International Journal of Multidisciplinary Research and Development
Online ISSN: 2349-4182, Print ISSN: 2349-5979; Impact Factor: RJIF 5.72
Received: 08-06-2020; Accepted: 17-06-2020; Published: 09-07-2020
www.allsubjectjournal.com
Volume 7; Issue 7; 2020; Page No. 80-83

Celiac Disease: An emerging health epidemic

Rashi Pant[1*], Kirti Joshi[2]
[1] PG Student, JVW University, Jaipur, Rajasthan, India
[2] Assistant Professor, JVW University, Jaipur, Rajasthan, India

Abstract
Celiac disease, also known as "celiac sprue," is a chronic inflammatory disorder of the small intestin, produced by the ingestion of dietary gluten products in susceptible people. This is a multifactorial disease that includes genetic and environmental factors. The environmental trigger is represented by gluten, while a genetic predisposition has been identified in the main area of the histocompatibility complex. Celiac disease is not a rare disorder, as previously thought, with a global prevalence of about 1%. Although it is clear that celiac disease (CD) develops in genetically predisposed subjects exposed to gluten, the degree of other environmental factors in the pathogenesis of the disease is an area of current research. Currently, the main therapeutic intervention for CD is a gluten-free diet; however, new non-food agents are under active research. An important milestone in the history of celiac disease was the identification of tissue transglutaminase as an autoantigen, confirming the autoimmune nature of this disorder. Currently, the only treatment for celiac disease is a strict lifelong gluten-free diet that improves the quality of life, improves symptoms and prevents the occurrence of refractory celiac disease, ulcerative nausea, as well as adenocarcinoma and lymphoma of the small intestine.

Keywords: disease, Celiac, disorder, pathogenesis

Introduction

Celiac disease is a condition in which your immune system attacks your own tissues when you eat gluten. This can damage the gut (small intestine), making it unable to absorb nutrients.

Gluten is a protein naturally found in certain grains such as wheat, barley, rye and spelt. It acts as a binder, holding food together and adding a “stretchy” quality like a pizza maker tossing and stretching out a ball of dough. Without gluten, the dough would rip easily.

Celiac disease is a very complex disease based on knowledge, pathophysiology, diagnosis, management, and treatment that change over the years. The relationship between celiac disease and wheat intake was not established until 1950 [1].

Celiac disease damages the villi, leaving your body unable to absorb the nutrients needed for health and growth. The villi help your body incorporate nutrients from food into the bloodstream. Without villi, your small intestine will not be able to get enough nutrients, no matter how much you consume [25].

Triggered by gluten intake in people with a genetic predisposition, celiac disease is the most genetically widespread food intolerance in the world, the prevalence of which is approximately 1% of the total population. This disease can occur at any age with various symptoms and manifestations.

In young children, gastrointestinal manifestations are Common and include chronic diarrhea, poor growth and bloating; however, extraintestinal manifestations are becoming increasingly common.

Celiac disease is associated with another autoimmune disease. Example: thyroid, diabetes, down's syndrome etc.

This is the most important milestone in the history of celiac disease: the genetic markers HLA-DQ2 and HLA-DQ8, as well as antibodies against transglutaminase, have been identified.

The high risk of mortality from all causes of death in the aggregate reflected, for the most part, disorders characterized by immune dysfunction.

After removing wheat gliadins (alcohol-soluble toxic gluten fractions) and equivalent food prolamins in barley, rye and oats from the diet, the biopsy of the small intestine quickly improves, along with better absorption of nutrients.

India (Prevalence of Celiac Disease)

Wheat, the staple food for millions of Indians, is also responsible for celiac disease, an autoimmune disorder, among lakhs of people. The numbers are growing, but most Indians are unaware of it.

In 2002, most of the subsequent CD reports were from North India (Punjab, Haryana, Delhi, Rajasthan, Uttar Pradesh), where wheat is the main cereal in the diet (Fig.1). There are reports of a Maharashtra CD (Mumbai, n = 13) among adults; however, the geographical origin of these patients is unknown [19].

