

The ultrastructural changes in brain parenchyma of BALB/c mice infected with 1000 *Toxocara canis* eggs

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Toxocarosis is caused by ingestion of embryonated *Toxocara canis* eggs by the host such as dog, cat and accidentally by human or mouse. After ingestion of eggs by paratenic host, larvae do not mature in the intestine, but migrate via the blood circulation to the liver, lungs, left heart, from where they disseminate via systemic circulation to brain or eye. The predominant clinical picture of neurological manifestations of *T. canis* is meningoencephalitis with eosinophilic pleocytosis, transverse myelitis or cerebral vasculities. Neurotoxocarosis remains a poorly understood phenomenon and an important differential diagnosis of various neurological disorders.

This work aimed to investigate the pattern of changes in brain parenchyma of immunocompetent mice experimentally infected with Toxocara canis with using transmission electron microscopy (TEM).

For this purpose ten 6-week-old male BALB/c mice were orally infected with 1000 *T. canis* eggs. In 14 or 21 day post infection, from mice under general anesthesia, the brains were fixed by perfusion technique with mixture of 2% paraformaldehyde and 2,5% glutaraldehyde administrated through the left ventricle under pressure in 100 mm Hg. The samples of frontotemporal lobes were obtained, postfixed in 1% OsO_4, and embedded in Spurr resin. Ultrathin sections were examined using a JEM 1200 EX transmission electron microscope (TEM).

The electron microscopic images showed destruction of neurons, glial cells, and vascular compartments of the brain parenchyma. In many micrographs we observed degenerative neurons with numerous of Golgi apparatus, accumulation of lipofuscin granules and swollen other organelles in cytoplasm of these nerve cells. Astrocytes were accompanied by a large amount of collagen fibrils. In microglia cells, numerous phagolysosomes and many pinocytotic vesicles inside them were present. The perivascular space was dilated and widely communicated with the enlarged extracellular space in the neuropil. We observed the proliferation of blood vessel endothelial cells and opening the tight endothelial junctions between two adjacent cells.

We concluded that alterations in the brain of mice infected with *T. canis* could increase the risk of aggregation of blood elements in the brain parenchyma. Altered expression of tight junction protein could cause a blood-brain barrier breakdown, what may lead to interstitial oedema in the brain.