Acute Q Fever Pneumonia

Amit Panjwani¹, Shashikala Shivaprakasha², Dilip Karnad³

Abstract

Q fever is a zoonotic disease caused by Coxiella burnetii which has a worldwide distribution. Pneumonia occurs in almost half of the patients who have an acute C. burnetii infection. Less than 5-6% of community acquired pneumonia (CAP) is found to be caused by this organism. Endemicity of C. burnetii infection has been recorded in various studies carried out in our country. However there is no mention about Q fever as a cause of CAP in the various studies done to identify the aetiological agent. We report a case of acute Q fever related pneumonia and this appears to be the first reported case of pneumonia due to C. burnetii infection in India.

Introduction

Q fever is a zoonotic infection caused by Coxiella burnetii. It has a worldwide distribution with the exception of New Zealand, Antarctica and some parts of Europe,¹ and presents as self limiting mild febrile illness, pneumonia or hepatitis.² Although C. burnetii infection is endemic among farm animals in India and human infections with C. burnetii has been documented in some epidemiological seroprevalence studies, we found no reports of acute Q fever from India.

Case Report

A 35 year old male painter developed fever with chills, rhinorrhea, headache, malaise and nausea after returning from his hometown in northern India. Over the next 10 days he developed shortness of breath and dry cough. There was no chest pain, wheeze, haemoptysis, abdominal pain or loose motions. On examination, he had fever (100.2°F), tachycardia, BP of 100/50 mm Hg and respiratory rate of 32 breaths/min with SpO₂ of 92% on room air. He also had an erythematous rash over the face, neck and the chest, suffused eyes, crackles in both infrascapular areas in the chest and mild hepatosplenomegaly. Other systemic examination was unremarkable.

Investigations showed hemoglobin of 11.2 g%, leucocyte count of 8150/ mm³, platelet count of 18000/ mm³. Serum bilirubin was normal with mild elevation of liver enzymes (AST-168 U/L (range: 8-61), ALT- 83 U/L (range: 0-41 U/L), alkaline phosphatase – 203 U/L (range: 40-130 U/L)), and renal function and electrolytes were normal; CRP was 10.95 mg% (range: 0.015- 2.0 mg%). Urine examination showed albuminuria (1 +), 6-8 WBCs/hpf and 15-20 RBCs/hpf. Chest radiograph showed reticular opacities in all zones bilaterally with alveolar opacities in both mid zones (Figure 1). Malaria antigen, Widal test and HIV were negative but dengue IgM antibody test was positive. He was treated with intravenous fluids, antipyretics, intravenous ceftriaxone and levofloxacin with a provisional diagnosis of atypical pneumonia, and required non-invasive ventilation for 4 days. Intravenous artesunate too was started empirically as he had mild anemia and significant thrombocytopenia.

He continued to remain febrile (upto 103 F), tachypneic but normotensive even after a week of therapy with persisting dry cough, bodyache and dyspnea. Platelet count too remained low and varied between 18,000 and 36,000/mm³. HRCT Thorax was performed, which revealed ground-glass opacities and interlobular septal thickening in both the lungs. Consolidation was seen in both upper lobes without significant mediastinal lymphadenopathy (Figures 2 and 3). Echocardiography was normal and pulmonary function test showed a mild restrictive defect with severely reduced single breath diffusion capacity for carbon monoxide. Blood culture was sterile and serum procalcitonin levels were normal.
were normal. In view of the clinical triad of persisting fever, hepatitis and atypical pneumonia in the background of significant thrombocytopenia a possibility of rickettsial infection, particularly acute Q fever was entertained and blood sample for Coxiella burnetii real time PCR assay was positive. Oral doxycycline was started and there was a remarkable response. The fever completely subsided and respiratory distress resolved in two days. Platelet count became normal and chest radiograph showed regression of infiltrates in both the lung fields over the next 5 days (Figure 4). Doxycycline was continued for 2 weeks. On retrospective questioning, the patient mentioned that he had goats in his household in the village and that he had witnessed a delivery of one of the goats during his recent stay there.

**Discussion**

Although uncommon, Q fever related pneumonia could mimic several other illnesses like atypical pneumonia, dengue, malaria or typhoid, and the diagnosis may be missed initially, as in our patient. However, the acute febrile illness improves dramatically accompanied by extra-respiratory symptoms like fever, headache and myalgia. Hepatosplenomegaly may occasionally occur and was seen in our case. The leucocyte count is usually normal while thrombocytopenia occurs in 25% of cases. Hepatic transaminases may be slightly raised with elevated alkaline phosphatase seen in almost 70% of patients. Almost all these features were present in our patient. Chest radiography commonly shows lower lobe predominance of opacities in segmental or subsegmental distribution unilaterally or bilaterally.

Isolation of Coxiella burnetii is difficult and hazardous. The indirect immunofluorescence assay is the most dependable and widely used serologic method for diagnosis. Recently PCR has become an important tool for detection of C. burnetii in biological samples and can be performed on serum, urine, genital swabs and other biological specimens. PCR assays (real-time and trans-PCR) have a good sensitivity (85%) and specificity (99.5%) in the detection of C. burnetii infection.

Doxycycline 200 mg daily for 14 days is the treatment of choice for acute Q fever and macrolides, fluoroquinolones, co-trimoxazole and rifampicin are other alternative drugs.

**Conclusion**

A diagnosis of C. burnetii infection should be considered in patients from rural areas with fever, thrombocytopenia, elevated liver enzymes and bilateral lung infiltrates. Although contact with parturient animals is common in rural India, acute Q fever has surprisingly not been reported before. This may be because fever and thrombocytopenia may often be attributed to malaria or other tropical diseases like scrub typhus and treated empirically with doxycycline.

**References**