CORRESPONDENCE

Acute Neuroschistosomiasis: A Cerebral Vasculitis to Treat with Corticosteroids Not Praziquantel

To the Editor-in-Chief:

We read with interest the article by Houdon and colleagues reporting two patients with imported acute neuroschistosomiasis due to *Schistosoma mansoni*.

Both patients presented with neurological signs revealing acute schistosomiasis (AS), and the diagnosis of acute disseminated encephalomyelitis (ADEM) was raised to explain these symptoms. However, the diagnosis of eosinophilia-induced cerebral vasculitis appears to be more likely than that of ADEM for many reasons: patient’s histories (which started with neurological signs), clinical presentation (association with other signs), high eosinophilia (1900 and 2100/mm³, respectively), and the brain magnetic resonance imaging aspects (suggesting border zone infarcts). Indeed, ADEM is considered as a postinfectious disorder because it is usually preceded (7–14 days, 2 days to 4 weeks, according to the authors) by a febrile episode (or an antigenic challenge), most commonly related to a viral or bacterial infection (mostly nonspecific upper respiratory tract infection) or sometimes a vaccination.

The hallmark of ADEM is a demyelinization process associating acute multiple lesions in the subcortical and central white matter of both hemispheres, cerebellum, brainstem, and spinal cord, with possible involvement of basal ganglia or thalamus. Cortical gray-white junction lesions when present are not isolated but are part of more widespread lesions. Therefore, the radiological abnormalities presented in the article are not characteristic of demyelinization. In contrast, and as underlined in the discussion, they are indeed close to the abnormalities reported in one of our cases, but the aspects of border zone infarcts led us to suggest the mechanism of cerebral vasculitis not ADEM.

Of note, similar neurological signs have been observed during the course of trichinellosis, another helminthic disease leading to high eosinophilia (as in these two previously reported cases of acute neuroschistosomiasis, and small vessel thrombosis is considered as the most likely pathophysiological mechanism leading to acute neuroschistosomiasis. And this mechanism may also explain the cardiac and pulmonary complications seen during AS.

Both patients were initially treated with praziquantel (which aggravated their neurological status) and finally recovered after corticosteroids (and praziquantel). This is concordant with other studies showing that praziquantel is associated with a clinical deterioration in about 40% of the patients treated during AS. In addition, praziquantel does not prevent the occurrence of the chronic phase of schistosomiasis when given during AS. Therefore, more and more authors now recommend the use of corticosteroids in AS. According to the authors, praziquantel may be used either in combination with corticosteroids (but there are pharmacokinetic interactions leading to a 50% decrease of praziquantel plasma levels) or after corticosteroids, whereas others (including ourselves) recommend to wait for egg laying before using praziquantel.

Therefore, similarly to other diseases giving rise to vasculitis, corticosteroids must be considered as the first-line treatment of AS when patients present with neurological, cardiac, or pulmonary life-threatening complications.

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References


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