

# Tryps after adventurous trips

F.A.P. Claessen<sup>1\*</sup>, G.J. Blaauw<sup>2</sup>, M.J.D.L. van der Vorst<sup>1</sup>, C.W. Ang<sup>2</sup>, M.A. van Agtmael<sup>1</sup>

VU University Medical Center (VUmc), Amsterdam, the Netherlands, <sup>1</sup>Department of Internal Medicine, <sup>2</sup>Department of Medical Microbiology & Infection Prevention, \*corresponding author: e-mail fap.claessen@vumc.nl

A 30-year-old previously healthy woman was admitted to the general medical ward because of a one-day history of high fever. Six to ten days before, she had visited Tanzania on her honeymoon and went on safari tours in open jeeps in Tarangire, Lake Manyara, Serengeti and Ngorongoro Crater National Parks, successively. She recalled multiple tsetse fly bites. Four days before admission she felt ill and noticed a chancre on her leg (*figure 1*). She was taking atovaquone/proguanil for malaria prophylaxis and had been vaccinated against yellow fever, hepatitis A and B and typhoid fever. On presentation, she looked ill and was jaundiced, not obtunded, with a temperature of 40°C, blood pressure 125/70 mmHg and pulse 120 beats/min. There was no nuchal rigidity and on auscultation discrete crackles were heard over the left chest. A thick smear revealed no malaria parasites but trypomastigotes of *Trypanosoma brucei* spp. Her laboratory results revealed pancytopenia (haemoglobin 6.6 mmol/l, leucocytes  $2.2 \times 10^9/l$ , thrombocytes  $37 \times 10^9/l$ ), diffuse intravascular coagulation, metabolic acidosis, elevated bilirubin (212  $\mu\text{mol/l}$ , conjugated fraction 0.66), ASAT (594 U/l) and ALAT (416 U/l), serum creatinine 55  $\mu\text{mol/l}$  and a mild proteinuria.

**Figure 1.** Chancre on the left calf 4 days after its first appearance due to infection with *Trypanosoma cruzi rhodesiense* after a bite by an infected tsetse fly



To exclude central nervous system infestation, a lumbar puncture was performed. Cerebrospinal fluid analysis was normal with no trypomastigotes. The electrocardiogram showed repolarisation abnormalities and her chest X-ray was normal. She was treated with suramine intravenously, first with a test dose of 200 mg and then 1000 mg on days 1, 3, 10, 17, 24 and 31. The following day, she developed progressive dyspnoea. Now the chest X-ray showed diffuse changes compatible with acute respiratory distress syndrome (ARDS) and she was transferred to the intensive care unit, where hydrocortisone was given from day 2 to 4 and supportive care. No intubation or vasoactive medication were required.

Clinical improvement started on day 3. On day 4 the blood smear was negative for trypomastigotes. The proteinuria disappeared during treatment. However, her serum creatinine gradually increased to 110  $\mu\text{mol/l}$  three months after the start of the treatment, with a creatinine clearance of 79 ml/min. No other side effects of the suramine were noticed (adrenal insufficiency, polyneuropathy). After six months she has fully recovered and the serum creatinine has normalised.

This patient presented with acute sleeping sickness or human African trypanosomiasis (HAT) with severe disease and multi-organ involvement four to five days after the first symptoms and after a remarkably short incubation time of less than seven days following visits to game parks in Tanzania.

In 2002, Jelinek *et al.*<sup>1</sup> reported nine cases of sleeping sickness among tourists travelling to the Tarangire and Serengeti National Parks in Tanzania. Three of them had multiple organ failure and one died. In travellers to endemic areas, blood smears for malaria should also be examined for trypomastigotes. Not all textbooks mention icterus as an early sign of HAT as we observed in this patient. A lumbar puncture in the diagnostic workup is controversial: meningo-encephalitis is unlikely in the first week of illness and false-positive results may occur, which could prompt unnecessary treatment with the toxic

melarsoprol. Theoretically, accidental contamination of the cerebrospinal fluid after a traumatic lumbar puncture is possible, although proven cases have not been described. HAT is caused by protozoa of the *Trypanosoma* genus. Transmission to humans occurs by the bite of a tsetse fly (*Glossina* genus), infected with either *T. brucei gambiense* or *T. brucei rhodesiense*. Less than 10% of cases are caused by *T. brucei rhodesiense* and can be found in Eastern and Southern Africa.<sup>2</sup> Since the year 2000, when the World Health Organisation reinforced surveillance and disease control measures, the number of reported cases of East African sleeping sickness has stabilised: 669 in 2000 and 486 in 2006.<sup>3,4</sup>

Tourists travelling to endemic areas are at risk, although the risk of acquiring trypanosomiasis is much lower than malaria. The number of tourists returning to Europe

yearly with HAT is not known, but presumably very low. Travellers to endemic areas should be made aware of the risk of acquiring trypanosomiasis and minimise exposure to the bite of the vector.

## REFERENCES

1. Jelinek T, et al. Cluster of African trypanosomiasis in travellers to Tanzanian National Parks. *Emerg Infect Dis.* 2002;8:634-5.
2. <http://www.who.int/mediacentre/factsheets/fs259/en/print.html>
3. WHO. Human African trypanosomiasis (sleeping sickness): epidemiological update. *Weekly Epidemiol Rec.* 2006;82:71-80.
4. Simarro PP, Jannin J, Cattand P. Eliminating human African trypanosomiasis: where do we stand and what comes next? *PloS Med.* 2008;5(2):174-180,e55.