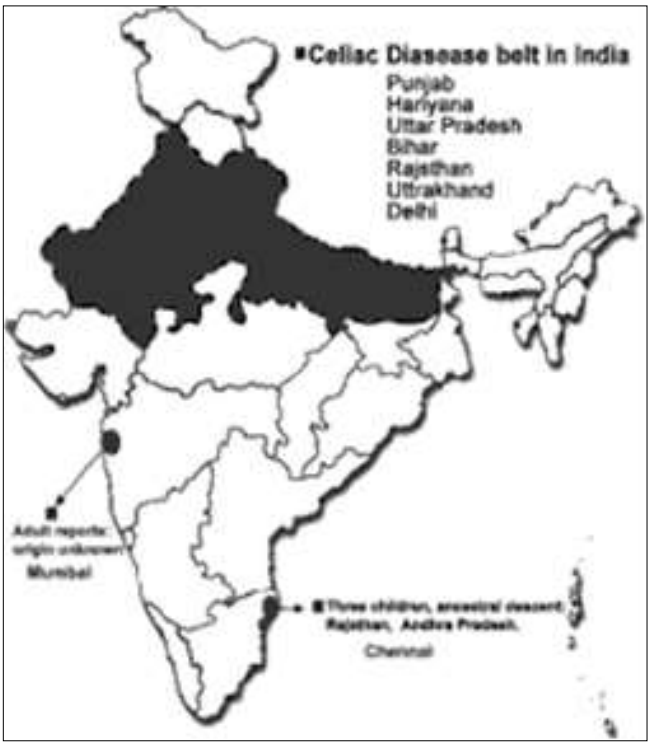

Fig 1

Three children with CD, having ancestral descent from Rajasthan and Andhra Pradesh, have been reported from Chennai. [20]

Thus, in India, there are clear regional differences in the incidence rate, which may be due to differences in the genetic predisposition to CD, differences in wheat consumption, or both [21].

In 2015, 23,331 healthy adults were selected from three regions of India: North (n = 6207), Northeast (n = 8149) and South (n = 8973) and tested for CeD using antibodies against IgG transglutaminase. Positive tests were confirmed using a second ELISA. CeD was diagnosed if the second test was positive.

The age-adjusted prevalence of celiac disease in autoantibodies was 1.23% in the north, 0.87% in the northeast and 0.10% in southern India ($p < 0.0001$). The prevalence of CeD and latent CeD, respectively, was 8.53 / 1000 and 3.70 / 1000 in the north, 4.66 / 1000 and 3.92 / 1000 in the northeast, and 0.11 / 1000 and 1.22 / 1000 in the southern part. The prevalence of the population of genes that determine the expression of HLA-DQ2 and / or -DQ8 was 38.1% in the north, 31.4% in the northeast and 36.4% in southern India [24].

In 2015, a multicenter study in which 23,000 people took part found that one out of 90 people had the disease in North India, while in South India the incidence was only 1 out of 1,000. Worldwide, from 40 to 60 million people suffer from this disease [22].

According to experts from the All India Institute of Medical Sciences, six to eight million people in India suffer from celiac disease. It is estimated that 1 in 100 people suffer from celiac disease.

Environmental Factors

Diets in the first year of life and possible viral infections (i.e. rotavirus) may be associated with the development of celiac disease. [2] A prospective study examined the role of a specific infectious agent in celiac disease, and the authors found that a higher rotavirus infection rate predicts an increased risk of autoimmune diseases in children with celiac disease. Gluten, one of the most common ingredients in human nutrition, mainly consists of prolamins and glutenin [3]. The prolamins in wheat are gliadins, rye sekalin and barley hordes.

Other key "environmental factors," such as the type of breastfeeding and the duration of lactation, can also play a role in influencing the intestinal microenvironment. In addition, an increase in the number of gram-negative and decreased intestinal bifidobacteria was found in subjects with celiac disease. Other environmental factors discussed may be represented by the heavy metals and bacterial TG present in foods [4].

Pathogenesis

Celiac disease is one of the most characteristic diseases of the immune system. All affected patients have the following:

- genetic predisposition (HLA-DQ2 or HLA-DQ8);
- clearly defined acceleration factor (gluten);
- Highly sensitive and specific autoantibodies to the natural human enzyme, tissue transglutaminase (TG2).

