Gluten-free diet and quality of life in celiac disease

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ABSTRACT

Many recent studies overshadow the effects of gluten-free diet. Gluten-free diet positive effects were observed in celiac disease patients: increase in body mass index, higher energy intakes, reducing adiposity gain, moderates the risk of the associated complications. However, adhering to a gluten-free diet is difficult for many people. A new solution is needed for quality of life of celiac disease patients, not for celiac disease treatment. Health education on gluten-free diet at home and in society seems to be the solution. The aim of our study is to evaluate the recent research on gluten-free diet as a nutritional therapy for patients with celiac disease. To achieve this purpose we have analyzed the published studies from 2008 to the present on nutrition in celiac disease.

Keywords: Celiac disease, Gluten-free diet, Quality of life.

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Introduction

Recent studies have shown that early diagnosis of celiac disease (CD) can improve quality of life (QOL) of these patients (1). From diagnosis to only therapy, which is gluten-free diet (GFD), CD is an interdisciplinary problem (2). Response to GFD is variable (3). What is the cause? Traces of gluten in GFD can produce damaging of the small intestine mucosa (4). For these reasons, recent research has focused on finding low or null toxicity wheat for CD patients.

Our aim was to discuss the problems related to QOL and GFD. We conducted the study on PubMed database using keywords nutrition, CD. Eligibility criteria consisted of data found in PubMed database with reference to nutrition in CD from nutrition journals.

Dietary strategies started from the clinical effects of GFD

Dietary strategies with regard to GFD and the research studies that analyze the gluten presence in wheat species occupy the largest share in the studied articles. Quality of life (QOL) of CD patients, the clinical effects of GFD, diagnostic problems in screening for gluten toxicity were also found in the studied articles. Our data shows that GFD positive effects were observed in CD patients: increase in body mass index, higher energy intakes, reducing adiposity gain, moderates the risk of the associated complications. Adhering to a GFD is difficult for many people.

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On 149 CD children followed for 16 years, a significant (p=0.008) increase in body mass index (BMI) z-score after GFD was found (5). The frequency of overweight (12 % vs. 23.3 %, p=0.014) and underweight (16 % vs. 4.5 %, p<0.001) in children under GFD were substantially lower than that reported in tertiary care centers (6). A recent study concluded that 79 % from 413 patients (77 % were female and mean BMI was 24.1) with biopsy-proven CD had seen a dietitian (7). Martin et al. concluded that there was no significant association of either socio-economic deficiency or co-morbidities with adherence to GFD in CD patients (8).

The researchers recognize long chain ω -3 fatty acids, plant flavonoids, and carotenoids as modulators for gene expression, oxidative stress and production of inflammatory mediators in CD (9). Therefore, adoption of dietary with these components could play a protective role against toxicity of gliadin peptides, keep intestinal barrier integrity and have a role in nutritional therapy of CD. Bergamo et al. evaluated the gliadin effects to DQ8 mice fed with a gliadin-containing diet with or without conjugated linoleic acid (CLA) supplementation and in differentiated human intestinal epithelial cell line (Caco-2). Gliadin was unable to generate oxidative stress and pathological consequences (10). Docosahexaenoic acid (DHA) counteracts many of the proinflammatory effect of arachidonic acid (AA). Vicentini et al. exposed a Caco-2 to gliadin peptides (PT-gl) (500 µg/ml) and DHA (2 µg/ml), both alone and simultaneously up to 24 h. The results showed that intestinal epithelial cells sustain the CD inflammation but cell culture studies have limitations (11).

A study revealed that CD patients are at the risk of having an inadequate intake of calcium, non-starch polysaccharides and vitamin D (12). Ohlund et al. aimed to assess the dietary intakes of energy and nutrients in children and adolescents on GFD. The conclusion was that children on GFD seem to follow the same trends as healthy children on a common diet, with high intakes of sucrose and saturated fat and low intakes of dietary fiber, vitamin D and magnesium (13). The dietary intake of CD children on a GFD and noncoeliac children were also evaluated. The observation was that CD children had higher energy intakes than controls, although BMI was comparable to the groups (14). Children and adolescents with CD are at risk for suboptimal bone health at time of diagnosis and after 1 year on GFD. This could be due in part to suboptimal vitamin D/K status. In conclusion, therapeutic dietary strategies for optimizing vitamin K/D intake are needed (15). Kautto et al. compared the energy and nutrient intakes among 13-year olds diagnosed with CD in early childhood with those of a non-coeliac (NC). The results revealed that both groups had low consumption of vitamin C, with 13% in the CD-group and 25% in the NCgroup below estimated average requirements (EAR), and 21% of boys in the CD-group below EAR for thiamine (16). Therefore, some nutrient deficiencies need specific management (17). Many organizations were found for providing a dietary support and advice to the CD patients. Laparra et al. concluded that oral administration of Bifidobacterium longum ameliorates gliadinmediated perturbations in liver iron deposition (18).

Other dietary proteins, such as those of cow's milk, were also investigated in CD. A study concluded that intolerance of CD patients to bovine milk is not due to CD + T cell stimulatory epitopes of gluten (19). Some other dietary proteins, such as alpha- and beta-caseins induce CD-like symptoms in some CD patients (20). Association between CD and primary lactase deficiency was also investigated. In CD patients, lactose intolerance could be owing to secondary lactase deficiency and to primary lactase deficiency but the hereditary lactase deficiency is frequent in CD children as in control population (21).

