LETTERS

Should calcified neurocysticercosis lesions be surgically removed?

To the Editors:

The recent article by Rathore et al.[1] concluded that "calcified neurocysticercosis lesions (CNLs) are potential cause for antiepileptic drug (AED)-resistant and surgically remediable epilepsy, as well as dual pathology." We would like to comment on some of the methodologic issues that limit the clinical validity of these findings in patients with neurocysticercosis (NC).

In the absence of a control group, the coexistence of CNLs and antiepileptic drug (AED)–resistant epilepsy may have been entirely coincidental. Furthermore, several patients of this highly selected sample had other neurologic pathologies (e.g., malformations of cortical development, mental retardation, benign rolandic epilepsy, and hippocampal sclerosis), making it difficult to determine the actual role of NC as an etiology of epilepsy in these patients.

It is also unclear why certain patients were selected for surgery and which procedure may have been beneficial. Forty-five patients with CNLs were identified from a database of 3,895 patients with AED-resistant epilepsy; however, only 15 of these patients were chosen for surgery. This may have introduced significant bias because the reasons that the remaining 30 patients were not chosen for surgery were not reported. Of the 15 patients who underwent surgery, only 6 patients had removal of the calcifications (i.e., lesionectomy) alone. The remaining nine patients underwent another procedure (e.g., lobectomy) with or without lesionectomy. Therefore, it is not reasonable to assume that surgical removal of calcifications resulted in a seizure-free outcome. Furthermore, the association between seizure type and location of CNL was not reported, so it is not known how surgery actually affected the incidence of specific types of seizures.

This study presents other broader issues such as the lack of validated diagnostic criteria for NC.[2] The definition for CNL used in this study only differentiates NC from other diagnoses by the patient residing in an endemic region. This is concerning because cerebral calcifications may have other etiologies.[3,4] In this study, the majority of patients who underwent surgery had only a single CNL, making this definition particularly problematic. In addition, the existence of a dual pathology may be coincidental, since it is unclear whether any relationship exists between hippocampal sclerosis and CNLs.[4]

In general, NC has a favorable prognosis in terms of seizure recurrence.[5] Our opinion is that CNLs should not be surgically removed, with the exception of CNLs located in the hippocampus, since we do not know if the benefits of surgery outweigh the risks. The current evidence regarding the epileptogenicity of NC, and specifically the role of CNLs in AED-resistant epilepsy, is scarce and based on anecdotal cases. We agree with Rathore et al.[1] and others[6,7] that if the presence of CNLs is a cause for AED-resistant epilepsy, it is exceptionally rare. We would propose conducting a prospective cohort study to assess the association of different evolutive phases of the parasite and the development of seizures, as well as the evolution of NC cysts after medical (and/or surgical) treatment.

DISCLOSURE

The authors declare no conflicts of interest. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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