CASE REPORT

Two rare manifestations of Q fever: splenic and hepatic abscesses and cerebral venous thrombosis, with literature review *ma non troppo*Manuel Mendes Gomes,¹ Andreia Chaves,¹ Ana Gouveia,³ Lèlita Santos¹

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SUMMARY

Q fever is a zoonosis caused by *Coxiella burnetii*. It often manifests as a flu-like syndrome; other common manifestations are pneumonia, hepatitis and endocarditis. Its course may be acute or chronic. The authors present two clinical cases of Q fever with rare manifestations. Case 1: A 55-year-old man admitted due to abdominal pain, diarrhoea and fever. Blood tests showed elevated transaminases, low platelets and elevated C reactive protein, with normal white cell counts; abdominal ultrasound showed splenic and hepatic abscesses. Serologies to *C burnetii* were positive (1:640), leading to the diagnosis of Q fever with splenic and hepatic abscesses. Case 2: A 47-year-old man admitted due to headache after sneezing, with unstable gait and vertigo. A brain tomography showed cerebral venous thrombosis. After an exhaustive investigation, antibodies to *C burnetii* were found and were undoubtedly positive (1:5120), leading to the diagnosis of Q fever. Both patients were treated with oral doxycycline.

BACKGROUND

Q fever is a zoonosis caused by *Coxiella burnetii*, which can cause acute or chronic infection, with many different manifestations affecting various organs.¹ Despite that, abscesses, namely hepatic and splenic ones, are very unusual in this setting.^{2,3}

Neurological manifestations of Q fever are rare (less than 1% of all cases).¹ Cerebral venous thrombosis (CVT), which is a thrombosis of the venous channels in the brain and an uncommon cause of cerebral infarction,^{4,5} was not included, as far as we know, on the list of possible neurological manifestations of Q fever.

CASE PRESENTATION**Case 1**

A 55-year-old man presented to our casualty department (CD) with 2 days of severe epigastric pain, without nausea or vomiting, diarrhoea or other symptoms; on physical examination, he was febrile (37.8°C) and haemodynamically stable, his abdominal palpation was painful, especially in the epigastrium, and he had hepatomegaly (2 cm below the costal margin). Blood tests showed $5.2 \times 10^9/L$ white cell count, platelets $122 \times 10^9/L$, C reactive protein (CRP) 3.33 mg/dL, sodium 130 mmol/L, aspartate aminotransferase (AST) 40 U/L, alanine transaminase (ALT) 45 U/L, γ -glutamyltransferase (γ GT) 136 U/L, alkaline phosphatase (AP) 64 U/L,

total bilirubin 1.3 U/L and serum and urine amylases were within normal ranges. Chest X-ray and 12-lead ECG were normal; abdominal ultrasound (US) showed liver steatosis only. After metemazole magnesium, he improved substantially and was discharged with request for further internal medicine outpatient clinic appointment.

After 3 days, he returned to CD with continuous, diffuse and very severe abdominal pain, diarrhoea (4–5 liquid stools/day, without pus, mucus or blood), vomiting and high fever (maximum 39.5°C). He denied weight loss, anorexia, rashes, tick bites or any other symptoms.

The patient's medical history included arterial hypertension (controlled with ACE-inhibitor), heavy alcohol consumption (about 100 g/day) and peptic ulcer many years ago. He lived in the countryside, had contact with chickens and denied any consumption of unpasteurised food or non-potable water, contact with patients with infectious diseases or any new medication. He had no recent trips abroad, namely to countries with endemic infectious diseases.

On physical examination, his blood pressure was 150/90 mm Hg, heart rate was 83 bpm and temperature was 39.4°C; he was eupneic, without jaundice, skin rashes or tick bites, but was dehydrated. Heart sounds were normal; pulmonary auscultation showed right basal crackles. His abdomen was tender, diffusely painful, but mostly in the right lower quadrant, with no masses but with painful hepatomegaly (3–4 cm below the costal margin). No adenopathies were found.

