Editorial

iMedPub Journals
www.imedpub.com

Pediatric Infectious Diseases: Open Access
ISSN 2573-0282

2021
Vol.6 No.3:12

Celiac Disease in Children

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Received: March 05, 2021; **Accepted:** March 15, 2021; **Published:** March 25, 2021

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Citation: Emma L (2021) Celiac Disease in Children.Pediatric Infect Dis Vol.6 No.3:12.

Editorial

The most prevalent genetically related food sensitivity is Celiac Disease (CD). Celiac disease is a complex autoimmune condition that affects people who are genetically predisposed to it. It's induced by a well-known environmental component (gluten and similar prolamins found in wheat, rye, and barley), and the autoantigen (the ubiquitous enzyme tissue transglutaminase) is well-known as well.

The condition primarily affects the small intestine, causing flattening of the mucosa of the small intestine.

Patients with celiac disease can be classified as having silent, prospective, or latent celiac disease. Patients with celiac disease who meet the above criteria but have no symptoms are referred to as having silent celiac disease. Asymptomatic persons who are at elevated risk for celiac disease are typically screened for such diagnoses. Patients with particular serum autoantibodies who may or may not have symptoms compatible with celiac disease but no evidence of an autoimmune insult to the intestinal mucosa are referred to as having possible celiac disease.

The so-called latent celiac disease is a last category of celiac patients: those who have normal mucosal morphology (like the prospective) but have had a gluten-dependent enteropathy at some point in their lives.

Well-known haplotypes in the Human Leukocyte Antigen (HLA) class II area (i.e, DR3 or DR5/DR7 or HLA DR4) confer genetic vulnerability to celiac disease.

Antigen-presenting cells of the mucosa (mainly dendritic cells) express such haplotypes; roughly 90% of patients express the DQ2 heterodimer, while approximately 7% of patients express the DQ8 heterodimer. Only half of the DQ2 heterodimer is present in the remaining 3% of cases.

Celiac disease can strike at any age; it's not uncommon for persons over 60 to be diagnosed. Undiagnosed celiac disease has increased considerably in the United States over the last half-century, according to data rising from 0.2% in the late 1940s to 0.9% 50 years later.

Celiac disease is an autoimmune disease in which the autoantigen tissue transglutaminase (tTG) has been identified as the target of the aberrant immune response. Gluten is the single most important environmental factor that causes celiac disease, which is linked to class II HLA DQ2 haplotypes (DR-17 or DR5/7) and, to a lesser extent, DQ8 haplotypes (haplotype DR-4). In the last few years, scientific understanding of the aetiology of celiac disease has vastly improved; the joint roles of innate and adaptive immunity are now more recognised.

Intraepithelial lymphocytes (IELs) play a crucial function in epithelial cell destruction. IELs identify non-classical Major Histocompatibility Complex (MHC)-I molecules generated on the surface of enterocytes by stress and inflammation via unique Natural Killer Receptors (NKR) expressed on their surface. These armed effector IELs become lymphokine-activated killer cells as a result of this contact, and they cause epithelial cell death in a TCR-independent way. The cytokine Interleukin (IL)-15, which is strongly expressed in celiac mucosa, contributes to this death. NKG2D has been discovered to have a critical function in celiac disease intestinal inflammation.

