African tick-bite fever (ATBF) is caused by *Rickettsia africana*, a spotted fever group rickettsia that is transmitted by infected cattle ticks (*Amblyomma hebraeum*) in rural areas of southern Africa. Although the distinctive clinical features of ATBF have been known to physicians practicing in southern Africa for approximately 60 years, it was erroneously assumed to be due to *R. conorii* until 1992 when Kelly and others isolated *R. africana* from a patient in Zimbabwe with ATBF. Since then, several investigators have re-examined the clinical and epidemiologic features of ATBF using case definitions based on modern and highly accurate laboratory methods.

Brouqui and others recently described seven French patients with ATBF who acquired their illness in southern Africa and returned to Europe during the incubation period of their illness. Once in their homeland, they manifested an illness that was initially unfamiliar to their French physicians. Brouqui and others also suggested that the authors of previous reports of *R. conorii* infection in American travelers to southern Africa may, in fact, have actually described *R. africana* infection. In May 1998, a similar cluster of ATBF cases involving nine persons returning to Oregon from a group trip to Swaziland was reported. We report a serologically confirmed case of ATBF in an American traveler to South Africa to confirm that imported cases of ATBF occasionally occur in the United States and to alert other physicians to the clinical and epidemiologic features of this distinctive illness.

**CASE REPORT**

On May 18, 1997, a 66-year-old previously healthy physician removed several ticks from his body while hunting near Kruger National Park in South Africa. The following day he returned to the United States. On August 20, 1997, he noticed five discrete red lesions on his arms and back. Over the next three days, these lesions ulcerated and formed discrete eschars (Figure 1). He developed minimal generalized aching and fatigue simultaneous with the appearance of these skin lesions, which he ascribed to the 24-hr flight home. He did not have fever, chills, anorexia, or significant malaise. When first seen on August 25, 1997, one new vesicular red lesion was present on his forearm, but the remainder of his physical examination showed normal results. African tick bite fever was suspected. Therapy with oral doxycycline (200 mg twice a day) was begun. During the next 72 hr, the skin lesions began to heal; full recovery occurred within five days. The skin lesions eventually healed without scarring. Acute and convalescent serum samples were tested for antibodies against *R. africana* using a microimmunofluorescent antibody technique. IgM antibodies were undetectable in the acute serum sample and increased to a titer of 1:64 in the convalescent serum sample; IgG antibody titers to the same antigens were 1:32 and 1:64, respectively. Antibodies specific for *R. africana* were detected in the convalescent serum but not the acute serum sample using a Western blot technique.

**DISCUSSION**

During the 1930s, Pipper and others described the clinical features of ATBF isolated a causative organism from ticks, and concluded that ATBF was a separate disease from Boutonneuse fever (which at the time was known to be caused by *R. conorii*). However, subsequent experiments by Gear failed to confirm these findings; thus, for the next 52 years, causes of ATBF were erroneously assumed to be due to *R. conorii*, a spotted fever group rickettsiae that is present in the same geographic areas as *R. africana*.

In 1990, Kelly and Mason demonstrated that spotted fever group rickettsiae isolated from *A. hebraeum* ticks in Zimbabwve were different from *R. conorii*. In 1992, Kelly and others isolated *R. africana* from a patient with an illness typical of ATBF. After using a variety of modern molecular methods such as 16S ribosomal RNA gene sequencing that compared their isolate to earlier isolates from *A. hebraeum* ticks, Kelly and others proposed that this organism be designated as a new species called *R. africana*. Since then, there have been several reports describing the clinical and epidemiologic features of a number of patients with laboratory-confirmed ATBF.

The authors of the above clinical reports emphasized that ATBF is characteristically a mild disease. Multiple ulcerated skin lesions (tache noires) as seen in our patient (Figure 1) are the hallmark of the illness. An eruptive maculopapular or generalized petechial rash, which appears after 1–5 days of specific symptoms, typical of most other spotted fever rickettsioses, is not present in most patients with ATBF. Some patients with ATBF also develop inflammatory papules that may vesiculate (Figure 1). Most, but not all patients with ATBF have regional lymphadenopathy and fever, findings that were not present in our patient. However, our patient was similar to many previously reported cases in that...


