Second stage African Trypanosomiasis presented as Non Convulsive Status Epilepticus

Eetadal Ibrahim[1], Mohammed Gasm Elseed M Elmahal[2], Khabab Mohamed Ahmed[3], and Mohammed Omer[4]

[1]Al-Neelain University Faculty of Medicine
[2]The National center for Neurological Sciences
[3]University of Khartoum Faculty of Medicine
[4]University of Gadarif Faculty of Medicine and Health Science

November 10, 2021

Abstract

Human African Trypanosomiasis also known as sleeping sickness is a common disease in South Sudan. There are two recognized sstage, The early hemolymphatic stage and The late encephalitic stage when the CNS is involved specially with Gambians infection, broad neurologic spectrum has been reported such as psychiatric, motor, sensory.

Second stage African Trypanosomiasis presented as Non Convulsive Status Epilepticus

Etedal Ahmed A Ibrahim[12,]

,Mohammed Gasm Elseed M Elmahal[3]

Khabab Abbasher Hussien Mohamed Ahmed[4]

Mohammed Eltahier Abdalla Omer 5

1/ consultant Neurologist, National Center of Neurological Sciences, Khartoum, Sudan

.

2/ Al-Neelain University, Faculty of Medicine, Neurology department, Sudan

, 3/National Center of Neurological Sciences,

4/ Medical student, University of Khartoum Faculty of Medicine, Khartoum, Sudan.

5/ Teaching Assistant, Internal Medicine Department, Gadarif University Faculty of Medicine and Health Sciences, Gadarif, Sudan.

Key message :

Our case highlights that late neurologic complications such as epilepsy should be considered in patients with long standing history of African Trypanosomiasis specially if they developed progressive mental and behavioral changes.

Abstract

Human African Trypanosomiasis also known as sleeping sickness is a common disease in South Sudan. There are two recognized sstage, The early hemolymphatic stage and The late encephalitic stage when the CNS is

involved specially with Gambians infection, broad neurologic spectrum has been reported such as psychiatric, motor, sensory.

INTRODUCTION

Human African trypanosomiasis (HAT), also known as sleeping sickness, is caused by protozoan parasites. There are two forms of the disease: an acute form which occurs mainly in East Africa and caused by Trypanosoma brucei rhodesiense, and a more chronic form which mainly occurs in West and Central Africa, caused by Trypanosoma brucei gambiense.

Human African trypanosomiasis (HAT) is characterized by an early stage, known as the hemolymphatic stage, during which trypanosomes circulate in the blood or lymphatics, and a late stage, in which there is involvement of the central nervous system (CNS). Trypanosoma brucei gambiense causes a slowly progressive infection, and an oligo symptomatic phase can last for months or years.

Early infection (stage I) — Early symptoms of HAT infection include intermittent headache, fevers, malaise, and arthralgia. These symptoms may correspond with successive waves of parasitemia and antibody production. Hepatomegaly and particularly splenomegaly may be observed, and generalized lymphadenopathy may also be present. Other nonspecific symptoms may be present including pruritus, rash, weight loss, and facial swelling. Neuroendocrine disturbances leading to amenorrhea in women or impotence in men may also occur. The duration of this phase is approximately three years in T. b. gambiense infection. In contrast, T. b. rhodesiense presents as an acute illness with poor demarcation between stages and leading to death within months.

Late infection (stage II) — Late infection refers to involvement of the central nervous system (CNS). The death rate is 100% in the absence of treatment. Stage II is defined by an increase in the number of white blood cells (> 5 cells / microL) in the cerebrospinal fluid. Activated plasma cells with eosinophilic inclusions containing IgM, called Mott morule cells, can be seen in cerebrospinal fluid (CSF), Following early T. b. gambiense infection, progressive diffuse meningoencephalitis and parenchymal edema of the brain develop, with perivascular and meningeal inflammatory infiltrates, cerebral micro hemorrhages, and widespread multifocal white matter demyelination (Stage II). Symptoms include headache, difficulty concentrating, difficulty performing complex tasks (2 or 3 step activities), personality changes, psychosis, sensory disturbances, tremors, and ataxia. Meningismus and focal neurologic signs may occur, but are unusual. Disturbed circadian sleep / wakefulness frequently develops a cycle leading to daytime sleepiness. Seizures can occur, especially in children. The deterioration progresses gradually until the patient is in a stupor or coma.

Case description

16 year old boy from South Sudan referred to Sudan National Centre for Neurological Sciences with mental deterioration and behavioral changes for the last 9 months, to degree that he became unable to communicate with his family and inattentive to his surroundings, he had poor school performance ended by leaving school. There were no motor or sensory symptoms, and no symptoms related to other systems.

His past medical history is only significant to African Trypanosomiasis 3 years back which had been treated by combination therapy.

He has no family history of similar condition or neurological diseases.

Physical Exam:

The patient was conscious disorientated to time ,place and persons .

Abbreviated mini mental score was zero. There was no signs of meningeal irritation, intact cranial nerves, normal fundal examination. Normal upper and lower limbs motor examinations. Sensations were intact. Stable vital signs Other systems examinations were unremarkable.

