

## Paediatric sleeping sickness in Kenya: A case report

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### SUMMARY

Sleeping sickness is often considered a disease of adults rather than children due to their reduced exposure to the vector. Presumptive diagnosis of sleeping sickness was however difficult since the clinical signs observed were non-specific. This makes clinical diagnosis difficult. Often the disease in children masquerades as a pulmonary infection that is undetectable on x-ray or auscultation. A male child aged two years and eight months was diagnosed with the disease in western Kenya. The patient presented with severe respiratory distress, hepatosplenomegaly and neurological symptoms. The disease transmission was associated with the socio-cultural habit of placing children under bushes whilst farming. The implications of delayed diagnosis on response to treatment are discussed.

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### Introduction

Human African Trypanosomiasis (HAT), also known as sleeping sickness is caused by haemoflagelates of the genus *Trypanosoma*. It has been reported that there remains 200 active foci for HAT between latitudes 15°N and 15°S. Sixty million people living in 36 countries are at risk. Of these, less than 4 million benefit from adequate surveillance, case-finding programs or from vector control activities [1]. Sleeping sickness has an insidious effect in the quality of life within the communities due to its debilitating symptoms. If left untreated, the final outcome of the disease for the individual is death [2]. Western Kenya remains one of the active focus for *T. b. rhodesiense*, sleeping sickness. The area is characterised by low-level endemicity with between 20 and 30 confirmed cases annually [3]. The foci are currently confined to Teso, Busia and Bungoma districts.

Children are not usually affected under normal circumstances by sleeping sickness mainly due to their reduced risk of exposure. The infection is especially rare below the age

of five years [4]. When it occurs, the disease takes a rapid course with early development of central nervous system signs [5]. The presenting clinical signs normally include high fever, dullness, lethargy, anaemia that borders on jaundice, liver and spleen enlargement, meningeal signs and behavioural changes [6]. Diagnosis is often delayed and the child finally presents with psychomotor retardation, seizures and or coma with trypanosomes demonstrated in both blood and cerebrospinal fluid [7,8]. There is a high risk of neurological sequel in the sensory and motor system with mental and intellectual retardation [5,6,9].

### Case report

The patient was attended to at the National Sleeping Sickness Referral Hospital-Alupe (NSSRH- Alupe) with patients derived from the HAT endemic foci. This is located approximately 500 km from Nairobi the capital city of Kenya.

**Definition of HAT case:** A person was considered to have HAT if he/she was positive for trypanosomes in blood or cerebrospinal fluid by haematocrit centrifugation technique (HCT) as described by Murray et al. [10].

### ***Clinical manifestation, diagnosis and treatment of HAT***

A male child aged two years and eight months was admitted to the National Sleeping Sickness Referral Hospital in Alupe in October 1999. He was from On'gariama sub-location in Teso district. This admission followed a referral from the Alupe sub district hospital where the child had been unsuccessfully treated for various conditions for a period of one month. The history of disease was intense lumbar headache, cough, fever, palpitations and loss of concentration for duration of 5 months. The child was unresponsive to malarial and typhoid therapy. During the final admission, the child had been observed with high fever, respiratory distress, and agitation for a period of six days. A presumptive diagnosis of pneumonia was made.

A visiting veterinarian made the diagnosis of sleeping sickness when asked to comment chest and head x-rays of the patient. The signs observed were a non-resolving cerebral oedema and grossly enlarged liver and spleen. With suspicion based on the area of origin of the patient and clinical history of the disease, a Giemsa-stained thin blood smear was suggested. This revealed high density of trypanosome in excess of ten per field. It was noted that the initial smear the trypanosomes were thought by the laboratory technician to be artefacts. The patient was subsequently transferred to the NSSH Alupe. When presented at the referral hospital, the child was comatose, with periodic convulsions, restless and a high but undulating fever of 39.4°C. Abdominal palpation of the liver and the spleen indicated that they were grossly enlarged, which impinged on the diaphragm and resulted in respiratory distress. The mucous membranes were jaundiced and the head pulled back to present a tender neck. On lumbar puncture, two trypanosomes per

microscope field and 6 white cell counts (WCC/ml<sup>3</sup>) were demonstrated in the cerebrospinal fluid. This confirmed the child to be in the late stage of the disease.

Therapy was instituted with suramin at 20mg/kg bwt increasing dosage to a maximum of 1gm on days 1, 3, 7, 14 and 21. Subsequently melarsoprol was administered in several series of three consecutive injections at 3.16 mg/kg bwt. These injections are usually given every twenty-four hours for three days with one-week rest between each series (WHO, 1998). Other signs observed as consciousness was regained in response to treatment were somnolence, psychosis, purities and a flaccid paralysis. The child was discharged after completing the course of treatment in 41 days. The child was, however, found to have relapsed during the second follow-up done three months from the date of discharge. He was subsequently re-admitted for a similar treatment regimen successfully carried to completion. During further follow-up, the child consistently presented with classical psychomotor dysfunction, dental problems, speech and locomotor disorders, a chronic phobia for the hospital and moderate arsenical encephalopathy (ARE) as indicated by abnormal behaviour.

### **Discussion**

Sleeping sickness has been described in children over the years. Several authors have reported a wide variety of clinical symptoms during disease [12,13,14,15]. However, this case suggests that children are candidates for ARE because of the delayed diagnosis of sleeping sickness. Diagnosis is often wrong due to the respiratory signs observed in children. In this case it would appear that the trypanosomes could have been missed or thought to be artefacts. Routine deworming with Albendazole in children is thought to increase risk of ARE as described by Ancelle et al., [16]. Prednisolone on the other hand reduces its effects described by Pepin et al. [17]. Miss-diagnosis of sleeping sickness often leads to a delay in the institution of disease specific therapy resulting in a neurological sequel. This has been previously

described among children in *T. b. gambiense* areas [6]. Such children were reported to have a poor prognosis of full recovery. They also exhibited prolonged debilitation even after treatment when compared to adults. Sociologists attribute transmission of sleeping sickness among children to the socio-cultural practices of the Iteso community. They lay children underneath *Lantana camara* thickets for shade while cultivating their gardens. This exposes them to tsetse.

In Western Kenya, even adult patients continue to report delayed diagnosis of sleeping sickness by the health care delivery system. Infected individuals may remain undiagnosed or even die due to inadequate surveillance, poor access to diagnostic or therapeutic facilities and limited knowledge of the disease by frontline health staff. Under diagnosis is made worse by the low sensitivity of diagnostic methods [3].

### Conclusion

It would therefore appear that although *T. b. rhodesiense* sleeping sickness is severe enough to cause patients to seek health care, the awareness among the intermediate health care providers of the clinical signs and parasitological methods of detection for the disease is limited. These can be addressed through targeted training and education. An improvement of the capacity of existing health provider facilities to correctly diagnose HAT will make meaningful contribution to sleeping sickness case detection. Disease control can be done through passive surveillance, regular active screening, prompt case treatment and ring treatment of livestock in affected villages as a more cost-effective method of control rather than waiting for emergency interventions during epidemics.

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## CASE REPORTS

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