Available online at www.elixirpublishers.com (Elixir International Journal)

Cardiology

Elixir Cardiology 110 (2017) 48593-48595

ISSN: 2229-712X

Q fever Infective Endocarditis: Case Report

G.El Ouazzani, S.Rahhali, A.Ghalem, N. El Ouafi and N.Ismaili

Department of cardiology, Mohammed VI University Hospital, University mohammed first of Oujda, 60049 Oujda, Morocco.

ARTICLE INFO

Article history:

Received: 21 August 2017;

Received in revised form:

20 September 2017;

Accepted: 30 September 2017;

Keywords

Coxiella burnetii,

Endocarditis,

Q fever.

ABSTRACT

Infectious endocarditis with Q fever is a rare and not very specific infection; Its diagnosis is difficult since it is usually a negative blood culture endocarditis. From a patient who was hospitalized in the cardiology department at the Mohamed IV University Hospital Center in Oujda; We describe the clinical and paraclinic presentation of this type of endocarditis while analyzing its lesional features; Therapeutic and evolutionary

Introduction

Q fever Infectious endocarditis (IE), a zoonosis caused by *Coxiella burnetii*, is a rare and non-specific infection. *Coxiella burnetii* is an obligatory intracellular bacteria whose growth is relatively slow; It requires culturing on specialized media. Diagnosis is usually made by serological tests and / or polymerase chain reaction (PCR) [1, 2].

We describe the case of a 56-year-old patient with a mitral stenosis, who was admitted for prolonged fever, in which work-up led to the diagnosis of Q fever IE [3].

Case report :

A 56 years-old male presented to our hospital with prolonged fever, nocturnal paroxysmal dyspnea , and poor general condition. Upon presentation, vitals were as follows: oral temperature of 37.5°C, blood pressure of 120/75 mmHg, heart rate of 80 beats/min , and respiratory rate of 20 breaths/min, with an oxygen saturation of 98 % on room air. The patient was alert and oriented. Physical examination revealed mitral valve rumble and left arm tremor. No signs of heart failure were found. Electrocardiogram was normal.

On admission, the patient's hemoglobin, white blood cell count, and platelet count were 8.6 g/dL, 9420/mm3, and 240.000/mm3, respectively. The mean corpuscular volume (MCV) and hemoglobin concentration (MCHC) were 86 fL and 31 g/dL respectively. Serum level of ferritin was 1150ng / mL, of C-reactive protein was 92 mg/l, and of erythrocyte sedimentation rate was 36 mm at 1 hour. Cytobacteriological examination of urine (CBEU) showed microscopic hematuria. Serial blood cultures were negative. Serum electrolytes, renal function, and liver function tests were unremarkable.

Transthoracic echocardiography (TTE) displayed thickened mitral valves, a moderate mitral stenosis, and a vegetation measuring 12 x 22 mm attached to the posterior mitral leaflet. Abdominal ultrasonography showed homogeneous splenomegaly along with renal and splenic infarctions. Cerebral computed tomography scan delineated two middle cerebral artery ischemic strokes.

Broad-spectrum antibiotic therapy with ampicillin, cloxacillin, and gentamicin was initiated. Given the absence of improvement of the inflammatory syndrome and the destruction of the mitral valve becoming regurgitant, endocarditis with atypical germs was strongly suspected. Serological tests for challenging microorganisms were performed, including *Coxiella burnettii, Legionella pneumophila, Mycoplasma pneumoniae, Bartonella henselae, Bartonellaquintana, Aspergillus spp., Brucella spp*. They were positive for *Coxiella burnettii*. We talked back to the patient who revealed he had been living with two cats.

The patient was referred to surgery given the embolic complications and mitral valve destruction. He was put on Doxycycline and Hydroxychloroquine for 18 months with a favorable course.

Discussion:

Blood culture–negative IE accounts for 10% of all IE. Except for prior antibiotic administration, they are most often due to fungi or fastidious bacteria, notably obligatory intracellular bacteria such as *Coxiella burnettii* for which serology and genome amplification techniques are available [1].

