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Triggering Agents for Transient Celiac Disease

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Abstract Celiac disease is a genetically determined gluten-sensitive auto-immune condition, mainly an enteropathy resulting in nutrient malabsorption. Its intestinal damages, with lymphocytic gastroduodenitis, can also be due to a myriad of gastro-intestinal infections that act as true "triggering agents". We present two pediatric patients with infectious associations leading to a transient positive celiac disease serology, with various agents and different genetic suscpetibility.

Keywords: *celiac disease, intra-epithelial lymphoctosis, HLA, Giardia, helicobacter pylori*

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1. Introduction

Celiac disease (CD) is a genetically determined gluten-sensitive auto-immune condition, mainly an enteropathy resulting in nutrient malabsorption. This latter enteropathy (with its histological lymphocytic gastroduodenitis) can also be due to a myriad of gastro-intestinal infections.

We present two pediatric patients with infectious associations leading to a transient positive CD serology.

2. Case Presentations

First Patient

A 3 years old girl is consulting for positive CD serology after family screening. Duodenal biopsy found a *Helicobacter pylori* duodenitis without CD stigma on histopathology. She is put on triple therapy and monitored clinically and immunologically every 3 months.

After 1 year of follow up, her primary serology (weakly positive IgA anti-Transglutaminase, 15 IU) returned negative, along with anti- IgG and IgA deamidated gliadin made at 12 months, also negative. She remained free of clinical signs.

Positive genetic test (presence of the haplotype HLA DQ2) made the close clinical/serological follow-up mandatory.

Second Patient

A boy of 13 years followed during 4 years in private practice for failure to thrive that would be due to CD (positive IgA anti-Transglutaminase = 18 IU, partial villous atrophy, grade: March 2).

Repeted endoscopy with histology confirmed the presence of giardiasis; and three courses of metronidazole were prescribed.

The subsequent negativity CD serology and the absence of HLA DQ2 or DQ8 allow free diet.

3. Discussion

CD is an inflammatory disease of the small intestine with a prevalence of roughly 0.5%-1% and occurs in genetically predisposed population (with Human Leukocyte Antigens HLA-DQ2 or HLA DQ8 haplotypes). It can present with both intestinal or extra-intestinal manifestations. [1]

CD can, by itself, induce lymphocytosis along the digestive tract; but duodenal lymphocytosis are more than 80% of cases due to other etiologies than true gluten intolerance; [2,3] while during the silent forms of CD (including serology positive without any clinical signs), several "triggering infectious agents" are implicated like Giardia and *H. pylori*:

Giardia lamblia is a worldwide protozoan parasite, with variable seroprevalence from 2% in developed countries to up to 30% in developing world [3]. Giardiasis may cause a clinical disease which mimics CD in both symptomatology and histopathology [4,5,6].

On the other hand, *H. pylori* is more frequently reported (and thus studied) during CD. Its controversial relationship ranges from proactive to passive infectious agent [7,8].

Particular emphasis should be given to such conditions that may induce intraepithelial lymphocytosis with preserved villosity like eosinophilic gastroenteritis, drugs, autoimmune enteropathies, different immune deficiencies, and, mostly, enteric infections. Most authors do not advocate gluten-free diet at first, but any confounding risk (i.e. HLA genetic risk) would require a tight monitoring [9,10].

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