Protozoan parasites of the genus Trypanosoma cause a variety of diseases in humans and animals. Sleeping sickness (human African trypanosomiasis) is a fatal disease caused by trypanosomes belonging to the species Trypanosoma brucei. Trypanosoma evansi is a widely distributed protozoan parasite that was first reported in 1885. This trypanosome occurs in Africa, the Middle East, Asia, and South America and infects a variety of large animals including horses, mules, camels, water buffaloes, cattle, and deer. The pathogenesis depends on the virulence of the trypanosome strain and on the species of host. Mortality can be high and infected animals die after fever, anemia, and emaciation.

Trypanosoma brucei rhodesiense, T. b. gambiense, and T. b. brucei are closely related and belong to one species, Trypanosoma brucei. Trypanosoma b. gambiense is genetically more distinct from the other two subspecies, which mainly differ in the presence of the serum resistance–associated gene in T. b. rhodesiense, which is absent in T. b. brucei. Trypanosoma evansi is closely related to those species that infect humans and morphologically identical to them. However, it lacks the developmental stages in the tsetse fly vector and is mechanically transmitted by biting flies. The absence of a vector stage in T. evansi is explained by the lack of maxicircles. Thus, phylogenetically T. evansi probably derived from T. brucei after having lost the maxicircles and thus cyclical transmission.

There were devastating epidemics of sleeping sickness at the beginning of the 20th century, but by the end of the 1960s the disease had been almost eliminated by large-scale control programs. However, after independence, wars and civil unrest led to a breakdown of control and a resurgence of the disease in many African countries. Today, the prevalence is estimated to be 300,000–500,000 with major outbreaks in Angola, the Democratic Republic of Congo, and southern Sudan. Sleeping sickness is restricted to the distribution of the tsetse fly vector in sub-Saharan Africa and the disease is not known outside Africa.

In this issue of the American Journal of Tropical Medicine and Hygiene, there is an important paper by Joshi and others entitled “Human trypanosomiasis caused by Trypanosoma evansi in India: the first case report.” If this proves not just to be an isolated case, the report may mark the beginning of a new chapter in the history of human trypanosomiasis. It describes not only the first case of trypanosomiasis on the Indian sub-continent, but the first case of a confirmed infection of a human by this parasite. The possibility of human infection needs to be taken seriously because T. evansi has a much wider distribution than T. brucei. The investigators conducted a careful study to confirm that the infection was by T. evansi. However, some questions have not been unequivocally answered.

How could the parasite survive in humans? Animal pathogenic trypanosomes are sensitive to human plasma components. Trypanosoma evansi would die off when introduced into the bloodstream of a human. In this first human case of a T. evansi infection, the trypanosomes had evidently developed the ability to resist this lytic activity. Two explanations can be given: 1) The parasite mutated to a form that can resist the lytic factor in the human plasma, or 2) the human host had a deficiency in the lytic factor in the plasma. In the case presented, the trypanosomes could not be isolated from the patient and thus were not available for specific biochemical and molecular investigations. However, it should be possible to test the plasma of the patient for lytic activity against different T. evansi isolates to investigate the possibility of a deficiency in the lytic factor. Such deficiencies have been shown for patients with Tangier disease who were infected with T. b. brucei. Patients with Tangier disease have very low levels of high-density lipoproteins and thus low levels of the trypanolytic factor that was reported to be apolipoprotein L-1.

Why could the parasite not be isolated in rodents? Trypanosoma evansi can usually be propagated in mice to high parasite densities. It would be expected that such T. evansi isolates from humans could also be grown in mice, especially in immunosuppressed animals. The report states that mice injected with the blood of the patient remained aparasitemic, but does not give enough detail (number of trypanosomes used for injection or the route of application) to assess why this human-infective strain of T. evansi was not able to grow in rodents.

What is to be expected in the future? The report describes one case. It is of paramount importance to find out if this was an isolated incident of infection, or the beginning of a new chapter in the history of human trypanosomiasis. The next step should be a survey of the affected district to examine blood smears for trypanosomes of people who were sick or febrile. If more cases are found, it will be important to obtain more information on the pathology of this new disease and to assess whether fatalities are likely to occur. Once the importance of the disease can be assessed, decisions can be made about control policies. However, the scenario for the future does not necessarily have to be comparable with that for human African trypanosomiasis. As long as these human-infective T. evansi strains do not invade the central nervous system and remain sensitive to standard drugs such as suramin, the new form of human trypanosomiasis will probably not develop into a major health problem. Nevertheless, the situation requires thorough investigation in the field and laboratory.

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