TG2 is an autoantigen that plays a central role in the pathogenesis of celiac disease: it enhances the immunogenicity of immunogenic gluten peptides through a chemical reaction in the small intestine (deamidation). [5-6]

Diagnosis and Treatment of Celiac Disease

Celiac disease is a chronic immune-mediated enteropathy that occurs due to dietary gluten in genetically predisposed people. The current diagnosis is based on a demonstration of enteropathy in small intestine biopsies, where histological examination shows villous atrophy, crypt hyperplasia and intraepithelial lymphocytosis, as well as the presence of circulating CeD-specific antibodies to tissue transglutaminase (tTG), deamidated gliadin peptides. (DGP) and endomysium (EMA) In children who have symptoms indicative of CeD, the very positive tTG antibody ttG (tTGA) and the HLA genotype associated with CeD, the diagnosis of CeD may be possible without a small bowel biopsy. Since gluten has been defined as a trigger for CeD since the 1950s, a strict gluten-free diet (GFD) has been the basis of treatment [7, 8].

Symptoms

Children

- Persistent diarrhoea or constipation
- Pale, fatty, foul-smelling stools
- Weight loss
- Vomiting
- No growth of height
- Abdominal bloating and pain

Adults

- Iron-deficiency anaemia
- Joint pain and stiffness
- Weak, brittle bones
- Fatigue and seizures
- Skin disorders
- Numbness and tingling in the hands and feet
- Tooth discolouration or loss of enamel
- Pale sores inside the mouth

- Irregular menstrual periods [23]

Diagnosis

The classic manifestation of celiac disease with symptoms related to the gastrointestinal tract may represent only a fraction of the cases. Now we understand that celiac disease is actually a multisystem disorder that varies greatly in clinical expression, can occur at any age and can manifest itself in various manifestations. The diagnosis is often delayed for these reasons. Gastrointestinal manifestations may include diarrhea, weight loss, stunted growth, stunted growth, abdominal pain, bloating and bloating, anorexia, vomiting and constipation. On the other hand, patients with celiac disease often have extraintestinal manifestations with minimal gastrointestinal symptoms. They may be presented to the appropriate general practitioner or sub-specialist with one or more manifestations, including iron deficiency anemia, stunted growth, osteoporosis, vitamin deficiency or fatigue. They may be presented to obstetrics / gynecology with delayed puberty, infertility or intermittent fetal loss [9, 10, 11].

Celiac disease can be associated with autoimmune endocrine disorders, such as thyroiditis and type 1 diabetes mellitus. It is associated with neuropsychiatric conditions such as depression, anxiety, peripheral neuropathy, ataxia, and epilepsy. These patients may experience an asymptomatic increase in transaminases, and an unusual association of celiac disease is associated with chronic liver disease and non-cirrhotic portal fibrosis [12, 13].

Diagnostic Tests of Celiac Disease

Serological Tests

Anti-tissue transglutaminase antibodies: The best strategy for serological diagnosis is the detection of antibodies against transglutaminase (tTGA) using an enzyme-linked immunosorbent assay (ELISA). These antibodies show sensitivity up to 97%, specificity about 96% and accuracy up to 98%, while anti-endomysial IgA antibodies (EMA IgA) are used as a confirmatory test in positive cases of tTGA due to its highest specificity (approximately 100% against 91% tTGA) [14].

Anti-gliadin antibodies: Antigliadin antibodies (AGA) (IgG and IgA) are no longer recommended due to their low sensitivity, specificity and low accuracy, with the exception of young children.

Deamidated gliadin peptides: The actual detection of antigliadin antibodies has been replaced by recently developed immunoassays using antibodies against deamidated gliadin peptides, IgA and IgG. To improve diagnostic accuracy, in recent years, doctors have usually prescribed serial tests [15].

Treatment

The cornerstone of celiac disease treatment is a gluten-free diet. Patients should avoid all offensive crops. The clinical response to gluten withdrawal is rapid, usually within the first month. The normalization of lesions of the mucous membrane of the small intestine may require longer periods. Failure to improve suggests resistant celiac disease and usually indicates that an unidentified or ubiquitous source of gluten is present in the diet. If compliance has been rigorous, the initial diagnosis may be incorrect or there may be a second partial cause of clinical symptoms. In some patients, especially those with an initial clinical response to a gluten-free diet, recurring symptoms may reflect an associated disorder (i.e., collagen or lymphocytic colitis) or a complication (i.e. lymphoma) [26].

