for persons with cryptococcal meningitis but without bacteremia was 30% (47/158); 1 patient was lost to follow-up. Thus, the estimated attributable mortality rate for bacteremia was 13% (odds ratio 1.8, 95% CI 0.78–4.0, p = 0.17) compared with patients without bacteremia during their initial hospitalization.

Case–control comparisons identified no risk factors for bacteremia (online Technical Appendix, wwwnc.cdc.gov/EID/article/20/4/13-1277-TechnicalAppendix.pdf). Although 21 (70%) of 30 bacteremia episodes were preceded by phlebitis at a peripheral intravenous site, phlebitis caused by amphotericin was also common in patients without bacteremia (49%), but these percentages did not differ statistically.

Accurate data regarding incidence of nosocomial infections in Africa are lacking. A systematic review by WHO in 2011 that assessed published data for 1995–2009 identified only 2 high-quality studies. WHO estimated a prevalence of 2.5%–14.8% for nosocomial infections and a cumulative incidence of up to 45.8% in some areas and a cumulative incidence of up to 14.8% for nosocomial infections estimated a prevalence of 2.5%–14.8% for nosocomial infections and prevalence of multidrug resistance among hospitalized persons in resource-limited areas.

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Severe Babesiosis in Immunocompetent Man, Spain, 2011

To the Editor: Babesiosis, a malaria-like illness, is transmitted through Ixodes ticks by the zoonotic parasites, Babesia spp. In humans, these parasites are transferred from mammalian animal reservoirs, and the rate of infection in humans is increasing. Babesiosis also potentially threatens the blood supply because asymptomatic infections in humans are common; such infections can be life-threatening in some recipients (1). Most human infection is caused by B. microti, but babesiosis caused by B. divergens, B. duncanii, and B. venatorum has been reported.

Human babesiosis can be clinically silent or progress to a fulminating malaria-like disease. The infection resolves spontaneously or after treatment with azithromycin/atovaquone or clindamycin/quinine. However, immunocompromised patients may respond suboptimally to these drug regimens (2). Given the death rate
associated with babesiosis, no treatment is fully satisfactory (3). Infection with *B. divergens* is particularly problematic and is associated with a high death rate in splenectomized or immunocompromised patients (3). In Europe, sporadic cases of babesiosis have also been reported in immunocompetent persons (4).

In October 2011, a 46-year-old man whose spleen was intact was hospitalized after 3 days of fever, severe abdominal pain, jaundice, and black and red deposits in his urine. The man lived in a rural area in Asturias, Spain, where he was employed as a forest ranger. He reported that he removed ticks from his dogs.

Laboratory findings included hemoglobin 12.3 g/dL (reference range 13.8–17.2 g/dL); creatinine 1.52 mg/dL (reference range 0.7–1.3 mg/dL); total and direct/conjugated bilirubin 18.4 and 12.8 mg/dL (reference ranges total 0.3–1.9 mg/dL; direct/conjugated 0–0.3 mg/dL), lactate dehydrogenase 822 IU/L (reference range 105–333 IU/L); and showed thrombopenia, low haptoglobina, and hematuria. A value of 822 IU/L (reference range 0.7–1.3 mg/dL); direct/conjugated bilirubin 13.8–17.2 g/dL; creatinine 1.52 mg/dL, and no parasites were detectable by microscopy. The treatment was extended for an additional 5 weeks, and the patient was free of parasites on subsequent visits.

We have described what appears to be the third case of human babesiosis in non-splenectomized patients in Europe. Human presence in tick, cattle, and domestic animal habitats could be responsible for this case. Martinot et al. (4) earlier pointed out that in Europe, babesiosis can also occur in persons with intact spleens. A combination of clindamycin and quinine is the recommended treatment of severe babesiosis (2,3). However, in this case, the recommended therapy failed, and therapy was switched to atovaquone/proguanil plus azithromycin. Other case reports have also related failure, ineffectiveness, adverse reaction, or persistent and relapsing babesiosis to clindamycin and quinine treatment in splenectomized patients infected by *B. divergens* or *B. microti* (3,6–9) or suspected *B. microti* drug resistance in immunocompromised patients (2). The recently sequenced *B. microti* genome reveals absence of proteases necessary to digest host hemoglobin and hemozoin formation by the parasite; this absence may explain the ineffectiveness of chloroquine, and perhaps other compounds of the aminoquinoline family used in babesiosis therapy (10).

