

# The relationship between neurocysticercosis and the development of mesial temporal lobe sclerosis

A relação entre neurocisticercose e o desenvolvimento de esclerose temporal mesial

To the editor:

The work by Oliveira, et al. published in the journal last July addressed once again the interesting question regarding the relationship of chronic calcifications suggestive of neurocysticercosis (NC) and the development of mesial temporal lobe epilepsy with hippocampal sclerosis (MTE-HS)<sup>1</sup>. The publication concluded that there is a higher incidence of brain calcifications suggestive of NC in patients with MTS-HS, when compared to a control of other epilepsy syndrome populations. This conclusion corroborates similar findings published by different groups previously<sup>2</sup>. Since neurocysticercosis is largely implicated as the cause of epilepsy in the developing world, including Brazil, our group considered this work very important as the catalyst for future discussions and research in this subject. As pointed out in the discussion session, many questions are still to be answered: What is the potential mechanism involved in the development of MTS-HS in these patients? More importantly, who is at increased risk of developing MTS-HS from NC infections? The predominant incidence of calcifications in the ipsilateral temporal lobe points towards a possible local mechanism, as mentioned in the article. In addition, repeated clinical and sub-clinical seizures, triggered during the acute NC phase appear as potential sources of initial precipitating injuries. Recent abstract presented by our group from Fortaleza-Brazil at the 2013 American Academy of Neurology meeting pointed out an increased incidence of white matter disease and lacunar

strokes in patients with calcified lesions suggestive of NC, which also raises the hypothesis that local inflammatory reactions may be associated to these findings<sup>3</sup>. It also revealed a prevalence of calcifications suggestive of NC of 2.7% in a general population who underwent brain CT scan for several different reasons, not only convulsions. Nevertheless, as stated by Oliveira et al., there is still a possibility that the calcifications identified by CT brain are not from NC. The current criteria developed by Del Brutto et al.<sup>4</sup> addresses potential active forms of NC, but we lack a more precise way of identifying intra-cerebral calcifications as “incomplete” or “halted” forms of infection by *Taenia solium*. As suggested, adding epidemiological criteria, such as contact with pigs or living in rural areas, might help. Serological and pathological studies would add to this quest. The answers for these questions will help identifying why some patients do not appear to develop the active form of NC, while others suffer from acute and late effects of this disease.

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