THE AMERICAN JOURNAL of MEDICINE®

## Chronic Q Fever Causing Aortitis

*To the Editor:*

A 64-year-old man with a history of rheumatoid arthritis, on hydroxychloroquine and low-dose steroid, and with a stable infra-renal abdominal aortic aneurysm, was admitted in November for a 2-week history of low back pain. He described 3 months of low-grade temperature elevations, night sweats, and a 10-pound weight loss. He lived in southern Iowa and spent most of the preceding summer outdoors working at a shooting range, in close proximity to sheep and cattle farms. He was afebrile, and physical examination revealed no vascular bruits or murmurs. There was no abdominal pulsatile mass or tenderness on back or abdominal palpation.

Laboratory data revealed an elevated erythrocyte sedimentation rate of 47 mm/h (0-15 mm/h) and C-reactive protein of 1.5 mg/dL (<0.5 mg/dL). Complete blood count and liver function tests were normal. Computed tomography scan of the abdomen/pelvis confirmed the stable infra-renal aortic and common iliac artery aneurysms, but detected new fat stranding and focal dilatation of the inferior mesenteric artery (**Figure**).

Positron-emission tomography−computed tomography showed a hyper-metabolic focus at the origin of the inferior mesenteric artery consistent with inflammation or infection. Blood cultures were negative. *Brucella*, Lyme, *Ehrlichia*, and *Bartonella* serologies and QuantiFERON-TB Gold results were negative. Q fever serology showed an immunoglobulin G phase I >1:32,768 and phase 2 >1:4096; immunoglobulin M phase 1 and 2 were both 1:32. Treatment was initiated with doxycycline, and his hydroxychloroquine dose was increased. He underwent surgical resection with placement of polytetrafluoroethylene graft 1 month later.

## DISCUSSION

*Coxiella burnetii* is an obligate intracellular pathogen responsible for Q fever, a worldwide zoonosis. Common animal reservoirs leading to human infections include cattle, sheep, and goats.[1,2] Following inhalation or ingestion of organisms, *C. burnetii* can cause acute or chronic infection.[1,3] Acute Q fever typically manifests as a flu-like illness often associated with pneumonia or hepatitis.[4]

Chronic Q fever develops in up to 10% of infections,[5] facilitated by host factors and organism virulence features. This patient's immunocompromised state due to rheumatoid arthritis and steroid use likely contributed to the evolution to

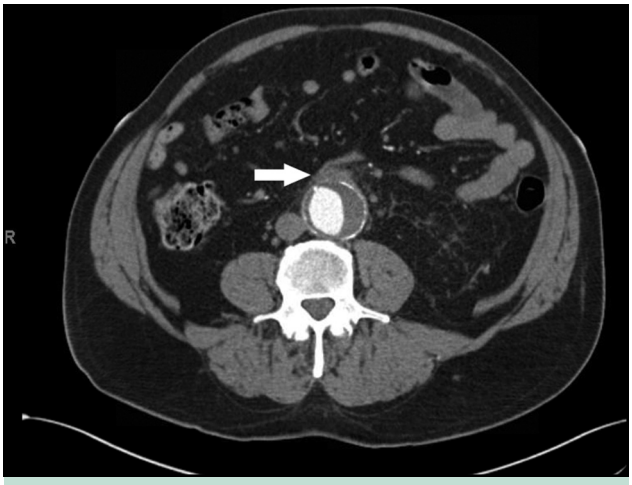


**Figure** Abdominal CT scan demonstrates fat stranding at the origin of the inferior mesenteric artery (*arrow*).

**Funding:** None.

**Conflicts of Interest:** None.

**Authorship:** Both authors had access to case information and a role in writing the manuscript.

Requests for reprints should be addressed to Judy Streit, MD, Department of Medicine, University of Iowa Hospitals and Clinics, 200 Hawkins Drive, Iowa City, IA 52242.

E-mail address: judy-streit@uiowa.edu

chronic infection. *C. burnetii* targets monocytes/macrophages and survives within the phagolysosome. Diseased vascular/endovascular tissues are predisposed to chronic infection, leading to further tissue damage.[1,6] Hence, chronic Q fever is manifest as endocarditis in the context of preexisting cardiac valvular abnormality, and less commonly, infected vascular grafts and aortic aneurysms, as in this case. Management of *Coxiella*-infected aneurysms includes surgical resection and long-term antibiotics. The addition of hydroxychloroquine can reduce the duration of treatment for endovascular infections.[7]

Because signs and symptoms of Q fever are nonspecific, the index of suspicion is often low without a compelling exposure history. Diagnosis usually rests on serology, given *C. burnetii's* fastidious nature. Polymerase chain reaction can be performed for diagnosis, but performance varies widely based on chronicity of infection and whether antibody titers are markedly elevated.[8] These factors likely contribute to the rarity with which *C. burnetii* is identified as the responsible agent in vascular infection.

Uchenna T. Ikediobi, MD, MPH[a]
Judy Streit, MD[a,b]
[a]*Department of Medicine*
[b]*Division of Infectious Diseases*
*Carver College of Medicine*
*University of Iowa Hospitals and Clinics*
*Iowa City, Iowa*

http://dx.doi.org/10.1016/j.amjmed.2013.02.004

## References

1. Woldehiwet Z. Q fever (coxiellosis): epidemiology and pathogenesis. *Res Vet Sci*. 2004;77:93-100.
2. Babudieri B. Q fever: a zoonosis. *Adv Vet Sci Comp Med*. 1959;5:81-181.
3. Raoult D, Marrie T, Mege J. Natural history and pathophysiology of Q fever. *Lancet Infect Dis*. 2005;5:219-226.
4. Kazar J. *Coxiella burnetii* infection. *Ann N Y Acad Sci*. 2005;1063:105-114.
5. Wegdam-Blans MCA, Vainas T, van Sambeek MR, et al. Vascular complications of Q fever infections. *Eur J Vasc Endovasc Surg*. 2011;42:384-392.
6. Mege JL, Maurin M, Capo C, Raoult D. *Coxiella burnetii*: the "query" fever bacterium. A model of immune subversion by a strictly intracellular microorganism. *FEMS Microbiol Rev*. 1997;19:209-217.
7. Raoult D, Houpikian P, Dupont HT, et al. Treatment of Q fever endocarditis: comparison of 2 regimens containing doxycycline and ofloxacin or hydroxychloroquine. *Arch Intern Med*. 1999;159:167-173.
8. Fenollar F, Fournier PE, Raoult D. Molecular detection of *Coxiella burnetii* in the sera of patients with Q fever endocarditis or vascular infection. *J Clin Microbiol*. 2004;42:4919-4924.