Blood tests were again performed, which showed the following results: CRP 16.62 mg/dL, white cell count $8.3 \times 10^9/L$, platelets $135 \times 10^9/L$, sodium 131 mmol/L, AST and ALT 47 U/L, AP 74 and γ GT 171 U/L. Chest X-ray showed a mild right pleural effusion but no images suggesting pneumonia (figure 1), and abdominal US showed hepatic steatosis. Hence, abdominal CT was performed, which showed a small right pleural effusion, an unremarkable liver and spleen with small infracentimetric hypodense nodules (figures 2–4).

The patient was started on ciprofloxacin and admitted to an internal medicine ward in order to be monitored, treated and to continue investigations. Further tests were performed in order to discard other infectious foci and possible causes to diarrhoea and splenic nodules. Blood cultures were negative, prostate-specific antigen was 1.1 ng/mL and immunoelectrophoresis showed a doubtful γ -monoclonal peak; echocardiogram was unremarkable.

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Figure 1 Case 1: chest X-ray, performed at the casualty department showing mild right pleural effusion.

On day 3 after admission, new abdominal US was performed to monitor spleen nodules, which showed not only the same splenic nodules but also small abscesses/inflammatory pseudotumors located at the right hepatic lobe; local adenopathies were present as well. At this point, the patient still had residual pain and fever, but had improved from diarrhoea. Viral, bacterial and parasitological serologies, along with stool cultures, were performed (table 1), highlighting positive results for *C burnetii*. Thus, diagnosis of Q fever with splenic and hepatic abscesses, pleural effusion and diarrhoea was reached. The patient was started on doxycycline 100 mg twice daily, with substantial subsequent improvement. Eleven days after admission (and 5 days after initiating doxycycline), he underwent a repeated abdominal CT, which showed the absence of hepatic nodules and reduction of splenic masses (figures 5–7). At day 15 after admission, he was discharged to internal medicine outpatient clinic and remained asymptomatic. After a few months, US was again performed, which was unremarkable.

Case 2

A 47-year-old man presented to our CD with frontal, pulsatile headache (of moderate intensity: 7/10 in the visual analogue scale) with vertigo and unstable gait. Symptoms had started 3 days earlier, following one sneeze. He could not identify any other precipitant, relieving or aggravating factors. He also had nausea but no vomiting. He denied any other symptoms. His medical history included community-acquired pneumonia 6 months ago and mild smoking habit. He had no usual medication. He lived in the countryside and had contact with dogs, chickens and other farm animals. He denied any consumption

Figure 2 Case 1: abdominal CT, performed at casualty department showing splenic infracentimetric hypodense nodules.

Figure 3 Case 1: abdominal CT, performed at casualty department showing splenic infracentimetric hypodense nodules.

of unpasteurised food or non-potable water, contact with patients with infectious diseases or recent trips abroad.

On physical examination, he was haemodynamically stable and afebrile; neurological examination showed rotatory nystagmus and careful gait because of vertigo, without any other remarkable findings.

Brain CT was performed, showing spontaneously hyperdense superior sagittal sinus and torcular filling defect (figure 8). In order to clarify these changes, brain CT venography was performed—it confirmed venous thrombosis of left longitudinal sinus and transverse sinus (figures 9 and 10). The patient was then admitted to a neurology ward to start treatment and to be submitted to aetiological investigation. He was started on enoxaparin 1 mg/kg twice daily; several blood tests were performed (table 2), since one isolated sternutation, without any history of trauma, was insufficient to justify CVT. Among his blood tests, we highlight positive antinuclear antibodies (1:640), antivimentine antibodies (1:640), negative antiphospholipid antibodies and mildly positive antibodies to *Rickettsia conorii* (1:160). He was discharged 14 days after admission, on warfarin, with request for a neurology outpatient clinic appointment for re-evaluation and an internal medicine—systemic autoimmune diseases appointment for further investigations.

After careful analysis of all the blood tests performed until then and clarification of symptoms and epidemiological context, a few more blood tests were performed at internal medicine outpatient clinic appointment (table 3): JAK2 mutations and antineutrophil cytoplasmic antibodies were negative; immunoelectrophoresis was normal. *R conorii* serology was repeated and decreased (now 1:80) but serology for *C burnetii* was also performed and results were

Figure 4 Case 1: abdominal CT, performed at casualty department showing splenic infracentimetric hypodense nodules.

