Q FEVER:A CASE REPORT IN URBAN AREA

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Abstract
Starting in 2007, Q fever incidence in the Netherlands has increased significantly, from 10-20 cases annually to 170 cases in 2007 and 808 cases in 2008 (1). Q fever is a zoonotic disease caused by Coxiella burnetii. Only one-half of infected people show signs of clinical illness, most frequent atypical pneumonia, 10% of patients have jaundice and Clinical Manifestations of Hepatitis. Q fever is diagnosed serologically: ELISA test detection of IgM is sensitive (95%) and highly specific. Early diagnosis and therapy may shorten the duration of illness and protect from chronic Manifestations with a high mortality. We present a case of a patient hospitalised for acute Q fever with liver involvement.

Keywords: Q fever, Coxiella Burnetti, anthropozoonoze

INTRODUCTION

Rickettsioses is anthropozoonotic diseases caused by Gram negative bacteries with intracellular development, generally transmitted by arthropod vector and clinically characterized by a febrile exanthema, eschar with inoculation, headache. Q fever is a special rickettsiosis by etiology (Coxiella burnetii is situated separately in the family Rickettsiaceae), by the transmission mode (directly without vector) and the clinical picture (fever is not accompanied by exanthema). Q fever, a zoonosis recognized as a clinical entity in 1937 (2), is caused by the obligate intracellular parasite, Coxiella burnetii. The disease is endemic worldwide, occurring in different geographic regions and climatic zones. New Zealand is probably the only large country without Q fever (3).
Coxiella burnetii cause symptomatic or asymptomatic infections at many animals, especially sheep, goats, cows, but also dogs, cats, rodents or birds. It is found in tissues, fluids, faeces, survives especially in placental tissue (4, 5). At humans the disease appears especially as professional disease (veterinarians, farmers, shepherds), but also to people without direct contact animals in endemic areas where exists the specific reservoir. Germ is spread by direct contact with the pathological product, by respiratory aerosols and rarely by digestive (milk underboiled). The extremely large reservoir, the multiple ways of propagation, the high resistance in the environment of this germ makes himself a potential biological weapon.
The forms of the disease in humans are diverse, acute or chronic, difficult to diagnose in the absence of epidemiological or clinical characteristic arguments. The combination of respiratory and liver determinations can facilitates the diagnosis of acute forms, as endocarditis lesions in chronic forms (6). The most characteristic acute manifestation remains pneumonia with radiological appearance of "gut glass"(7, 8).

History:
The case presents a young, MRC, aged 28 years, the sales agent profession in Oradea, with 2 episode of acute articular rheumatism in 6 and 9 years in pathological history, recently returned from a training conducted in Bucharest, admitted in 2008/10/28 in Oradea Infectious Diseases with suspicion for acute viral hepatitis and a tracheo-pharyngeal Acute bronchitis.
The patient was in the 7th day of the tell-onset with fever 38-40 degrees, chills, subsequently dry cough, nausea, generalized myalgia, vomiting bile content, fatigue, loss of appetite and dark urine emissions and biological investigations performed on ambulatory reveals:thrombocytopenia: platelet 59.000/mm3, moderate inflammatory syndrome: ESR 29/58 mm, hepatocytolisis TGP 71 IU / l (limit <39 IU / l), TGO 56 IU / l (limit <36 IU / l). It is follows treatment with Clarithromycin 2x500 mg. / day 3 days and then Zinnat 2x1 tb. / day 3 days.

On admission:
The patient presented coughwith expectoration reduced , nausea, loss of appetite, pale skin and easy scleral jaundice, pulmonary: harsh vesicular murmur in both bases, sabural language , elastic abdomenn with mild sensibility in epigastrium, hepatomegaly at 2 cm below rebord, elastic consistency, spleen palpable lower pole.
Biology: thrombocytopenia 128.000/mm3, normal leucocytes WBC 5000/mm3, with normal leucocytes formula, Hb = 12,5g / dL, moderate hepatocytolisis TGP = TGO = 2 x VMN, Total bilirubin=2,2 mg% (limit <1,1 mg%), easy inflammatory syndrome: ESR = 40 mm / hour, C-reactive protein (CRP) pozitive. Radiological examination has revealed a pre and infrahilar bilateral interstitial drawing type reticular emphasized the aspect of interstitial pneumonia.

