The Silent Threat: *Bartonella quintana* Endocarditis Unveiling Heart Failure and Severe Pulmonary Hypertension

**Patient:** Male, 51-year-old

**Final Diagnosis:** Endocarditis • pulmonary hypertension

**Symptoms:** Aphasia • confusion

**Clinical Procedure:** —

**Specialty:** Cardiology • Infectious Diseases • Pulmonology

**Objective:** Rare coexistence of disease or pathology

**Background:** *Bartonella quintana* is a slow-growing gram-negative bacterium that can cause severe culture-negative endocarditis. In many cases, its insidious onset can be difficult to diagnose given the variable symptoms in the early phases of the disease. This delay in detection and thus treatment can cause advanced consequences of the disease, including heart failure and severe pulmonary hypertension.

**Case Report:** A 51-year-old man presented to the Emergency Department with signs and symptoms indicating an acute stroke. Further investigation showed that the source was cardioembolic, and despite negative blood cultures, endocarditis was suspected due to echocardiogram findings. *Bartonella* endocarditis was diagnosed based on serology results. Further testing indicated severe pulmonary hypertension, a sequelae of chronic heart failure in the setting of endocarditis. This caused a significant delay in valvular repair surgery. This case illustrates the progression from acute to chronic infection, the sequelae of this disease process, and the considerations involved in management.

**Conclusions:** *Bartonella* is an under-appreciated cause of endocarditis and can evolve into chronic disease with clinical consequences requiring nuanced management. We described a case of chronic culture-negative endocarditis that presented with acute embolic stroke and the sequelae of severe multi-valvular disease in a patient with recent incarceration and unstable housing. This case provides clinicians with valuable insight into the recognition of *Bartonella* endocarditis, the variable clinical presentations of this pathology, the nuanced and multifactorial approaches to medical management, and the indications for surgery.

**Keywords:** *Bartonella quintana* • Embolic Stroke • Endocarditis • Heart Failure • Hypertension, Pulmonary

**Full-text PDF:** https://www.amjcaserep.com/abstract/index/idArt/942160
Background

*Bartonella quintana* is a slow-growing gram-negative bacterium carried by arthropod vectors [1]. Transmitted via the *Pediculus humanus* body louse, *B. quintana* associates with conditions such as decreased hygiene and overcrowding. It is thus more commonly found in individuals experiencing homelessness or incarceration and causes an array of diseases, including endocarditis. *B. quintana* is one of the most frequent causes of culture-negative endocarditis, with a prevalence of up to 4.5% of all endocarditis [2], yet it was only first described as a cause in the early 1990s [3]. Of the 7 *Bartonella* species, *B. quintana* is the most frequently implicated [2]. Due to the insidious course of *Bartonella* endocarditis, infections can produce minimal to no symptoms for months. This makes its diagnosis challenging and potential consequences severe [4].

Case Report

A 51-year-old man with no significant past medical history presented to the hospital with acute aphasia and confusion. He was recently incarcerated in the central valley of California for 2 years and previously was unstably housed in the San Francisco Bay area.

On arrival, he was afebrile and hemodynamically stable. His cardiovascular examination revealed a II/VI holosystolic murmur at the apex, a II/VI diastolic murmur at the right upper sternal border, distended jugular veins, and pulmonary crackles. He was disoriented and aphasic but had no other localizing neurologic findings. Laboratory studies were significant for a hematocrit level of 30.2% (baseline 40% two years prior) and a white blood cell count of 7.0 k/ul (reference range 3.9-11.7 k/ul). No inflammatory markers were collected on admission, given his acute stroke presentation. Computed tomography of the head revealed an intracranial hemorrhage in the left parieto-occipital region with mild local mass effect. Magnetic resonance imaging of the brain with gadolinium contrast showed a 5-mm pseudoaneurysm in the M4 parietal branch of the middle cerebral artery (dark blue arrow) and surrounding vasogenic edema (light blue arrow).

Transthoracic and transesophageal echocardiography showed concentric hypertrophy of the left ventricle, with an ejection fraction of 66%, vegetations on the aortic and mitral valves (Figures 2, 3) associated with moderate-to-severe regurgitation, and elevated estimated right ventricular systolic pressure (100 mmHg). The estimated size of the vegetation on the anterior leaflet of the mitral valve was 0.3×0.4 cm (by transesophageal echocardiography) and on the aortic valve was 1.2×0.5 cm (by transthoracic echocardiography). Although blood cultures remained negative, empiric antibiotic therapy was initiated with intravenous ceftriaxone, doxycycline, and rifampin. Indirect immunofluorescent antibody testing (ARUP laboratories) revealed *B. quintana* immunoglobulin G at a titer of 1: 4096 and *Bartonella henselae* immunoglobulin G at a titer of 1: 32 786. The patient denied a history of injuries from cats or contact with flea-infested cats. Antibiotics were narrowed to doxycycline and rifampin. The patient underwent left heart catheterization, confirming pulmonary hypertension with a mean pulmonary artery pressure of 49 mmHg, pulmonary vascular resistance of 8.5/6.1 Wood units (WU) (thermodilution/Fick), pulmonary capillary wedge pressure of 26 mmHg, right atrial pressure of 9 mmHg, and no obstructive coronary disease. He was subsequently diuresed, with hemodynamic improvement, with repeat right heart catheterization showing a mean pulmonary artery pressure of 44 mmHg, pulmonary vascular resistance of 5.0/4.1 WU (thermodilution/Fick), pulmonary capillary wedge pressure of 21 mmHg, and right atrial pressure of 1 mmHg.

