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Mini review

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The primary nutritional approach in pediatric celiac disease: a mini-review

Abstract

Background: Celiac disease (CD) is chronic small bowel inflammation with villous atrophy, consequently, an immune-mediated disorder triggered by ingesting Gluten in wheat, barley, and rye in genetically susceptible individuals.

Aims: Summarize the fundamental principles for managing the diet of patients with CD. This Mini-Review using relevant articles in PubMed's online databases followed the medical keywords Celiac Disease, Gluten, gluten-free diet, nutritional approach, and diet adherence. The institution's Ethics Committee has waived the need to review the ethical aspects of this text. The cornerstone of the treatment is the lifelong adherence to a Gluten-Free Diet (GFD). This dietary regimen, which forms the basis of the patient's treatment, requires eliminating all food components containing Gluten and its derivatives from the diet. However, the treatment approach has changed from focusing on food avoidance to a wide-ranging evaluation of the various factors influencing the dietary choices in the patient's life. The Global Approach to the Follow-up of Celiac Disease is based on 1) control of symptomatology and dietary adherence; 2) improving emotional and social wellness in treating CD patients, with the active involvement of a psychologist; and 3) ensuring successful education of both the child and the family about its management. Indeed, the impact of CD extends beyond the patient and affects the well-being of caregivers and 4) evaluate socioeconomic burden, considering that Gluten-free food products remain significantly more expensive than gluten-containing equivalents with variable availability. So, the GFD is the primary treatment for CD, and difficulties in managing the Disease can lead to impaired Quality of Life. **In conclusion**, strict adherence to the GFD is imperative, requiring a multidisciplinary team involving physicians, dietitians, and psychologists on a regular visit schedule to assess dietary adherence and avoid long-term complications.

Keywords: celiac disease, gluten, gluten free diet, nutritional approach, diet adherence

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Abbreviations: CD, celiac disease; CDAT, coeliac dietary adherence test; FDA, food and drug administration; GFD, gluten-free diet; SDE, standardised dietitian evaluation; SGA, subjective global assessment; tTG, tissue transglutaminase

Introduction

Background: Hippocrates' famous quote, "Let food be thy medicine and medicine be thy food," has a profound historical significance. It reminds us that since ancient Greek and Roman times, 'disease' has been used to denote physical imbalance, and food and diet have been considered crucial to restoring this imbalance. CD is chronic small bowel inflammation with villous atrophy, consequently an immune-mediated disorder. It is generated by Gluten, which is part of the composition of the wheat, barley, and rye and their by-products in genetically susceptible children/adults carrying the HLA-DQ2 and HLA-DQ8 gene, with the highest risk observed in individuals who are homozygous for DQ2. The estimated 1% prevalence occurs more in children than in adults.[1,2] An increasing prevalence has been observed in the previous thirty years.[3] Early diagnosis of CD is based on clinical and serological tests, villous atrophy on duodenal histology, and the positive response to a GFD. The early diagnosis empowers healthcare professionals to intervene effectively and improve patient outcomes. In pediatric patients, CD is associated with gastrointestinal clinical symptoms (diarrhea, steatorrhea, flatulence, abdominal pain, and distension and malabsorption-associated complications) and extraintestinal symptoms (mainly malnutrition, failure to thrive, anemia). The clinical symptoms correlate with histological findings and Anti-Tissue Transglutaminase Antibodies.[4–7]

Aims: This mini-review will explore the basic principles of the nutritional approach in Celiac Disease (CD), focusing on the specific dietary interventions that offer promising benefits of a Gluten-Free Diet (GFD). This Mini-Review using relevant articles in PubMed's online databases followed the medical keywords Celiac Disease, Gluten, gluten-free diet, nutritional approach, and diet adherence. The institution's Ethics Committee has waived the need to review the ethical aspects of this text.

