African trypanosomiasis: lessons for occupational health practitioners

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INTRODUCTION

Occupational medical practitioners in non-endemic areas globally, not only in southern Africa, should be aware of the exposure to, and the symptoms, diagnosis and treatment of, trypanosomiasis in workers from endemic areas in Africa. Human African trypanosomiasis (HAT) comprises two insect vector-borne protozoan diseases. Morphologically, the pathogenic trypanosome species are indistinguishable, but the two diseases differ in their tsetse fly vector species, geographic distribution, maintenance host reservoirs, and clinical features. After an initial haemolymphatic stage, without treatment, the disease invariably progresses to central nervous system invasion and death; the rapidity of progression varies according to the type of HAT. Although 97% of cases in Africa are caused by *Trypanosoma brucei gambiense* (West African trypanosomiasis (WAT) – the chronic variety), which is found in 24 countries in west and central Africa,[1,2] this report focuses on the more severe, acute type (incubation period < 1–3 weeks, occasionally longer) caused by *Trypanosoma brucei rhodesiense*. This is East African trypanosomiasis (EAT), sporadically transmitted in 13 countries in east Africa.[1] Uganda is the only country that has both types of HAT. While WAT typically has a long incubation period of several months to years in endemic populations, it is important to note that travellers acquiring this form of disease almost invariably have a more acute presentation, with an incubation period of less than a month in 75% of cases.[3]

OCCUPATIONAL RISK OF HUMAN AFRICAN TRYPANOSOMIASIS

Cases of trypanosomiasis acquired in sub-Saharan Africa by travellers from non-endemic areas in Africa, Australasia, Europe, and North and South America have been described.[4-7] A review of 68 cases reported in the literature and from personal communication over a 20-year period (1990–2010) comprised travellers to, and immigrants and occupational cases from, endemic countries in Africa.[4] The majority of cases of HAT occur in local residents in endemic areas, although these can be occupational in origin. As outlined by the World Health Organization (WHO), those most exposed to the tsetse fly and, thus, trypanosomiasis live in rural areas and are engaged in agriculture, fishing, animal husbandry, or hunting.[1] A case-control study of WAT in Guinea indicated that workers were more likely to be infected with *Trypanosoma brucei gambiense* than non-working residents, since they walked twice as far and had more occupational sites where exposure could occur, mostly in mangrove forests.[8] Although an earlier case-control study in Zambia did not show an association of infection by *Trypanosoma brucei rhodesiense* (EAT) with main occupation, there was an increased risk of infection with fishing as an auxiliary occupation.[9] The review of people returning to non-endemic areas with HAT from 1990 to 2010 revealed that, of 19 cases of WAT, eight were occupational (seven expatriates and one military case) while, of 44 cases of EAT, four were listed as occupational (three soldiers and a researcher).[4] In a review of 21 cases of EAT acquired in five African countries, who were medically evacuated to Johannesburg from 2004 to 2018, seven (33%) were occupationally exposed: two game ranchers, two involved in nature conservation, one UK soldier on a military exercise, one pilot, and one church-related traveller.[7] Amongst 49 cases of WAT and EAT diagnosed in non-endemic countries from 2011 to 2020, four (28%) of the 14 WAT and 15 (43%) of the 35 EAT cases were occupationally exposed.[10] To date, the only case of WAT seen in Johannesburg had worked in the forestry industry in Gabon.[11] She had been referred for investigation of a suspected brain tumour and the diagnosis of WAT was serendipitous.

DISCUSSION

All recent clinical and laboratory experience around trypanosomiasis in South Africa relates to evacuated EAT cases. In Johannesburg, there is a WHO-supplied regional stock of the relevant medicines, and the high level of clinical care and laboratory expertise required for successful management are available. There have been some recurring problems in the areas of initial clinical assessment, laboratory investigation, and treatment, here summarised.[7]

- Lack of local awareness of trypanosomiasis, and non-recognition of the pathognomonic skin lesion (the trypanosomal chancre) leads to persistence with ineffective treatment, such as antibiotics and antimalarials. Malaria, tick bite fever, and bacterial sepsis are frequent clinical misdiagnoses. Patients often rapidly progress to multi-organ failure during prolonged delays.

- Laboratory diagnosis problems include false-negative microscopy in early stages of illness when the parasites may be scanty, but also because suspected malaria is often misleadingly anticipated by clinicians and the laboratory.
- Clinical management problems frequently start with evacuation administrative delays, despite the need for urgent treatment; this is sometimes because medical insurance companies resist authorising emergency evacuation due to misdiagnosis, or misassessment of clinical severity.
- The medicines currently used for treatment are toxic, and ideally should be given in an intensive care unit by clinicians experienced in treating the disease; patients are often extremely ill on arrival, and ventilation and haemodialysis may be required.

Fifty years ago, three cases of EAT were described in miners who came to work in South Africa from areas in the tsetse fly belt of northern Botswana and Malawi.[12,13] The mine medical practitioners initially suspected and treated them for malaria, typhoid, or tuberculous meningitis until trypanosomes were identified in blood, bone marrow and cerebrospinal fluid (CSF), and the appropriate treatment was given. The conclusion that, in a non-endemic area, *"the diagnosis of trypanosomiasis is not always considered, because it is rare to occur in our environment"*, is still relevant for occupational physicians today.[12] What can be done to reduce the occupational risk of HAT? Working in conservation, hunting, and tourism in game reserves in transmission areas is the major work-related exposure risk for EAT, given its zoonotic nature. Tsetse flies are aggressive and bites are not easily prevented, but use of insect repellents and avoiding dark blue or black clothing, which is attractive for the flies, is advised; fortunately, only a small percentage of tsetse flies are infected. Not all EAT patients remember a tsetse bite, and not all develop the characteristic chancre. The most important advice to impart to those occupationally at risk is to be aware that disease transmission exists in that geographical location, and if they become ill with malaria-like symptoms, but have negative malaria test results, to remind the healthcare professional about the possibility of trypanosomiasis. East African trypanosomiasis is a dangerous and rapidly progressive disease, treatment is not widely available, and health insurance that will cover emergency evacuation, if required, is highly recommended.

CONCLUSION

The WHO's global health goal includes the interruption of transmission of trypanosomiasis (zero cases) by 2030, with controlling flies as the primary strategy.[1] Recently, Ebrahim and colleagues identified volatile sex pheromones in tsetse flies that control their mating behaviour, and could thus be used in tsetse fly traps to make them more effective in controlling transmission of trypanosomiasis in sub-Saharan Africa.[14] Given that it is a zoonosis involving wild game, EAT will never completely be eliminated, but reducing the already low burden of human disease is desirable. Effective drug treatment will also contribute to accelerated elimination of WAT. A major development is the replacement of the toxic injected agents, which have been the only treatment options available since the 1920s, with an effective, orally administered and safe compound, fexinidazole. The WHO now recommends this medicine for treatment of first-stage and non-severe second-stage WAT.[15] Clinical trials of fexinidazole for EAT are expected to be concluded soon.

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