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Perspective

Epidemiology of African Trypanosomiasis in Human Being

Xiaoming Zhang*

Department of Medicine, North Sichuan Medical College, Sichuan, People's Republic of China

DESCRIPTION

African trypanosomiasis is caused by parasites of genus *Trypanosoma* and transmitted by infected tsetse flies and is endemic in 36 sub-Saharan African countries where there are tsetse flies that transmits the disease. Without treatment, the disease is considered fatal. *Trypanosoma congolense* causes syndrome in sub-Saharan African animals. Although trypanosomiasis is an acute form, the underlying mechanism of this severity remains unclear. We developed a mouse model of brain trypanosoma using *T. congolense* strain 1/148 and characterized the cellular, behavioural, and physiological consequences of this infection. Using in vivo imaging, we show large-scale parasite isolation in cerebral blood vessels over a long period of time (up to 8 hours), with extensive brain partially caused by ICAM1-mediated T cell recruitment and accumulation causes damage. Antibodies-mediated blocking of ICAM1 and lack of lymphocytes reduce the isolation of parasites in the brain and prevent the development of trypanosomiasis in the brain. Here, we establish a new mouse model of trypanosomiasis brain and propose mechanisms by which parasite isolation, host ICAM1, and T cells play a central role in mammalian hosts. *Trypanosoma congolense* the most common and pathogenic African trypanosoma species in African domestic animals utilizes cell adhesion isolation to endothelial cells in some mammals. It is a complete intravascular parasite. Understanding the molecular basis of animal African trypanosomiasis (AAT) disease severity is important because natural infections show wide variability in pathogenesis and clinical outcomes. The disease is acute and can have a per-herd mortality rate of 70% or less or chronic. Signaling can range from mild fever, anemia and weight loss to cachexia, acute inflammatory syndrome, disseminated intravascular coagulation, and neuropathy, ultimately leading to systemic organ failure and death. In animals, necrotic and haemorrhagic lesions can occur in major organs, especially the brain, liver, and spleen, but remain chronically infected with mild signals, may be asymptomatic. The reason for this large phenotypic variation is unknown, but the discriminatory isolation of parasites may play an important role. It has been observed with other parasitic infections, including: *B. Plasmodium falciparum*, a causative agent of malaria and by causing cerebral malaria when it occurs in the brain, isolation is directly associated with the severity of the disease, malaria-related Acute Respiratory Distress-Pulmonary Syndrome. Parasite isolation usually causes an inflammatory response. Adhesion of Congolense cells to the host cell membrane causes an antibody complement cascade, increases vascular permeability and suggests endothelial damage. It has also been reported that the parasite itself releases soluble molecules such as trans- sialidades, which activates the endothelium and increases inflammation. Therefore, the physical damage caused by parasite cell adhesion and the resulting immune response of the host can affect the progression of the disease. The first mouse model of trypanosomiasis in animals to investigate the mechanism of disease in living animals characterized the distribution of parasites by taking mice vasculature, vascular diameter preference, duration of interaction between parasites and endothelial cells. In addition, the effect of infection on the development of brain endothelium and trypanosomiasis. Trypanosomiasis in the brain is associated with increased parasite isolation in the brain and T cell activation by up regulation of intercellular adhesion molecule 1 (ICAM1) expression in both endothelial and circulating bone marrow cells shown to be caused by the combination. The importance of parasite isolation provides a cellular mechanism for the development of trypanosomiasis in animals. The sequestration rate of *Trypanosoma congolense* depends on the strain and tissue, regardless of the parasite load. *T.congolense* strain 1/148, in this strain premature death was caused by the deposition of parasites in the cerebrovascular disease. Cell adhesion and isolation are often used interchangeably. Here, cell adhesion is called the physical action of parasite adhesion to the endothelium, and isolation is called the mechanism.

Correspondence to: Xiaoming Zhang, Department of Medicine, North Sichuan Medical College, Sichuan, People's Republic of China, E-mail: zhang@smc.edu.cn

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