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AFRICAN TICK-BITE FEVER IN A RETURNING TRAVELER

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☐ **Abstract—Background: African tick bite fever (ATBF) is an emerging infection endemic to sub-Saharan Africa and increasingly noted in travelers to the region. Case Report: We present a case of ATBF in a 63-year-old man who presented with complaints of a rash and fever to the emergency department. Why Should an Emergency Physician be Aware of This?: Rickettsial diseases are increasingly common and are seen on every continent except Antarctica. Many factors are contributing to their prevalence, and they have become the second most common cause of fever behind malaria in the traveler returning from Africa. Due to the global distribution of rickettsial diseases, as well as increasing international travel, emergency physicians might encounter ill and febrile travelers. A careful travel history and examination will enable the emergency physician to consider spotted fever group rickettsial diseases in their differential diagnosis for single and multiple eschars.** © **2015 Elsevier Inc.**

☐ **Keywords—African tick bite fever; rickettsia**

INTRODUCTION

Fever and rash are common complaints of returning travelers. Epidemiology, geography, time course, physical diagnosis, and laboratory tests are important components of a successful emergency department diagnosis and are crucial in differentiating between the many emerging illnesses facing travelers throughout the world.

CASE PRESENTATION

A 63-year-old man presented to the emergency department with fever, myalgias, and a rash 2 weeks after returning from travel to India and South Africa. He stayed primarily in large cities in India for 3 weeks, followed by 1 week on a private game reserve safari in Kwa-zulu Natal. He was well during the trip, but did notice multiple mosquito bites on his legs. He did not notice any tick bites. Five days after his return, he was moving boxes and noticed generalized stiffness and soreness but without fever. Two days later, he developed fevers, chill, and nightsweats, as well as arthralgias. One week after the onset of symptoms, and 12 days after returning from South Africa, he noticed a quarter-sized erythematous lesion on his left hip. The following day he was febrile to 101°F, and he presented to our emergency department.

The patient states that he spent about 4 h each morning on the game reserve, and then would retire to his hotel, and then would again go out on safari in the evening. He did not get out of his vehicle or have direct contact with animals. He denies drinking unpasteurized milk, he did not swim in local water, and he drank only bottled water. He was not using malaria prophylaxis.

Medical history includes hypertension, left retinal artery occlusion, and his only medications are aspirin, hydrochlorothiazide, and lisinopril. He has no known drug allergies. The patient has an extensive travel

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history—he had served in the merchant marines in his 20s—and he has made > 30 trips to Africa and has visited this particular game reserve about 10 times.

On physical examination, the patient had a temperature of 100.5°F, pulse 94 beats/min, and his blood pressure was 126/67 mm Hg. He was alert, well appearing, and conversant, and his HEENT (head, ears, eyes, nose, and throat) examination was unremarkable, with anicteric sclera, no conjunctival injection, and a clear oropharynx. His neck was supple and without adenopathy. His lungs were clear to auscultation, his heart examination demonstrated a normal s1s2 and he had a regular rate and rhythm, with no murmurs or rubs. His abdomen was soft, nontender, and there was no hepatosplenomegaly. His skin examination was remarkable for a 2-cm erythematous lesion with a black center on his left lateral hip. There was no fluctuance, it was minimally tender, and there was no associated lymphadenopathy in the inguinal area. He had several erythematous papules on his chest wall and neck. He had several scattered crusted papules on his lower extremities.

Laboratory examination revealed a white blood cell count of 5600 with 48% polymorphonuclear leukocytes, 20% bands, 21 % lymphocytes, 10% monocytes, and no eosinophils. His chemistry examination was normal with a blood urea nitrogen of 21 mg/dL, and creatinine of 1.05 mg/dL. His liver enzymes were mildly elevated with an aspartate aminotransferase of 88 IU/L, and an alanine aminotransferase of 72 IU/L. A thick and thin smear was negative for malaria and babesia. A human immunodeficiency virus test was nonreactive. The patient had a rickettsial spotted fever group titer sent (*Rickettsia rickettsii, Rickettsia akari,* and *Rickettsia conor*ii), which were initially negative, but at 4 weeks were IgM positive at 1:64 and IgG 1:256. *R. Conorii* cross reacts with *Rickettsia africae* and the final diagnosis of African tick bite fever (ATBF) due to *R. africae* was made in the patient. He was treated with doxycycline and his symptoms abated.

DISCUSSION

Rickettsia are classified primarily into the spotted fever group *rickettsia* and the typhus group *rickettsia* (1). While other rickettsiae groups exist as well, the tickborne rickettsioses, which belong to the spotted fever group of the *rickettsia,* have increased dramatically over the past several decades and numerous studies have postulated reasons for the increase in tickborne disease. Better detection methods, climate change, land use patterns, and widespread travel have led to an unprecedented resurgence in tickborne illness in every continent except Antarctica (2). In North America, the preceding factors, as well as an increased deer population, and the decreased population of land-based mammals feeding on mice have all likely led to the increase in tick and human interaction, causing an increase in recognition of tickborne diseases (3).

R africae, the causal agent of ATBF, is an obligate intracellular gram-negative bacteria. It is carried by the ticks of the genus, *Amblyomma,* and is distributed broadly across sub-Saharan Africa, where it has been detected in ticks as well as humans in > 20 sub-Saharan African countries (1). The disease was recognized in the early 20th century primarily as a rural illness and was associated with the ticks of cattle and game. It was isolated as an organism in 1990 and the first proven case of human infection was noted in 1992 (4). Before the organism's isolation, it was believed that ATBF was caused by *R. connori,* the causal agent of Mediterranean spotted fever (5). The disease has had a global impact—it has been reported in numerous North African countries and has been noted in Australia, New Caledonia, and in Asia (6,7). Although the tick vector was imported into Guadalupe in the 1800s via Senegalese cattle, the first case was not reported in the Western hemisphere until 1998 (1,8). Since this initial description, the organism has been detected in *Amblyomma variegatum* ticks throughout the Caribbean (1).

ATBF in North America and Europe is usually acquired by tourists, hunters, or deployed military members returning from areas where the disease is endemic. A study of travelers from sub-Saharan Africa reported by the GeoSentinel network found that it was the second most common cause of systemic fever in travelers returning from sub-Saharan Africa (9). Like many tickborne diseases, it begins about 5−7 days after a tick bite, with initial onset of myalgias, fatigue, headache, and fever. Inoculation eschars, the *tache noire*—an ulcerated black-crusted eschar with a red halo—are common, and are the sine qua non of the disease (Figure 1). After the bite of an infected tick, the rickettsia proliferate within the endothelial cells at the site of the original bite, leading to a localized vasculitis and the formation of an eschar (10). Multiple eschars are typical of the aggressive biting habits of the *Amblyomma* genus of ticks.

An eschar is also commonly seen in Mediterranean (*Bouttoneuse*) spotted fever. Although not highly specific, the eschar is quite suggestive of ATBF, given the appropriate epidemiologic setting. Anywhere from 50%−100% of patients are affected. Other infectious and noninfectious causes of eschar are noted in Table 1 (1,11−14). Once again, time course of disease onset, severity of illness, and travel history are vital in helping to differentiate between these eschar-forming diseases. Other common features include a generalized maculopapular, or perhaps a papulovesicular, rash, as well as