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A Case of *Bartonella* Endocarditis and Torrential Aortic Regurgitation Leading to Cardiac Arrest

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INTRODUCTION

Infective endocarditis can be difficult to diagnose, especially when blood culture-negative. We describe a case of a patient who presented with signs and symptoms of new, acute decompensated heart failure who was found to have culture-negative endocarditis, a large, mobile aortic valve mass, and torrential aortic regurgitation. Although the patient remained clinically stable during early admission and was planned for surgical correction of the valvular pathology, he suffered abrupt clinical decompensation which resulted in cardiac arrest. Postmortem serologies were found to be positive for *Bartonella henselae* and *Bartonella quintana*.

CASE PRESENTATION

Subjective:

A 53-year-old male with a past medical history of opioid use disorder on suboxone, anxiety, and normocytic anemia, was transferred to Thomas Jefferson University Hospital with a week and a half of progressive dyspnea with exertion and lower extremity edema. He endorsed orthopnea, paroxysmal nocturnal dyspnea, and intermittent substernal chest pain while recumbent. He denied subjective fevers or chills, current chest pain, or shortness of breath at rest. He denied a history of smoking or alcohol use. He endorsed prior fentanyl insufflation, last use about one month prior, but denied having ever used intravenous drugs. The patient had been undomiciled for the past few months.

Objective:

Upon presentation, the patient was afebrile, with a heart rate of 77, blood pressure of 121/60, and saturating 94% on 2L nasal canula (not on home oxygen). Physical exam showed a slightly anxious and unkempt male, who appeared older than stated age. Cardiac exam was notable for a III/VI diastolic murmur and II/IV systolic murmur, both loudest at the right upper sternal border, and jugular venous distension. Lungs were clear to auscultation bilaterally. He had trace bilateral lower extremity edema.

Table 1. Notable labs and trends

	Admission, 7 days prior to code	Day of code
Hgb (baseline 7-10)	7.4	7.9
WBC	11.6	13.9 (w/ neutrophilic predominance)
Na	134	126
BUN	22	61
Cr (baseline 0.8)	1.1	1.5
AST	63	1129
ALT	77	960

Platelets and bilirubin were normal. Urine drug screen was negative. Multiple sets of blood cultures had no growth.

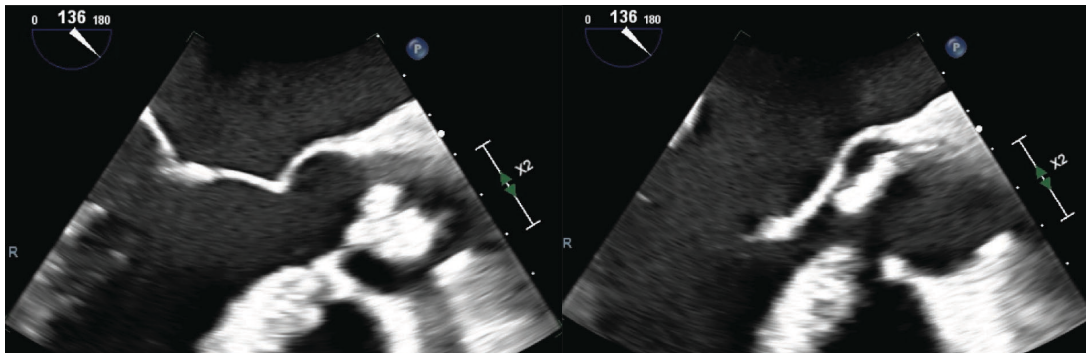


Figure 1: TEE, midesophageal aortic valve long axis view: with aortic vegetation (1.3cm x 0.6cm) extending into the aorta (left), and prolapsing into the left ventricular outflow tract (right).

A transthoracic echocardiogram (TTE) showed new, severe aortic regurgitation and possible bicuspid aortic valve, and could not rule out the possibility of a vegetation. A subsequent transesophageal echocardiogram (TEE) confirmed a bicuspid aortic valve and showed torrential aortic regurgitation (an extreme variety of severe regurgitation) and a large, calcified, mobile mass (1.3 x 0.6 cm) which prolapsed in and out of the left ventricular outflow tract (**figures 1-3**).



Figure 2: TEE, 3D reconstruction of aortic valve, with vegetation extending into the aorta.

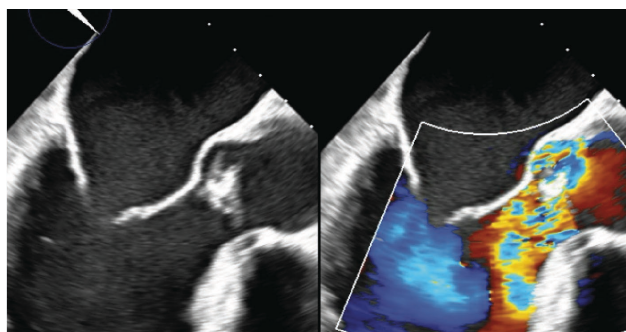


Figure 3: TEE, midesophageal aortic valve long axis view: with aortic valve vegetation and color doppler depicting torrential aortic regurgitation.

The patient was aggressively diuresed and underwent surgical evaluation for valve replacement, which included infectious disease consultation for the mobile density on the aortic valve. Given negative blood cultures, the infectious disease team recommended holding antibiotics and beginning the workup for culture-negative endocarditis, including *Bartonella* serologies.

The Code:

The night prior to his cardiac arrest, the patient was fully oriented, with stable vital signs on 3L oxygen via nasal cannula. He was noted to have intermittent episodes of shortness of breath, and panic attacks that resulted in brief oxygen desaturations when he removed his nasal cannula. Anxiety had been an ongoing issue throughout the hospitalization and was routinely remedied with 1mg oral lorazepam. He also developed his first fever that night, to 101.1 F. He was started empirically on intravenous vancomycin and piperacillin-tazobactam.