Gluten and the development of celiac disease

Table 1 displays the main components of the development of CD. The central environmental factor is Gluten, a mixture of prolamin proteins composed of gliadins and glutenins. They are predominantly found in wheat but in other cereals with specific designations: barley (hordeins), rye (secalins), and oats (avenins). They are highly resistant to degradation and activate the immune system in patients with a genetic predisposition.[8] Due to increased intestinal permeability, peptides interact with antigen-presenting cells in the lamina propria. However, oats consumption seems safe for most celiacs.[4–6,9]

CD encompasses gastrointestinal, musculoskeletal, dermatological, reproductive, cardiovascular, endocrine, and neurological symptoms. These symptoms can be attributed to both intestinal malabsorption and immune activation. The continuing effects of villous atrophy and inflammation result in malnutrition. However, managing CD through a GFD prevents severe nutritional consequences and significantly improves the patient's Quality of Life.[10]

Table 1 Development of celiac disease (CD)

Susceptible children carrying the Haplotypes HLA-DQ2 and HLA-DQ8 gene that are necessary but not sufficient for the development of CD	
Gluten is a complex mixture of water-insoluble proteins composed of gliadins and glutenins, enriched in the amino acids glutamine and proline, which are highly resistant to proteolytic digestion. ⇨	Resistance is a critical factor in the persistence of gluten-derived peptides in the intestinal lumen. **Gluten as an environmental trigger**
Gluten-derived peptides persist in the intestinal lumen, releasing many immunogenic peptides. Triggers gliadin to bind to intestinal epithelium's receptor. GALT (Gut-Associated Lymphoid Tissue) Interaction and activation of the critical component of the immune system housed within the gut mucosa ⇨	The **activation of GALT by these gluten-derived peptides precipitates an inflammatory response, which is the root of the pathogenesis in CD.**
Gliadin upregulates the production of the intestinal peptide zonulin. ⇨	**Zonulin increases tight junctions' permeability**, allowing increased paracellular and transcellular peptide transport into the lamina propria.
Once presented by antigen-presenting cells, these antigens are recognized by T-cell receptors on CD4+ T helper cells, activating and proliferating them. Activated T cells produce proinflammatory cytokines, including IFN, IL-21, and IL-2. ⇨	**Gluten activates immune response: serum autoantibody response in IgA and IgG class** • anti-transglutaminase IgA • anti-endomysial antibodies IgA • deamidated gliadin-related peptide IgA and IgG **Which results in pathological changes in the small intestine**
The villous atrophy reduces the intestinal absorptive surface area. It results in malabsorption and nutritional deficiencies.	

Deficiency of minerals and vitamin D

At diagnosis, the most prevalent mineral, higher in adults than children and comprising over half of the CD patients, is iron deficiency anemia.[11–14] Indeed, the duodenum is the coexistence of villous atrophy and the maximum site of iron absorption, calcium, and Vitamin D. Accordingly, Vitamin D is involved in the hormonal regulation of bone remodeling and calcium absorption. It is of concern, considering the probable impaired bone health. Zinc deficiency is prevalent in untreated and treated patients, mainly related to malabsorption, and is partially responsible for skin lesions.[10,15] Also, Copper deficiency is related to anemia, thrombocytopenia, and neurologic impairment.[16–18] Therefore, Pediatric patients with CD are at risk of short stature, impaired peak bone mass achievement, and delay of puberty.[8,19]

Treatment

Gluten-free diet

The basis of CD treatment is permanent adherence to a GFD that excludes food and non-food products from wheat, rye, and barley. This dietary regimen, which forms the basis of the patient's treatment,[20] stimulates the Immune system when Gluten is ingested, making strict adherence to a GFD critical. The significant improvement in symptoms and the promotion of healing of the duodenal mucosa observed with a GFD underscore its efficacy and the importance of patient supervision to ensure adherence and promote healing.[5,21–23]

Dietary counseling

The treatment approach has changed from focusing on food avoidance to evaluating the various factors influencing the dietary choices in the patient's life. Dietary and behavioral counseling should accurately assess the individual's skill to implement the GFD. It is important to note that diet restriction and compliance can be challenging for many patients. The dietary counseling model must address the complex aspects of life, considering the effects of GFD on physical, emotional, and social well-being. The dietitian should identify barriers and motivators in their life and assist in developing strategies to navigate these situations.[24, 25] Table 2 displays the primary approach of patients with CD, and Table 3 presents Education (Discuss, Ensure, Explain, Encourage, Give, Highlight, Provide) about the GFD.