Table 1 Some of the laboratory tests requested for case 1**Case 1: Laboratory tests obtained during admission in internal medicine ward**

Urine and stool parasitological examination	Negative	Blood, urine and stool cultures	Negative
Serology for <i>Giardia lamblia</i> , <i>Entamoeba histolytica</i> and <i>Fasciola hepatica</i>	Negative	<i>Rickettsia conorii</i>	1:320
IDR	Anergy	<i>Coxiella burnetii</i>	1:640
Rose Bengal test for Brucellosis	Negative	Phase II IgG for <i>C. burnetii</i>	400
<i>Leishmania</i> serology	Negative	Phase II IgM for <i>C. burnetii</i>	50

Phase antigens for *C. burnetii* were requested posteriorly and to an external laboratory, because our hospital's laboratory is not able to identify specifically the phase antigens.
IDR, PPD intradermorreaction.

positive, with a titre of 1:5120, confirming Q fever as the cause of the CVT. Then, the patient was started on doxycycline, which he was due to continue for at least 18 months. Echocardiogram and abdominal US were performed, which were unremarkable, and MR venography was performed 2 and 12 months after the event, to re-evaluate the changes (figures 11 and 12). The patient remained asymptomatic.

DIFFERENTIAL DIAGNOSIS

Case 1

Our patient presented with diarrhoea, vomiting, abdominal pain and fever; he also had splenic and hepatic abscesses and mild pleural effusion. Splenic abscesses are very rare; its cause may be *Salmonella typhi* and *Salmonella paratyphi* infections, pneumococcal bacteraemia, malaria, infective endocarditis or other infections with the potential to cause haematogenous spread or systemic embolisation.^{3 6} Other bacteria, such as *Escherichia coli*, *Yersinia* spp or *Campylobacter* spp can cause infections associated with splenic abscesses. Hepatic abscesses/inflammatory pseudotumors are very rare but can occur again in the setting of infections with the potential to cause haematogenous spread or systemic embolisation.^{7–10} Regarding our patient, most of these infections were discarded. As discussed later, one previous report of splenic abscesses associated with Q fever has been made²; however, we did not find any report of *Coxiella* infection with hepatic abscesses. The combination of all these

Figure 5 Case 1: control abdominal CT, performed during admission at internal medicine ward, 5 days after initiating doxycycline showing the absence of hepatic nodules and reduction of splenic masses.

Figure 6 Case 1: control abdominal CT, performed during admission at internal medicine ward, 5 days after initiating doxycycline showing the absence of hepatic nodules and reduction of splenic masses.

signs and symptoms, in the setting of a patient with suggestive epidemiological context, fits well in a zoonosis scenario.

Case 2

The patient's differential diagnosis includes all the possible causes of CVT. Since it is a rare condition, less common causes must be discarded from the beginning as well. Moreover, association between different causes is also a possibility.^{4 5 11} Major trauma, sinusitis, medications associated to CVT and most of the hypercoagulable states were discarded from the beginning. Trivial trauma, as sneezing, was present in this case and its role as the cause of CVT has been studied^{11 12}; evidences show that it may act as a trigger, but usually in the presence of other pro-thrombotic factors.¹¹ In our patient, the sneeze was immediately followed by mild symptoms of headache and vertigo; so, it may have acted indeed as the trigger for a CVT in a setting of an endothelitis caused by *Coxiella* infection, as discussed later.

TREATMENT

Both patients were treated with doxycycline 100 mg twice daily. Regarding case 1, duration of treatment was approximately 21 days. In case 2, because this was a very uncommon manifestation of Q fever and antibodies titre was excessively high, making it less clear whether this was an acute or chronic infection, we decided to extend treatment and monitor these serologies, thus keeping treatment for 18 months. Subject will be discussed later.

Figure 7 Case 1: control abdominal CT, performed during admission at internal medicine ward, 5 days after initiating doxycycline showing the absence of hepatic nodules and reduction of splenic masses.

