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A Case Report: Q Fever with Acute Hepatitis

Elif Ertan[1], Talha Özüdoğru[1], İsmail Demir[1]

[1].Bozyaka Training and Research Hospital, Department of Internal Medicine, Izmir, Türkiye.

ABSTRACT

Q fever is a zoonotic bacterial infection caused by *Coxiella Burnetii*. The primary sources of this pathogen are small and large livestock. The causative agent is disseminated into the environment through the birth products, milk, feces, and urine of infected animals. This bacterium is highly resistant to environmental conditions. Transmission from animals to humans and/or from humans to humans occurs via inhalation. The acute disease presentation can be asymptomatic or resemble influenza-like symptoms, and it can also lead to more severe manifestations such as fever, pneumonia, and hepatitis. In cases with severe clinical courses, Q fever should be considered in the differential diagnosis, especially considering epidemiological risks. This paper discusses a case presenting with fever, jaundice, and hepatitis, subsequently diagnosed as Q fever.

Keywords: Fever, Hepatitis, Jaundice

INTRODUCTION

Q fever is a zoonotic systemic disease caused by the Gram-negative intracellular coccobacillus *Coxiella Burnetti*.[1] Q fever was first reported in 1937 by Edward Holbrook Derrick, who was investigating a febrile illness among abattoir workers in Australia.[2] Subsequently, during World War II, it manifested as atypical pneumonia in the United States, Greece, and Germany.[3] The first Q fever outbreak in Turkey was reported by Payzın in Aksaray in 1947.[4]

The main source of the pathogen is livestock, including cattle and sheep. Farmers, veterinarians, and butchers are at risk for Q fever. The pathogen spreads through inhalation; due to its resistance to environmental conditions, it can be carried up to 10 kilometers away by water and wind. After settling in the lungs, it spreads throughout the body via the bloodstream, leading to systemic illness. The incubation period is from 3 to 30 days, and depending on the bacterial load, it can present in various forms from asymptomatic to severe systemic disease.[1, 3]

Acute disease is usually mild. It can cause fever, severe retro-orbital headache, muscle pain, and a non-productive cough. Atypical pneumonia is detected in 40-50% of cases, and approximately 35% of these cases are accompanied by hepatitis.[3] In nearly all cases, the disease is self-limiting within two weeks. However, 5-8% progress to the chronic form. In chronic Q fever, symptoms persist for more than six months; prolonged fatigue and muscle pain may be observed even after full remission. Endocarditis, valvulitis, and osteomyelitis are frequently observed complications.[1, 3]

Although bacterial serology is most commonly preferred for diagnosis, the gold standard method is the polymerase chain reaction(PCR) test. The indirect immunofluorescence assay(IFA) is the best diagnostic

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Address for correspondence: *Elif Ertan, University of Health Sciences, Bozyaka Training and Research Hospital, Department of Internal Medicine, Izmir, Türkiye*
E-mail: elifertan998@gmail.com

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method in acute cases. In this test, a fourfold or greater increase in Phase 2 IgG titer (IgG>1:128) is considered significant. In chronic disease, a Phase 1 IgG titer greater than 1:800 is considered significant.

For the treatment of acute disease, 14-21 days of doxycycline therapy is sufficient. Alternatively, macrolide and quinolone antibiotherapy can be preferred. In chronic disease, it is recommended to use doxycycline and hydroxychloroquine together for at least 12 months.[1, 3] In selected cases, such as pregnant women and those with endocarditis, the treatment regimen and duration may vary.[3]

CASE REPORT

A 46-year-old male patient presented to the district state hospital with a three-day history of abdominal pain and newly onset jaundice, which began a day ago. Upon evaluation in the emergency department, his blood pressure was within normal limits, pulse was 104 bpm, subfebrile fever (37.8 ^{0}C), and fingertip oxygen saturation was measured at 97%. On physical examination, his skin and sclerae were icteric, lung auscultation sounds normal, and abdominal palpation revealed tenderness, particularly in the right upper quadrant, with voluntary guarding. No palpable lymphadenopathy or hepatosplenomegaly was found. In his complete blood count, leukocyte and hemoglobin levels were normal, but thrombocytopenia was present. Biochemical tests showed that transaminase levels were 4,5 times above the upper limit, cholestasis enzymes were normal, total bilirubin was elevated with direct bilirubin dominance. C-reactive protein was elevated, and INR was normal. Abdominal ultrasonography revealed a contracted gallbladder, normal intrahepatic and extrahepatic bile ducts, and normal liver and spleen sizes. Based on these outcomes, the patient was referred to our internal medicine clinic for further investigation and treatment, with the knowledge of our gastroenterology specialist.

Upon admission to our clinic, initial tests showed elevated transaminase levels, hyperbilirubinemia with direct bilirubin dominance, normal cholestatic enzymes, elevated C-reactive protein, increased urea and creatinine, and thrombocytopenia without any abnormalities in the erythrocyte and leucocyte series (Table 1). Additionally, cultures and serological tests for viral hepatitis were sent for the detection of an infectious focus. The patient was empirically started on intravenous (IV) ceftriaxone 2x1 g and metronidazole 3x500 mg antibiotics along with hydration support.

