Blood-brain barrier impairment with enhanced SP, NK-1R, GFAP and Claudin-5 expressions in experimental cerebral toxocariasis

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摘要

Abstract

Infection by Toxocara canis in humans may cause cerebral toxocariasis (CT). Appreciable numbers of T. canis larvae cross the blood-brain barrier (BBB) to invade the brain thus causing CT. In the present studies, we evaluated the BBB permeability and BBB injury as assessed by the cerebral Evans blue (EB) concentration as well as by pathological changes and glial fibrillary acidic protein (GFAP) expression in T. canis-infected mice monitored from 3 days (dpi) to 8 weeks post-infection (wpi). The vasodilation neuropeptides, the expressions of substance P (SP) and its preferred binding neurokinin-1 receptor (NK-1R) as well as claudin-5 of tight-junction proteins associated with BBB impairment were also assessed by Western blotting and reverse-transcriptase polymerase chain reaction. Results revealed that BBB permeability increased as evidenced by a significantly elevated EB concentration in brains of infected mice. BBB injury appeared due to enhanced GFAP protein and mRNA expressions from 4 to 8 wpi. Leukocytes might have been unrelated to BBB impairment because there was no inflammatory cell infiltration despite T. canis larvae having invaded the brain; whereas markedly elevated SP protein and NK-1R mRNA expressions concomitant with enhanced claudin-5 expression seemed to be associated with persistent BBB impairment in this experimental CT model.