Table 2 Assessment of patients with celiac disease

Explain the objectives of the consultation.
Take a current diet intake.
Check the evolution of symptoms (before and after diagnosis).
Consider any dietary restrictions, such as lactose intolerance, diabetes, vegetarian or vegan diet, food taboos, religious aspects, and food preferences according to age group and economic condition.
Assess intake of macronutrients (particularly saturated fats), minerals (iron and calcium intake), and vitamins (especially Vitamin D).
Clear the concept of a Gluten-Free Diet, and teach how to read packaged food labels.
Involve the entire Family in Education about the Disease and its therapeutic plan.

Table 3 Education (discuss, ensure, explain, encourage, give, highlight, provide) about gluten-free diet

What is coeliac Disease, and how does it affect the body?
The risks of not following a lifelong strict gluten-free diet.
Gluten-free diet –where to find it and tools to select safe foods (gluten-free labeling, allergen labeling).
Which foods are naturally gluten-free
Gluten-free alternatives.
High-fiber gluten-free products.

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Table 3 Continued...

How to read labels
Cross-contamination with Gluten; cross-contamination in medications
Eating out: discuss the strategies
Gluten-free recipes.
Join coeliac support groups.

The primary nutritional approach in pediatric celiac disease

The four different viewpoints on the primary nutritional approach in CD, brilliantly explained by Perez-Junkera et al.,[26] are briefly presented below:

Regular observation of symptoms and adherence to a GFD

The initial assessment requests medical conditions, weight, physical activity, diet, allergies, food intolerances, nutrient deficiencies, and the individual's knowledge of CD. Food obtaining, preparation, food preparation facilities, and cooking skills must be assessed individually before beginning patient education. So, read food labels and understand the importance of avoiding gluten exposure.[26] On the contrary, nonadherence. to diet can aggravate intestinal damage, sustain symptoms, and raise the risk of small intestinal cancer.[25]

Routine to be adopted at each visit

Clinical signs/symptoms valuation and dietetic interview are the basis associated with laboratory exams (Hemogram, serum electrolytes, ferritin, vitamins (D and B12), alkaline phosphate, aspartate aminotransferase, and alanine aminotransferase levels.[25] For monitoring GFD adherence, stool and urine markers and measurement of ELISA- IgA antibodies to Tissue Transglutaminase (tTG) are essential.[4,5,27] Hence, a follow-up biopsy may be performed in patients diagnosed at an older age and with severe initial presentations.

Dietary adherence

Strict adherence to a GFD is difficult, considering that many processed foods can hide Gluten. Dietitian assessment, education, and counseling are the cornerstones of dietary management and require close follow-up. The basic actions would be monitoring dietary history, investigating nutritional deficiencies, and evaluating body composition. Explore gluten transgressions and educate for deficiency of knowledge about reading labels and the risk of cross-contamination.

Tools to evaluate the adherence to a GFD

Combining questionnaires with other procedures expands the assessment of dietary adherence, and two main tests have been proposed to measure GFD.

1) The Standardised Dietitian Evaluation (SDE). It involves a skilled dietitian assessing gluten exposure and adherence and analyzing a 3-day food record. Consequently, it is time-consuming.[28]
2) The Coeliac Dietary Adherence Test (CDAT) is a 7-item survey identifying poor adherence. Higher scores denote worse GFD adherence.[29]

Refractory celiac disease

The refractory CD is characterized by persistent symptomatology despite adherence to GFD for 12 months. Suppose suspected incorrect diagnoses; the first step is to revise the serology, histology, and HLA typing results. Once the diagnosis is confirmed, gluten inadvertent exposure should be re-evaluated.[30,31] The duodenal biopsies may classify patients into First) If persistent symptoms with normal duodenal histology, consider CD in remission or explore other causes. Second) Persistent symptoms and villous atrophy are considered slow responses to GFD, cross-contamination, small amounts of Gluten, or other reasons for villous atrophy.[32,33]

