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LETTER

Experimental neurocysticercosis and demyelination

Neurocisticercose experimental e desmielinização

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Dear Editor,

We read with great interest the recent article by Moura et al, titled "Demyelination in experimental intraventricular neurocysticercosis"[1]. The authors are well-experienced with experimental neurocysticercosis, having previously published papers demonstrating inflammatory features in this model, as well as the effects of anti-parasitic drugs on the energetic metabolism of *Taenia crassiceps*[2,3,4].

In this article, the authors demonstrated the occurrence of demyelination in periventricular regions, including alterations in the hippocampus' architecture, accompanying inflammatory patterns throughout the brain. The authors proposed good insights concerning the link between inflammation and neurodegeneration. We would like to add to the discussion the fact that dementia and cognitive impairment have been well documented in patients with neurocysticercosis, including changes in episodic verbal memory, executive functions, naming, verbal fluency, constructive praxis, and visuospatial orientation[5]. Besides that, regardless of the phase of neurocysticercosis (active or inactive), the disease may lead to a broad spectrum of cognitive abnormalities, from impairment in a single domain to dementia.[6]

The authors have achieved a good laboratory system of a commonly observed clinical condition, and this welcome model may help the current knowledge of neurocysticercosis and mesial temporal lobe changes. However, a point should be observed: the ventricle enlargement induced by the presence of cysts resembling the hydrocephalus may also have played a role in the development of demyelination.

This concern is especially important because patients with neurocysticercosis and hydrocephalus may have remission of symptoms after ventricle derivation, including reversible dementia. It is well-recognized that, in experimental models, hydrocephalus itself can cause demyelination and morphological changes in the corpus callosum and hippocampus[7]. Alternatively, the study's observed demyelination may be a combination of both factors: inflammation and mechanical compression of the periventricular regions.

Finally, the authors referenced five experimental models of neurocysticercosis, but they omitted our model, in which cysts of *Taenia crassiceps* injected into the subarachnoid space of rats lead to progressive hydrocephalus and enlarged basal cisterns. In this model, the immunopositivity of aquaporin-4 is increased to compensate for cerebrospinal fluid absorption[8,9].

In both models, understanding the role of inflammation in the development of demyelination and hydrocephalus provides a roadmap for further elucidation of neurocysticercosis, a neglected but still relevant disease.

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