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-Techapp1.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 (4) and recommended surveillance to estimate the rates of nosocomial infection. WHO acknowledges that health care–associated infections are causes of prolonged hospitalizations, increased antimicrobial drug resistance, financial burdens on health care systems, and causes of excess illness and death (5).

Limitations of our study include the retrospective design and inability to identify predictive risk factors for bacteremia. Given the differences in bacteremia incidence between our 2 sites, findings are probably not generalizable to all clinical settings in Africa. However, these findings identify a clinical problem.

The incidence of nosocomial bacteremia was 15% in our hospitalized cryptococcal meningitis cohort at a median time of 14 days after hospitalization. The most frequent etiologies were *S. aureus* and *K. pneumonia*. Less than 25%

of isolates were sensitive to ceftriaxone, a standard empiric drug used throughout Africa. Further prospective studies are needed to determine the prevalence and risk factors 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. duncani*, and *B. venatorum* has been reported.

Human babesiosis can be clinically silent or progress to a fulminant 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 35% CD4+ T cells (reference range 30%–60%) indicated normal immune status. Results of serologic tests for hepatitis; HIV; and *Bartonella*, *Brucella*, *Leishmania*, *Leptospira*, and *Borrelia* spp. and of blood cultures were negative. Abdominal ultrasound scan revealed mild hepatomegaly and cortical echogenicity compatible with acute kidney failure. Howell-Jolly bodies were identified in blood, and functional splenic studies were conducted. Scintigraphic parameters showed a normal deposit of radioactive hepato-splenic material, compatible with a normal-sized spleen of 13.4 cm.

Giemsa-stained blood smears showed intra-erythrocytic parasites, mainly observed in the 2-celled dividing pyriform stage, leading to the diagnosis of babesiosis with a parasitemia level of 10% (Figure). The complete *B. divergens* 18S rRNA gene was amplified
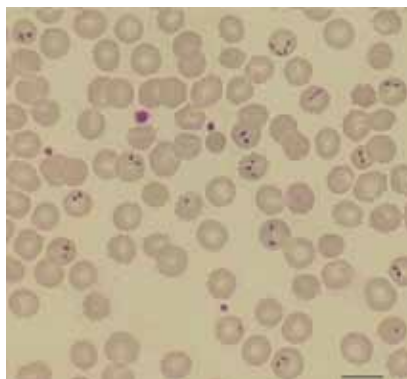

Figure. A Giemsa-stained thin film of blood from a 46-year-old man showing *Babesia divergens*. Double pear shaped intraerythrocytic parasites are indicated by arrows. Slides were examined with a Nikon microscope (Nikon Instruments, Inc., Melville, NY, USA) at 60× magnification. Scale bar indicates 500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

from the patient's blood (*5*), and the nucleotide sequencing (GenBank accession no. KF533077) showed 100% homology with *B. divergens* human strains (GenBank accession nos. FJ944822 and FJ944823) (*5*) and with 2 babesiosis cases reported previously (*4*). Indirect immunofluorescent assays of *B. divergens* cultures showed specific antibodies against *B. divergens* in the patient's serum (online Technical Appendix Figure, wwwnc.cdc.gov/EID/article/20/4/13-1409-Techapp1.pdf). The patient was treated with 650 mg oral quinine every 8 hours and 600 mg intravenous clindamycin every 6 hours. The parasitemia diminished gradually and resolved 10 days later, but the hemolytic anemia remained severe, as evidenced by hemoglobin of 7.2 g/dL.

The man's illness unexpectedly relapsed on day 18 after treatment. His reticulocyte count was elevated, and parasites were once again detected in blood samples. Thus, treatment was changed to a combination of atovaquone/proguanil 250/100 mg administered every 8 hours plus azithromycin 500 mg every 24 hours. Two weeks later, the patient's hemoglobin was 8.7

g/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 nonsplenectomized 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 babesisosis 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.

Acknowledgments

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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