Clinical remission of psoriasis during fingolimod therapy for multiple sclerosis: a case report

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Introduction: Psoriasis is a chronic skin disease resulting from the dysregulated interplay between keratinocytes and immune cells. It has not only been associated with several comorbidities like metabolic syndrome and cardiovascular disease, but also with neurologic disorders like multiple sclerosis (MS). MS and psoriasis are thought to be triggered by autoreactive T cells.

Case report: a 27-year-old Caucasian woman developed paresthesia in her left upper limb associated with Lhermitte's sign in 2007. MRI of her central nervous system revealed the presence of multiple hyperintense lesions in T2- weighted images of her brain and spinal cord consistent with a diagnosis of MS. No lesion showed enhancement following intravenous infusion of gadolinium. Cerebrospinal fluid did not contain oligoclonal bands. A new MRI performed in 2008 showed new hyperintense lesions in T2- weighted images of her brain and spinal cord and treatment with IFN- β-1a once/week was started. In 2010 she had tingling sensation in the left arm associated with strenght impairment. Two new lesions were found at brain and spinal cord MRI. The symptoms resolved with a five-day course of intravenous methylprednisolone. Neutralizing antibodies to interferon were absent and she started treatment with IFN- β-1a three times/week. In 2013 an MRI showed a new cervical cord lesion with gadolinium enhancement on T1-weighted images.She felt strenght impatiment in her left arm that resolved with steroid treatment. In that period red, scaly, raised plaques appeared at different body sites consistent with a diagnosis of psoriasis. She suspendend IFN treatment with no improvement of the psoriatic lesions. Dimethyl fumarate was not available at that time and she started treatment with fingolimod after 3 months. She noted a remission of psoriatic lesions within two months of treatment. From the MS point of view she remained clinically and radiologically stable.

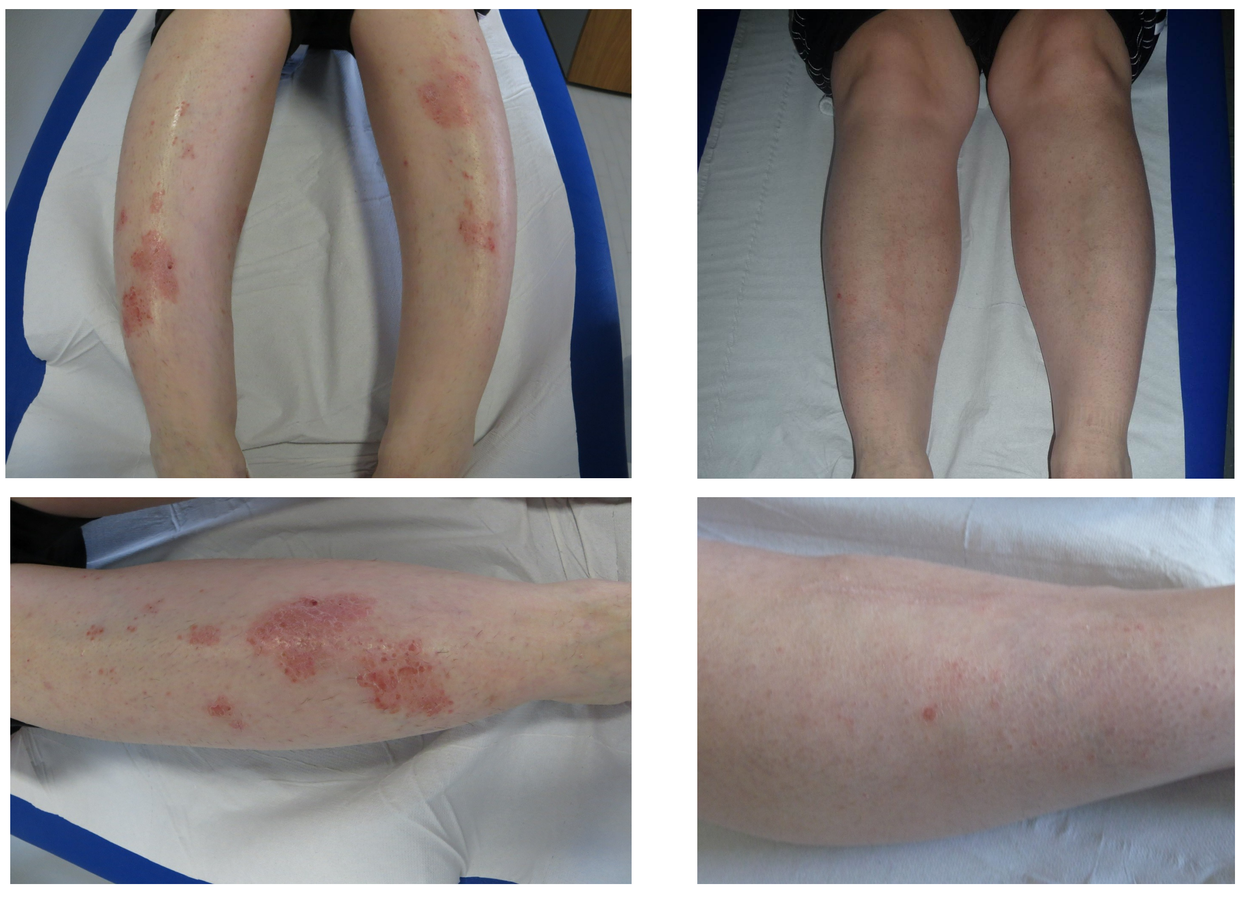

Figure 1. Psoriatic lesions during treatment with interferon-beta (left) and during treatment with fingolimod (right).

