Chronic Q fever is most commonly associated with culture-negative endocarditis and less frequently with infection of vascular grafts, infection of aneurysms, hepatitis, pulmonary disease, osteomyelitis, and neurological abnormalities. We report a case of chronic sternal wound infection, polyclonal gammopathy, and mixed cryoglobulinemia in which Q fever endocarditis was subsequently diagnosed. Polymerase chain reaction analysis of the wound tissue was positive for *Coxiella burnetii* DNA, and treatment of the endocarditis resulted in prompt healing of the wound. Chronic Q fever can occur without epidemiological risk factors for *C. burnetii* exposure and can produce multisystem inflammatory dysfunction, aberrations of the immune system, and persistent wound infections.

Q fever is a zoonotic infection caused by the intracellular pathogen *Coxiella burnetii*. This infection has been reported worldwide, and the most common animal reservoirs for *C. burnetii* are cattle, goats, and sheep [1], although transmission to humans by parturient cats or wild rabbits has also been reported [2–4]. Two forms of the infection, acute and chronic, have been recognized [5]. The acute illness is commonly manifested as self-limited febrile illness, granulomatous hepatitis, or pneumonia [6–12]. The chronic form, frequently misdiagnosed, may have a variety of presentations, the most common of which is culture-negative endocarditis [13–16].

Other reported presentations of chronic Q fever are infection of a vascular prosthesis, infection of aneurysms, osteomyelitis, hepatitis, purpuric skin eruptions, and interstitial pulmonary fibrosis [16–19]. To our knowledge there has never been a reported case of surgical wound infection by *C. burnetii*. We report a case of chronic Q fever with a surgical (sternotomy) wound infection, granulomatous hepatitis, and endocarditis.

**Case Report**

A 66-year-old businessman without a history of agricultural exposure presented to a southwestern Wisconsin medical center in July 1997 with a sternal wound infection. The first event in his medical history, extending over 9 years and involving four medical centers, occurred sometime after January 1989, when he underwent a St. Jude aortic valve replacement and thoracic aortic aneurysm repair in Houston. He recovered from his surgery uneventfully and did well until early 1994, when he developed intermittent fevers (temperatures up to 102°F), weight loss, malaise, and night sweats. Physical examination revealed moderate hepatosplenomegaly, and laboratory evaluations showed microcytic anemia, polygammopathy, and elevated erythrocyte sedimentation rates of 120 mm/h.

Between early 1995 and January 1997 he developed renal insufficiency with mild proteinuria, progressively enlarging splenomegaly, purpuric skin eruptions, leukopenia, severe headaches, and intermittent fevers. Diagnostic evaluations over the 3-year period included a liver biopsy, positive for granulomatous hepatitis; three bone marrow biopsies, showing diminished iron stores and nonspecific increases in cellularity and plasma cells; a temporal artery biopsy, negative for giant-cell arteritis; an abdominal CT, confirming splenomegaly; a skin biopsy, revealing leukocytoclastic vasculitis; serum electrophoresis, of which findings were consistent with polyclonal gammapathy; analysis of cryoglobulins, which were positive for mixed type; transesophageal echocardiography (TEE), showing no valvular vegetations; and numerous blood cultures, which were sterile.

The patient was empirically treated with prednisone for a presumed autoimmune process, but no improvement occurred. Over the same time period he underwent repair of an aortic aneurysm and splenectomy (which showed no malignancy or granuloma).

In January 1997 culture-negative endocarditis was diagnosed while he was vacationing in Florida, and he was treated with a 25-day course of intravenous vancomycin and ceftriaxone. Shortly after completion of the antibiotic course, his fevers, chills, and night sweats returned. TEE was repeated and showed no obvious valvular vegetations but moderate to severe left ventricular hypokinesia. His health continued to decline with development of congestive heart failure and polyarthritis.

In July 1997, swelling, erythema, drainage, and granulation tissue at the site of his old sternotomy incision developed. A
underlying heart or vascular disease, and (3) chronic Q fever without cardiovascular involvement. The latter category includes isolated fevers, isolated hepatitis, pulmonary disease, osteomyelitis, osteoarthritis, neurological abnormalities, and cutaneous lesions [8–11, 16, 19, 22–25]. The cutaneous manifestations of chronic Q fever are rare and usually limited to purpuric rash (which is due to an immune vasculitis [16]) and erythema nodosum [26]. Our patient displayed features of all three of the above categories: fevers, culture-negative endocarditis, aortic aneurysm and vascular prosthesis (both of which are presumed to have been infected), severe headaches, arthritis, purpuric eruptions, and possible osteomyelitis of the sternum.

The exact interval between the onset of infection and the diagnosis in this case, as is typical of chronic Q fever, is impossible to determine, but the clinical presentation and serological data suggest that the infection went untreated for at least 4 years. This prolonged period of untreated infection is often seen because of the confusing and polymorphic clinical presentation, with *C. burnetii* infection in multiple tissue sites. CT scan of his chest showed a 5-cm fluid collection overlying the sternum (figure 1) but no fluid collection, gas in tissues, or inflammatory changes within the sternal bone. A technetium scan demonstrated increased uptake above the sternum, consistent with the overlying tissue inflammation. The patient had no sternal pain, sternal instability, or wound dehiscence to suggest osteomyelitis.

This area was incised but continued to drain seropurulent fluid. Multiple cultures of specimens from his persistently draining wound (figure 2) remained negative for 2 months, after which time only one of multiple mycobacterial cultures yielded *Mycobacterium terrae*; he was treated with azithromycin and trimethoprim-sulfamethoxazole. With this therapy his sternal wound infection healed transiently, but it relapsed when trimethoprim-sulfamethoxazole was discontinued secondary to development of a skin rash.

In November 1997, Q fever endocarditis was diagnosed on the basis of serology for antibodies to Q fever. Titers of IgG were >1:1,024 to phase I and phase II antigens, and titers of IgM were >1:1,024 to phase I antigens and >1:256 to phase II antigens. Vegetations were now seen around the aortic valve by TEE. The patient was treated with doxycycline (100 mg twice daily) and ofloxacin (400 mg every 12 hours); his sternal wound healed promptly, and the fevers, chills, sweats, and headaches resolved. The wound specimens from biopsies that were performed in late 1997 were positive by PCR for *C. burnetii* DNA. The PCR testing, performed at the Molecular Diagnostics Laboratory of the U.S. Army Medical Research Institute of Infectious Diseases (Fort Detrick, MD), utilized a nested primer set against a highly conserved and specific insertion sequence of *C. burnetii* [20, 21].

Discussion

Brouqui et al. [16] recommended classifying chronic Q fever as follows: (1) Q fever endocarditis, (2) chronic Q fever with

![Figure 1. CT scan of chest showing fluid collection overlying the sternum (arrow) of a patient who was found to have chronic sternal wound infection and endocarditis with *Coxiella burnetii.*](image1)

![Figure 2. Petechial rash and granulation tissue overlying the sternal wound infection of the patient in figure 1.](image2)
negative cases, including wound infections, can be evaluated for Q fever with appropriate serological and/or PCR testing.

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