Commentary

The report by Chauhan *et al*. describes the rather peculiar and rare presentation of neurocysticercosis (NCC) in brainstem location with isolated nuclear IIIrd nerve palsy, and interestingly with rapid spontaneous clinical improvement.[1] This case highlights again the reputation of NCC as the great imitator of other neurologic disorders in endemic countries. Thus, it is important in these countries to rule out other neurologic disorders before making the diagnosis of NCC when the clinical presentation is atypical like in this case with brainstem involvement. A vascular disorder (TIA or minor stroke) is a reasonable differential or complication of NCC with such transient presentation. However, the latest hypothesis is more suggestive with the association of subarachnoid cystic lesions (particularly at the suprasellar cistern) or abnormal enhancement of basal leptomeninges,[2] which is not the case in the report by Chauhan *et al*.

NCC diagnosis can be challenging when there is no "hole-with-dot" appearance on brain imaging. A solitary ring-enhancing lesion differential diagnosis can then be very difficult especially in many developing countries where imaging and biological investigations are limited. It is therefore difficult in this setting to differentiate this lesion from brain abscess or even tumor. This is the reason why diagnostic criteria have been developed for NCC.[3] Clinicians should also keep in mind the possibility of NCC co-morbidity with other neurologic disorders in endemic countries.

Transient and recurrent CNS disorders, especially seizures and headache, are common manifestations of NCC as shown in epidemiological and clinical studies.[4-6] The pathomechanisms of such presentations are far to be well understood. Larval stage of taenia solium which is responsible of NCC, unlike other infectious agents is a complex multicellular organism which induces immunotolerance of their host, explaining the relative low perilesional edema in contrast with an often heavy infectious load. It has been shown that seizure activity is correlated to perilesional blood brain barrier breakdown, and this was attributed by authors to intermittent antigen presentation to host immune cells during remodeling of chronic lesions.[7] Brain inflammation can modify cortical excitability through biochemical and glial changes in the neuronal environment resulting from breakdown of the blood–brain barrier.[6] IL-1β and monocyte chemoattractant protein-1 (MCP-1) can powerfully modulate synaptic transmission by enhancing excitatory synaptic transmission and suppressing inhibitory synaptic transmission.[6] Therefore, we can hypothesize that dynamic and complex immune and inflammatory interactions between the parasite and his host, with long-term neuronal networks remodeling may be at the center of processes explaining transient or recurrent neurological symptoms in NCC. Further research is mandatory to clarify this issue.

NCC is considered by WHO as a neglected tropical disease, with less concern from developed countries for research on this disorder despite it is the most prevalent CNS parasitic infection worldwide with around 50,000 deaths/year. With increasing intercontinental immigration and travels, added to the fact that cysticercosis is mostly transmitted from person to person,[8] NCC tends to become a global disease.

It is of paramount interest at global and regional levels, to increase awareness among populations and health personnel about this condition. There is a reason of hope because NCC can be prevented. NCC is the only infection or process where large numbers of normal persons are commonly infected with a seizure- or headache-causing agent. For researchers in these fields, NCC represents a very interesting human model for the study of these disorders for many reasons: (1) The high prevalence of NCC in endemic areas, potentially allowing investigators to evaluate large series of patients; (2) the defined localization of the NCC lesions; (3) the existence of symptomatic and asymptomatic NCC cases; (4) the overall good prognosis of patients with single degenerating lesions or a few intraparenchymal cysts; (5) the feasibility of intervening with specific antiparasitic drugs; (6) the chronic nature of residual brain calcifications; and (7) the presence of NCC in western countries, owing to increased travel and immigration. It is important for scientists and medical professionals dealing with NCC in endemic countries to built research and collaborative networks to help reduce the burden of this disorder.

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