The nutritional status evaluation

Assuming the specific nutritional needs of patients with CD, a nutrition assessment is recommended to provide a primary approach to evaluating dietary intake and nutrient deficiencies and determining the severity of malnutrition. The CD patient presents malabsorption, resulting in multiple nutritional deficiencies.[34] Screening for malnutrition needs to be performed carefully at each visit. The Subjective Global Assessment (SGA) is the most utilized tool for evaluating intake, weight, functional status, and body composition, allocating into three malnutrition grades: no malnutrition (SGA A), mild/moderate (SGA B), and severe (SGA C).[35]

Psychological primary approach

Improving emotional and social well-being is vital in approaching CD patients, considering the risk of depression, anxiety, stress, and fatigue with consequent impairment on Health-Related Quality of Life. In addition, eating disorders such as limitations when eating outdoors. GFD "extreme vigilance" may feel annoyance, distress, disgrace, and sadness. The impact of CD extends beyond the patient and affects the well-being of caregivers and the wider family. Indeed, the family must also be considered when educating and supporting these patients. The psychologist must assist in promoting adherence to the GFD. One of the critical strategies is the provision of coping skills, such as social skills, acceptance, and control.

Primary social approach and education

GFD is connected with a substantial socioeconomic burden. Gluten-free food products are more expensive than regular products, compromising the adherence to a GFD. So, providing patients with education and treatment information is crucial. Indeed, social limits when eating out, participating in social events, or traveling could impact their psychological well-being. Traveling when no GFD food is available is challenging. In addition, the parents' and general population's knowledge and consciousness about CD are low. Promoting Celiac patients' integration in the CD Society is essential to encourage social inclusion.[36]

Labeling gluten-free food and cross-contamination

The labels of food products must be routinely checked to guarantee complete gluten-free products. Foods known not to contain Gluten may present gluten traces resulting from cross-contamination.[37] Thus, cross-contamination might occur from planting to the final preparation and lead to apprehension. Celiac patients are at risk at home and eating outside the home. Shared kitchen utensils and equipment may not increase gluten cross-contamination if appropriate directions are followed.[38,39]

Alternative non-dietary treatments for celiac disease

Table 4 presents the current and future alternatives proposed for the non-dietary treatment of CD, according to Wessels et al.[40]

Table 4 Alternative non dietary treatments for celiac disease, according to Wessels, et al.[40]

The following strategies for CD treatment are considered:
Eliminating toxic gluten peptides before they reach the intestine
Regulating the immune stimulatory elements of toxic gluten peptides
Modifying intestinal penetrability
Modulating the immune system, forming gluten tolerance
Controlling the imbalance in the intestine microbiota via immunotherapy
That could be obtained with:
Using genetically modified wheat grains
Thermally modified wheat
Sequestering Gluten in the gut lumen before absorption
Decreasing gut permeability
Anti-zonulin therapy
Using a selective oral transglutaminase inhibitor
Silencing RNA therapies
Enzyme therapies
Using DQ2/DQ8 inhibitors
Monoclonal antibodies
Celiac vaccines

Prevention

Prevention could be achieved based on three points.

1) Primary strategies such as avoiding disease development are impossible (until now). There is no evidence that the time of breastfeeding or the time of introducing Gluten into the infant's diet has an impact on the risk of developing CD.[4]

2) Secondary: serological screening of high-risk groups (first-degree relatives, patients with Down, Turner, or Williams syndrome, autoimmune diseases or selective IgA deficiency),

3) Tertiary: improved treatment and avoiding complications[7]

Marketing and media

The benefits of a GFD have been widely publicized, leading to a significant increase in food companies' production of gluten-free options, allowing CD patients to reproduce the dietary habits and patterns of the general population.[21] To guarantee a GFD, the Food and Drug Administration (FDA) has endorsed a gluten-free labeling of a product as gluten content (<20 mg/Kg or 20 ppm) or declaring Free, Without, or No Gluten.

