Neuroinflammatory and Cognitive Impacts of Toxocara canis Infection in a Murine Model

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Toxocara canis causes toxocariasis, a disease that primarily infects dogs. In paratenic host, such as humans and other animals the parasite does not complete its life cycle but instead undergoes larval migration, a condition known as “larva migrans”. The larvae migrate through various organs, including the brain. Here we sought to investigate how T. canis lodges in the central nervous system (CNS) of C57BL/6 mice infected with 1,000 larval eggs after 14 days post infection. We aimed to assess larval migration patterns, characterize neuroinflammatory responses using spectral flow cytometry, evaluate neurotrophic factor alterations by ELISA, and analyze behavioral changes. Larval recovery via the Baermann-Moraes method confirmed preferential brain lodging over the cerebellum. Neuroimmunological profiling using flow cytometry revealed significant systemic leukocytosis without peripheral cell activation but pronounced inflammatory infiltration in the CNS, predominantly by activated neutrophils and eosinophils. Microglial activation indicated a local inflammatory response to larval presence. Additionally, decreased CX3CL1 levels both systemically and in the brain, along with reduced GDNF in brain tissue, suggested impaired inflammatory regulation and neuroprotection. Infected mice also exhibited signs of memory deficit. Our findings suggest that infection with T. canis induced a disruption in neuroimmune homeostasis and impaired neuroprotection, which may contribute to the observed memory deficits. These results highlight the potential for lasting neurological consequences of T. canis infection and reinforce the need for further studies to elucidate its impact on brain function and behavior.

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Key-Words: *Toxocara canis*, Neuroinflammation, Neurotrophic factors