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The Superiority of Cerebral Spinal Fluid Examination to Peripheral Blood Examination in Detecting Trypanosomes in Clinical Stage-I of Rhodesiense Human African Trypanosomiasis: A Case Report from Zambia

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ABSTRACT

Human African Trypanosomiasis (HAT) is one of the Neglected Tropical Diseases (NTDs), as classified by the World Organisation (WHO). It is transmitted by the bite of an infected tsetse fly of the genus Glossina spp. There are two forms of the disease namely, Gambiense HAT (gHAT) that is found in west, central, and parts of east Africa, and Rhodesiense HAT (rHAT), which is found in east and southern Africa.

We present a case of a patient that presented with symptoms and signs of clinical stage-I of rHAT at a rural hospital in Zambia, southern Africa. Routine laboratory investigation by examining peripheral blood from the patient revealed no trypanosomes by stained blood film light microscopy. Laboratory diagnosis of rHAT was confirmed by findings in a laboratory test for stage-II of rHAT. We give possible explanation for this observation and its relevance in the laboratory diagnosis and therefore the management of HAT in endemic area. This case underscores the relevance of cerebral spinal fluid examination for HAT diagnosis in cases where light microscopy blood film examination is negative for trypanosomes and yet clinically the patient is in stage-I of rHAT.

Keywords
Fluid, Examination, Trypanosomes.

Introduction
Human African Trypanosomiasis (HAT), also known as African sleeping sickness, is one of the diseases classified by the World Health Organisation (WHO) as Neglected Tropical Disease (NTDs) [1-3]. It is a vector-borne parasitic disease caused by a protozoan parasite, the trypanosomes, belonging to the species *Trypanosoma brucei.*_The two subspecies responsible for disease in humans are *Trypanosoma brucei rhodesiense* (*Tbr*), causing Rhodesiense HAT (rHAT) or East African HAT and *Trypanosoma brucei gambiense* (*Tbg*), causing *Gambiense* HAT (gHAT) or West African HAT. The disease, regardless of the subspecies responsible, presents clinically in two stages namely stage-I and stage-II [4-6]. In stage I, also called Hemo-lymphatic stage, the trypanosomes are confined to blood and the lymphatics. In stage-II, also Meningo-encephatilic stage, the trypanosomes have invaded the CNS. The disease progresses from stage-I (early stage) to stage II (late stage). Stage-I is confirmed by demonstrating the presence of trypanosomes in blood and lymphatics by various laboratory diagnostics methods [7-10]. Stage-II is confirmed by finding trypanosomes in the cerebral spinal fluid (CSF) and/or haematological and immunological abnormalities in the CSF [10,11]. The disease is usually fatal if left untreated [12].

The stage-I symptoms and signs of HAT are non-specific and include fever, headache, arthralgia, myalgia, malaise, pruritus, lymphadenopathy (posterior cervical for *Tbg* and generalised, if present, for *Tbr*). The Stage-II symptoms and signs include

neurological symptoms such as confusion, psychosis as in altered behaviour, disturbed sleep, tremors, convulsions, gait disturbance, ataxic dyskinesia, stupor, coma, and finally death, if no treatment instituted [5,13].

We present a case of rHAT whose clinical symptoms and signs were that of stage-I but diagnosis was confirmed with a laboratory test for stage-II of the disease.

The Case

An eight years old female patient was admitted to a rural hospital in Zambia. Her presenting complaints were headache, fever, body weakness and generally not feeling well. There was no history of convulsions, insomnia, somnolence, and personality changes. On examination, the patient was fully conscious. The general condition was good and all the body systems were normal. Since the patient came from a malaria and HAT endemic area laboratory investigations were requested on the patient to confirm or rule out these infections. A thick blood film was made from the patient and stained with Giemsa and examination under the light microscope. The blood film examination revealed no malaria parasites and no human African trypanosomes. Thereafter a lumbar puncture was done and cerebral spinal fluid collected and sent to the laboratory for parasitological and haematological examinations. Parasitological examination revealed presence of trypanosomes. A diagnosis of *r*HAT in stage-II was made. The patient was put on a 10 days course of intravenous Melarsoprol at 2.2 mg/kg body weight once per day. The patient made a full recovery after the course and was discharged from the hospital thereafter.

Discussion

The laboratory diagnosis of HAT is based on demonstrating the presence of trypanosomes in body fluids by various laboratory investigations. In stage-I of the disease trypanosomes are present in blood and the lymphatics. The laboratory investigations here includes chancre aspirate light microscopy, wet mount blood smear light microscopy, thick/thin giemsa stained blood film light microscopy, micro haematocrit centrifugation method (Woo's method), Quantitative Buffy Coat analysis (QBC), erythrocyte lysis and centrifugation, and miniature Anion Exchange Centrifugation Technique [7-10]. Stage- II of the disease is established by CSF examination after double centrifugation and detecting trypanosomes and/or finding white blood cell count equal or greater than 5 white blood cells/µL of CSF and high levels of Immunoglobulin M.

Several reasons have been brought forward to explain why trypanosomes are more easily detected in CSF than in blood in stage-II of HAT [15]. One reason is the active penetration of the blood Brain Barrier (BBB) by the trypanosomes where they cross the fenestrated endothelium of the blood vessels and enter the CSF leading to infection of the Central Nervous System (CNS). The second reason is the diagnostic method for CSF that involves double centrifugation which increases significantly the sensitivity for detection of the trypanosomes, often finding them when they are not detectable in blood. The other reason is the reduced immune clearance of trypanosomes in the CSF. The CSF lacks the high concentration of immune cells such as lymphocytes and macrophages, found in blood, which destroy the trypanosomes. For all these reasons, the CSF acts as a reservoir for trypanosomes in stage-II of HAT. Specialised centrifugation techniques of CSF make it easier to detect trypanosomes in CSF than in blood.

The observation in our case implies that CSF examination is more sensitive than Giemsa stained thick/thin blood film light microscopy in detecting trypanosomes of rHAT in patients who are clinically in stage-I and where the trypanosomes have already crossed into the CNS but without symptoms and signs of stage-II. There is, however, no direct evidence in literature indicating that CSF examination is generally more sensitive than peripheral blood examination specifically for detecting trypanosomes in stage-I of HAT. CSF examination, especially by double centrifugation, remains essential for the staging of the disease. CSF examination may detect trypanosomes when they are not detectable in blood by laboratory tests such as thick/thin giemsa stained blood film light microscopy during the transition period, from late stage-I to early stage-II of the disease, when typical symptoms and signs of stage-II have not yet developed and the patient is still exhibiting typical stage-I symptoms and signs.

Recommendation

We recommend double centrifugation of CSF examination for the detection of trypanosomes in situations where a patient has symptoms and signs of stage-I rHAT but thick/thin giemsa stained blood film light microscopic examination doesn't detect trypanosomes in rHAT endemic areas. This is especially for resource constrained rHAT endemic countries lacking more sensitive laboratory diagnostic techniques such as MAECT, molecular techniques such as Polymerase Chain Reaction (PCR) and Loop isothermal mediated Amplification (LAMP) test, and immunological tests such as immunotrypanolysis [16].

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