Reversible Alopecia Associated With Glatiramer Acetate

We describe a 42-year-old woman with relapsing-remitting multiple sclerosis (MS) who developed alopecia while receiving pharmacotherapy with glatiramer acetate (GA).

The patient was diagnosed with relapsing-remitting MS in 1998 based on her clinical presentation, positive surrogate disease markers on magnetic resonance imaging, and the presence of multiple oligoclonal bands in her cerebrospinal fluid. The patient was initially treated with interferon β-1b. In 2004, GA therapy was initiated. Findings on neurological examination had been stable for the past 4 years, and her score on the Expanded Disability Status Scale1 was 0 in March 2009. The patient has tolerated GA well and repeatedly denied any adverse effects typically associated with this agent. Her medical history is otherwise significant for hypothyroidism, which was diagnosed in 1997. While receiving levothyroxine therapy, the patient has maintained euthyroid during our care for the past 4 years based on the measurements of thyroid stimulating hormone and free thyroxine serum levels.

In August 2006, the patient first noticed substantial progressive hair loss over her scalp, pubic area, axillae, and extremities. Specifically, she was concerned about her scalp hair falling out in tufts when brushing. There was no recent illness, menorrhagia, or hematochezia. Her father has a history of male-pattern balding.

The patient was referred to a dermatologist for evaluation, who documented diffuse hair loss over the scalp, largely in density, and some miniaturizing of the hair follicles anteriorly. Eyelashes and eyebrows were not affected. No hair was noted on the upper extremities. A hair pull test was negative, and a diagnosis of a non-scarring alopecia was established. A diagnosis of alopecia areata was thought highly unlikely.

The patient stopped taking GA for 3 months and reported complete remission of her alopecia. Treatment with GA was reinitiated for 6 months, and there has been no recurrence of alopecia.

This is the first report of alopecia during GA therapy. There is no evidence that alopecia is more common in patients with MS than in the general population. While an association between MS, alopecia universalis, and autoimmune thyroiditis was described by Alviggi et al,2 there has not been any laboratory evidence of inflammatory thyroid disease in this patient. Hair loss is also relatively common observed in patients with MS who receive immunosuppressive agents,3,4 which is thought to be a consequence of toxicity to the hair follicle. The fact that the patient’s alopecia was partial, localized, and fully reversible would argue against a similar underlying mechanism with GA. This case report suggests that temporary cessation of GA treatment may be sufficient in patients with MS who experience hair loss but are otherwise clinically stable.

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COMMENTS AND OPINIONS

Anxiety as Initial Diagnosis in Opsoclonus Myoclonus Syndrome

We read with interest the article by Kurian et al1 describing a young woman with opsoclonus-myoclonus syndrome (OMS) in the context of an anti–N-methyl-D-aspartate receptor (NMDAR) encephalitis. The clinical signs at presentation were speech difficulties, hypophonia, anxiety, and tremulous eye and head movement followed by increasing gait instability. She was diagnosed with depression and initially hospitalized in the psychiatric division.

We would like to describe our related experience of a young woman with the same syndrome. Four months