Main conditions associated with celiac disease

Many conditions may be associated with celiac disease. The term "concomitant conditions" refers to the conditions most commonly found in patients suffering from celiac disease. These conditions include "genetic disorders" such as Down syndrome, Turner syndrome and Williams syndrome, as well as "autoimmune" or "neurological" disorders [16].

Type 1 Diabetes: One of the most recognized and widely studied disorders associated with celiac disease is type 1 diabetes. Approximately 5% to 10% of patients with type 1 diabetes had antibodies associated with celiac disease and up to 75% had tissue disorders small bowel biopsy. The prevalence of celiac disease varies from 1% to 19% in patients with type 1 diabetes [27].

Autoimmune thyroid diseases: A higher prevalence (almost 2% -5%) of thyroid diseases (i.e. hyperthyroidism, Graves' disease or hypothyroidism, Hashimoto's thyroiditis) diagnosed before or after the diagnosis of celiac disease has been reported. celiac enteropathy [28].

Autoimmune hepatitis and other forms of liver damage: Hepatic involvement is common in patients with celiac disease. Hypertransaminasemia occurs in approximately 40% of adults and 54% of children with a classic diagnosis of celiac disease when diagnosed. Conversely, celiac disease occurs in approximately 9% of patients with unexplained chronic hypertransaminasemia [29].

Neurological Disorders: A possible association between celiac disease and various neurological disorders has been reported. Data on the relationship between neurological conditions and celiac disease remain scarce. Although ataxia is a neurological disorder indicated in some patients with celiac disease, the most common neurological condition in subjects with celiac disease is epilepsy, the prevalence of which ranges from 1.2% to 5% [30].

Products for inclusion in the diet

Foods such as meat, fish, fruits, vegetables, rice and potatoes without additives or seasonings are gluten-free and are part of a well-balanced diet. You can eat types of bread, pasta, and other gluten-free products that are now easier to find in stores, restaurants, and food companies. Instead of wheat flour, you can also eat potato, rice, soy, amaranth, quinoa, buckwheat, or bean flour.

In the past, doctors and nutritionists did not recommend eating oatmeal if you had celiac disease. Available evidence suggests that most people with this disease can safely eat moderate amounts of oats, if they do not come in contact with wheat gluten during processing, ask a nutritionist about whether to include oats in your diet.

Gluten-free foods are generally more expensive than those containing gluten [17].

Conclusion

Celiac disease is undoubtedly still a very controversial and complex human disease. Various serological tests are available to help diagnose with high sensitivity and specificity; However, screening for asymptomatic patients is not yet recommended. Obviously, the only effective way to

improve currently unsatisfactory diagnostic indicators is through wider detection. Since there is no obvious biological reason for age and territorial differences in criteria, it would be desirable to develop more uniform evidence-based global guidelines for celiac disease. Dietary management improves symptoms.

