

## Celiac Disease and Allergic Rhinitis: is there any Interlink?

Kajal S<sup>1</sup>, Anam Ahmed<sup>2</sup> and Alka Singh<sup>2\*</sup>

<sup>1</sup>Department of Otolaryngology and Head-Neck Surgery, All India Institute of Medical Science, India

<sup>2</sup>Department of Gastroenterology and Human Nutrition, All India Institute of Medical Science, India

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### Corresponding author:

Alka Singh,  
Department of Gastroenterology and  
Human Nutrition, All India Institute of  
Medical Science, India,  
Email: singhalka34@gmail.com

### ABSTRACT

Celiac Disease (CeD) is an autoimmune like T-cell mediated intestinal inflammatory condition caused due to the ingestion of immuno-dominant gluten protein found in wheat barley and rye [1]. It occurs in genetically predisposed individuals who display specific Human Leukocyte Antigen (HLA) molecule (HLA-DQ2 and/or HLA-DQ8). CeD is a common condition, a recent systematic review from our research group, estimated 0.7% (95% CI, 0.5–0.9%) biopsy proven CeD prevalence all over the world [2]. Only effective and accepted treatment for CeD available so far is a life-long strict elimination of gluten from the diet [3]. Rather than a primary gastrointestinal disease, CeD is a multi systemic condition that remains associated with different conditions like gluten ataxia, type 1 diabetes, thyroid rheumatized arthritis, and several other similar diseases [4]. Rhinitis is defined clinically as having two or more symptoms of anterior or posterior rhinorrhoea, sneezing, nasal blockage and/or itching of the nose during two or more consecutive days for more than an hour on most days [5]. It is called Allergic Rhinitis (AR) when these symptoms arise due to IgE mediated reaction following allergen exposure. The association between CeD and AR is not fully explored but is speculated due to shared pathophysiological mechanisms involved in the disease process [6].

In this article we have explored articles published until the year 2019 on the related issue from different electronic database like PubMed and Google scholar with the key words, ‘Rhinitis and celiac disease, Allergic Rhinitis and celiac disease’, Allergic rhinitis with coeliac disease, Allergic rhinitis in coeliac disease, Prevalence of allergic rhinitis in celiac, Rhinitis and its association with Celiac disease’, ‘Celiac disease and its association with other diseases’, ‘Association between celiac disease and Rhinitis’. Gluten is a complex protein that contains repeated sequence proline and glutamine, human’s intestinal and pancreatic enzymes lack the ability to digest these amino acid arrangements. Hence partially digested larger gluten fragments cross the lamina propria where these modified gluten fractions are deamidated by Tissue Trans-Glutaminase-2 (TGM2) enzymes. These negatively charged (de-amidated) gluten recognized HLA heterodimer that remains present at the surface of Antigen-Presenting Cells (APCs) in the gut. HLA presents these peptides, where T-cells get activated and these activated T-cells produce mainly TH1 cytokines that eventually causes villous atrophy [7].

In Allergic Rhinitis, APCs process allergens and present some peptides from allergens on the Major Histocompatibility Complex (MHC) class II molecule. This MHC class-II

molecule and antigen complex bind to T-cell receptors on naive CD4<sup>+</sup> T-cells. This results in the differentiation of naive CD4<sup>+</sup> T cells to allergen-specific Th2 cells. Activated Th2 cells secrete several cytokines [8]. Regulatory T cells are implicated in the function of both Th1 and Th2 cells. The function of these regulatory cells is deficient in allergic diseases. Patients with CeD have Vitamin D deficiency which is required for the functioning of regulatory T cells [8]. Defective functioning of these regulatory T cells in CeD may be implicated in allergic symptoms such as rhinitis in some patients.

Most T-cells express an antigen receptor integrated by  $\alpha$ - and  $\beta$ - chain. A small population of T-cells expresses a different type of T-Cell Receptor (TCR) known as the TCR- $\gamma\delta$ . These cells are seen in epithelia where they are known as  $\gamma\delta$  intraepithelial lymphocytes. An increase in the  $\gamma\delta$  lymphocytes has been implicated in intestinal hypersensitivity processes like Alimentary Allergy and characteristically CeD and also in allergic airway diseases like Allergic Rhinitis [9].

Allergic rhinitis can also be a rare manifestation of IgE mediated food allergies. In study by Mohammed W showed that patients with a history of allergic rhinitis and clinical presentation underwent specific IgE test with inhalants and food allergens. It was seen that 14% of patients were allergic to gluten [10]. In patients with Paediatric Acute-Onset Neuropsychiatric Syndrome (PANS), elimination of gluten and dairy is associated with perceived improvement of PANS symptoms. In another study conducted by Rosa JS, et al., The authors have observed that prevalence and impact of allergic and immune-mediated food disorders in PANS showed that Allergic rhinitis is more prevalent in the PANS cohort [11]. This again points towards some interlinked pathophysiology between CeD and AR. Another study by Krishna MT, et al., showed higher incident rate ratio of CeD and other autoimmune disorders in patients allergic diseases like allergic rhinitis/conjunctivitis, atopic eczema and asthma [12]. There are few studies which showed no statistically significant association between CeD and AR as compared to healthy controls [13,14]. But interlinked pathophysiology of both diseases point towards some association between two; although the prevalence of such association may be low.

Through this communication, we have given shreds of evidence that showed, there is an association between CeD and AR.

However, further bigger studies are required to confirm this association.

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