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Trypanosomiasis in an infant from India

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Human trypanosomiasis is endemic in Africa and South America. Human African Trypanosomiasis (HAT) also known as sleeping sickness is caused by *Trypanosoma brucei rhodesiense* (in East and South Africa) or *Trypanosoma brucei gambiense* (in West and Central Africa)[1]. Disease due to *T.b. rhodesiense* progresses rapidly over weeks while disease due to *T.b. gambiense* tends to be chronic. In both the cases, infection leads to CNS involvement and if untreated even death. American trypanosomiasis known as Chagas disease is caused by *T. cruzi*[2]. HAT is transmitted by tsetse fly while the American variety is transmitted by reduviid bugs. Human trypanosome infections like the ones seen in Africa and South America are unknown in India[3]. There have been only three documented cases of Trypanosomiasis reported from India. Two cases of self-limiting febrile illness due to *Trypanosoma lewisi* were reported in 1974[4] of an adult couple who lived in a rat-infested village and symptoms resolved without specific treatment after two to three days. Repeat blood films taken eight weeks later were found negative. Another unusual case of *Trypanosoma evansi* was reported from rural parts of Chandrapur district in Maharashtra in an adult male farmhand with recurrent febrile episodes and he responded to suramin[2].

Human infection by animal species of Trypanosoma is not seen due to presence of trypanolytic factor in human serum[2]. A one and a half month old girl staying in a rat infested house in Mumbai presented with fever, hepatosplenomegaly, thrombocytopenia and blood films teeming with multiple trypanosomes morphologically similar to *Trypanosoma lewisi*.

Case report: A one and a half month old girl born of non-consanguineous marriage presented with fever since 5 days on 1 September 2006. There was no cough, diarrhea, vomiting, refusal of feeds or lethargy. She was a full-term normal delivery with no antenatal or postnatal complications. She was on breast feeds and had received boiled cow's milk once. She had received only one dose of oral polio vaccine. She was a resident of Andheri, Mumbai and stayed in a flat that was infested by rodents. The parents were well. On examination, she was well-nourished and her vital parameters were normal. She was febrile and had hepatosplenomegaly. Other systemic examination was normal. Investigations showed hemoglobin of 8.5 g% with WBC count of 8800/mm^3 (38% polymorphs, 60% lymphocytes and 2% eosinophils). Platelet count was reduced (77,000/mm^3). Peripheral smear showed presence of trypanosomes which were suggestive of the *T. lewisi* species in view of large size of kinetoblast, pointed posterior of cell and lack of undulating membrane (Fig. 1). Serum aspartate transminase (SGPT) was 171 IU/L and total proteins were 6.8 g/dl with serum albumin of 3.3 g/dl. Total bilirubin was 0.7 mg/dl and both prothrombin time (PT) and partial thromboplastin time (PTT) were prolonged (16 & 47 sec respectively). Renal function tests and serum electrolytes were normal. In view of symptomatic trypanosomiasis, an HIV ELISA test was done to rule out immunocompromised state which was negative. After hospitalization, the child continued to have fever for 4 days which then subsided on its own. Repeat investigations after 12 days of presentation showed normal platelet count (3,78,000/mm^3) and SGPT decreased to 73 IU/L with albumin increasing to 3.5 g/dl and normalization of PT (15 sec) and PTT (33 sec). Repeated peripheral smear examination over a period of 15 days showed no trypanosomes

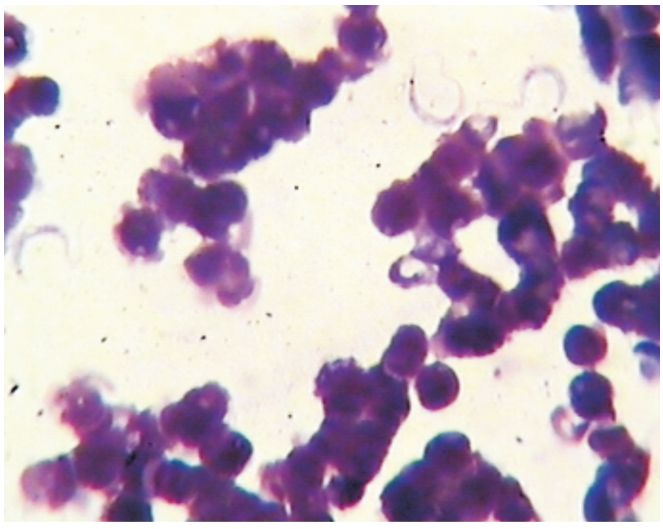

Fig. 1: Trypanosomes on peripheral blood film.

and child continued to remain asymptomatic and had regression of liver and spleen enlargement. The child was discharged and continues to do well on follow up.

Trypanosomiasis is confined to animals in India and *T. lewisi, T. evansi* have been found in camels, dogs and rodents in India[5-7]. *Trypanosoma lewisi* is a trypanosome of the sub-genus Herpetosoma and is a parasite of rats (*Rattus rattus* and *Rattus norvegicus*) tramsmitted by fleas. *Trypanosoma lewisi* has stringent species-specificity and cannot grow in other rodents such as mice. Rats are infected principally by oral route, through contamination by flea faeces or ingestion of fleas[8]. All trypanosomes of the sub-genus Herpetosoma are non-pathogenic to their vertebrate host[8]. There are only 4 cases of human *T. lewisi* around the world; 2 adults from India[4], one from Malaysia[9] and a child from The Gambia[1]. The Gambian infant had CNS infection also and responded to malarsoprol whereas the child from Malaysia who was also four month old responded without any specific treatment. In our patient also, the parasitemia resolved on its own without any specific treatment.

Humans are naturally resistant to infection by animal trypanosomes possibly because of a trypanolytic factor in the serum[10]. Also recent research suggests that for *T. brucei* an apolipoprotein leads to resistance to infection in human serum[11]. Whether similar factors are important in controlling infections with *T. lewisi* is unknown. In our patient, we tested the child for a possible infection with HIV which was negative.

The usual duration of the incubation period of *T.b. gambiense* is months whereas Herpetosoma infections develop detectable parasitemia typically within 7 to 14 days of inoculation in rodents[1]. This suggests that in our patient, transmission probably occurred due to exposure to the excreta of infected fleas in the environment in which the child was living. For early stage HAT, pentamidine is the drug of choice[12] whereas suramin is the drug of choice in *T.b. rhodesiense*. For CNS involvement, eflornithine or malarsoprol have been used[13].

Thus, to conclude, animal trypanosomiasis can infect humans given the right combination of environmental, host-related and organism-related factors and these patients should be managed diligently. Also, it will be necessary to determine whether other people in Mumbai also harbor trypanosoma infections.

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