References

1. Dicke WK. Coeliac Disease: Investigation of the Harmful Effects of Certain Types of Cereal on Patients Suffering from Coeliac Disease. M.D. Thesis, 2nd English translation; Utrecht University, The Netherlands, 1950, pp 97.
2. Troncone R, Auricchio S. Rotavirus and celiac disease: clues to the pathogenesis and perspectives on prevention. J Pediatr Gastroenterol Nutr, 2007; 44:527-528.
3. Stene LC, Honeyman MC, Hoffenberg EJ, Haas JE, Sokol RJ, Emery L, *et al*. Rotavirus infection frequency and risk of celiac disease autoimmunity in early childhood: a longitudinal study. Am J Gastroenterol, 2006; 101:2333-2340.
4. Pinier M, Fuhrmann G, Verdu EF, Leroux JC. Prevention measures and exploratory pharmacological treatments of celiac disease. Am J Gastroenterol, 2010; 105:2551-2561. quiz 2562. [PubMed] [Google Scholar]
5. Detlef Schuppan, Prof. Dr. med. Dr. rer. nat.1 and Klaus-Peter Zimmer, Prof. Dr. med.*,
6. Stefano Guandalini, MD1,2,3; AsaadAssiri, MD2 (JAMA Pediatr. 2014; 168(3):272-278. doi:10.1001/jamapediatrics.2013.3)
7. Ludvigsson JF, Leffler DA, Bai JC, Biagi F, Fasano A, Green PH, *et al*. The Oslo definitions for coeliac disease and related terms. Gut, 2013; 62:43-52. 10.1136/gutjnl-2011-301346 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
8. Husby S, Koletzko S, Korponay-Szabo IR, Mearin ML, Phillips A, Shamir R, *et al*. European society for pediatric gastroenterology, hepatology, nutrition guidelines for the diagnosis of coeliac disease. J Pediatr Gastroenterol Nutr, 2012; 54:136-60. 10.1097/MPG.0b013e31821a23d0 [PubMed] [CrossRef] [Google Scholar]
9. Celiac disease: Can we avert the impending epidemic in India? BS Ramakrishna Department of Gastrointestinal Sciences, Christian Medical College, Vellore 632 004, India
10. Di Biase AR, Colecchia A, Scaioli E, Berri R, Viola L, Vestito A, *et al*. Autoimmune liver diseases in a paediatric population with celiac disease - a 10-year single-centre experience. Aliment Pharmacol Therm 2010; 31:253-60. [PUBMED] [FULLTEXT]
11. Eapen CE, Nightingale P, Hubscher SG, Lane PJ, Plant T, Velissaris D, *et al*. Non-cirrhotic intrahepatic portal hypertension: associated gut diseases and prognostic factors. Dig Dis Sci, 2010. [Epub ahead of print] PubMed PMID: 20499175.
12. The diagnosis of celiac disease is based on the presence of serologic tests (ELISA) confirming the presence of antibody to tissue transglutaminase (anti-tTG).
13. NIH consensus development conference on celiac disease. NIH Consens State Sci Statements, 2004; 21:1-23.
14. Volta U, Villanacci V. Celiac disease: diagnostic criteria in progress. Cell MolImmunol, 2011; 8:96-102. [PMC free article] [PubMed] [Google Scholar]
15. Rubio-Tapia A, Hill ID, Kelly CP, Calderwood AH, Murray JA. ACG clinical guidelines: diagnosis and management of celiac disease. Am J Gastroenterol, 2013; 108:656-676. quiz 677. [PMC free article] [PubMed] [Google Scholar]
16. Pietzak M. Celiac Disease, Wheat Allergy, and Gluten Sensitivity: When Gluten Free Is Not a Fad. Journal of Parenteral and Enteral Nutrition, 2012; 36:68S-75S.
17. Marcason W. Is there evidence to support the claim that a gluten-free diet should be used for weight loss? Journal of the American Dietetic Association. 2011; 111(11):1796.
18. Prevalence of Adult Celiac Disease in India: Regional Variations and Associations**.**
19. Ramakrishna BS, Makharia GK, Chetri K, Dutta S, Mathur P, Ahuja V, *et al*. Adult celiac disease in northern India**.** Indian J Gastroenterol. 2003; 22(4):124-6.
20. Sathiyasekeran M, Shivbalan SO. Celiac disease in Chennai. Indian Pediatr, 2005; 42:1171-2
21. Celiac disease in India: Surender Kumar Yachha, Ujjal Poddar Department of Pediatric Gastroenterology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow
22. 'One in 90 people in north India suffer from celiac disease' By Namita Kohli | September 11, 2017.
23. Celiac disease more common in North India as compared to Southern, Eastern regions by Somitra Ghosh. Published: 16th February 09:06 am, 2020.
24. Thakur B, Mishra P, Desai N, Thakur S, Alexander J, Sawant P, *et al*. Profile of chronic small-bowel diarrhea in adults in Western India: a hospital-based study. Trop Gastroenterology.
25. https://www.hopkinsmedicine.org/health/conditions-and-diseases/celiac-disease
26. BCMJ. Clinical Articles By: Hugh Freeman, MD, FRCPC, FACP, FACG. 2001; 43(7):390-395
27. Ludvigsson JF, Ludvigsson J, Ekbom A, Montgomery SM. Diabetes Care. 2006; 29(11):2483-8.
28. Celiac disease and autoimmune thyroid disease. Ch'ng CL, Jones MK, Kingham JG Clin Med Res. 2007; 5(3):184-92. [PubMed] [Ref list]
29. The liver in celiac disease. Rubio-Tapia A, Murray JA Hepatology. 2007; 46(5):1650-8. [PubMed] [Ref list]
30. Neurological complications of coeliac disease: what is the evidence? Grossman G Pract Neurol. 2008; 8(2):77-89. [PubMed] [Ref list]