive [4]. The correlation between CGN and Bartonella endocarditis may be one where, in settings of unexplained renal failure and concordant findings on biopsy, the diagnosis of Bartonella should be considered.

To further assess this correlation, we reviewed 17 case reports between 2010 and 2020 (Table 2). These cases were selected based on diagnosis of infectious glomerulonephritis via renal biopsy and/or positive blood cultures. In 7 of the 17 cases the patient underwent renal biopsy, revealing features characteristic of crescentic glomerulonephritis, with treatment of Bartonella resulting in restoration of renal function. Concurrent infectious and auto-immune work up was completed in 10 cases given symptoms and negative blood cultures.

In axenic media, Bartonella species appear to be susceptible to many antimicrobials, including penicillin, cephalosporins, fluorquinolones, macrolides and aminoglycosides, but the correlation between in vivo and in vitro is lacking [17]. Gentamicin is the aminoglycoside that appears to be most studied [18]. There appear to be limited or no prospective studies to support treatment guidelines for endocarditis, and most recommendations are largely based on retrospective, observational data. Many experts recommend dual therapy for effective treatment, at least in the initial 2 weeks [18]. The AHA (2005 edition) recommends using doxycycline for 6 weeks after valve surgery or 12 weeks total if the valve is retained and gentamicin for at least 14 days. Alternatives to doxycycline are macrolides with recommendation for 12 weeks minimum with valve surgery and 6 months without surgery. Valv e replacement seems to be central to appropriate therapy, having occurred in 80% of past cases [17]. If gentamicin cannot be used, notably in settings where renal function is a concern, the alternative is rifampin for at least 14 days. Gentamicin is chosen because aminoglycosides are bactericidal, and doxycycline has been shown to penetrate erythrocytes. The combination works well to eradicate the bacteria in different niches within the host [18,19]. In a retrospective study of 101 patients, at least 14 days of aminoglycosides (in combination with another antimicrobial) demonstrated higher rates of recovery as compared to non-aminoglycoside monotherapy [20]. The question remains whether bactericidal activity is required for clearance of the organism. Some data shows Bartonella residing within erythrocytes may afford protection from gentamicin, hence the recommendation to use more than one agent [19]. Interestingly, there is in vitro data to support the use of azithromycin/ciprofloxacin and rifampin/ciprofloxacin combinations to completely eradicate biofilms after 6 days and kill stationary phase Bartonella after day 1 of exposure [21]. The total duration of antimicrobial treatment is not clearly defined, leaving room for future guidelines to assist with clarification.

Conclusion
Bartonella endocarditis remains difficult to identify and manage. Several concurrent presenting symptoms may lead the clinician towards this diagnosis, including renal failure with an auto-immune-like component. NGS may have an up-and-coming role in both diagnosis and treatment. Treatment remains largely surgical, with improved outcomes seen with the use of combination antimicrobial therapy.

Ethical approval
Not applicable-no studies conducted on the patient.

Consent
Not applicable-no studies conducted on the patient/no patient identifying information used in the case report.

Funding
There are no funding sources to declare.

Author contribution
Roshni Patel, Kansas Koran and Amanda Schnee contributed conception and composition of the work. Mark Call assisted with critical edits.

Disclosure
No conflict of interest to disclose for all authors. Our case report does not include factors necessitating patient consent.