Figure 8 Case 2: Brain CT performed at casualty department showing spontaneously hyperdense superior sagittal sinus and torcular filling defect.

OUTCOME AND FOLLOW-UP

In case 1, the patient improved of his major symptoms after 21 days of treatment. Despite that, fatigue lasted longer, over a few months, with progressive improvement. We continued to monitor serologies, which kept low. He continued with regular follow-up at internal medicine outpatient clinic and is, so far, asymptomatic.

In case 2, the patient was followed up at internal medicine outpatient clinic, while continuing his treatment with warfarin and doxycycline. Anticoagulation was stopped after 12 months, because his repeated brain MRI showed complete permeability of the venous sinus. Echocardiogram was also repeated, and was, once more, unremarkable. We decided to maintain treatment with doxycycline for at least 18 months and monitor serologies, because initial values were extremely high and such manifestation was of uncertain behaviour. Associating hydroxychloroquine was a possibility, since doxycycline/hydroxychloroquine regimen is usually used in chronic Q fever endocarditis; despite that, we

Figure 9 Case 2: Brain CT venography performed at casualty department to confirm changes identified in brain CT showing venous thrombosis of left longitudinal sinus and transverse sinus.

Figure 10 Case 2: Brain CT venography performed at casualty department to confirm changes identified in brain CT showing venous thrombosis of left longitudinal sinus and transverse sinus.

decided to continue with doxycycline alone. Subject will be discussed later.

DISCUSSION

Q fever is a zoonosis caused by the obligate intracellular Gram-negative bacterium *C burnetii*, which is a member of the γ group of *Proteobacteria*, with great similarity to *Legionella pneumophila* (although initially classified as a species of the genus *Rickettsia*).¹ It has a wide variety of reservoirs, but the main ones are cattle, sheep and goats. Transmission occurs primarily through inhalation of aerosols from contaminated

Table 2 Some of the laboratory tests requested for case 2 during his admission in a neurology ward

Case 2. Laboratory tests obtained during admission in neurology ward			
Homocysteine	15 μ mol/L	Haemoglobin	17.4 g/dL
vWF	205%	ESR	15 mm/h
C3	1.19 g/L	CRP	0.09 mg/dL
C4	0.31 g/L	Prothrombinemia	78%
Parathormone	35 pg/mL	Lupus inhibitors	Negative
SACE	42 U/L	TSH	1.3 uUI/mL
Functional C protein	61%	Leiden V factor	Normal
Functional antithrombin III	92%	Factor V H1299R	Normal
Free S protein	77%	Prothrombin 20210 GA	Normal
APC resistance	3.07	MTHFR C677T	Normal
HIV 1/2	Negative	VDRL	Negative
Antibodies to CMV, HSV1, EBV	IgM negative, IgG positive	Anti- <i>Rickettsia conorii</i>	1:160
Antibodies to <i>Mycoplasma</i>	Negative		

Parameters classified as 'normal': no mutation identified. Positive IgG serologies for CMV, HSV1 and EBV related to previous infections
APC, active protein C; CMV, cytomegalovirus; CRP, C reactive protein; EBV, Epstein-Barr virus; ESR, erythrocyte sedimentation rate; vWF, von Willebrand factor; HSV, Herpes simplex virus; MTHFR, methylenetetrahydrofolate reductase gene mutation; SACE, seric aldosterone conversion enzyme; TSH, thyroid stimulating hormone; VDRL, venereal disease research laboratory.

Table 3 Some of the laboratory tests requested for case 2 in his first internal medicine outpatient clinic appointment**Case 2. Laboratory tests obtained during internal medicine appointment**

c-ANCA/p-ANCA	Negative	JAK2	No mutation
Anti-MPO	Negative	Anti- <i>Rickettsia conorii</i>	1:80
Antiproteinase 3	Negative	Anti- <i>Coxiella burnetii</i>	1:5120
Anticardiolipin	Negative	Phase II IgG for <i>C burnetii</i>	800
Anti-B2-GP	Negative	Phase II IgM for <i>C burnetii</i>	800

Phase antigens for *C burnetii* were requested posteriorly and to an external laboratory, since our hospital's laboratory is not able to identify specifically phase antigens.

