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Latent Celiac Disease or Low-Gluten-Containing Diet?

[Letters to the Editor]

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To the Editor:

We read with interest the article of Lewis et al. (1) that points out the phenomena of “self-diagnosis” of celiac disease (CD) and of “self-prescription” of a gluten-free diet in family members of a described patient.

As they correctly stated, serologic screening and intestinal biopsy today are advisable for individuals at

increased risk, including first-degree relatives of patients with CD. Nevertheless, such a policy has led to the problem of people who have, despite positive tests, normal intestinal mucosa. In a number of them, if maintained on a gluten-containing diet and undergo subsequent biopsy months or years after, histologic deterioration will develop, confirming latent CD (2). Until now, there has been no agreement on how to manage such a wait, because the natural history of subjects who screen positive and have normal histology is not completely known. Do they deserve aggressive gluten challenge to accelerate mucosal damage? Would either the trend in antibody titers or worsening symptoms suggest the timing of a second biopsy? What is the minimal degree of mucosal involvement necessary to diagnose CD when family, genetic, and immunologic data are consistent? Some of these questions are relevant to the case of a girl we recently observed.

The girl was the firstborn of a woman with CD that was diagnosed at 18 years. Under the influence of her mother's diet, the child was fed a gluten-free diet until the age of 18 months and then fed a low-gluten-containing diet (< 0.5 g/d). At the age of 3.6 years, after a positive antiendomysial test, she underwent intestinal biopsy. At admission, she complained of occasional loose stool and mild recurrent abdominal pain. She had iron deficiency and a 1-hour blood xylose test was 12 mg/dL. Immunoglobulin A and IgG antigliadin antibodies were normal. Morphology of the jejunal mucosa was normal and intraepithelial lymphocytes were $<10\%$. Despite the prescription of unrestricted gluten-containing diet, her gluten assumption remained low. Two intermediate controls in a different hospital (at the age of 4 and 4.6 years) showed regular growth (> 50 th percentile for both weight and height) and confirmed the persistence of mild gastrointestinal symptoms and iron deficiency. Immunoglobulin A, antigliadin antibodies, and antiendomysial tests were positive. Intestinal permeability test (lactulose/mannitol) was abnormal. HLA typing was DR3-DQ2 positive. On both occasions, her parents refused to give permission for a second biopsy. At the age of 7 years, she began an unrestricted gluten-containing diet, and during the following 8 months the child experienced increasing abdominal distension and complaints, perioral dermatitis, and loss of weight. When she was admitted to our institution, weight was above the 75th percentile and height was above the 50th percentile. Antiendomysial tests were clearly positive, whereas IgA and IgG antigliadin antibodies titers were borderline. Hemoglobin and serum iron levels were normal. Intestinal biopsy showed subtotal villous atrophy, increased intraepithelial lymphocyte infiltration (35%), and enlarged and hypertrophic crypts (Marsh IIIb) consistent with the diagnosis of CD. A gluten-free diet was prescribed.

In our patient, despite persistent low gluten intake and normal mucosa, subtle disturbances, serologic findings, permeability tests, and HLA typing suggested gluten intolerance. The parents' eventual adoption of an unrestricted diet for their daughter may have caused the acceleration of the disease process that allowed histologic confirmation of CD.

The amount of gluten intake is probably important in determining clinical, serologic, and histologic features of CD in genetically predisposed individuals. Population-based studies suggest that quantity and quality of cereals and the time of introduction of gluten into infants' diet may influence the severity of symptoms, offering an explanation for differences in the epidemiology of CD in neighboring countries (3). Serology correlates well with the amount of gluten intake (4) and with the degree of mucosal damage (5), and histologic effects of gluten ingestion have been reported as clearly dose-dependent in children with CD (6).

In our opinion, this case addresses two important issues. First, in evaluating close relatives of a patient with CD, one should be aware that induced dietary habits may lead to unjustified (either intentional or unintentional) gluten restriction. Second, a low gluten intake in predisposed individuals may cause diagnostic uncertainties in the histologic assessment and further prolong the “latent” phase of CD. Because some evidence suggests that diagnostic delay may increase the risk of CD-associated diseases (7), we suggest that in first-degree relatives of patients with CD, the amount of gluten intake should be carefully assessed (and, according to the circumstances, brought to an unrestricted regimen) to avoid harmfully prolonging subclinical disease.

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