

Thrombocytopenia as A Complication of Rhodesian Human African Trypanosomiasis in A Non-Indigenous Caucasian Patient in Zambia: A Case Report

Victor Mwanakasale^{1*}, Peter Songolo², James CL Mwansa³ and Moheb M Labib⁴

¹Copperbelt University, School of Medicine, Ndola, Zambia.

²World Health Organization, Country Office, Lusaka, Zambia.

³Apex Medical University, Lusaka, Zambia.

⁴Coptic Hospital, Lusaka, Zambia.

*Correspondence:

Victor Mwanakasale, Copperbelt University, School of Medicine, Ndola, Zambia.

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Keywords

Diseases, Caucasian patients, Zambia.

Introduction

Rhodesian Human African Trypanosomiasis (rHAT) is an invasive protozoan Neglected Tropical Disease (NTD) caused by the subspecies *Trypanosoma brucei rhodesiense*. It is a zoonosis that is transmitted to humans from infected wild and domestic animals by the bite of an infected tsetse fly belonging to the genus *Glossina* spp. Rhodesian HAT is endemic in eastern and southern Africa. Animal studies have demonstrated that thrombocytopenia is a universal complication of HAT [1]. However, only few cases of thrombocytopenia as a complication of HAT have been reported in literature [2]. We present a case report of thrombocytopenia as a complication of rHAT in a Caucasian patient and discuss possible mechanisms for this complication.

Case Report

A 19 years old female Caucasian patient was admitted to a private hospital in Lusaka, Zambia, in June 2019 with complaints of fever, headache, vomiting, and general body pains for four days. The patient gave a history of having returned from a safari in one of the game parks in Zambia 10 days prior to the onset of the symptoms. On physical examination the patient was fully conscious but febrile to touch. All systems were normal. There was an erythematous eruption on one of the legs (Figure 1). A thick blood film Giemsa stain examination was requested which revealed presence of moderate to heavy trypanosomes (Figure 2). A diagnosis of Rhodesian HAT was thus made. In addition, a Full blood count was requested on the patient which revealed a blood picture of thrombocytopenia. (platelet count less than 150,000/ μ L of blood). The patient was commenced on a full course of Suramin

(Intravenous) and made a full recovery. No antiplatelet agents were administered to the patient.



Figure 1: An erythematous eruption (Trypanosomal chancre) on one of the legs.

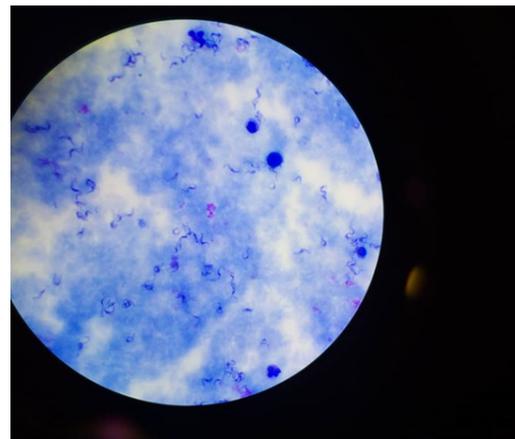


Figure 2: A thick blood film stained with Giemsa stain showing moderate to heavy trypanosomes.

Discussion

Platelets are part of the innate immune system in parasitic infections [3]. They interact with the invading parasites in the initial stages of the infection leading to the destruction of some of the parasites. However, in the late stages of the parasitic infection there is platelet activation which may lead to pathology. In both stages of parasitic infection there is depletion of platelet count.

Thrombocytopenia is a rare complication of rHAT in indigenous black Africans. The patient in our case report was a Caucasian from Europe who had lived in Zambia for about one year before contracting rHAT. From animal experiments, there are a number of mechanisms that are responsible for thrombocytopenia in rHAT. One is Disseminated Intravascular Coagulation (DIC) due to platelet activation in some patients [1,4,5]. Another possible cause of thrombocytopenia is immune damage to platelets. It has been demonstrated that thrombocytopenia is a feature of rHAT infection and is due mainly to hypertrophy of the reticuloendothelial system that accompanies this infection. Another postulated mechanism from animal experiments of thrombocytopenia in rHAT is by direct injury of platelets by the trypanosomes [1]. By this mechanism the trypanosomes might contain a protein with thrombin, fibrinogen, or collagen activity or a trypanosomal protein unrelated to other aggregating substance [1]. This protein has been demonstrated to be heat-labile and is inactivated by sonication lasting as little as 20 seconds. This protein causes remarkable platelet aggregation in animal experiments. The platelet injury in these animal experiments is independent of hemolysis, DIC, or immunologic injury related to immune adherence, complement-mediated lysis, or release of kinins. It is believed that platelet effect could be due to direct toxic effect of trypanosome protein toxin or enzyme on

the platelets without DIC. Further, release of procoagulants from injured platelets might then result in intravascular clotting and account for the DIC observed in some Caucasian patients with rHAT.

Conclusion

Caucasian patients that acquire rHAT while on safari in endemic areas of Africa should be investigated and managed appropriately for thrombocytopenia which seems to be a uniform accompaniment of this infection in this race. This is because severe thrombocytopenia may result which might lead to death due to gastrointestinal hemorrhage.

Acknowledgement

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References

1. Davis EC, Weller DR, Braude IA. Thrombocytopenia in experimental Trypanosomiasis. *J Clin Invest.* 1974; 53: 1359-1367.
2. Ottoman P, Zumwalt J, Chin J, et al. African trypanosomiasis-California. *Morbidity Mortality.* 1970; 19: 233.
3. Ana Lopez Alonso, Demot Cox. Platelet interactions with Viruses and Parasites the non-thrombotic role of platelets in Health and Disease. *Intech Open.* 2015.
4. Robin-Browne RM, Schneider J, Metz J. Thrombocytopenia in trypanosomiasis. *Am J Trop Med Hyg.* 1975; 24: 226-231.
5. Barrett-Connor E, Ugoretz RJ, Braude AI. Disseminated Intravascular Coagulation in trypanosomiasis. *Arch Intern Med.* 1973; 131: 574-577.