ANCA, antineutrophil cytoplasmic antibodies; MPO, myeloperoxidase antibodies; GP, glycoprotein.

materials, such as urine, faeces, milk (and unpasteurised dairy products) or birth products, but other forms of contamination are also described. Its average incubation period is of 20 days. *C burnetii* has a worldwide distribution (except New Zealand) and has a higher incidence during the Spring and early Summer (the acute form, in Europe). In 2006, 169 cases of Q fever were reported in the USA; the UK reports about 100 cases each year and Portugal reports about 14 cases each year (probably underestimated).¹³

Macrophages are the main host for *C burnetii*, which lives inside acidic lysosomes; this fact is thought to have therapeutic implications.^{1 14} *C burnetii* has the ability to change the properties of its lipopolysaccharide, resulting in a phase variation, being that phase I is highly infectious and is the natural phase found in infected animals, whereas phase II is less infectious and is obtained only in laboratory.

Clinically, *C burnetii* can cause acute or chronic infection.¹ Regarding the acute form of the disease, about 60% of patients are asymptomatic. The three most common clinical presentation forms are: (1) flu-like syndrome, with high fever, myalgia,

Figure 11 Case 2: MRI venography (three-dimensional reconstruction images), performed 1 year after the event showing normal permeability of venous sinus. **Figure 12** Case 2: MRI venography (three-dimensional reconstruction images) performed 1 year after the event, showing normal permeability of venous sinus.

headache and fatigue; (2) pneumonia, with pleuritic chest pain, dry cough and pleural effusion and (3) hepatitis (predominant in Europe^{1 15}), with mild elevation of transaminases, jaundice, nausea and vomiting, right upper quadrant abdominal pain and, rarely, diarrhoea. Other manifestations are possible, such as pericarditis, myocarditis, meningoencephalitis, erythema nodosum and other non-specific rashes. Chronic Q fever, defined as an infection lasting for more than 6 months,¹ can develop in 0.2–1.4% of patients with acute infection. The major clinical manifestation is endocarditis with negative blood cultures, which occurs mostly in patients with pre-existing valve defects or prosthesis, especially of the aortic valve. It accounts for about 60–70% of all cases of chronic Q fever. Other possible manifestations are osteomyelitis, abortion, granulomatous hepatitis, interstitial pulmonary fibrosis, glomerulonephritis and chronic fatigue syndrome, the latter responding for about 10% of all cases of chronic infection. In the presence of epidemiological context, the association of fever without tachycardia, normal white cell count, low platelets and elevated liver enzymes is considered to be highly suggestive of Q fever.

Diagnosis of Q fever is based on detection of antibodies to phase antigens, for which indirect immunofluorescence is the method of choice. A titre of anti-phase II IgG $\geq$ 200 and IgM $\geq$ 50, a fourfold increase in IgG between two samples with 2–4 weeks interval and seroconversion (rise in IgG and IgM against phase II antigen) are diagnostic features of acute Q fever. Serological findings in chronic Q fever are: rise in IgG and IgM against phase I antigen and phase I IgG $\geq$ 800 (if $>$ 1600, 100% predictive of chronic infection). A phase I IgG of 1:800 is one of the major modified Duke' criteria for endocarditis.

Other laboratory and imaging studies are required to assess organ involvement, such as full blood count, liver enzymes, renal function, chest X-ray and abdominal US, depending on clinical findings.

Treatment of choice for Q fever is doxycycline 100 mg twice daily, usually for 10–14 days in acute infection. Treatment of chronic infections is complex, usually requiring long-term antibiotics (ranging from 1 to 4 years); recommendation for endocarditis is combination treatment with doxycycline and hydroxychloroquine, at least for 18 months. Fluoroquinolones can be used as an alternative to doxycycline.^{1 16}

Regarding that Q fever may affect virtually any organ and with many different clinical syndromes, it is expected to find unusual presentation forms. Despite that, diarrhoea and splenic and hepatic abscesses are exceedingly rare, so our first case is a real finding; Cathébras *et al*² described a case of splenic abscesses only. As for the second case, we did not find any descriptions in the literature of such manifestation of Q fever or such cause for CVT, making this the first report of its kind.