In agreement with guidelines from the American Association of Thoracic Surgery (AATS), the patient was considered for urgent surgical replacement of his aortic and mitral valves. Given the medical complexity, a multidisciplinary team, including the cardiology, cardiothoracic surgery, neurology, and infectious disease services, was enlisted to balance the significant perioperative risks against the hemodynamic and microbiologic
valve replacement 8 weeks after admission. He was discharged to an acute rehabilitation center for 2 weeks, with improvement in motor function, but no change in aphasia. Since returning home, his course was complicated by 3 recurrent presentations for a supraventricular tachycardia, thought to be new-onset atrial flutter. At the time of publication, the patient was 7 months out from his surgical repair and was undergoing appropriate treatment for atrial flutter.

Figure 2. Transesophageal echocardiogram image of the aortic valve in the mid-esophageal long-axis view. The yellow arrow denotes the vegetation on the aortic valve.

Figure 3. Transesophageal echocardiogram image of the aortic and mitral valves in the mid-esophageal aortic long axis view. The yellow arrow denotes the vegetation on the aortic valve; the red arrow denotes the vegetation on the mitral valve.

Discussion

The Duke criteria, published in 1994 (and later modified) have guided the management of infectious endocarditis for the past 3 decades. However, our understanding of the epidemiology and diagnostics for infectious endocarditis has since evolved. Earlier this year, the International Society for Cardiovascular Infectious Diseases proposed modified ISCVID-Duke criteria to include specific microbiological testing for Bartonella species and consideration of intraoperative inspection as a “major criterion,” when compared with the traditional Duke criteria [7]. As in this case, it can be difficult to distinguish between different Bartonella species, as serologies often cross-react [1]. For this reason, Bartonella speciation requires advanced molecular techniques, such as polymerase chain reaction testing of the 16S rRNA, ribC, rpoB, or gltA genes [8], which was not performed in this case as it would have added additional cost without changing management. Furthermore, Bartonella serologies can also cross-react with Coxiella species, although this was not seen in this case. Bartonella henselae can also still be the culprit in patients with no exposure to cats, although this is less common [8]. Given the patient’s risk factors, including lack of feline contact and known history of incarceration and housing instability, B. quintana was determined the presumed culprit of infectious endocarditis in this case.

Infectious endocarditis is one the most common etiologies of acute aortic regurgitation of the native valve [9], with the other being aortic dissection. Infectious endocarditis drives aortic regurgitation via 2 potential mechanisms. First, vegetations can directly disrupt the leaflets, causing either malcoaptation or frank perforation. Second, vegetations can perturb the mechanical integrity of the aortic annulus, leading to leaflet prolapse. Retrograde flow of blood across the aortic valve during diastole increases left ventricular volume. Acutely, this rapidly increases end-diastolic pressure and, in severe cases, causes clinical heart failure. However, because the symptoms of heart failure are nonspecific (eg, dyspnea and fatigue), nonfulminant cases can be difficult to identify. Such cases progress over time, as the ventricle remodels and dilates to accommodate the increased volume. This compensatory remodeling does not produce symptoms per se, but as the left ventricle dilates, its compliance decreases, causing filling pressures to
rise. The subsequent left heart failure and pulmonary congestion can then raise pulmonary pressures and precipitate right heart failure. The rise in filling pressures leads to progressive symptoms of congestion, which often sparks the clinical presentation.

The timing of surgical intervention in this case posed a challenge. The American Heart and Stroke Associations (AHA/ASA) recommend deferring elective surgery for at least 6 months from the time of a stroke [10]. However, in cases of infectious endocarditis complicated by major stroke or intracranial hemorrhage requiring valve surgery, the recommendation is shorter: a delay of at least 4 weeks [11]. This difference, based on higher observational mortality rates for surgery performed during this 4-week window [12], attempts to balance the risk of the high-dose anticoagulation required for cardiopulmonary bypass with definitive source control for further cardioembolic events. In this case, a delay would also risk further deterioration in cardiac function, particularly if the disease could not be medically stabilized. However, expedient surgery would fix the proximal cause of heart failure but would still pose high perioperative risk.