Q fever, a zoonosis caused by *Coxiella burnetii*, is an ubiquitous infection. However, some regions seem to have a preponderance for the disease, such as the Basque area of Spain and the south of France [4]. Domestic ruminants (cattle, goats, and sheep), cats and dogs are the main reservoirs. Humans are infected by inhalation of aerosols from contaminated soil or animal waste [5, 6].

Clinical presentation of Q fever IE, which accounts for approximately 3% of IE, is insidious and atypical [7,8]. As a result, there is often a significant diagnosis delay with an average from the onset of symptoms to diagnosis of 7 months [9].

The majority of cases have congestive heart failure due to valvular dysfunction [10]. Unlike other cases of endocarditis, fever is absent in a significant proportion and is frequently intermittent or low-grade [11]. Although embolic events have been reported in one-third of cases, they are limited to advanced disease processes [12].

Tele: 00212 628 428 64

E-mail address: elouazzanighizlane@gmail.com

Immune complex glomerulonephritis is common and usually manifests as microscopic hematuria [13].A purpuric eruption can also be seen in a minority of patients [9]. The presence of hepatomegaly and splenomegaly are suggestive of the disease [14]. Numerous hematological abnormalities can be seen in up to 50 % of Q-fever IE, the most frequent being anemia and the least frequent being leukocytosis, leukopenia, and thrombocytopenia [4, 9].

While TTE reveals abnormalities in only 12% of cases, due to the –usually- small size and nodular shape of the vegetation, transesophageal echocardiography is more sensitive [9,14,15].

From a microbiological standpoint; the diagnosis of Q fever IE is hampered by the inability to cultivate *Coxiellaburnetii,*an obligatory intracellular bacteria, using usual media. Therefore, diagnosis is generally established by serological tests, which reveals elevated IgG level at 1: 800 (specificity: 99.6%) [16, 17, 18]. In fact, the Duke criteria were modified to include this serological threshold as a major criterion for the diagnosis of endocarditis [19]. Immunohistology and polymerase chain reaction (PCR) are last resort options used to search for *Coxiella burnetii* in blood cultures and surgical material; however, PCR has a sensitivity of only 75% [20].

The Optimal treatment for Q fever IE is not completely defined. In vitro studies using cell cultures have shown that doxycycline, chloramphenicol, rifampicin, and quinolones are active against *Coxiella burnetii* [21, 22]. These antibiotics are bactericidal or simply bacteriostatic depending on the cellular culture model used. In the model developed by Raoult et al., doxycycline in combination with chloroquine exhibited bactericidal activity [22]. This combined therapy using two active antibiotics against *Coxiella burnetii* in vitro appears to be successful. The combination of fluoroquinolone with doxycycline or rifampicin was successful in controlling the infection, although relapse rates were 50% when antibiotics were discontinued despite prolonged therapy (three years) [23, 24]. A comparison between the association of doxycycline/chloroquine and doxycycline/ ofloxacin [25] has shown that the group with doxycycline/chloroquine combination experienced much less relapse, further no relapse was seen after 18 months of treatment.

Conclusion :

Q-fever IE is a rare and severe condition whose diagnosis remains difficult and is based mainly on serological tests. When managing prolonged fever, the Clinician must consider the diagnosis if there is a history of contact with cats, dogs, and domestic ruminant. Given the destructive and mutilating character of *Coxiella burnetii*, surgery is often required.

