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Case Report Open Access

Human African Trypanosomiasis: Challenges in the Diagnosis of *Trypanosoma Brucei Rhodesiense* – Case Report

Tedious Sokesi[1], **Peter Songolo**[2], **Freddie Masaninga**[2*] **and Olusegun Babaniyi** [3]

[1]*Ministry of Health, Lusaka, Zambia*

[2]*World Health Organization, Lusaka, Zambia*

[3]*Freelance Consultant, Abuja, Nigeria*

[*]**Corresponding author:** Freddie Masaninga, World Health Organization (WHO), P.O. Box 32346, Lusaka, Zambia, Tel: +260 211 255322; E-mail: Masaningaf@who.int

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Abstract

Introduction: *Trypanosoma brucei rhodesiense* has been known to be responsible for transmission of Human African Trypanosomiasis (HAT) in Zambia for decades, affecting predominantly rural districts and communities. Despite a decline in cases of *T.b. rhodesiense* HAT in the country over the years, sporadic cases are still being reported in some rural areas. Despite years of dealing with the disease challenges are still being experienced on diagnosis. This article on a HAT case highlights challenges in the diagnosis of *T.b. rhodesiense*.

Case history: A 27 year old black African male developed malaria-like symptoms and signs, followed by palpable swellings in the neck and skin rash. The patient was initially diagnosed of malaria and treated with *artemether lumefantrine*, according to national guidelines. The patient later developed central nervous system (CNS) manifestations and was diagnosed as bacterial meningo-enchephalitis and put on several antibiotics. Thirty five days (35) after admission *Trypanosoma brucei rhodesiense* parasites were confirmed in the spinal fluid with fatal outcome despite commencing the patient with definitive treatment.

Conclusions: The diagnosis of *Trypanosoma brucei rhodesiense* continues to experience challenges resulting into delayed treatment. Improved capacities to diagnose treat, and map the disease are vital elements to effective control in endemic areas.

Keywords: *Trypanosoma brucei rhodesiense*; Human african trypanosomiasis (HAT); Diagnosis; Central nervous system; Zambia

Introduction

Human African Trypanosomiasis (HAT) is a re-emerging parasitic infection in Africa. The causative parasite *Trypanosoma brucei rhodesiense* (*T.b. rhodesiense*); tsetse flies of the genus Glossina transmit the disease to humans through their bite [1,2]. The disease predominantly affects rural communities [3,4]. Although there has been a decline in the cases of rhodesiense HAT in the country, sporadic case are still being reported [5]. *Trypanosoma brucei rhodesiense* causes an acute form of the disease if untreated. HAT cases have been reported in the Eastern and Southern Africa. The rhodesiense form is predominantly a zoonosis, with occasional infection of humans [6]. The clinical presentation and disease progression caused by *T.b. rhodesiense* is influenced by its virulence resulting in either an acute and severe or a mild and chronic form of the disease [7]. The disease is usually acute in *T.b. rhodesiense*, with central nervous system (CNS) invasion by the parasite occurring early, within a few months after initial infection [8].

This article presents challenges in the diagnosis of *T.b. rhodesiense* leading to delayed identification of the causative organism and late commencement of definitive treatment. In this case treatment was only started in the second stage of the disease. This case presented as an acute and severe form of the disease resulted into fatal outcome within seven months following onset of symptoms.

Case History

Chief complaint: A 27-year-old male was admitted to a tertiary hospital in the country because of reduced level of consciousness.

History of presenting complaint: The patient presented to one urban health centers in Lusaka on 13th July 2015 and two days later he was referred and admitted to one of the three tertiary hospitals for severe anaemia. Initial history obtained at admission suggested that the patient had been unwell for two weeks with the following presenting complaints: body weakness, backache, loss of memory, and fever for two weeks and a cough for one day. He was reported to have stopped talking, but was groaning and was unable to walk. The patient was noted to have come from a rural district a week prior to admission. History and clinical examination revealed no previous history of diabetes mellitus, hypertension, asthma, epilepsy; no history drugs, alcohol or smoking.

The patient was started on Penicillin G and Chloramphenicol for presumed meningoencephalitis. Five days after admission, Glasgow Comma Scale was 7/15. An HIV test was performed and was non-reactive.

Ten days after admission, treatment was switched to ceftriaxone 2 g IV BD; 12 days after admission metronidazole was added to cover for possible aspiration.

Fourteen days after admission, he was noted to have diarrhea and the condition was not improving. Treatment for tuberculosis (TB) was

recommended but was not commenced because family refused the treatment; ceftriaxone was stopped. Pressure sores were noted on day 15 after admission.

Thirty five days after admission, patient was seen by the Infectious Diseases team upon request from the primary team. Detailed history was obtained which revealed that the patient had been unwell with fever, general malaise and malaria-like symptoms which gradually progressed into altered mental status and reduced responsiveness over a period of 8 months. Symptoms began in December 2014, three months after he had moved to Lukwipa village, a hard-to-reach rural area which is a known focus of HAT in Rufunsa district, Lusaka province. He had spent two of those 3 months working as a bush clearing labourer for a construction company that was putting up overhead electricity power lines in Lukwipa, Rufunsa District. Seven months prior to admission, he was treated for malaria at the local rural health center, four months prior to admission; he had developed swellings in the neck and was treated with injections at the local rural health center. He developed confusion a month prior to admission. Two weeks prior to admission, he had developed stool and urinary incontinence. HAT was suspected and a buffy wet prep and Giemsa stain confirmed trypanosomiasis. LP was repeated and many motile trypanosomes were seen in the cerebral spine fluid (CSF) on day 35. The patient was started on melarsoprol but died 7 days after commencement of the medicines. He had been started on steroids prior to initiating melarsoprol. His level of consciousness had improved slightly during treatment but he never gained full consciousness.