In addition to intracranial hemorrhage progression, the other major risk consideration was the pulmonary hypertension, as severe pulmonary hypertension of any etiology is associated with perioperative mortality rates up to 25% [13]. This is in part because high pulmonary pressures limit the capacity of the right ventricle to weather hemodynamic shifts. Perioperatively, right ventricular preload can fluctuate from blood loss, fluid administration, or reduced venous return from positive pressure ventilation. Similarly, right ventricular afterload can vary as changes in alveolar oxygen tension modulate hypoxic vasoconstriction and pulmonary vascular resistance. Under the stress of severe pulmonary hypertension, these acute hemodynamic changes can reduce cardiac output and precipitate cardiogenic shock, arrhythmias, or sudden cardiac death [13]. Thus, efforts to maintain the systemic blood pressure (ie, mean arterial pressure) must be finely balanced against the pulmonary hemodynamics.

The etiology of pulmonary hypertension is important to consider, as reversible causes of pulmonary hypertension offer an opportunity for risk reduction. Pulmonary hypertension due primarily to left-sided heart disease (WHO group II) can be greatly improved with diuresis, which decreases pulmonary pressures provided that the pulmonary vasculature has not yet remodeled. Practically speaking, this can be assessed with a repeat right heart catheterization to reassess the filling pressures and

---

**Table 1.** Class I American Association of Thoracic Surgery (AATS) recommendations for early valve replacement in infective endocarditis [14].

<table>
<thead>
<tr>
<th>Indication for surgery (regardless of completion of full antibiotic course)</th>
<th>AATS Class of recommendation</th>
<th>Present in this patient?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe heart failure in the setting of valve dysfunction</td>
<td>I</td>
<td>Yes</td>
</tr>
<tr>
<td>Left-sided endocarditis caused by highly resistant organisms (as well as Staphylococcus aureus or fungi)</td>
<td>I</td>
<td>No</td>
</tr>
<tr>
<td>Infective endocarditis causing complete heart block</td>
<td>I</td>
<td>No</td>
</tr>
<tr>
<td>Infective endocarditis causing annular or aortic abscess</td>
<td>I</td>
<td>No</td>
</tr>
<tr>
<td>Presence of a destructive or penetrating vegetation</td>
<td>I</td>
<td>No</td>
</tr>
<tr>
<td>Presence of persistent bacteremia or fever for at least 5 days</td>
<td>I</td>
<td>No</td>
</tr>
</tbody>
</table>

**Table 2.** Summary of the risks and benefits of early surgery in our case.

<table>
<thead>
<tr>
<th>Risks of early surgery</th>
<th>Benefits of early surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticoagulation peri- or intraoperatively in the presence of known intracranial hemorrhage</td>
<td>Definitive treatment of the underlying cause of severe heart failure</td>
</tr>
<tr>
<td>Perioperative fluid shifts risking worsening of decompensated heart failure and/or pulmonary hypertension</td>
<td>Source control for bacteremia and prevention of further embolic strokes originating from the vegetations</td>
</tr>
<tr>
<td>Positive pressure ventilation in the setting of known pulmonary hypertension and right heart strain</td>
<td>Treatment of the cause of severe pulmonary hypertension and prevention of further remodeling</td>
</tr>
<tr>
<td>Acute right ventricular failure, arrhythmia or cardiogenic shock</td>
<td>Prevention of vegetation progression, reducing risk of penetrating vegetations, heart block, or abscess formation</td>
</tr>
</tbody>
</table>
pulmonary vascular resistance after diuresis, with the aim to reduce both, as done and observed in the present case.

To the extent possible, early identification of infectious endocarditis-induced aortic regurgitation, before the emergence of pulmonary hypertension, decreases perioperative complications. Accordingly, AATS guidelines (Table 1) suggest timing of surgery “within days” when indicated, but this should be weighed against potential complications of early intervention [14], as described above. In our patient, the class I AATS indication was severe heart failure in the setting of valve dysfunction, and the complications of both severe pulmonary hypertension and intracranial hemorrhage required multidisciplinary input to determine the optimal timing for valve intervention. After 4 weeks of antibiotics and hemodynamic optimization, given the risks and benefits discussed (Table 2), the patient underwent aortic and mitral valve replacement, with a complication presumed to be atrial flutter. Notably, the management of this case was limited by a diagnostic delay from the incomplete history caused by the aphasia and a prolonged search for next of kin. Further, the patient’s socially complex situation, including a lack of stable housing, created barriers to post-hospital follow-up.

### References:


### Conclusions

Cases of probable *B. quintana* endocarditis are becoming more common due to increasing exposure to unstable housing and urban crowding. The main educational point in this case is to maintain a high index of suspicion for endocarditis when the clinical presentation suggests it, even in the absence of positive blood cultures. As we explored in this case, early recognition of the disease can be challenging, and consequences of advanced disease are often devastating. Furthermore, the available clinical guidance is complex and nuanced, as clear risks and benefits of intervention must be considered. Early recognition of this pathology and the subsequent clinical management often requires a diligent, thorough, and multidisciplinary approach.

### Department and Institution Where Work Was Performed

Department of Medicine, University of California San Francisco, San Francisco, CA, USA.

### Declaration of Figures’ Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.