CASE REPORT

African Tick Bite Fever

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Physicians may encounter unfamiliar diseases as a result of international travel. Fever with rash is an important sign that usually represents initial manifestation of infectious disorders. A 62-year-old Taiwanese woman presented with multiple eschars, a papulovesicular rash, and fever 5 days after returning from South Africa. A biopsy specimen of an eschar had wedge-shaped tissue necrosis, hemorrhage, necrotizing vasculitis of the small venules and arterioles, and a dense perivascular lymphocytic infiltrate in the dermis. Serologically, there was cross reaction with both *Rickettsia conorii* and *R. rickettsii*. DNA sequencing demonstrated *R. africae*, confirming the diagnosis of African tick bite fever. The patient responded well to minocycline. Recognition of the symptoms and signs, and diagnostic tools for different types of rickettsiosis are essential for correct diagnosis and treatment. [*J Formos Med Assoc* 2008;107(1):73–76]

Key Words: African tick bite fever, *Rickettsia africae*

Many physicians are unfamiliar with African tick bite fever (ATBF), a rickettsiosis caused by *Rickettsia africae*[1] and transmitted by ticks in rural sub-Saharan Africa. We report the case of a patient who was infected with ATBF whilst in South Africa.

Case Report

A 62-year-old Taiwanese woman presented with fever and two mild tender skin nodules 5 days after returning from a 2-week trip to South Africa. She denied a history of insect bites or close contact with animals. There were two erythematous pustular nodules on her left inner thigh and left shoulder. Oral cephalexin was prescribed for presumed furunculosis. However, 10 days later, the nodules became two black eschars, about 1 cm in diameter (Figures 1A and 1B), and with scattered 2–3-mm asymptomatic erythematous papules and vesicles near the eschars (Figures 1C and 1D). She also had a low-grade fever but no myalgias or lymphadenopathy.

Histologically, an eschar specimen had wedge-shaped tissue necrosis and diffuse inflammation extending to the subcutis (Figure 2A). There was necrotizing vasculitis of small venules and arterioles with a dense perivascular lymphocytic infiltrate and hemorrhage (Figure 2B). Gram and periodic acid-Schiff stains were negative for bacteria and fungi. Serial indirect immunofluorescence serology for rickettsiae by the Taiwan Centers for Disease Control found a four-fold increase (160–1280×) in immunoglobulin G titers for both *R. rickettsii* and *R. conorii*, but was negative for other rickettsioses.

Nested polymerase chain reaction (PCR) of eschar material was performed as previously described.[1] The first suicide PCR assay used the primers AF3F, AF3R, AF4F and AF4R for nested amplification (Figure 3). The PCR product was then amplified by using AF5F, AF5R, AF6F and

