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Gray matter (GM) damage, in terms of focal lesions,[1] "diffuse" tissue injury, and atrophy is a well-known feature of multiple sclerosis (MS). Recently, T1-hyperintensity on unenhanced T1-weighted sequences has been found in the dentate nuclei of patients with MS with severe disability and high T2 lesion load.[2] Such an abnormality has been interpreted as an additional sign of the neurodegenerative processes known to occur in the course of MS. This report describes a patient who, despite being mildly disabled and having a low T2 lesion load and no evident brain atrophy, showed a bilateral dentate nucleus T1 hyperintensity.

The patient was a 44-year-old man who had a diagnosis of relapsing-remitting MS (RRMS) in September 1997, after 3 relapses that occurred in June 1995, March 1997, and September 1997. Brain and cord MR imaging and CSF examination were suggestive of MS. After the diagnosis, he started treatment with interferonβ-1α, with clinical stability until January 2009, when he complained of vertigo, which gradually resolved after 5 days of steroid treatment (methylprednisolone, 1 g daily intravenously). In September 2010, he entered a research protocol and underwent neurologic and neuropsychologic (Rao Brief Repeatable Neuropsychological Battery) evaluations and brain MR imaging on a 3T scanner. The neurologic examination showed bilateral nystagmus, increased deep tendon reflexes, and bilateral Babinski sign (Expanded Disability Status Scale [EDSS] score, 1.5). The neuropsychological evaluation showed a moderate decline in working memory, information processing speed, and verbal learning. MR imaging revealed an abnormality of the dentate nuclei bilaterally, which was hyperintense on unenhanced T1-weighted and hypointense on T2-weighted sequences (Fig 1*A*). Multiple hyperintense lesions in the periventricular and deep white matter were also identified on the T2-weighted scan (lesion load, 2196 mm^3) (Fig 1*B*) in the absence of gross atrophy.

T2 hypointensity in GM structures, including the thalami, basal ganglia, and dentate nuclei, has been shown to occur in patients with MS. In MS, T2 hypointensities are thought to reflect pathologic iron deposition[3] and have been related to the clinical stage of disease, the degree of clinical impairment, and the presence of cognitive deficits. Hyperintensity of the dentate nucleus on unenhanced T1-weighted MR images in these patients has been observed rarely. Recently, Roccatagliata et al[2] estimated the prevalence of this finding in 119 patients with MS and described this feature in 19.3% of them. Remarkably, T1 hyperintensity of the dentate nucleus was more frequently seen in patients with secondary-progressive MS (46% versus 8% in RRMS), in patients with severe disability (EDSS score, $\geq$4.0), and in patients with a high lesion load on T2-weighted scans and brain atrophy.

Different from these previous findings and despite the relatively long disease duration (15 years), our patient had only a mild clinical impairment, a low T2 lesion load, and no evident brain atrophy. Nevertheless, moderate cognitive deficits of the working memory, information processing speed, and verbal learning were found. The role of the dentate nucleus in cognitive processing is well known. Together with the neocerebellum, this structure is part of the executive and affective networks of the brain, which subserve attention, working memory, procedural reasoning, and salience detection.[4] This case indicates that it is also worth assessing the presence of hyperintensity on unenhanced T1-weighted scans in patients with minimally disabling MS (and possibly with benign MS), in whom it may be a sign of impaired cognition, which would call for a comprehensive neuropsychological assessment.

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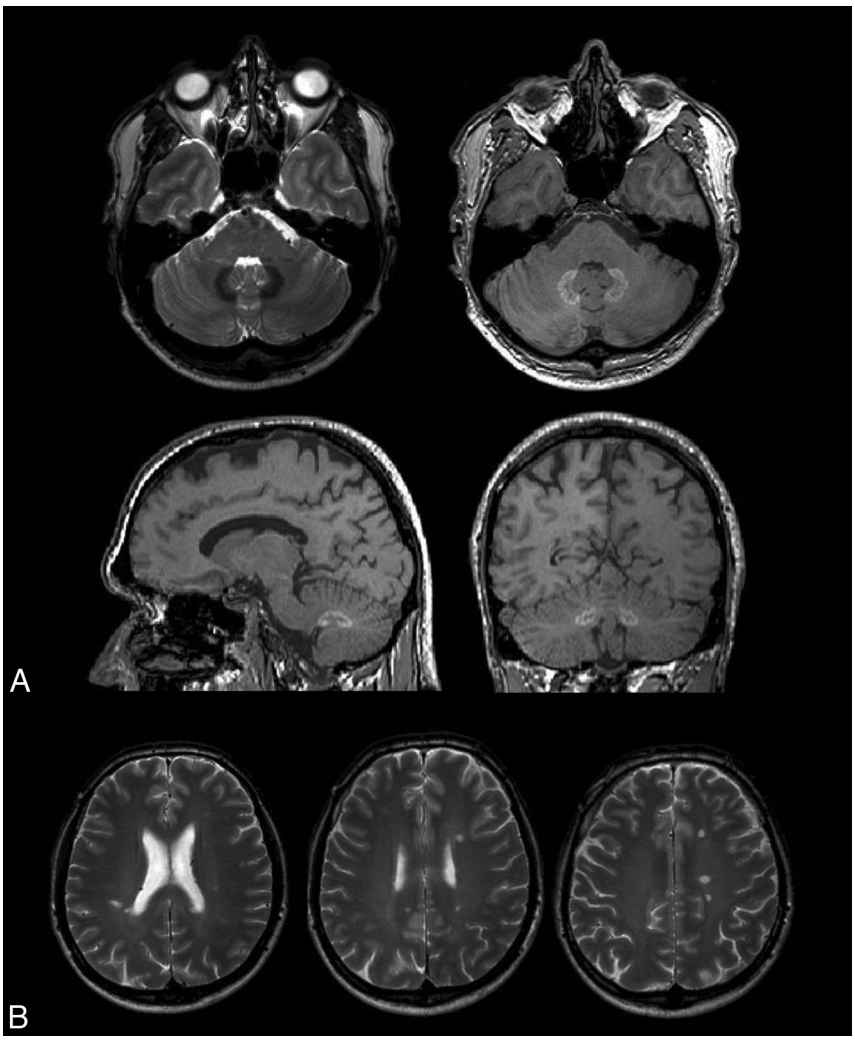

Fig 1. *A*, Hypointensity on T2-weighted and hyperintensity on unenhanced T1-weighted scans of the dentate nuclei, bilaterally, in a patient with RRMS. *B*, Multiple hyperintense lesions, mainly located in the periventricular and deep white matter, are visible on the T2-weighted sequence. These findings are congruent with the clinical diagnosis.