

CASE REPORT

An unusual presentation of acute Q fever

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Learning points for clinicians

Acute Q fever is a common zoonotic infection with a worldwide distribution. Even rare manifestations may be encountered and need to be recognized.

Barring parasitic infestations, peripheral blood eosinophilia in the course of infectious diseases is distinctly uncommon. Acute Q fever may very rarely present with significant peripheral blood or pleural fluid eosinophilia.

Acute Q fever may cause granulomatous lymphadenitis with mediastinal lymphadenopathy which may mimic lymphoma or sarcoidosis.

Oral doxycycline constitutes an effective treatment and these rare manifestations are entirely reversible.

A healthy 68-year-old man presented with fever (38.3°C), dyspnea, dry cough and 5 kg weight loss over 10 days. On admission right lung rales and splenomegaly were found, without lymphadenopathy (LN). Chest X-ray showed right middle lobe (RML) infiltrate, small pleural effusion and bilateral hilar LN. Laboratory tests showed: Hb 12 g/dl, platelets 186 x 10^9/l, WBC 12 x 10^9/l with 4.6 x 10^9/l neutrophils and 5.1 x 10^9/l (42%) eosinophils. C-reactive protein (CRP) 154 mg/l (N < 6). Serum albumin 31 g/l, globulins 41, globulins 41 g/l - polyclonal increase; transaminases 160–170 IU/l; Lactate dehydrogenase 1030 IU/l; alkaline phosphatase 240 IU/l and gamma-glutamyl transpeptidase 170 IU/l. Serum glucose, electrolytes, urinalysis, renal function, coagulation tests and autoantibody screen were normal. Cultures were negative. Pneumococcal/ legionella antigens were not found and mycoplasma/chlamydia serology was negative. Serology for fungi, HIV, Cytomegalovirus, Epstein-Barr virus, treponema, bartonella, brucella and rickettsiae was also negative. Immunofluorescence for Q fever phase II IgG was 1:64 and for IgM >1:256. Chest CT revealed significant mediastinal and hilar LN (Figure 1a, b). Mediastinoscopy and LN biopsy ruled out lymphoma demonstrating extensive granulomatous reaction without necrosis or Mycobacterium tuberculosis (M. TB). Unlike sarcoidosis, serum angiotensin converting enzyme levels were normal and he was not anergic (purified protein derivative 6 mm). Repeated serology confirmed acute Q fever (phase II IgG >1:1024). The patient was an avid cat feeder. On 3 weeks doxycycline (100 mg, bd), he became afebrile, asymptomatic with normal examination and tests (0.3 x 10^9/l eosinophils, CRP 10 mg/dl, normal liver enzymes). Chest CT showed a substantial decrease of LN. Follow-up over 5 years was uneventful.

Q fever is a worldwide zoonotic infection caused by the strict intracellular bacterium coxiella burnetii. Acute disease typically presents as pneumonia and asymptomatic hepatitis in a febrile patient who may have a rash.1 Our patient’s presentation was compatible but the significant eosinophilia and thoracic LN were distinctly unusual and suggested other diagnoses.

When the most common causes of eosinophilia worldwide - parasitic infestations and atopic/allergic disorders are excluded, eosinophilia in a hospitalized patient is mostly reactive (polyclonal) and can be a useful diagnostic clue. Important associations include neoplastic (mostly hematologic) or autoimmune diseases (especially vasculitis) but also adrenal insufficiency, sarcoidosis or inflammatory bowel disease. Infections are almost always associated with conspicuous eosinopenia.2 Very few infectious agents may cause eosinophilia, predominantly fungi, retroviruses and possibly M. TB. C. burnetii was involved in four cases of eosinophilic pleural effusion3 but peripheral blood eosinophilia was not reported. Possibly thoracic infection led to the local production of eotaxins,3 chemokines responsible for eosinophil recruitment.

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Atypical clinical manifestations of acute Q fever include isolated fever and rarely perimyocarditis, neurological or hematological involvement. Granulomas have been detected in the liver (‘granulomatous hepatitis’) and bone marrow, as well as LN of rare patients who had granulomatous lymphadenitis. Thus, reticuloendothelial system involvement may be detected clinically (e.g. splenomegaly), by laboratory testing (e.g. hepatitis) and histologically (e.g. granulomas). Although unmentioned in a recent review of granulomatous lymphadenitis, acute Q fever should be recognized as an additional established cause. Of 10 patients reported, 4 had peripheral granulomatous lymphadenitis (cervical, axillary, inguinal); 3 had internal lymphadenitis (mediastinal, abdominal); and 3 had both. The prevalence of lymphadenitis in acute Q fever had been estimated at 0.7% and 5/10 (50.0%) had mediastinal LN with granulomas. Typical ‘doughnut’ granulomas may be seen. These poorly recognized manifestations of acute Q fever were often interpreted as lymphoma, sarcoidosis or cat scratch disease. However, acute Q fever was well-established in all cases by any combination of serology, polymerase chain reaction of LN tissue (two cases) and response to tetracycline. In our patient, serology, treatment response and the uneventful follow-up were diagnostic. Thus, our report stresses eosinophilia and mediastinal granulomatous lymphadenitis as important to recognize, albeit rare, manifestations of acute Q fever.

Conflict of interest: None declared.

References