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Reply:

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Reply:

We thank Dr L. Celso Hygino da Cruz, Jr and colleagues for their interest in our article.[1] We evaluated 56 alcoholic (43%) and nonalcoholic (57%) patients affected by Wernicke encephalopathy (WE),[1] showing that signal-intensity alterations in areas considered atypical for the disease were noted only in nonalcoholic patients and always in association with the typical findings. Furthermore, we noted that contrast enhancement of the thalamus and mamillary bodies was significantly associated with alcohol abuse. However, as described in our article, contrast enhancement was also present in nonalcoholic patients.

Thiamine deficiency leads to brain lesions selectively involving brain regions with high thiamine turnover within 14–21 days.[2] The blood-brain barrier breakdown as depicted by contrast enhancement begins 7–10 days after thiamine deficiency.[3] We postulated that the higher incidence of contrast enhancement in the mamillary bodies and thalami observed in alcoholic compared with nonalcoholic patients underlines a selective anatomic susceptibility to the toxic effects of alcohol.[1] In alcoholism, the low thiamine absorption rate at the mucosal level, the impaired hepatic function, and the alcohol-related raised thiamine metabolism together may lead to the development of chronic thiamine deficiency. Thus, in alcoholism, a pre-existing status of chronic subclinical thiamine deficiency may accelerate the blood-brain barrier breakdown, resulting in a higher incidence of contrast enhancement at the time the patient is imaged.[1]

Most interesting, each of the 2 patients presented by Dr Hygino da Cruz, Jr and colleagues had peculiar patterns for WE. In fact, the first patient showed involvement of the pulvinar of the thalami associated with lesions typical of the disease, confirming that not only the medial nuclei but also the posterior (pulvinar) nuclei of the thalami may be involved in WE.[4] The second patient, a 13-year-old boy, demonstrated involvement of the putamina, which is a typical finding in pediatric patients affected by WE.[5] To explain basal ganglia selective involvement in pediatric patients with WE, we speculated that during development, the rate of thiamine-dependent metabolism is increased.[5]

In conclusion, WE shows a wide spectrum of neuroradiologic presentations, with the constant presence of typical alterations involving the mammillary bodies, thalami, and periaqueductal gray matter. At least 1 of the typical findings is invariably present in alcoholic and nonalcoholic patients.[1] Although a prevalence of contrast enhancement is observed in the alcoholic population,[1] this may be related to a pre-existing state of chronic thiamine deficiency, not just to the toxic effects of alcohol. In the clinical setting of WE, basal ganglia involvement does represent a typical finding of the disease in the pediatric age group.[5]

References

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