

and spreading of lesions to other parts of the body are frequently seen and may sometimes require systemic steroid therapy. The successful use of cyclosporine has also been reported (2). Topical PUVA photochemotherapy has been successfully applied; however, in order to achieve persistent remission, long-term topical PUVA treatment is necessary which is associated with the possibly increased risk of skin cancer development (3). High doses of UVA1 (340–400 nm) have been successfully integrated into the therapeutic approaches of treating severe atopic eczema. The marked improvements experienced in these cases were attributed to effects on IgE-bearing epidermal Langerhans' cells as well as modified secretory patterns of dermal mast cells and keratinocytes (4).

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Accepted February 5, 1998.

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Failure of Gluten-free Diet in Celiac Disease-associated Alopecia Areata

Sir,

Alopecia areata (AA) is quite a common disorder and occasionally may coincide with internal diseases. Celiac disease is the last of such alleged associations. Even its improvement with gluten-free diet has been advocated (1, 2).

A 32-year-old woman had had AA for 14 years. Initially, she had been treated with intralesional corticosteroids with temporary benefit. Scalp hairs regrew to shed again when the treatment was stopped, eventually resulting in alopecia universalis. In 1993, high titres of IgA antiendomysial (EmA) and anti-gliadin antibodies (AGA) were found. The patient, though asymptomatic, underwent endoscopy and jejunal biopsy, and celiac disease was diagnosed. Since then, she has been on a gluten-free diet for 4 years. The raised EmA and AGA titres became normal within a few months. All other laboratory tests have been within normal limits. Despite the good control of the celiac disease, no improvement of alopecia was obtained.

Her brother had high titres of IgA EmA and AGA and villos atrophy at the jejunal biopsy. For years, he had also had vesicular lesions on both elbows and back, which had been interpreted as atopic dermatitis. Direct immunofluorescence showed granular deposits of IgA at the top of dermal papillae consistent with the diagnosis of dermatitis herpetiformis (DH). DH improved with a gluten-free diet, which he followed steadily for 4 years. He was still on diet when AA developed on his legs, wrists and neck. IgA EmA and AGA were then negative; routine laboratory tests were within normal limits or negative and DH lesions were not flaring up. AA improved spontaneously about 2 months later.

DISCUSSION

The association between AA and celiac disease has been reported with 1% of patients having IgA EmA and AGA. IgG AGA were also found in 4% and 10% of AA patients tested with indirect immunofluorescence and ELISA, respectively. According to Volta et al. (2), the prevalence rate of celiac disease in patients with AA is 1 : 116, a figure higher than that of celiac disease in general population (1 : 305). In addition, three patients had a complete or partial regrowth of the hair after being submitted to the gluten-free diet (1, 2).

We confirm the possible association of the two diseases, but fail to do so for the effect of the diet. In fact, one of our patients developed AA when on diet, the other one deriving no benefit at all.

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Accepted February 16, 1998.

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