Differential diagnosis:
In these conditions, the differential diagnosis should elucidates the cause of a moderate hepatocytolisis syndrome, who began feverish and accompanied by uncharacteristic radiological manifestations of interstitial pneumonitis.

1. A liver disease, where the inflammatory syndrome could be explained by concurrent respiratory infection (caused by respiratory viruses or atypical germ-Chlamydia psittaci, Chlamydia pneumoniae, Mycoplasma pneumoniae), namely:

-An acute infectious hepatitis, situated at onset (A-E liver viruses), for which pleads the acute onset, partially the clinical picture and hepatocytolisis associated with cholestasis, no fit only for moderately elevated values of the liver function tests; best fit had an infection with EBV (but Ig M mononucleosis was negative) or an infection with CMV (but lacked of the epidemiological arguments);

-A more acute infectious hepatitis (B, C, B+D) for which pleads liver tests, thrombocytopenia, but lacked of the antecedents and the risk factors for chronic B or C infection and also, lacked of the hepatomegaly with characteristic clinical changes at the objective examination;

-A noninfectious liver disease (alcohol, drugs, autoimmune, steatosis or obstructive process satellite): lacked of the antecedents, repeated consumption of alcohol or drugs, or other autoimmune manifestations or factors associated with any steatosis creates less likely probability for this hypothesis; abdominal ultrasonography without changes of the liver or biliary tree can also rule out an obstructive process or steatosis.

2. A respiratory infection associated with hepatocitoliza in the context of the same disease (possibly with atypical germ Ch psittaci, Coxiella burnetii) this hypothesis is supported by the concurrent onset of the manifestations, the biological changes, the favorable trends in the treatment with ciprofloxacin.

3. A febrile disease with myositis and moderate hepatic impairment (leptospirosis): for which pleads the hepatic cytolysis and cholestasis, the inflammatory, but missing epidemiological arguments.

Treatment:
The Ciprophloxacyn antibiotics treatment was instituted after hospitalization because of the potential involvement of atypical germ and thanks very good therapeutic response obtained.

Diagnosis:
Investigations and serological markers have denied immunity to determine viral etiology of autoimmune or hepatic (liver negative markers for hepatic AE viruses, CMV, EBV) have refuted the possible damage leptospirotica or chlamydiae, but reported a slight increase in Complement Reaction Coxiella burnetii=1/80, raising the suspicion diagnosis of Q fever.

The second serological detection for C. burnetii (10 days from onset) revealed an apparent growing strength Complement Reaction Coxiella burnetii=1/320, which confirmed the initial diagnosis of Q fever. Then were determined and Ig M Coxiella Burnett were in a positive titer.
The patient was discharged 3 weeks after onset and the biological changes are fully remitted and radiological appearance of lung was normal. Because of high infectivity were performed serological from patient contacts. When his wife Coxiella Burnett Ig M were positive.

The case is interesting because this remember that disease in patients from urban and less typical forms (in this case lack of the characteristic radiological "gut glass". Due to low frequency who the disease is encountered in the absence of contact with animals, shapes decisively isolated liver are difficult to recognize. Also, thrombocytopenia which occurs in 25% of patients with Q fever can guide wrong diagnosis to other diseases.

CONCLUSIONS

The importance of diagnosis is high, although the disease is generally self-limiting (mortality in acute forms <1%) because of the risk of chronic, with determinations endocarditis or liver (granulomatous hepatitis), measurements are difficult to treat and have high mortality (30-60 %). Furthermore, if this signals an outbreak veterinarian diagnosed to be addressed and need contacts prevention considering the extremely high contagionisity of this pathogen.

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