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An unusual presentation of a known condition: Q fever manifesting as an acute abdomen

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ABSTRACT

Q fever is an endemic zoonotic infection in Australia caused by *Coxiella burnetii*. It has been recognised in other parts of the world, especially among livestock rearing occupations, stock yard and abattoir workers. Majority (65%) of patients infected with *C. burnetii* are asymptomatic while symptoms similar to those of respiratory and hepatitis are the most common making diagnosis difficult in the early stages.

We report a case of a young man who was exposed to and infected with Q fever as an occupational hazard. He presented in an unusual way with the predominant initial symptoms of abdominal pain, fever, hepatitis and sterile peritonitis necessitating an emergency surgical procedure to explore a suspected surgical abdomen. Respiratory involvement ensued only several days later. The diagnosis of Q fever was confirmed with positive convalescent serology phase II IgM and IgG antibodies to *Coxiella burnetii*. A marked clinical response to doxycycline pending serological confirmation was supportive of this highly suspected diagnosis in an at-risk patient.

Keywords: Q fever, acute abdomen

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Description

A previously healthy 23-year-old male presented with a one-week history of fevers, lethargy, nausea, headaches and progressively severe abdominal pain. He denied any past medical history or regular medications, and was physically active, working full time in the transportation of ruminant animals in Queensland, in the North-Eastern part of Australia. He was a smoker of ten cigarettes a day for 8 years prior. Of relevance he denied any arthralgias, myalgias, rashes, respiratory or urinary symptoms. He had not travelled overseas recently and did not identify any known infectious exposures, however reported that a colleague at work had recently been hospitalised with a febrile illness. He had come in contact with this colleague two weeks prior when they both cleaned a truck used to transport goats, using a high pressure water hose. This pertinently demonstrated a shared exposure to aerosolized mud particles contaminated by goat faeces.

On examination, he was febrile to a temperature of 38.7°C but otherwise hemodynamically stable. Abdominal examination revealed a very tender epigastrium and right upper quadrant, with hepatosplenomegaly. Investigations demonstrated an elevated C-reactive protein of 250 mg/L, leucopenia with a white cell count of 2.8/nL, and thrombocytopenia with a platelet count of 69/nL. There were no other clinical or biochemical features to suggest disseminated intravascular coagulation or microangiopathic hemolytic anemia. Liver function tests revealed a transaminitis with an alanine aminotransferase (ALT) level of 333 IU/L, and impaired synthetic function with hypoalbuminaemia (albumin 24 g/L) and reduced total protein (51 g/L). Hyperferritinaemia (1229 ug/L) was also present. A computed tomography (CT) scan of his abdomen demonstrated hepatosplenomegaly with periportal oedema, free fluid within the peritoneal cavity, and fluid around the gallbladder with mild wall thickening, suggestive of hepatobiliary sepsis.

He was commenced on ceftriaxone and metronidazole, and referred to the surgical team. Given the clinical examination concerning for an acute abdomen, he subsequently underwent an exploratory laparoscopy which revealed no obvious viscus perforation. Microscopic examination of the peritoneal fluid showed a lymphocytosis, however no organism was isolated on microbiological culture.

On the second day of his admission he was transferred to the medical team for further management. At this stage the patient reported development of a dry cough and left-sided pleuritic chest pain suggestive of a respiratory tract infection. A chest X-ray showed left lower infiltrates and a small pleural effusion (figure1). The differential diagnoses at this stage were Q fever and melioidosis considering the geographical part of Australia where he resided, and the nature of his occupational exposures. He continued to have febrile episodes, so multiple blood cultures were sent, and Q fever serology was requested. His antibiotics were then changed to meropenem and doxycycline. Serological testing was also performed for hepatitis, HIV, leptospirosis, Epstein-Barr virus, Cytomegalovirus, toxoplasmosis and Brucella. Normal peripheral blood flow cytometry excluded lymphoma, and a normal echocardiogram revealed no features suspicious for infective endocarditis. Three sets of blood cultures returned no growth, and the other aforementioned tests returned negative. The patient improved significantly within 72 hrs of the change in antibiotics, which were then continued for one week. Meropenem was ceased at this point as the negative blood cultures and peritoneal fluid cultures excluded the presence of *Burkholderia pseudomallei*, the cause of melioidosis. Doxycycline was continued and a convalescent Q fever serology was requested. Marked clinical improvement was seen on doxycycline, enabling the patient to be discharged home and followed up in the outpatient clinic. The convalescent Q fever serology returned positive for phase II IgG and

IgM antibodies to *Coxiella burnetii*. The patient was treated for 3 weeks in total with doxycycline and on review in the outpatients clinic two weeks after discharge he was clinically well, with a normal repeat chest X-ray. The plan following this was for a further review at the 12 week mark with a repeat echocardiogram to assess for endocarditis or valvular damage.

Discussion

We report a case of a young man who was exposed to and infected with Q fever as an occupational hazard. He presented in an unusual way with the predominant initial symptoms of abdominal pain, fever, hepatitis and sterile peritonitis necessitating an emergency surgical procedure to explore a suspected surgical abdomen. Respiratory involvement ensued only several days later. The diagnosis of Q fever was confirmed with positive convalescent serology phase II IgM and IgG antibodies to *Coxiella burnetii*. A marked clinical response to doxycycline pending serological confirmation was supportive of this highly suspected diagnosis in an at-risk patient.