Conclusions

The primary nutritional approach in Pediatric Celiac Disease is a GFD. Difficulties in managing the Disease and all difficulties can lead to psychosocial distress and impaired Health-Related Quality of Life. Despite the essential role of diet, the risk of nutritional imbalances, metabolic syndrome, cardiovascular diseases, and obesity is higher in CD patients than in the average population. Nevertheless, strict adherence to the GFD is imperative. Therefore, managing CD requires a multidisciplinary team involving physicians, dietitians, and psychologists on a regular visit schedule. Lastly, it is vital to emphasize that caring for a pediatric patient with Celiac Disease must cover all aspects of the patient's life with a generous dose of empathy.

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Conflicts of interest

The authors declare no conflict of interest.

References

1. Lebwohl B, Sanders DS, Green PHR. Coeliac disease. *Lancet*. 2018;391:70–81.
2. Lebwohl B, Rubio-Tapia A. Epidemiology, presentation, and diagnosis of celiac disease. *Gastroenterology*. 2021;160(1):63–75.
3. Singh P, Arora A, Strand TA, et al. Global prevalence of celiac disease: systematic review and meta-analysis. *Clin Gastroenterol Hepatol*. 2018;16(6):823–836.e2.
4. Al-Toma A, Volta U, Auricchio R, et al. European society for the study of coeliac disease (ESsCD) guideline for coeliac disease and other gluten-related disorders. *United European Gastroenterol J*. 2019;7(5):583–613.
5. Rubio-Tapia A, Hill ID, Semrad C, et al. American college of gastroenterology guidelines update: diagnosis and management of celiac disease. *Am J Gastroenterol*. 2023;118(1):59–76.
6. Ray S, Gillett PM, Le Huray V, et al. Updated European guidelines for coeliac disease in children. *Nurs Child Young People*. 2023;35(4):16–21.
7. Husby S, Koletzko S, Korponay-Szabó I, et al. European society paediatric gastroenterology, hepatology and nutrition guidelines for diagnosing coeliac disease 2020. *J Pediatr Gastroenterol Nutr*. 2020;70(1):141–156.
8. Biesiekierski JR. What is Gluten? *J Gastroenterol Hepatol*. 2017;32 Suppl 1:78–81.
9. Raiteri A, Granito A, Giamperoli A, et al. Current guidelines for the management of celiac disease: A systematic review with comparative analysis. *World J Gastroenterol*. 2022;28(1):154–175.
10. Kreutz JM, Adriaanse MPM, van der Ploeg EMC et al. Narrative review: nutrient deficiencies in adults and children with treated and untreated celiac disease. *Nutrients*. 2020;12(2):500.
11. Harper JW, Holleran SF, Ramakrishnan R, et al. Anemia in celiac disease is multifactorial in etiology. *Am J Hematol*. 2007;82(11):996–1000.
12. Leffler DA, Green PHR, Fasano A. Extraintestinal manifestations of coeliac disease. *Nat Rev Gastroenterol Hepatol*. 2015;12:561–571.
13. Seidita A, Mansueto P, Compagnoni S, et al. Anemia in celiac disease: prevalence, associated clinical and laboratory features, and persistence after gluten-free diet. *J Pers Med*. 2022;12(10):1582.
14. Talarico V, Giancotti L, Mazza GA, et al. Iron deficiency anemia in celiac disease. *Nutrients*. 2021;13(5):1695.
15. Wierdsma NJ, van Bokhorst-de van der Schueren MA, Berkenpas M, et al. Vitamin and mineral deficiencies are highly prevalent in newly diagnosed celiac disease patients. *Nutrients*. 2013;5(10):3975–3992.
16. Halfdanarson TR, Litzow MR, Murray JA. Hematologic manifestations of celiac disease. *Blood*. 2007;109(2):412–421.
17. Henri-Bhargava A, Melmed C, Glikstein R, et al. Neurologic impairment due to vitamin E and copper deficiencies in celiac disease. *Neurology*. 2008;71(11):860–861.