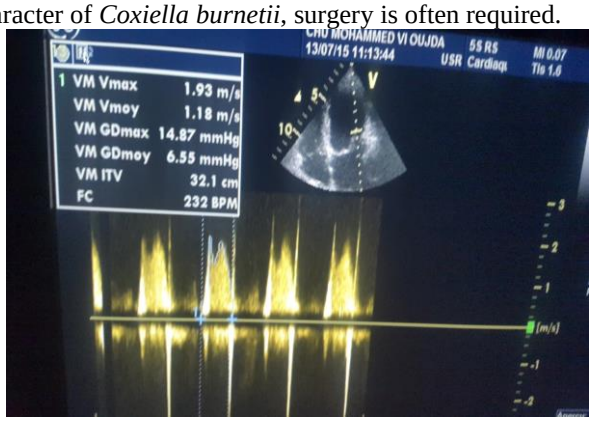

Figure1. A moderate mitral stenosis.

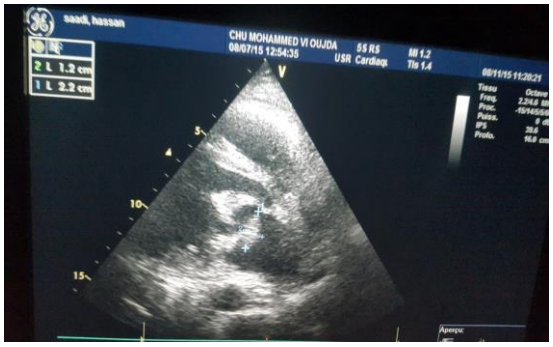

Figure 2. A vegetation measuring 12 x 22 mm attached to the posterior mitral leaflet.

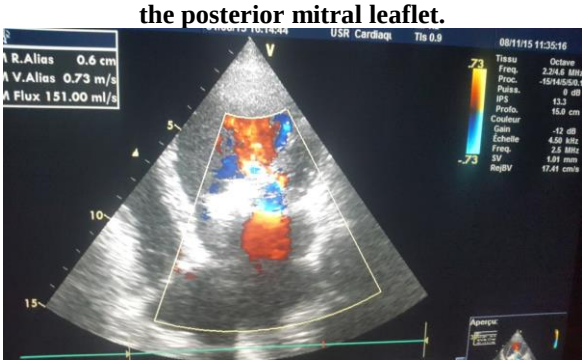

Figure 3. TTE: Destruction of mitral valve and appearance of mitral insufficiency.

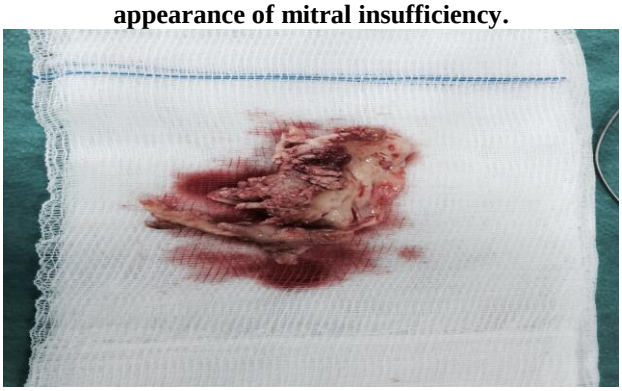

Figure 4. A mitral operative part.

References:

1. **Mohammed Chakour , Jérôme Maslin , Mariama Chadli, Jean-Jacques Chomel [c], Elisabeth Nicand** Endocardite infectieuse à hémocultures négatives (L'étiologie virale comme diagnostic d'exclusion) - 27/11/08 REVUE FRANÇAISE DES LABORATOIRES

2. **Raoult D, Fournier PE, Drancourt M, Marrie TJ, Etienne J, Cosserat J, et al.** Diagnosis of 22 new cases of Bartonella endocarditis. Ann Intern Med. 1996;125(8):646–52. [PubMed]

3.**Perloff JK**. The clinical recognition of congenital heart disease. 4th ed. Philadelphia: Saunders; 1994. p. 323-8.

4.**Maurin M, Raoult D**. Q fever. ClinMicrobiol Rev 1999;12:518-53.

5.**Marrie TJ, Durant H, Williams JC, Mintz E, Waag DM**. Exposure to parturient cats: A risk factor for acquisition of Q fever in Maritime Canada. J Infect Dis 1988;158:101-8.

