Bithalamic Inflammation in Relapsing-Remitting Multiple Sclerosis

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Figure 1. A, T2 hyperintensities of the bilateral thalami evident at presentation. Although not pictured, subtle gadolinium enhancement was noted. B, Three months later, after treatment for MS relapse, the T2 hyperintensities had nearly resolved (and were no longer enhancing).

A 27-year-old gentleman recently diagnosed with relapsing-remitting multiple sclerosis, awaiting insurance authorization for disease-modifying therapy initiation, presented with 4 days of numbness, paresthesias, and mild left hemibody weakness, followed by dysphagia and stuttering. Examination demonstrated normal mental status, slowed and hesitant speech, mild left nasolabial flattening, decreased light touch and temperature over the left hemibody, and mild left hemiparesis. Magnetic resonance imaging (MRI) revealed prominent T2/FLAIR lesions of the bilateral thalami (Figure 1A), plus multiple new white matter lesions, without evidence of deep cerebral venous thrombosis. Due to concern that the thalamic lesions were atypical of demyelinating disease, further evaluation was performed. Cerebrospinal fluid showed 3×10^6 WBC/L, protein 31 mg/dL, and glucose

65 mg/dL. Bacterial culture of the CSF revealed no organisms. Cytology and flow cytometry of the CSF were normal. Immunoglobulin index was 0.5 and there were greater than 5 unique oligoclonal bands in the CSF. Computed tomography of the chest with contrast was also normal. He received high-dose steroids and transitioned to fingolimod as an outpatient. Repeat MRI

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3 months later showed near resolution of the bithalamic lesions (Figure 1B).

Deep gray matter lesions have been documented in acute disseminated encephalomyelitis cohorts.^{1,2} In the pediatric population, thalamic lesions have been associated with monophasic demyelinating illness more so than MS.³ Gray matter and thalamic atrophy is well described in MS, but T2/FLAIR hyperintensity is less typical.^{4,5} In fact our team had considered infection or neoplasm on the differential diagnosis. Ultimately, the patient was treated for a multiple sclerosis relapse and showed appropriate clinical improvement.

Authors' Note

Andrew R. Romeo contributed to conception of work, first draft and critical revision of the manuscript, and final approval. Maulik Shah contributed to conception of work, critical revision of the manuscript, and final approval.

Declaration of Conflicting Interests

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