18. Webster J, Vajravelu ME, Choi C, et al. Prevalence of and risk factors for low bone mineral density in children with celiac disease. *Clin Gastroenterol Hepatol*. 2019;17(8):1509–1514.
19. Laurikka P, Kivelä L, Kurppa K, et al. Review article: Systemic consequences of coeliac disease. *Aliment Pharmacol Ther*. 2022;56 Suppl 1(Suppl 1):S64–S72.
20. Haines ML, Anderson RP, Gibson PR. Systematic review: the evidence base for long-term management of coeliac disease. *Aliment Pharmacol Ther*. 2008;28(9):1042–1066.
21. Itzlinger A, Branchi F, Elli L, et al. Gluten-free diet in celiac disease-forever and for all? *Nutrients*. 2018;10(11):1796.
22. Aljada B, Zohni A, El-Matary W. The Gluten-free diet for celiac disease and beyond. *Nutrients*. 2021;13(11):3993.
23. Pinto-Sanchez MI, Silvester JA, Lebwohl B, et al. Society for the study of celiac disease position statement on gaps and opportunities in coeliac disease. *Nat Rev Gastroenterol Hepatol*. 2021;18(12):875–884.
24. Dennis M, Lee AR, McCarthy T. Nutritional considerations of the gluten-free diet. *Gastroenterol Clin North Am*. 2019;48(1):53–72.
25. Lee AR. Review article: Dietary management of coeliac disease. *Aliment Pharmacol Ther*. 2022;56 Suppl 1:S38–S48.
26. Perez-Junkera G, Ruiz de Azua L, Vázquez-Polo M, et al. Global approach to follow-up of celiac disease. *Foods*. 2024;13(10):1449.
27. Leffler DA, Schuppan D. Update on serologic testing in celiac Disease. *Am J Gastroenterol*. 2010;105:2520–2524.
28. Leffler DA, Edwards George JB, Dennis M, et al. A prospective comparative study of five measures of gluten-free diet adherence in adults with coeliac disease. *Aliment Pharmacol Ther*. 2007;26(9):1227–1235.
29. Leffler DA, Dennis M, Edwards George JB, et al. A simple validated gluten-free diet adherence survey for adults with celiac disease. *Clin Gastroenterol Hepatol*. 2009;7(5):530–536.e2.
30. Dewar DH, Donnelly SC, McLaughlin SD, et al. Celiac disease: management of persistent symptoms in patients on a gluten-free diet. *World J Gastroenterol*. 2012;18(12):1348–1356.
31. Penny HA, Rej A, Baggus EMR, et al. Non-responsive and refractory coeliac disease: experience from the NHS England national centre. *Nutrients*. 2022;14(13):1–12.
32. Baggus EMR, Hadjivassiliou M, Cross S, et al. How to manage the adult coeliac disease: a perspective from the NHS England rare diseases collaborative network for non-responsive and refractory coeliac disease. *Frontline Gastroenterol*. 2020;11(3):235–242.
33. Schiepatti A, Maimaris S, Raju SA, et al. Persistent villous atrophy predicts the development of complications and mortality in adult patients with coeliac disease: a multicentre longitudinal cohort study and development of a score to identify high-risk patients. *Gut*. 2023;72(11):2095–2102.
34. Downey L, Houten R, Murch S, et al. Recognition, assessment, and management of coeliac disease: summary of updated NICE guidance. *BMJ*. 2015;351:1–5.
35. Detsky AS, McLaughlin JR, Baker JP, et al. What is a subjective global assessment of nutritional status? *JPEN J Parenter Enteral Nutr*. 1987;11(1):8–13.
36. Burden M, Mooney PD, Blanshard RJ, et al. Cost and availability of gluten-free food in the UK: in-store and online. *Postgrad Med J*. 2015;91(1081):622–626.
37. Hartmann C, Hieke S, Taper C, et al. European consumer healthiness evaluation of 'Free-from' labeled food products. *Food Qual Prefer*. 2018;68:377–388.
38. Saturni L, Ferretti G, Bacchetti T. The gluten-free diet: safety and nutritional quality. *Nutrients*. 2010;2(1):16–34.
39. Wieser H, Segura V, Ruiz-Carnicer Á, et al. Food safety and cross-contamination of gluten-free products: a narrative review. *Nutrients*. 2021;13(7):2244.
40. Wessels M, Auricchio R, Dolinsek J, et al. Review on pediatric coeliac disease from a clinical perspective. *Eur J Pediatr*. 2022;181(5):1785–1795.

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