Work up:

Routine laboratory investigations:

Complete haemogram was normal apart from low Hb% 8.9g\dl M.C.V 65.5fl

ESR 61mm/hour

Renal and liver function tests were normal

Blood for trypanosomiasis:

Buffy coat preparation: no trypanosome seen

Wet preparation: no trypanosoma seen

Thin blood film: no trypanosoma seen

CSF Fluid Analysis:

Cells : less than 5 cells/ml,Glucose : 8.7 mg/dl,LDH : 27 u/l

Protein : 29.3 mg/dl

Card Agglutination Trypanosomiasis Test was positive

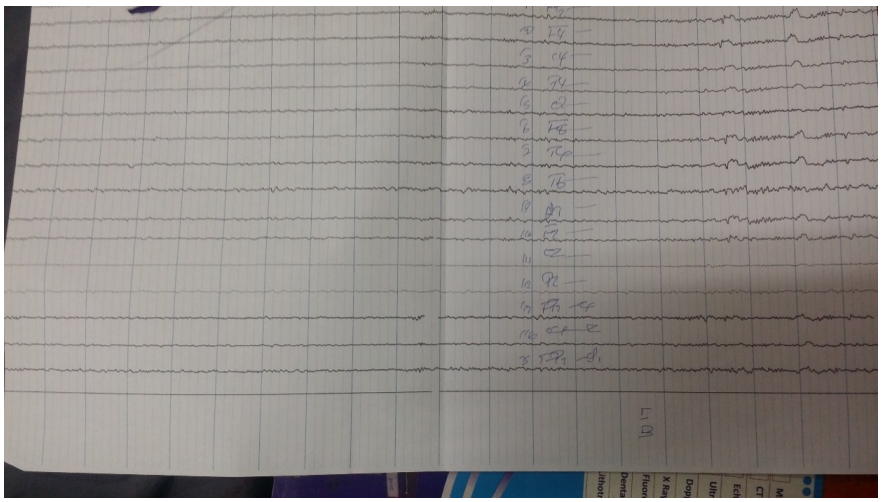
Radiological investigations:

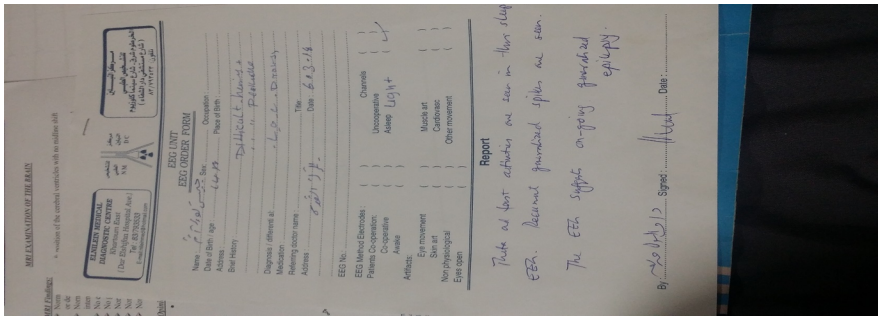
MRI Brain was normal

US abdomen: was normal apart from slightly enlarged spleen with normal texture

Electro-encephalogram EEG:

Was suggestive of on-going generalized seizure activity





Management

-Intravenous Phenytoin loading and maintenance, Oral Carbamazepine 400mg twice/day and tonics

Hospital course:

The patient showed remarkable improvement in his cognitive function and started to communicate with his family.

He had been referred back to his country as the combination therapy Nifurtimox-Eflornithine for African Trypanosomiasis treatment is not available in Sudan after separation.

Discussion

In our case we suspected late second stage African Trypanosomiasis with neurological complications such as non convulsive state as the patient presented with progressive decline of his cognitive function and our suspicious was confirmed by EEG, and supported more by remarkable cognitive improvement with Anti Convulsant thearpy.

Conclusion

Our case highlights that late second stage African Trypanosomiasis with neurological complications such as non convulsive status epilepticus should be suspected in any patient who developed progressive cognitive decline and behavioral changes following long standing history of African Trypanosomiasis and routine Electro-encephalogram EEG is the best tool to diagnose non convulsive status epilepticus.

Medical health professionals should suspect second stage African Trypanosomiasis with neurological complications such as non convulsive status epilepticus in any patient who have history of African Trypanosomiasis and developed progressive cognitive decline.

Early referral of the patients with second stage African Trypanosomiasis with progressive cognitive decline to Neurology Centers with Elector-encephalogram EEG facility might help to diagnose non convulsive status epilepticus in conjunction with early clinical suspicion of the condition will enhance the improvement of the prognosis.Further reporting of such cases is recommended.

Declarations:

Ethics approval:

Not applicable

Consent to participate:

Verbal and written consents were obtained from the patient before writing the case or using investigations.

Consent for publication:

Written consent to publish this information was obtained from the patient. The patient gave written consent for his personal clinical details along with his MRI images to be published in this study. This patient has not been reported in any other submission by the authors or anyone else.

Availability of data and material s:

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests:

The authors have no conflict of interest to declare.

Funding:

The study was funded by the authors themselves.

Authors' contributions:

EIA: The first author collected the data, analysed the results and wrote the manuscript. MGE, KH, MEO:authors wrote the manuscript, revised the manuscript and did editing. All authors read and approved the final manuscript.

References

1-WHO. Epidemiology and control of African Trypanosomiasis.

2 -Blum J.Schimds C, Burri C.Clinical aspects of 2541 patients with second stage human African Trypanosomiasis.

3-Buguet A, Bentivoglio M.Human African Trypanosomiasis: ahighly neglected neurological disease.

4-Campbell WR.Hanna MI.The clinical estimation of protein in the Cerebrospinal fluid.

5-Buscher P.Cecchi G.Jamonneauv, Priotto G.Human African Trypanosomiasis. Lancet.

.