The morning of his cardiac arrest, the patient was seen by the physician team at bedside. He was lethargic and only oriented to self. He endorsed shortness of breath, despite normal oxygen saturation on 3L nasal cannula. STAT labs showed a normal lactate and VBG of 7.35/37, but his overnight labs revealed an acute rise in transaminases, as well as an increase in white blood cell count and a decrease in serum sodium (**Table 1**). Shortly after, the patient became acutely agitated and could not be reoriented. He attempted to stand up, visibly short of breath, became bradycardic to the 30s, and lost consciousness. Found pulseless, chest compressions were promptly initiated, and a code blue was called. Telemetry monitoring revealed pulseless electrical activity (PEA) and advanced cardiovascular life support (ACLS) protocol was followed. Ventilation via bag-valve mask was difficult, and the first two attempts at intubation were unsuccessful, due in part to blood in the airway. With both anesthesia and otolaryngology teams present, the third attempt was successful with two-attending intubation using video laryngoscopy, and was confirmed by bilateral breath sounds, capnography, and robust color change of the patient.

The anesthesia team expressed their continued concern for active pulmonary hemorrhage, however, and bronchoscopy was pursued, which showed copious bright red blood originating from the left mainstem bronchus. An attempt was made to bypass the bleeding with right mainstem intubation, though he was found to have right lower bronchus bleeding as well. Greater than 1 liter of blood was suctioned during laryngoscopy and bronchoscopy. The endotracheal tube was ultimately retracted to the level of the carina while ACLS continued. Though resuscitation was attempted for over 30 minutes, the patient continued to have PEA arrest without successful return of spontaneous circulation (ROSC). Blood transfusion was thought to be of low utility given rapid bronchopulmonary hemorrhage, lack of ROSC, and time it would take to begin transfusion. No other reversible causes were identified, and patient death was pronounced. Three days post-mortem, the patient's blood serologies returned positive for *B. henselae* and *B. quintana* IgG and IgM, at which point he met the diagnostic threshold of "definite" infective endocarditis by Duke criteria. His major criterion was evidence of endocardial involvement on TEE; and his minor criteria were fever, predisposing heart condition (bicuspid aortic valve), serologic evidence of active infection with organism consistent with infective endocarditis, as well as (suspected) vascular phenomena.

DISCUSSION

Cat scratch disease and Trench fever may come to mind when one thinks of *Bartonella*, however it is a rapidly rising cause of culture-negative endocarditis, which often goes undiagnosed due to difficulties in detecting the species. The organism is characterized by indolent growth and the ability to form biofilms which, together with its characteristic intraerythrocytic propagation, increases the risk for infection of heart valves, and the ability to evade the immune system and systemic antibiotics. The patient in this case had both of the two most common causes of *Bartonella*-induced endocarditis: *B. quintana*, and *B. henselae*, with the former accounting for approximately 75% of cases of *Bartonella* endocarditis.

B. henselae often occurs in patients who have an underlying valvular condition, while *B. quintana* most frequently occurs with louse exposure in the homeless population. Interestingly, the patient had an underlying valvular condition and was recently unhoused, and subsequently tested positive for both species of *Bartonella*. The patient had denied any cat exposure, and no known louse or flea bites, though did endorse previously having “mites” in his bedding. Though traditionally reported in patients with HIV and in other immunocompromised states, *B. quintana* has since been well-documented in people without any known immunodeficiency. Of note, the patient had previously tested negative for HIV one year prior to admission, but had not been retested at the time of the present admission.

Current treatment recommendations vary and are not based on randomized trial data. Suggested treatments include a two-drug regimen, often rifampin and doxycycline for three months. Given the risk of nephrotoxicity with aminoglycosides (as well as immune-complex glomerulonephritis seen in *Bartonella* endocarditis), a less preferred alternative is an aminoglycoside in combination with either a beta-lactam, a macrolide, or a tetracycline.

We can only speculate what may have occurred in this intriguing case, as the patient’s family declined autopsy. Multiple signs suggest that the patient may have had showering of septic emboli from his endocarditis, such as his acute hepatocellular pattern of liver injury within 24 hours, and rapid-onset encephalopathy. However, metabolic confounders such as hyponatremia and azotemia may have also contributed to his change in mental status. Regarding his pulmonary hemorrhage, again, we can only speculate. CT scan of his chest two days prior to cardiac arrest showed “mild background pulmonary edema” and “lower cervical and mediastinal

lymphadenopathy, nonspecific and likely reactive.” The lymphadenopathy may have been related to *B. henselae*, as seen in cat scratch disease, perhaps with a more disseminated infection than anticipated, leading to alveolar hemorrhage. Though rare, there have even been reports of bronchopulmonary manifestations of bacillary angiomatosis.

A case such as this prompts providers to ask what could have been done differently. Could it have been detected earlier? Should any unhoused individual with endocarditis have *Bartonella* higher on their differential? Specific to this case, would earlier antibiotic management have made any difference? It seems unlikely given the extensive treatment course and the fastidious nature of the *Bartonella* species. The patient did develop an acute kidney injury, so had his results come back sooner, rifampin and doxycycline may have been considered as a treatment for him while awaiting definitive valvular surgery. Given the vegetation size and degree of symptomatic valvular dysfunction, he was tentatively planned for surgery, but unfortunately was still undergoing preoperative evaluation at the time of his rapid clinical decompensation. Earlier surgical intervention may have been the only way to increase the chance of survival, though time did not allow for that in this patient.

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