To screen for potential hepatobiliary pathologies, upper abdominal dynamic magnetic resonance imaging (MRI), MR cholangiopancreatography (MRCP), and portal Doppler ultrasonography (USG) were performed under the consultation of the gastroenterology clinic. These examinations revealed no signs of hepatobiliary pathology in the patient.

Although the patient presented with isolated thrombocytopenia in the complete blood count, further tests were conducted to evaluate disseminated intravascular coagulation (DIC) and hemolytic processes, even though no signs of bleeding were observed. The patient's prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen, and LDH levels were normal, and the peripheral blood smear showed no evidence of DIC or hemolysis. The peripheral blood smear confirmed true thrombocytopenia (Plt:15.000-20.000/mcL) with normochromic normocytic erythrocytes. The white blood cell formula is consistent with normal findings, and no atypical cells are observed. Based on these tests, the thrombocytopenia was primarily considered secondary to infection.

No growth was detected in the cultures sent during the patient's hospitalization. The viral hepatitis serology was unremarkable, except for past hepatitis A infection. Additionally, tests for Toxoplasmosis, Rubella, Cytomegalovirus (CMV), Herpes virüs(HSV), Brucella, Epstein-Barr virüs(EBV), and Syphilis were also conducted, but they all came negative. As the patient showed a decreasing trend under IV ceftriaxone and metronidazole, we had a further consultation with the Infectious Disease. Samples sent to the Public Health Laboratory to test other potential infectious agents, including West Nile virus, Leptospira, Hantavirus, Coxiella, and Crimean-Congo Hemorrhagic Fever virus. In addition, doxycycline 2x100 mg was added to the patient's treatment regimen to cover up these pathogens.

On the ninth day of hospitalization, during the ongoing treatment, follow-up tests showed regression in transaminases and hyperbilirubinemia, decreased infectious parameters, resolution of acute kidney injury, and normal platelet levels (Table 1). Significant improvement was also observed in his symptoms and clinical findings. While treatment was still in progress, the patient chose to leave the hospital against medical advice. He was prescribed doxycycline tablets 100 mg twice daily as a continued treatment on an outpatient basis.

After the patient left the hospital, test results from

Table 1. Follow-up tests of the patient

Test	Day 1	Day 9
AST	138 mg/dL	23 mg/dL
ALT	92 mg/dL	43 mg/dL
Total bilirubin	24 mg/dL	7 mg/dL
Direct bilirubin	19 mg/dL	2,45 mg/dL
ALP	79 U/L	43 U/L
GGT	30 U/L	28 U/L
INR	1,05	1,01
Urea	80,3 mg/dL	16,4 mg/dL
Creatinine	1,46 mg/dL	0,95 mg/dL
CRP	128 mg/L	5 mg/L
WBC	8.370/µL	4.170/µL
PLT	21.000/µL	235.000/µL
Hb	12,4 g/dL	12,6 g/dL

AST: aspartate aminotransferase, ALT: alanine aminotransferase, ALP: alkaline phosphatase, GGT: gamma glutamyl transferase, INR: international normalization rate, CRP: c-reactive protein, WBC: white blood cell count, PLT: platelet count, Hb: Hemoglobin

the Public Health Laboratory were received. Results showed that the patient's *Coxiella Burnetti* Phase 2 IgG was positive at a titer of 1:512. It was concluded that the patient's current clinical presentation was consistent with Q fever.

DISCUSSION

Acute Q fever can range from being asymptomatic to presenting with a severe clinical course involving multiple organ involvement.[4,5] In our patient, the disease manifested with subfebrile fever accompanied by jaundice, elevated transaminases, acute kidney injury, and thrombocytopenia. Pneumonia, which is commonly observed in Q fever, was not present in our patients. The prominent finding of severe jaundice at the time of presentation initially led to consideration of common hepatitis causes and cholestatic diseases in differential diagnosis. The exclusion of these primary diagnoses through initial testing prompted further investigation into less common etiologies and the initiation of empirical treatment targeting these.

Indeed, the patient, who responded dramatically to empirical ceftriaxone and doxycycline therapy, was later confirmed to have Q fever through subsequent testing.

Given that Q fever is a zoonotic disease, it is essential to consider Q fever in the differential diagnosis of patients presenting to hospitals with clinical pictures such as acute hepatitis, pneumonia, kidney injury and heart failure, especially if these patients have a history of living in rural areas, farming or livestock handling and have elevated acute phase reactants. Considering the potential delay in diagnosis and the possibility of severe clinical outcomes, such as liver failure and endocarditis, which could lead to higher mortality if left untreated or if the diagnosis is delayed, empirical antibiotherapy should be considered in these cases.

CONCLUSION

Caffeine, which is also the main ingredient of energy drinks, can cause syndromes such as addiction and withdrawal. Hence, we advise that daily energy drink intake should not be above the caffeine safety limitations defined by regulatory authorities, and should even be lowered based on the information available in the literature. Furthermore, additional systematic investigations are necessary to clarify the long-term effects of energy drink intake on human health.

Author Contributions:

All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version. Ali Can Memiş is the article guarantor.

Conflict of Interest

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Ethical Statement

In accordance with ethical standards, all patient information was anonymized, and no formal ethics approval was necessary.

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