

## East African Trypanosomiasis in a Pregnant Traveler

**To the Editor:** Human African trypanosomiasis (HAT) results in a large number of deaths and considerable illness in sub-Saharan Africa. Although the disease is uncommon in returning travelers from that region, awareness of it is important for medical practitioners in areas where the disease is not endemic. The disease can be categorized geographically into West and East African trypanosomiasis caused by *T. brucei gambiense* and *Trypanosoma brucei rhodesiense*, respectively, and clinicopathologically into hemolymphatic (stage I) disease and meningoencephalitic (stage II) disease (1). The East African form of the disease is less common than the West African form and accounts for 10% of the global incidence of trypanosomiasis.

Relative stability in East African nations may have contributed to the lower incidence of the disease in these countries, but drought and increasing pressure on water sources may lead to an upsurge in East African disease. The increasing ease of global travel and attraction of game safaris and hunting may also lead to increasing exposure in travelers. HAT is treated with toxic drugs in regimens that have changed little for decades. Few published data exist on the treatment of HAT in pregnancy, particularly for East African disease. We describe a case of *T. brucei rhodesiense* infection occurring in a pregnant traveler.

A 32-year-old woman, 20 weeks pregnant, returned from a 9-day safari trip to Tanzania 8 days before coming to a hospital in London. She described a short history of fever, headache, and soft-tissue swelling of the forehead with severe regional adenopathy. She had evidence of skin necrosis (chancre) (Figure) but no history of tsetse fly bite. Blood tests showed anemia

(hemoglobin 9.5 g/dL), leukopenia ( $1.8 \times 10^9$  cells/L), and thrombocytopenia ( $60 \times 10^9$  cells/L). A blood film showed trypomastigotes of *T. brucei rhodesiense*. Suramin was initially unavailable for treatment, but because of her deteriorating clinical state, she was treated with 1 dose of pentamidine (4 mg/kg) before suramin was obtained. Suramin was begun 36 hours after admission, initially at 5 mg/kg and increased over the next 2 doses up to 1 g. During the next 48 hours, her fever resolved, and serial blood films showed clearance of the parasites from the blood. A cerebrospinal fluid sample showed no signs of stage II disease, and the patient continued on suramin, completing a standard course as an outpatient. Her pregnancy was closely monitored, and she gave birth at term to a healthy baby girl.

The treatment of choice for stage I HAT caused by *T. brucei gambiense* is parenteral pentamidine (1). No published trials compare pentamidine and suramin in East African trypanosomiasis, but longstanding consensus suggests that suramin is more likely to be efficacious in stage I East African disease (1). The basis for this difference in efficacy is unexplained.

Theoretically, pentamidine may be teratogenic because it inhibits protein and nucleic acid synthesis in vitro (2). However, studies in rats found pentamidine to be fetocidal but not teratogenic (3). Pentamidine has been used extensively for HAT prophylaxis without reported problems (4) and has had limited use in pregnant women with *Pneumocystis jirovecii* pneumonia. It continues to be recommended in pregnant women with stage I HAT originating in West Africa. Suramin is known to cause a syndrome similar to preeclampsia in pregnant rats (5), yet it too has been used in large-scale treatment programs for onchocerciasis, and no fetal or placental effects have been reported in humans (2,6). We found 1 case report describing successful use of suramin, followed by melarsoprol, in a pregnant woman with HAT (7).

The treatment of stage II disease in pregnancy is problematic, and published information to guide therapy is lacking. Although the effect of arsenicals on fetuses is a concern, case reports have described the successful use of melarsoprol during pregnancy (7,8); if left untreated, the disease is fatal. Thus, if our patient had stage II disease, use of melarsoprol, which is



Figure. Chancre at site of tsetse fly bite on forehead of pregnant patient with trypanosomiasis. A color version of this figure is available online ([www.cdc.gov/EID/content/15/11/1866-F.htm](http://www.cdc.gov/EID/content/15/11/1866-F.htm)).

often given with prednisolone, would have been necessary.

In pregnant women with West African (*T. brucei gambiense*) stage II disease, either melarsoprol or eflornithine can be used, but neither is effective for East African disease. Although eflornithine can abort early pregnancies and cause disordered organogenesis (9), the severe encephalopathy associated with melarsoprol makes eflornithine a preferable option for single-agent treatment. However, nifurtimox–eflornithine combination therapy will soon replace single-drug regimens for stage II *T. brucei gambiense* cases (10).

We believed evidence was insufficient to withhold suramin therapy for this highly fatal disease. Because of the uncertainty about effects of pregnancy on the ability to clear trypanosomes, the patient will be followed up for signs of relapse. The danger of HAT should be specifically highlighted for all travelers to trypanosomiasis-endemic regions, particularly pregnant travelers because of potential harm to unborn children.

P.L.C. is supported by the University College London Hospitals Comprehensive Biomedical Research Centre Infection Theme.

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DOI: 10.3201/eid1511.090384

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## ***Rickettsia africae* Infection in Man after Travel to Ethiopia**

**To the Editor:** The first human case of African tick-bite fever was described in 1992 as occurring in Zimbabwe. The causative agent was identified as a new serotype of the spotted fever group (SFG) rickettsiae and named *Rickettsia africae* (1). These findings confirmed observations made by Pijper in the 1930s which suggested that there were 2 different kinds of human SFG rickettsioses in sub-Saharan Africa: Mediterranean spotted fever caused by *R. conorii* and transmitted by *Rhipicephalus* species, ticks of dogs, and African tick-bite fever caused by *R. africae* and transmitted by *Amblyomma* species, ticks of cattle and wild ungulates. African tick-bite fever has subsequently been diagnosed in patients from several other sub-Saharan countries and also from the West Indies (2,3).

In a recent analysis of the spectrum of diseases among returning travelers, tick-borne spotted fever was (after malaria) the second most frequent cause of systemic febrile illness among those returning from sub-Saharan Africa. It occurred more frequently than typhoid fever and dengue fever (4). The following case description reports an infection with *R. africae* in a man in France who recently returned from Ethiopia.

On November 4, 2005, a 62-year-old French man sought care at the Medical Center of the Institut Pasteur in Paris for fever, along with chills, headache, neck and shoulder pain, and fatigue over the previous 4 days. At the onset of these symptoms he had noticed dark nodular lesions on his neck and his left groin followed 2 days later by a slightly painful eruption on his arms and his trunk. He had spent a month in southwest Ethiopia, north of Kelem near the Sudanese bor-

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