

Post-Infective Glomerulonephritis and Acute Heart Failure as Manifestations of *Bartonella henselae* Infective Endocarditis: A Case Report

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Abstract

Although blood culture (BD) is the cornerstone of Infective Endocarditis (IE) diagnosis, up to 31% of all cases of IE are Blood Culture - Negative Infective Endocarditis (BCNIE). We were encountered with a case of BCNIE involving the aortic native valve, who was initially admitted with acute nephritic syndrome (due to a glomerulonephritis) and some days later developed acute heart failure due to severe valve insufficiency. It was finally identified *B. henselae* as the causative agent. The patient was referred for urgent surgery, where he had an aortic valve replacement, followed by 4 weeks of Doxycycline 100 mg/12h plus Gentamycin 3 mg/Kg/d during the first 2 weeks, with a successful outcome.

Keywords: Infective Endocarditis; Blood Culture; *Bartonella henselae*; Nephritic Syndrome

Introduction

Infective Endocarditis (IE) is usually suspected in a patient with fever and a new or changing cardiac murmur and is diagnosed based on the presence of vegetation on echocardiography and positive blood cultures. Even though, up to 31% of all cases of IE are Blood Culture - Negative Infective Endocarditis (BCNIE) [1]. Previous antibiotic treatment is likely the most frequent cause of BCNIE, but there are also so called "fastidious" bacteria, that don't grow using the usual blood culture methods and can also cause BCNIE. The two most prevalent etiologic agents of BCNIE are *C. burnetti* and *Bartonella* spp [2], whose diagnosis is usually carried out serologically, and there treatment is based on different antibiotics from those used empirically for IE. Sometimes, these treatments must be prolonged for months.

Case Presentation

58 years old man, currently smoker (40 py), with past medical history of severe obese hypoventilation syndrome (OHS), high blood pressure, hypercholesterolemia and peripheral arteriopathy with previous iliofemoral bypass, treated respectively with: CPAP, Olmesartan/Amlodipine/HCTZ, Rosuvastatin and Clopidogrel.

He presented to the Emergency Department (ED) with nycturia (up to 12 - 13 times per night) of some weeks duration, with no macroscopic hematuria. His vital signs were as follows: 171/77 mmHg, 93 bpm, 36.9°C, 98% oxygen saturation. Physical examination didn't show anything relevant. Laboratory data were: creatinine 1.8 mg/dl, urea 180 mg/dl, CRP 36,1 mg/l and urinalysis that showed proteinuria, microhematuria and leukocyturia. He was finally admitted to the ward with a diagnosis of nephritic syndrome.

During hospitalization additional laboratory results revealed thrombocytopenia, hypocomplementemia, and positive ANCA (MPO and PR3) antibodies. In the urine culture grew up only 20,000 CFU/ml of *E. coli* sensitive to all antibiotics tested, even though he started antibiotic treatment with an oral cephalosporine (for a total of 7 days) and was discharged pending a renal biopsy.

One week later, once removed anti-aggregation from daily treatment, the patient was readmitted for a programmed kidney biopsy to confirm the suspected glomerulonephritis. Then, he referred orthopnea for the previous three days and a new physical examination revealed a panfocal systolic murmur and basal crackles. Laboratory test showed BNP 1551 pg/ml and bilateral pleural effusion was observed in thorax radiography. The transthoracic echocardiogram showed a 12 mm vegetation in the aortic valve's right coronary veil which resulted in a severe valve insufficiency.