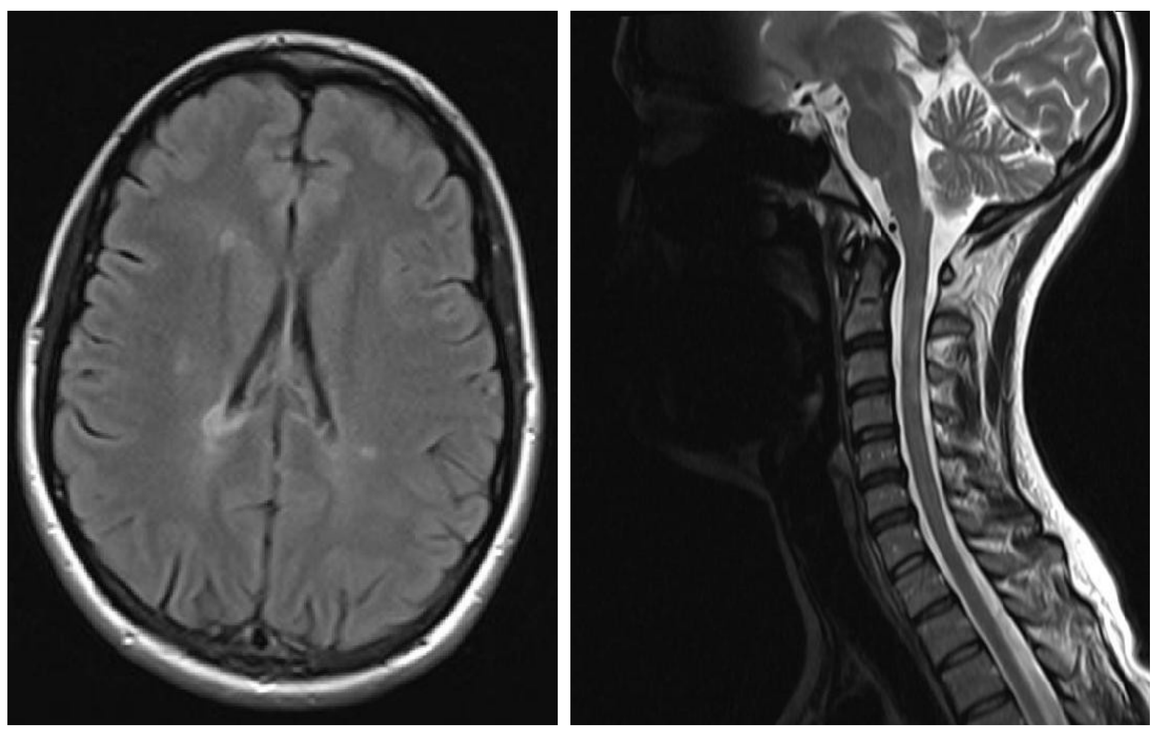

Figure 2. **Brain and cervical spine MRI showing typical MS lesions.**

Discussion: New-onset psoriatic lesions and exacerbations have been described after treatment with interferon-beta (INF-β) and natalizumab. We report the case of a patient that had a remission of psoriatic lesions with fingolimod. MS and psoriasis are both associated with pathogenic Th1 and Th17 cells. Fingolimod initially activates lymphocyte sphingosine-1-phosphate (S1P) via high-affinity receptor binding, yet subsequently induces S1P down-regulation that prevents lymphocyte egress from lymphoid tissues. Fingolimod has been shown to reduce the number of Th17 cells in peripheral blood, which are implicated in MS and psoriasis pathogenesis. Therefore fingolimod could be a good therapeutic option when MS and psoriasis coexist.

References:

1. Lowes MA, Suárez-Fariñas M, Krueger JG. Immunology of psoriasis. Annu Rev Immunol 2014;32:227-55.
2. Chun J, Hartung HP. Mechanism of action of oral fingolimod (FTY720) in multiple sclerosis. Clin Neuropharmacol 2010;33:91-101
3. Millán-Pascual J, Turpín-Fenoll L, Del Saz-Saucedo P, et al. Psoriasis during natalizumab treatment for multiple sclerosis. J Neurol 2012;259:2758-60.

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