According to Maurin and Raoult,¹ diarrhoea can occur in 5–22% of patients with acute Q fever; hepatitis, with mild elevated transaminase levels, is very common and may be present in up to 45–85% of these patients. Pneumonia and other pulmonary signs, as pleural effusion, are very common as well. Although the patient of case 1 had these relatively common manifestations, he also had two other manifestations only seen in few case reports, which are the splenic and hepatic abscesses. Cathébras *et al*² described one case of a splenic abscess related to *Coxiella* infection. As for the hepatic abscesses/inflammatory pseudotumors, which are very rare per se, we did not find any report of such finding related to Q fever.^{7–10}

In case 1, the patient improved substantially after initiating antibiotics; since the liver abscesses disappeared at day 5 after initiating doxycycline (and the splenic ones were almost gone), we decided to stop treatment after 21 days, with the compromise of a close watch, namely of the liver enzymes and liver sonographic appearance. He persisted with mild fatigue for a few months, undoubtedly related to this infection, but his overall and progressive improvement was consistent enough to justify not prolonging the treatment.

Neurological manifestations of *Coxiella* infection are among the less common: meningoencephalitis is the main one and accounts for about 1% of all cases of Q fever. Maurin and Raoult¹ state that such manifestations must be differentiated from embolic manifestations related to Q fever endocarditis. In a setting of chronic Q fever with endocarditis, embolisation phenomena can occur in up to 20% of patients^{1 17}; stroke is one of the possible findings.

CVT is, as said before, a rare form of cerebral infarction^{4 5}; estimated ratio of venous to arterial strokes is 1:62.5. Daif *et al*⁴ reported a frequency of 7 cases/100 000 hospitalised patients. Regarding its aetiology, some conditions are identified as risk factors, such as inflammatory bowel disease, connective tissue diseases, hyperhomocysteinaemia, sarcoidosis and spontaneous intracranial hypotension. Other conditions identified as causative agents of CVT are sinusitis, head trauma and neurosurgical procedures, hypercoagulable states (antiphospholipid syndrome, protein S and C deficiencies, etc) and some medications (as oral contraceptives and corticosteroids).^{4 5 18} The role of minor trauma as the cause of CVT is controversial: only a few cases have been reported and all of them included other prothrombotic factors. Fonseca *et al*¹² and Röttger *et al*¹¹ described one case each of CVT after sneezing, in patients with thrombotic risk factors.

Given that, the rationale for CVT in case 2 patient is a prothrombotic state induced by Q fever, wherein sneezing was the trigger for the vascular event. This manifestation can be considered an embolic phenomenon, even though there was no evidence of endocarditis or infection of any other sites.

When the patient presented to the internal medicine outpatient clinic, he already had the diagnosis of CVT, and our mission was to clarify the aetiology. In the setting of a highly suggestive epidemiological context, the *red flag* we found was the positive serology for *Rickettsia*, since it is known that serological cross-reactions between *Rickettsia* and *Coxiella* can occur.¹⁹

In this case, duration of treatment was very difficult to establish. On the one hand, we had phase antigens suggesting acute infection and no evidence of endocarditis; on the other hand, we had a previously unknown manifestation of Q fever, with probable relation with embolic phenomena, and very high serology titres. So, we decided to prolong the treatment for 18 months, while monitoring serologies and clinical evolution, without adding hydroxychloroquine, because, once again, there was no evidence of endocarditis or other site infection. Good clinical evolution and normal control echocardiogram strengthened our decision.

Learning points

- ▶ Q fever can cause splenic and hepatic abscesses.
- ▶ Q fever can be the cause of cerebral venous thrombosis.
- ▶ Assessing epidemiological context is of great value in patients with unclear diagnosis, since it may have a key role in interpreting signs, symptoms and test results in order to unveil their aetiology.
- ▶ *Coxiella burnetii* can cause acute or chronic infection with a wide variety of manifestations, affecting almost every organ and in multiple possible ways.

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Competing interests None.

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