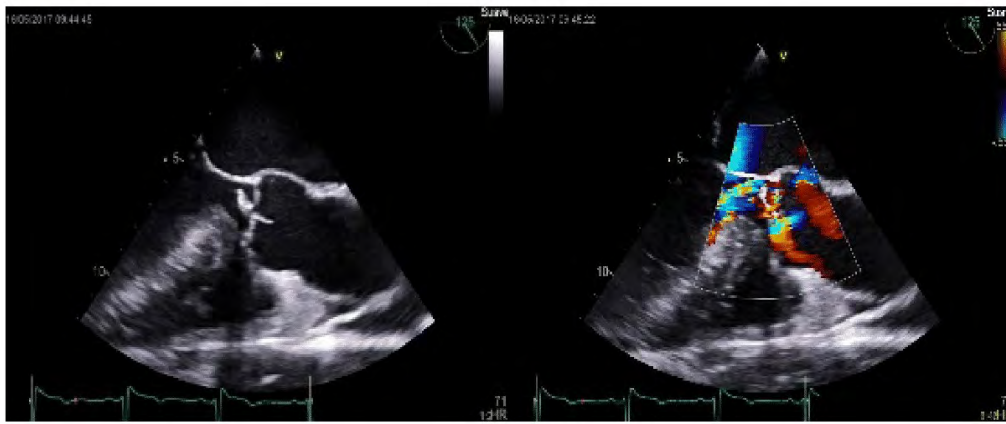


Figure 1: Transesophageal echocardiogram: Aortic valve vegetation and aortic valve insufficiency.

According to previous results, with a suspected IE diagnosis, blood cultures were obtained and empiric antibiotic treatment with Ampicillin, Cloxacillin and Gentamicin (adjusted dose according to creatinine levels) was initiated.

A transesophageal echocardiogram was performed, which confirmed previous findings and ruled out other local complications. A coronarography discarded coronary artery disease and the patient was forwarded to the reference Cardiac Surgery Department. 48 hours later he underwent an aortic-valve replacement surgery with mechanical valve prosthesis.

Due to all blood cultures negative results, an extended microbiological research was performed, with a positive *B. henselae* IgG in a 1/2560 title. After surgery, causative diagnosis could be confirmed with a positive PCR16S for *Bartonella* spp in the native aortic valve.

With a *B. henselae*'s IE diagnosis, ampicillin and cloxacillin were discontinued and a Doxycycline 100 mg/12h regimen was initiated and maintained for a total of 4 weeks, associated with Gentamicin for the first 2 weeks.

Clinical evolution was satisfactory and the patient was discharged with a nearly normalized kidney function.

Regarding the previous kidney biopsy, anatomopathological results showed a post-infectious glomerulonephritis, with positive immunofluorescence against multiple antibodies.

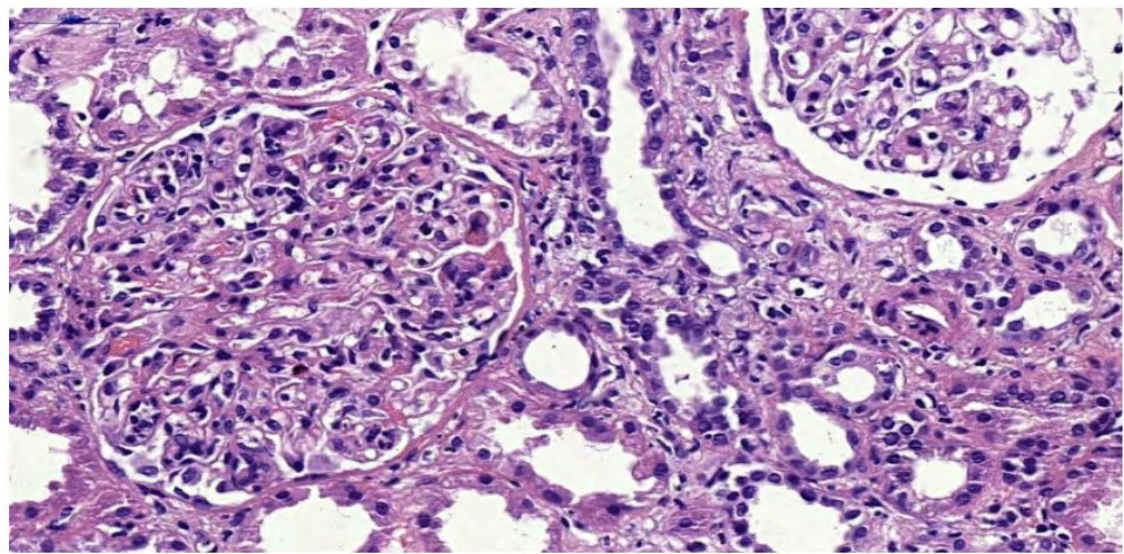


Figure 2: Optical microscope: half-moon which initially suggested rapidly progressive glomerulonephritis.