Q fever is an endemic zoonotic infection in Australia cause by *Coxiella burnetii*. It has been recognised in other parts of the world, especially among livestock rearing occupations, stock yard and abattoir workers [1-3]. *Coxiella burnetii* can be harboured in most herd and domestic animals including cattle, goats, sheep and dogs, Macropods such as kangaroos and wombats, and also in ticks that are associated with some of these animals [2,3]. *Coxiella burnetii* is particularly concentrated in the placenta, so although it is shed into bodily secretions including faeces, most human infections occur through contact with the placenta of infected animals [2,3]. Transmission can occur through inhalation of aerosolised particles contaminated by the secretions from infected animals, and ingestion of contaminated poultry or uncooked raw eggs [3,4]. The suspected mode of transmission in the above case was inhalation of aerosolised infected goat faeces when cleaning the contaminated truck with a high power jet

water hose. Most patients infected by *Coxiella burnetii* are asymptomatic [5], as reported by Bacci et al in their study where 64% infected individuals were asymptomatic, while only 31% experienced symptoms compatible with existing descriptions of Q fever. Acute *Coxiella burnetii* infection is typically mild and self limiting within two weeks. Even when present, the clinical manifestation of Q fever are nonspecific with no pathognomonic symptoms or signs, but with features seen in respiratory disease and hepatitis from other causes, thereby rendering the diagnosis a difficult one to convincingly arrive at clinically [1,5,6]. Our reported case demonstrated both respiratory and abdominal symptoms, but the abdominal pain predominated in the initial stage of his illness, resulting in an invasive investigative procedure indicated by the query of possible peritonitis.

The common acute symptoms of Q fever are asthenia, myalgia, fever, rigors, chills, cough, arthralgia, extreme fatigue, drenching sweats, weight loss and headache, in conjunction with abnormal liver function tests [1,3,5,6]. Deranged liver function due to hepatitis is a very common finding in Q fever, which we also observed in our case. Graves and Islam [3] performed a study in patients with Q fever and reported abnormal liver function in 65%, respiratory symptoms in 37%, and both hepatitis and respiratory symptoms in 23%.

Another possible presentation of acute or chronic Q fever is culture negative endocarditis. Jang et al [7] report a positive PCR for *Coxiella burnetii* in 41% of patients with culture negative endocarditis. Thus an important part of the diagnostic work up when Q fever is suspected or diagnosed is at least a transthoracic echocardiogram. Echocardiography during the acute stage in our patient revealed no features suggestive of endocarditis, however we intend to pursue a follow up echocardiogram to ensure this is a complication that does not develop.

The diagnosis of acute Q fever is made by a positive culture or PCR demonstrating *Coxiella burnetii* in blood or other bodily secretions.

Alternatively, as in our patient, the diagnosis is confirmed with positive serology, with positive phase II IgM or IgG titres over 100. Chronic Q fever is diagnosed with a single positive phase I IgG titre > 100, phase II Ig A or phase I IgA in the absence of IgM [3,8]. Symptomatic acute Q fever is treated with doxycycline, or if not tolerated, a quinolone, macrolide or trimethoprim-sulfamethoxazole for 2 to 3 weeks [8,9].

This case serves as a reminder of the variable way in which this treatable infection can present.

References

1. Eastwood K, Massey PD, Hutchinson P, et al. Q fever: A rural disease with potential urban consequences. *Aust J Gen Pract.* 2018;47(3):5555
2. Goyette M, Bouchard J, Poirier A, et al. Importance of Q fever in community acquired pneumo-nia. *Can J Infect Dis.* 1996; 7(6): 370–373.
3. Graves SR, Islam A. Endemic Q fever in New South Wales, Australia: a case series (2005-2013). *Am J Trop Med Hyg.* 2016;95:55–9.
4. Raoult D, Marrie T. Q fever. *Clin Infect Dis.* 1995;20(3):489-495.
5. Bacci S, Villumsen S, Valentiner-Branth P, et al. Epidemiology and clinical features of human infection with *Coxiella burnetii* in Denmark during 2006–07. *Zoonoses Public Health.* 2012; 59(1), 61–68.
6. Bernit E, Pouget J, Janbon F et al. Neurological involvement in acute Q fever: a report of 29 cases and review of the literature. *Arch Intern Med.* 2002;162(6):693.
7. Jang YR, Song JS, Jin CE, et al. Molecular detection of *Coxiella burnetii* in heart valve tissue from patients with culture-negative infective endocarditis. *Medicine (Baltimore).* 2018;97(34):e11881.
8. Anderson A, Bijlmer H, Fournier PE, et al. Diagnosis and management of Q fever - United States, 2013: Recommendations from CDC and the Q Fever Working Group. *MMWR Recomm Rep.* 2013;62:1-23.
9. Hartzell JD, Wood-Morris RN, Martinez LJ, Trotta RF. 2008. Q fever: epidemiology, diagnosis, and treatment. *Mayo Clin. Proc.* 83:574–579. 10.4065/83.5.574.
10. Lindsay PJ, Rohaila S, Miyakis S. Q Fever in Rural Australia: Education Versus Vaccination. *Vector Borne Zoonotic Dis.* 2018 Nov;18(11):632-634.

It is important to consider the diagnosis in an at risk patient as it will enable focused investigations, timely management and potential prevention of further spread of infection. In view of the recent termination of Q fever vaccination in Australia, one of the few countries that had a Q fever vaccination program, it is imperative that health care providers maintain an awareness of this diagnosis as a differential, and contribute to public health education on recognition and management of Q fever [8,10].