This clinical case report, together with the failure of clindamycin and quinine to successfully eliminate the parasite *Babesia*, again opens the debate about the limitations of conventional treatment for severe human babesiosis in immunocompetent and immunocompromised patients. The capability of *Babesia* spp. to invade erythrocytes is the key step of the disease process. Focusing on *Babesia* spp. molecules involved in the invasion steps may offer new targets for the development of new prophylaxis and treatment for human babesiosis.

![Figure. A Giemsa-stained thin film of blood from a 46-year-old man showing *Babesia divergens*. Double pear shaped intraerythrocytic parasites are indicated by arrows. Slides were examined with a Nikon microscope (Nikon Instruments, Inc., Melville, NY, USA) at 60× magnification. Scale bar indicates 500 nm. A color version of this figure is available online (wwwnc.cdc.gov/EID/article/20/4/13-1409-F1.htm).](image)
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Q Fever Endocarditis and New Coxiella burnetii Genotype, Saudi Arabia

To the Editor. Q fever is a worldwide zoonosis caused by an obligate intracellular bacterium, Coxiella burnetii (1). Q fever endocarditis is associated with surgery for 15%–73% of patients, causes death for 5%–65% of patients, and induces a large number of relapses when the endocarditis is inadequately treated (1). The most serious risk factor for endocarditis is a substantial underlying valvulopathy, but progression to endocarditis is also found in patients with clinically silent, previously undiagnosed, valvulopathies (1). Since the 1960s, Q fever has been recognized as a public health problem in Saudi Arabia, and studies have shown that coxiellosis occurs in livestock (2,3). Only a few cases of Q fever endocarditis in Saudi Arabia have been reported (4–6). We report 2 new cases of Q fever endocarditis and detection of a new C. burnetii genotype in this country.

The first case was detected in 2007 in a 45-year-old man in Saudi Arabia who had fever, pneumonia, and asthenia. A transesophageal echocardiogram showed endocarditis. Results of an immunofluorescence assay were positive for C. burnetii; phase I titers for IgG, IgM, and IgA were 51,200, 100, and 25, respectively, and phase II titers were 102,400, 200, and 50, respectively. Serum and blood samples were negative for C. burnetii by real-time PCR for the IS1111 and the IS30A spacers (7). For each sample, the quality of DNA extraction was verified by real-time PCR for a housekeeping gene encoding β-actin (7). The aortic valve was surgically replaced, and C. burnetii–specific PCR results for the valve were positive. According to multispacer sequence typing (8), this C. burnetii isolate was a new genotype, MST51 (Figure). A C. burnetii isolate was cultured from the valve of this patient by the shell-vial method that used human embryonic lung cells (7). IgG anticardiolipin testing results were negative (9). The patient was given 200 mg oral doxycycline daily and 200 mg oral hydroxychloroquine 3 times daily for 18 months.

The second case was detected in 2012 in a 13-year-old boy in Saudi Arabia who had tetralogy of Fallot, a prosthetic pulmonary valve, 2 intracardiac stents, and long-term fever. Serologic testing results were positive for C. burnetii; phase I titers for IgG, IgM, and IgA were 51,200, 400, and 200, respectively, and phase II titers were 102,400, 800, and 400, respectively. Whereas serum and blood
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Technical Appendix

Technical Appendix Figure. Immunofluorescent assays of intra-erythrocytic and free parasites were performed by using antibodies and Babesia divergens smears of a 46-year-old man that were prepared from in vitro human erythrocyte cultures (Bd Rouen 1986 strain). Bound antibody was detected by using fluorescein isothiocyanate-conjugated anti–human IgG. The preparations were counterstained with DAPI and examined by confocal microscopy. Samples were laser-stimulated at 488 and 405 nm. Intraerythrocytic parasites probed with patient IgG are shown in panels A and B and isolated free extracellular merozoites in panel C. Panels D, E, and F show the DNA of these same parasites stained with DAPI. Panels G, H, and I show both images overlaid. The immunofluorescent assays, using serum from healthy volunteers, were negative (data not shown).