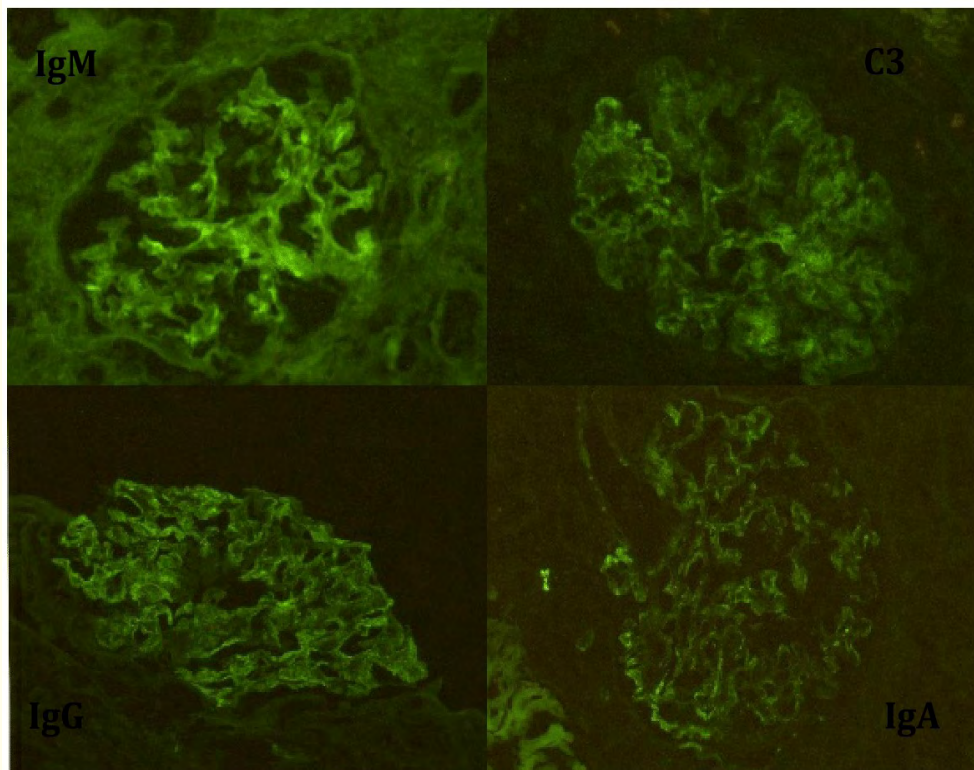


Figure 3: Immunofluorescence: positive for IgA, IgG, IgM and C3 which suggested post-infectious glomerulonephritis rather than rapidly progressive glomerulonephritis.

Discussion

Bartonella spp. are small facultative intracellular bacteria that stain gram negative. They are slow growth bacteria difficult to isolate with usual culture methods, so to identify them we must carry out serologic and molecular microbiologic tests.

These organisms cause various clinical syndromes in immunocompetent and immunocompromised hosts. *B. henselae* is transmitted to humans by a cat scratch or bite or by cat fleas and cause a subacute insidious endocarditis, so that the diagnosis is usually considerably delayed, which may explain why most patients present with acute cardiac failure.

Conclusion

IE is a deadly disease. Despite improvements in its management, IE remains associated with high mortality and severe complications. Early diagnosis must be our main objective, which becomes even more complicated in BCNIE. Therefore, uncommon causes of BCNIE should always be in mind when doing differential diagnosis.

Disclosure

The authors declare no conflict of interest.

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