The chronology of laboratory testing is as follows from the day of admission at a tertiary hospital.

Day 8 post admission: Blood culture conducted; no organisms on Gram stain, no growth after 9 days, urine mixed growth 2-4 pus cells.

Day 10: Toxoplasma gondii immunoglobulin (Ig) G negative; CSF fluid blood stained, cell count of white blood cells (WBC) 48 per mm^3, polymorphs 70%, lymph 30%, Red blood cells (RBCs) 2360 per mm^3, Gram stain no organisms, no yeast cells on India ink, no Cryptococcus species seen, glucose 2.7 g/dl, protein 2.37 g/dl, chloride 130 mmol/l, cerebral spinal fluid culture no growth, Cryptococcus antigen non-reactive.

Day 14: Urgent brain Computerized Tomography (CT) scan was requested and patient was booked for day 13 but eventually done on day 3 with and without contrast. CT scan revealed multiple patchy non-enhanced lesions in the bilateral thalamus and cerebral stem; there was meningeal enhancement, bilateral lateral ventricles decreased in size, no bone lesions were seen but right maxillary and sphenoid sinusitis. Based on CT scan, it was concluded that the patient had meningoencephalitis associated with multiple abscess formation.

Day 21: The following readings were obtained; phosphorous 3.2 mg/dl, potassium 3.8 mg/dl, sodium 130 mg/dl, magnesium 1.4 mg/dl.

Day 22: Wound pus swab results showed proteus species resistant to gentamycin, chloramphenicol, and ceftriaxone but sensitive to Imipenem and cefepime. Laboratory examination of the blood slide showed the presence of *Plasmodium falciparum* malaria parasites in the blood.

Day 27: The following results were obtained; sodium 134, potassium 3.3, chloride 95, urea 7.8, creatinine 22, lactate dehydrogenase 154 IU/liter, total bilirubin 31 mg/dl, alkaline phosphatase (ALP) 97 U/L, alanine transaminase (ALT) 48, aspartate aminotransferase (AST) 21 U/L, total protein (TP) 72 g/liter, white blood cells 9.52, red blood cells 2.8%, hemoglobin 8.5%, hematocrit 26%, mean Corpuscular Volume 92.9 FL, mean corpuscular hemoglobin 30.4, mean corpuscular hemoglobin concentration (MCHC) 32.7 g/dl, platelets 71%, neutrophil 80.5%, lymph 11.8%, monophils 3.2%, eosinophil 4.6%, basophils 0.3%.

Day 35: Buffy coat wet preparation made for trypanosomes showed presence of Trypanosoma species which were identified into *Trypanosoma brucei rhodesiense* in Giemsa stain.

Discussion

Our patient may be regarded as a classical presentation of *T.b. rhodesiense* case. All the clinical features, including fever, cervical lymphadenopathy, and the rash should have provided a clue for early diagnosis. And yet, the diagnosis proved to be elusive despite the fact the Lukwipa village in Rufunsa district has been generating almost all cases of *T.b. rhodesiense* since 2013.

A classical case of HAT goes through clinically distinct stages starting with development of a chancre at the site of the tsetse fly bite accompanied by regional lymphadenopathy. As the disease progresses from local symptoms to the first stage of a generalized infection, fever is one of the commonest, albeit nonspecific sign. The second stage of the disease is associated with chronic encephalopathy associated with headache and mental changes [9]. Untreated, *rhodesiense* HAT is uniformly fatal, and causes significant morbidity while mortality may ensue even with appropriate therapy [10].

The symptoms in HAT can be picked up or identified with adequate capacities in diagnosis of HAT. However, such capacities may not be readily available in resource constrained settings. Diagnostic capacities in our case were complicated by the non-availability of other diagnostic tests such as blood concentration techniques and/or polymerase chain reaction in health facilities.

This study was limited by inadequate information of the detailed history and findings about the patient.

Reliable, rapid and accurate diagnostic tools and surveillance are important in addressing the current challenges in HAT diagnosis [11,12]. There is need to build capacities both among clinicians and laboratory staff in identification of Human African Trypanosomiasis.

Conclusion

Availability of capacities, tools and expertise for accurate and rapid diagnosis coupled with information on geographical, epidemiological distribution of HAT are important aspects needed to assist health workers in making the right diagnosis for effective prevention and control of HAT in endemic areas. In addition, there is need for collaboration between heath and veterinary departments for impact, given that both domestic and wild ruminants serve as reservoirs of *T.b. rhodesiense*. There is also need to strengthen interventions which will have impact in reducing human and fly contacts, including avoidance of encroachment of human settlement sites into game parks.

Competing interests

The authors declare that there are no competing interests.

Authors' contributions: All the authors have reviewed and approved the manuscript.

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