SOLITARY SCLEROSIS: PROGRESSIVE MYELOPATHY FROM SOLITARY DEMYELINATING LESION

Simona Lattanzi, Ancona, Italy: We observed patients with characteristics similar to those described by Schmalstieg et al.¹ and we also referred to them as having a variant of multiple sclerosis (MS). However, we also encountered cases of progressive myelopathy in the setting of demyelination of spinal cord which, despite an extensive diagnostic workup and a follow-up period of at least 5 years, did not meet the diagnostic criteria for a defined nosologic entity nor were suggestive for MS variants. There are differences between these cases and those described by Schmalstieg et al.: higher age at onset of symptoms (median 59 years, range 50–72); absence of CSF findings characteristic of MS; single or multiple spinal lesions respectively in 4 and 6 cases; and positive response to immunosuppressive treatment. We hypothesized an idiopathic progressive myelopathy of inflammatory-demyelinating nature as an entity inside the spectrum of the noninfectious idiopathic inflammatory demyelinating disorders (IIDDs) but different from those currently known. Differential diagnosis of chronic progressive myelopathy presents a major diagnostic challenge because of the many conditions in the differential diagnosis.² IIDDs classification probably could not currently be considered completed and new nosologic entities or variants

Author Response: William F. Schmalstieg, Rochester, MN: Dr. Lattanzi discusses a group of patients with progressive myelopathy apparently related to inflammatory demyelinating disease of the spinal cord. As in our series, the patients she describes did not meet diagnostic criteria for MS. In our series of patients with a single radiologically stable lesion, 4 of 7 received some type of immunosuppressive or immunomodulatory therapy and there was no history of any convincing response to therapy as described by Dr. Lattanzi. However, it is possible that patients with more recently acquired or recently active lesions could respond differently to therapy. Longitudinal follow-up in cases carefully characterized beyond the imprecise label of “progressive MS” will hopefully better inform understanding and ability to prognosticate in these cases and determine which patients might respond to existing and future disease-modifying therapies. Given the incomplete understanding of the pathogenesis of CNS inflammatory demyelinating diseases, we agree that current diagnostic criteria are “incomplete.”

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1. Schmalstieg WF, Keegan BM, Weinshenker BG. Solitary sclerosis: progressive myelopathy from solitary demyelinating lesion. *Neurology* 2012;78:540–544.
2. Canellas A, Rovira Gols A, Izquierdo Rio, et al. Idiopathic inflammatory-demyelinating diseases of the central nervous system. *Neuroradiology* 2007;49:393–409.

CORRECTION

Transcobalamin 2 variant associated with poststroke homocysteine modifies recurrent stroke risk

In the article “Transcobalamin 2 variant associated with poststroke homocysteine modifies recurrent stroke risk” by F.-C. Hsu et al. (*Neurology*® 2011;77:1543–1550), the panel labels in figure 1 are correct but there is an error in the curves. Panel A, the low dose arm, should have the curves from panel B, and panel B, the high dose arm, should have the curves from panel A. The editorial staff regrets the error.

CORRECTION

Cognitive effects of one season of head impacts in a cohort of collegiate contact sport athletes

In the article “Cognitive effects of one season of head impacts in a cohort of collegiate contact sport athletes” by T.W. McAllister et al. (*Neurology*® 2012;78:1777–1784), there is an error in the abstract. The percentage of athletes demonstrating lower than expected performance on the California Verbal Learning Test (CVLT) should read 22% rather than 24%; however, the correct percentage is reported in both the text and table 3. The authors regret the error.

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