References
[1] Rose SR, Koehler JE. Bartonella, including Cat-scratch disease. In: Mandell, Douglas and Bennett’s principles and practices of Infectious Diseases, 2020, 2825–2843.
[2] Okaro U, Addisu A, Casanas B, Anderson B. Bartonella species, an emerging cause of blood-culture-negative endocarditis. Clin Microbiol Infect 2017;33(3):709–46.
[3] Fournier PE, Mainardi JL, Raoult D. Value of microimmunofluorescence for diagnosis and follow-up of Bartonella endocarditis. Clin Diagn Lab Immunol 2002;9(4):795–801. https://doi.org/10.3122/CCLIM.9.795-801.2002
[4] Rodino KG, Stone E, Saleh OA, Theel ES. Closing the brief case: Bartonella henselae endocarditis - a case of delayed diagnosis. J Clin Microbiol 2019;57(9):1–4.
[5] Gu W, Miller S, Chiu CY. Clinical metagenomic next-generation sequencing for pathogen detection. Annu Rev Pathol Mech Dis 2019;14:319–38. https://doi.org/10.1146/annurev-pathmechdis-012418-012751
[6] Camargo JF, Ahmed AA, Lindner MS, Morris MI, Anjan S, Anderson AD, et al. Next-generation sequencing of microbial cell-free dna for rapid noninvasive diagnosis of infectious diseases in immunocompromised hosts. F1000Research 2019;3:1–27. https://doi.org/10.26888/f1000research.19766.1
[7] Hauger S, Fernandez M, Murphey D, et al. Detection of Bartonella species in culture-negative endocarditis using the Karius test, a plasma next-generation sequencing test for pathogen detection. In: ASM Microbe 2010 – Session P548, 2019.
[8] Lesprit P, Noel V, Chazouillères P, Brun-Buisson C, Deforges L. Cure of Bartonella henselae endocarditis in four dogs. J Vet Intern Med 2005;19(6):796–803. https://doi.org/10.1111/j.1939-1676.2005.tb00662.x
[9] Pouilly P, Fournier PE, Raoult D. Quantitative analysis of valvular lesions during Bartonella endocarditis. Heart 2009;95(1):1–6. https://doi.org/10.1136/heart.2008.163852
[10] Varga Z, Gowda SN, Styx A. Myotic aneuroidia of the middle cerebral artery leading to subarachnoid hemorrhage, as the initial presentation of Bartonella henselae endocarditis. South Dak Med J South Dak State Med Assoc 2019;72(2):68–70.
[11] Pesavento PA, Chomel BB, Kasten RW, Mcdonald KA, Mohr FC. Pathology of Bartonella endocarditis in six dogs. Vet Pathol 2005;42(3):370–3. https://doi.org/10.1177/0300985805042003370
[12] Lepidi H, Fournier PE, Raoult D. Quantitative analysis of valvular disease during Bartonella endocarditis. Am J Clin Pathol 2000;114(6):880–9. https://doi.org/10.1093/ajcp/114.6.880
[13] Chomel BB, Kasten RW, Williams C, Wey AC, Henn JB, Magri R, et al. Bartonella endocarditis: a pathology shared by animal reservoirs and patients. Ann N Y Acad Sci 2009;1166(1):120–6.
[14] Raybould JE, Raybould AM, Morales MK, Zeaheer M, Lipkowitz MS, Timpone JG, et al. Bartonella endocarditis and pauci-immune glomerulonephritis: a case report and review of the literature. Infect Dis Clin Pr 2016;24(5):254–60. https://doi.org/10.1016/j.idcp.2016.09.003
[15] Jennette JC, Thomas DB. Crescentic glomerulonephritis. Nephrol Dial Transpl 2001;16(SUPPL. 6):S80–2. https://doi.org/10.1093/ndt/16.suppl.5.S80
[16] Vercellone J, Cohen L, Mansuri S, Zhang PL, Kellerman PS. Bartonella endocarditis mimicking crescentic glomerulonephritis with PR3-ANCA positivity. Case Rep Nephrol 2018;2018:1–4. https://doi.org/10.1155/2018/9607582
[17] Brosgu P, Raoult D. Endocarditis due to rare and fastidious bacteria. Clin Microbiol Rev 2001;14(1):177–207. https://doi.org/10.1128/CMR.14.1.177-207.2001
Rolain J, Brouqui P, Koehler J, Maguina C, Dolan M, Raoult D. Recommendations for treatment of human infections caused by bartonella species. Antimicrob Agents Chemother 2004;48(6):1921–33. https://doi.org/10.1097/01.acm.0000144910.19687.1f

Angelakis E, Raoult D. Pathogenicity and treatment of Bartonella infections. Int J Antimicrob Agents 2014;44(1):16–25.

Raoult D, Fourrier PE, Vandenesch F, Mainardi JL, Erykin SJ, Nash J, et al. Outcome and treatment of Bartonella endocarditis. Arch Intern Med 2003;163:226–30.

Zheng X, Ma X, Li T, Shi W, Zhang Y. Effect of different drugs and drug combinations on killing stationary phase and biofilms recovered cells of Bartonella henselae in vitro. BMC Microbiol 2020;20(1):1–9. https://doi.org/10.1186/s12866-020-01777-9

Shmuely H, Chernin G, Giladi M, Zimhony O, Miller EB. A cat’s scratch or a wolf’s bite? Lupus 2020;29(11):1469–71. https://doi.org/10.1177/096120332093632

Yoshifuji A, Hibino Y, Komatsu M, Yasuda S, Hosoya K, Kobayashi E, et al. A Case of Glomerulonephritis Caused by Bartonella spp. infective endocarditis: the difficulty and importance of differentiation from anti-neutrophil cytoplasmic antibody-related rapidly progressive glomerulonephritis. Intern Med Adv Publ 2021;60:1899–906. https://doi.org/10.2169/internalmedicine.5608-20

Bannon L, Choshen G, Giladi M, Ablin J. Bartonella endocarditis masquerading as systemic vasculitis with rapidly progressive glomerulonephritis (aka ‘Löhein nephritis’). BMJ Case Rep 2019;12(12):30–3. https://doi.org/10.1136/bcr-2019-231413

Kuan W, Dulnian K, Guglin ME, El Haddad H, Kolodzie AR, Leventhal A, et al. A “Cat”-astrophic case of Bartonella henselae infective endocarditis followed by cardiac transplantation salvage therapy. Transpl Infect Dis 2019;21(6):13179.