6.**Buhariwalla F, Cann B, Marrie TJ**. A dog-related outbreak of Q fever. Clin Infect Dis 1996;23:753-5.

7.**Marrie TJ**. A comparison of Q fever endocarditis with native valve endocarditis. Ann N Y AcadSci 1990;590:61-7.

8.**Marc W Deyell, Brian Chiu, David B Ross , Nanette Alvarez** .Q fever endocarditis: A case report and review of the literature. Can J CardiolVol 22 No 9 July 2006

9.**Houpikian P, Habib G, Mesana T, Raoult D**. Changing clinical presentation of Q fever endocarditis. Clin Infect Dis 2002;34:E28-31.

10.**Levesque B, De Serres G, Higgins R, et al**. Seroepidemiologic study of three zoonoses (leptospirosis, Q fever, and tularemia) among trappers in Quebec, Canada.ClinDiagn Lab Immunol 1995;2:496-8.

11**. Houpikian P, Habib G, Mesana T, Raoult D**. Changing clinical presentation of Q fever endocarditis. Clin Infect Dis 2002;34:E28-31.

12. **LeBer C.** Q fever, 1982-1986 – Ontario. Can Dis Wkly Rep 1988;14:70-1.

13.**Fenollar F, Fournier PE, Carrieri MP, Habib G, Messana T, Raoult D.** Risks factors and prevention of Q fever endocarditis.Clin Infect Dis 2001;33:312-6.

14.**Korman TM, Spelman DW, Perry GJ, Dowling JP**. Acute glomerulonephritis associated with acute Q fever: Case report and review of the renal complications of Coxiellaburnetii infection. Clin Infect Dis 1998;26:359-64.

15.**Maurin M, Raoult D**. Q fever. ClinMicrobiol Rev 1999;12:518-53.

16**. Albrich WC, Kraft C, Fisk T, Albrecht H**. A mechanic with a bad valve: blood-culture negative endocarditis. Lancet Infect Dis **4**: 777-784, 2004.

17.**Raoult D, Vestris G, Enea M**. Isolation of 16 strains of Coxiellaburnetii from patients by using a sensitive centrifugation cell culture system and establishment of the strains in HEL cells.JClinMicrobiol 1990;28:2482-4.

18**.Fournier PE, Marrie TJ, Raoult D**. Diagnosis of Q fever.JClinMicrobiol 1998;36:182334.

19.**Dupont HT, Thirion X, Raoult D**. Q fever serology: Cutoff determination for microimmunofluorescence. ClinDiagn Lab Immunol 1994;1:189-96.

20.**Lepidi H, Houpikian P, Liang Z, Raoult D.** Cardiac valves in patients with Q fever endocarditis: Microbiological, molecular, and histologic studies. J Infect Dis 2003;187:1097-106.

21.**Yeaman MR, Mitscher LA, Baca OG.** In vitro susceptibility of Coxiellaburnetii to antibiotics, including several quinolones.Antimicrob Agents Chemother 1987;31:1079-84.

22.**Raoult D, Drancourt M, Vestris G**. Bactericidal effect of doxycycline associated with lysosomotropic agents on Coxiellaburnetii in P388D1cells. Antimicrob Agents Chemother 1990;34:1512-4.

23.**Cacoub P, Wechsler B, Chapelon C, Thibon M, Godeau P**. Q-fever endocarditis and treatment with the fluoroquinolones.Arch Intern Med 1991;151:816,818

24.**Levy PY, Drancourt M, Etienne J, et al**. Comparison of different antibiotic regimens for therapy of 32 cases of Q fever endocarditis.Antimicrob Agents Chemother 1991;35:533-7.

25.**Raoult D, Houpikian P, TissotDupont H, Riss JM**, Arditi-DjianeJ,Brouqui P. Treatment of Q fever endocarditis: Comparison of 2 regimens containing doxycycline and ofloxacin or hydroxychloroquine. Arch Intern Med 1999;159:167-73.