Case Report

Fever of unknown origin due to African trypanosomiasis: A case report

Yann A. Meunier*

Honorary Member of Brazilian Academy of Medicine, Fellow of Australasian College of Tropical Medicine, Director of Stanford Health Promotion Network, USA

Abstract. We report the case of a 28 year old Greek sailor who presented first with a fever of unknown origin and then with the classical symptoms of trypanosomiasis in Ivory Coast, Greece, the USA and France but who was diagnosed late in the evolution of his disease and suffered incapacitating neurological sequelae despite appropriate treatment.

Key words: Fever, unknown origin, African trypanosomiasis

1. Introduction

African trypanosomiasis is due to *T.gambiense* South of the Sahara in West and Central Africa up to the Eastern Rift Valley and *T.rhodesiense* to the East of the Rift Valley North and South of the Great Lakes region to about 200 South latitude. It is transmitted by bites of tsetse flies (Glossina species). *T. rhodesiense* is mainly a zoonosis but humans are the principal mammalian host for *T. gambiense. T. rhodesiense* usually causes a more severe disease than *T. gambiense.* Untreated, both forms of African trypanosomiasis lead to coma and death.

2. Case report

Mr. M. D. is a 28 year old Greek sailor who is referred from a psychiatric department to rule out the possibility of trypanosomiasis because of erratic behavior with neurological symptoms and the notion of a stay in Africa in his past.

In fact, his symptoms had started 3 years earlier with febrile episodes which were treated locally as malaria bouts after a safari in Ivory Coast where he stayed for one month.

*Correspondence: Yann A. Meunier, MD
Director, Stanford Health Promotion Network
Stanford Health Prevention Research Center
1070 Arastradero Road, 3C03G
Palo Alto, CA 94304-1334, USA
Tel: (650) 721 2802 Fax: (650) 723 6450
Email: ymeunier@stanford.edu

Back in Greece, he noted some swollen lymph nodes in his neck and armpits. Hence, he consulted his general practitioner who also detected an enlarged spleen but did not make any specific diagnosis. The patient's general condition was good and he continued to work normally for another year. At that point he was admitted into a hospital in Athens for check-up. A lymph node biopsy was performed and showed atypical hyperplasia but still no diagnosis was established. Mr. M. D. was feeling somewhat tired and flew to the U.S.A. for further exploration. After a stay in a hospital he was diagnosed with atypical adenopathy. At this time more lymph nodes were enlarged, the spleen remained hypertrophied and a diffuse papular erythema emerged. At the end of the same year, another lymph node biopsy was made in Greece yielding the same result. Now psychiatric and neurological symptoms had appeared including: Erratic behavior, sleep disturbances (with diurnal sleepiness), urinary and fecal incontinence and slow ideation. For no apparent reason, Mr. M. D. was given corticosteroids. Upon the suggestion of a psychiatrist, he was referred to a tropical diseases specialist in France. At the Pitie-Salpetriere hospital, his biological check up reveals the following:

* ESR: 125 mm (first hour)
* IgM: 424 mg/dL
*Lumbar puncture: Albumin and glucose are elevated with leukocytosis
* EKG: Sinusal tachycardia and repolarization disturbances
*Bone marrow biopsy: Presence of *T. gambiense*

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Consequently, the diagnosis of African tryponosomiasis is established. The patient is treated with melarsoprol. His biological parameters all come back within normal limits but clinically both incontinences subsist.

3. Discussion

African trypanosomiasis is due to *T. gambiense* in West Africa and T. *rhodesiense* in East Africa (1). African trypanosomiasis is transmitted by glossin flies (2).

T. rhodesiense has an animal reservoir (impalas and many other domesticated and wild animals) making its eradication impossible, whereas *T. gambiense* does not (3).

In Africa, many patients with trypanosomiasis used to be found in psychiatric hospitals and asylums (4). The clinical evolution of trypanosomiasis is faster with *T. rhodesiense* than with *T. gambiense* (5). Corticosteroids accelerate the multiplication rate of trypanosomes.

In a febrile patient with a past stay in Africa after ruling out malaria a screening for trypanosmiasis should always be carried out (6).

Trypanosomiasis can be found in Ivory Coast but in West Africa it is more common in Cameroon, Gabon and the Congos (7).

The clinical picture of this patient was suggestive of trypanosomiasis with the combination of fever, diffuse lymph node enlargement, splenomegaly and skin lesions which can be seen in the first stage of the disease and psychiatric disorders and neurological troubles (in particular nyctemeral inversion) characteristic of the second phase.

The clinical differential diagnosis includes kala azar.

Even without neurological symptoms when the following biological association is present: very accelerated ESR and IgM hyperglobulinemia, one must aggressively pursue typanosoma in the blood (in particular using triple centrifugation) (8).

In trypanosomiasis IgMs are particularly elevated in the spinal fluid (9). Immunological tests include: Immunofluorescence, hemagglutination and direct agglutination or CATT (Card Agglutination Trypanosomiasis Test) (10).

Diagnosis must be made as soon as possible during the parasite hemolymphatic diffusion stage because: (a) Symptoms totally regress post treatment unlike in the second phase where sequelae can occur and (b) Drugs used at the first stage (I.V suramine or eflornithine (11, 12) and I.M. pentamidine (13) are less toxic than those used at the second stage (melarsoprol in particular) (14, 15).

Control of African trypanosmiasis is possible in West Afrika as shown by Jamot and his mobile team strategy in Cameroon (1926-1932).

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