Congenital Trypanosomiasis in an 11-Year-Old Girl at the Brazzaville University Hospital

Ossibi Ibara BR1,2,3, Adoua Doukaga T2,3, Ekat M3, Bendett P3, Mvoumbo G3, Kinga F3 and Kitembo L1

1National Control Program for Human African Trypanosomiasis.  
2Université Marien Ngouabi.  
3Brazzaville University Hospital.

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ABSTRACT

Human African trypanosomiasis is a neglected tropical disease about to be eliminated as a public health problem. It afflicts rural populations, particularly adults engaged in hunting and fishing activities in endemic homes. Left untreated, the disease is 100% fatal in an array of dormant cachexia. Vertical transmission from mother to child is rare but proven. We report a case of Human African Trypanosomiasis in an 11-year-old girl who has never been to an endemic area and who has no other risk exposure factor apart from the fact she was born from mother who was affected by the disease and treated for 10 years during pregnancy. The diagnosis was made after the analysis of the LCS notifying 144 elements with the presence of numerous trypanosomes. The patient was classified into the second period and treated with NECT for 10 days.

Keywords
Trypanosomiasis, Congenital, CHU, Brazzaville

Introduction

Human African trypanosomiasis, also known as sleeping sickness, is a neglected tropical disease that has long posed a public health problem in Africa due to high morbidity and mortality [1,2]. Adults living in endemic foci where tsetse flies proliferate are most at risk, mainly those engaged in fishing, hunting and farming. Children who usually accompany parents during these activities are also prone to the disease. Transmission occurs mainly through the daytime and painful bite of the tsetse fly. The old treatment in 2009 included a combination of drugs [3]. Cases of mother-to-child transmission during pregnancy are rare but proven (4, 50LBp124). We report an authentic case of human African trypanosomiasis in an 11-year-old girl who has never been to an endemic area for sleeping sickness and who has, as a risk factor, the mere fact of being born from a mother who had suffered from trypanosomiasis in Ngabé for more than 10 years and treated in Brazzaville in the second phase during the pregnancy of this patient.

Observation

This is an 11-year-old girl N, M, female admitted to the infectious diseases department of Brazzaville University Hospital on January 25, 2021 for the treatment of sleeping sickness. The current symptoms have been reported for 3 years with the occurrence of a disarticulated fever, resistant to antipyretics and antimalarial associated with persistent frontotemporal headaches without associated vomiting. This picture was the subject of several consultations in the medical offices where the patient was treated several times for malaria without success. It is finally before the appearance of sleep disorders such as daytime sleepiness and nocturnal insomnia (Figure 1) that parents decide to consult the National Control Program for Human African Trypanosomiasis where, after investigations, the diagnosis of sleeping sickness was selected and the patient was referred to the Brazzaville CHU for a better care. In her history, she has never been transfused. The patient was born vaginally without trauma. Her father is alive in an apparent good health. Her mother is a former second-stage trypanosome when she was pregnant carrying the teenager. She is the only daughter of her siblings.
The clinical examination permits to note a patient in a fairly good general condition with good skin-mucous staining, anicteric without folds of dehydration or undernutrition.

**Figure 1:** Hypersomnolence avant traitement.

The temperature is estimated at 38.9 °C, the heart rate at 80 beats per minute. The weight calculated at 21 kg for a size of m is a body mass index of Kg / m².

The neurological examination found a conscious patient, misguided in time and space. There is a change in the character of the adolescent who is sad associated with sensory disorders of the type of hyperesthesia. The patient has difficulty performing the pronosupination maneuver, explaining the sign of the key, that is, the inability of the patient to turn a key, so painful is the pressure. The finger-nose test is difficult to perform, indicating dysmetria related to cerebellar ataxia.

The ROTs are lively the neck is supple. At the spleno-ganglionic level, there is no large spleen or palpable lymphadenopathy. The heart is regular without added noise and without breath. The rest of the physical exam is normal. TDR was positive, as did CATT in whole blood and after dilution to 1/32. The lymph node puncture was not performed. The CTC was positive, as did the lumbar puncture, which found 144 elements on cytology with the presence of numerous trypanosomes. The phase diagnosis (Figure 2) as currently notified by WHO allowed the patient to be classified into the second phase. The patient was treated with NECT 200 mg / kg of Eflornithine intravenously every 12 hours for 7 days and 15 mg / kg of Nifurtimox to be taken orally every 8 hours for 10 days. The evolution showed the disappearance of the fever and physical asthenia with resumption of appetite and normalization of the nycthemeral rhythm and the patient was very quickly awakened from her cachectic sleep (Figure 2). After prescribing a post-treatment follow-up schedule, the patient was discharged from the hospital on the 15th day with a post-treatment follow-up schedule.

**Discussion**

Human African trypanosomiasis (HAT) is a neglected tropical disease that affects rural populations in Africa. It is currently being eliminated like a public health problem according to WHO [2]. Adult populations engaged in farming, fishing and hunting are the most affected as reported in the DRC, the most endemic country in the world, by Tshimungu and colleagues [5]. Childhood trypanosomiasis is also relatively common in endemic areas, but sometimes underestimated due to lack of documentation. Four signs dominate in this area: fever, physical asthenia, hypersomnia and neurological impairment as reported in our patient and already reported by Andjingbopou Y in CAR, another HAT endemic country [6]. Congenital trypanosomiasis is rare but proven, as was the case in this 11-year-old adolescent, born to a trypanosome mother who left the endemic area (the Ngabé focus) 5 months before childbirth and treated at the National Control Program for Human African Trypanosomiasis in the second phase for 10 days. The fact that the child presented symptoms related to sleeping sickness such as disarticulated and permanent fever, drowsiness from the age of 6, all in a context of failure to thrive when she has never lived in an endemic area of HAT testifies the contamination by vertical route at the time of childbirth.

The diagnosis of sleeping sickness is well codified according to the algorithm decreed by WHO and adapted by each endemic country such as in Congo (Figure 3). The sensitivity of CATT as a diagnostic orientation serological test has already been reported by several authors [7,8]. Sleep disorders signify the presence of trypanosomes having crossed the hemomeningeal barrier and located at the level of the supra-chiasmatic nuclei thus disrupting the biological clock, which gives all the interest of the lumbar puncture with analysis of the cerebrospinal fluid in order to pose...
both parasitological and phase diagnosis [9,10]. There are several treatments for human African trypanosomiasis caused by T. b. gambiense whose use of the molecules obeys diagnostic criteria as indicated in the therapeutic algorithm updated by WHO taking into account their mechanisms of action and resistance [11]. However, the presence of more than 100 cells on LCS cytology with the identification of trypanosomes in children weighing 21 kg requires the use of the combination of Eflornithine –Nifurtimox for a total duration of 10 days. Well treated and on time, trypanosomiasis is cured after several post-treatment follow-up sessions to confirm cure or detect relapse or sequelae as reported in Congo and DRC [12,13].

**Conclusion**

Congenital trypanosomiasis remains an epidemiological reality in endemic areas such as Congo, but often underestimated because of the low rate of passive screening. The most common signs are a disarticulated fever associated with neurological disorders. The treatment depends on the site, the cytology of the LCS and also the phase of the disease.

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