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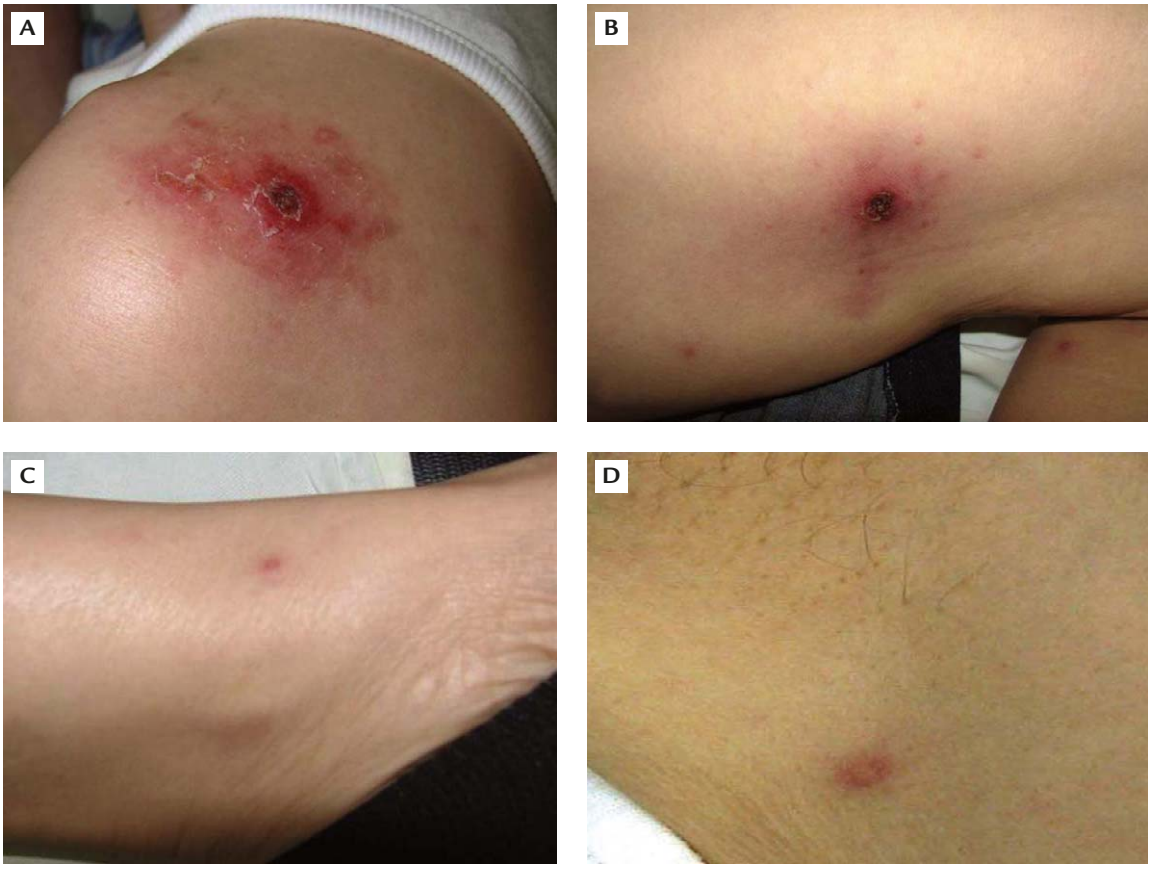

Figure 1. Black eschars on the (A) left shoulder and (B) left thigh, with a few surrounding red papules. (C) Erythematous papule on dorsal foot. (D) Vesicle in the axilla.

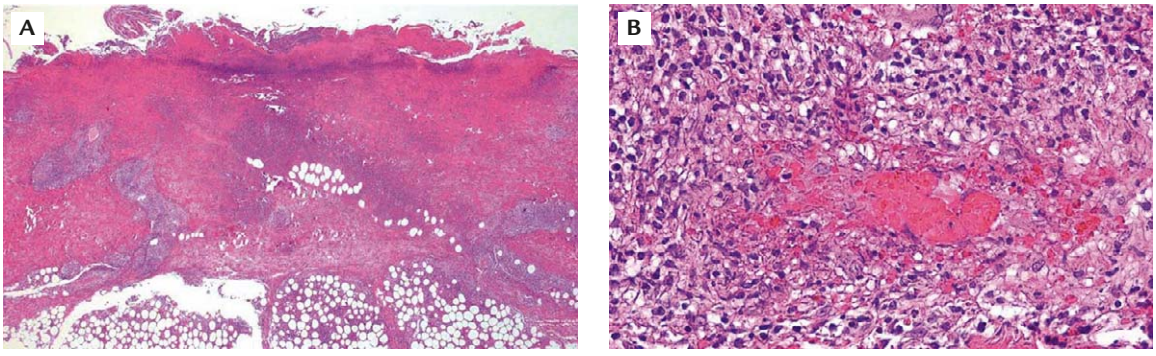

Figure 2. (A) Histopathologic examination shows wedge-shaped tissue necrosis deep in the subcutis (hematoxylin & eosin, 40×). (B) Necrotizing vasculitis of small venules with perivascular lymphocytic infiltration with some leukocytes, nuclear dust and hemorrhage (hematoxylin & eosin, 400×).

AF6R as second primers. The PCR product was then sequenced by restriction fragment length polymorphism and found to be homologous to *R. africae* DNA.

Initial treatment before confirmation of the diagnosis included ceftazidime, ciprofloxacin and minocycline, but ceftazidime was discontinued once ATBF was diagnosed. The patient had already received a total of 14 days of ciprofloxacin and minocycline before the diagnosis was made. She recovered without sequelae other than hyperpigmented scars at the eschar sites. None of the other members in her tour group had a similar disease.