It is commonly accepted that breastfeed is a protective factor against CD (22). It is still not fully known whether breast-feed protects with permanent tolerance acquisition (23). At present, two schools declared that changing early feeding regimens in at-risk infants could either prevent the onset of the disease or merely delay it (24). Data on the impact on infant feeding are inconsistent (25). Soares et al. evaluated whether gluten exclusion can prevent adipose tissue expansion and its consequences. They feed the C57BL/6 mice with a high-fat diet containing 4.5% gluten or no gluten. Gluten-free animals presented a decrease in body weight gain and adiposity, without changes in calorie intake or lipid excretion. Their data suggested the beneficial effects of GFD in reducing adiposity gain, inflammation and insulin resistance. Therefore, GFD has been proposed as an option to the prevention of obesity (26).

Analysis of the QOL of patients with CD

Our data show that following a GFD is difficult for many people. GFD involves restriction of food choice (27). A group of researchers aimed to investigate the impact of CD and GFD on the lifestyle of patients and their families, together with recommendations for improvement of OOL. Their conclusion was that CD children have low compliance with the GFD: poor palatability (32 %), dining outside home (17 %), poor availability of products (11 %), and asymptomatic disease diagnosed by screening (11 %) (28). Better education about the disease, available gluten-free products, and proper food labelling could improve compliance and QOL (29). Unfortunately, adhering to a GFD is practically difficult. CD affects many of daily activities and gluten consumption is more common with possible consequences on health (30). CD patients have a diminished QOL in the social aspects of life. Women reported greater emotional responses to a GFD (31). About 90% from 98 adult patients with CD said they transgress the dietary pattern and about 58% consumed without knowing gluten products (32). The patients with non-celiac gluten sensitivity reported a slightly more difficulty in following GFD compare to CD patients (33). However, QOL studies suggest that CD patients benefit from a GFD. Furthermore, the GFD moderates the risk of the associated complications (34).

Conclusion

A new solution is needed for QOL of CD patients, not for CD treatment. Health education on GFD at home and in society seems to be the solution.

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References

- 1. Khoshbaten M, Rostami Nejad M, Sharifi N, Fakhari A, Golamnejad M, Hashemi SH, Collin P, Rostami K. Celiac disease in patients with chronic psychiatric disorders. Gastroenterol Hepatol Bed Bench 2012;5:90-93.
- 2. Makovicky P, Samasca G. Present View of the Management and Tasks in the Celiac Disease Field: From Diagnosis to Therapy. International Journal of Celiac Disease 2013;1:3-5.

3. Mousavi M, Rostami Nejad M. A 27 year-old female patient with chronic watery diarrhea, weight loss, ascites, arthropathy and evidence of vitamin K deficiency. Gastroenterol Hepatol Bed Bench 2010;3:142-45.

4. Rostami Nejad M, Karkhane M, Marzban A, Mojarad EN, Rostami K. Gluten related disorders. Gastroenterol Hepatol Bed Bench 2012;5:S1-S7.

5. Valletta E, Fornaro M, Cipolli M, Conte S, Bissolo F, Danchielli C. Celiac disease and obesity: need for nutritional follow-up after diagnosis. Eur J Clin Nutr 2010;64:1371-72.

6. Brambilla P, Picca M, Dilillo D et al. Changes of body mass index in celiac children on a gluten-free diet. Nutr Metab Cardiovasc Dis 2013;23:177-82.

7. Mahadev S, Simpson S, Lebwohl B, Lewis SK, Tennyson CA, Green PH. Is dietitian use associated with celiac disease outcomes? Nutrients 2013;5:1585-94.

8. Martin U, Mercer SW. A comparison of general practitioners prescribing of gluten-free foods for the treatment of coeliac disease with national prescribing guidelines. J Hum Nutr Diet 2014;27:96-104.

9. Ferretti G, Bacchetti T, Masciangelo S, Saturni L. Celiac disease, inflammation and oxidative damage: a nutrigenetic approach. Nutrients 2012;4:243-57.

10. Bergamo P, Gogliettino M, Palmieri G, Cocca E, Maurano F, Stefanile R, et al. Conjugated linoleic acid protects against gliadin-induced depletion of intestinal defenses. Mol Nutr Food Res 2011;55:S248-56.

11. Vincentini O, Quaranta MG, Viora M, Agostoni C, Silano M. Docosahexaenoic acid modulates in vitro the inflammation of celiac disease in intestinal epithelial cells via the inhibition of cPLA2. Clin Nutr 2011;30:541-46.

12. Kinsey L, Burden ST, Bannerman E. A dietary survey to determine if patients with coeliac disease are meeting current healthy eating guidelines and how their diet compares to that of the British general population. Eur J Clin Nutr 2008;62:1333-42.

13. Ohlund K, Olsson C, Hernell O, Ohlund I. Dietary shortcomings in children on a gluten-free diet. J Hum Nutr Diet 2010;23:294-300.

14. Zuccotti G, Fabiano V, Dilillo D, Picca M, Cravidi C, Brambilla P. Intakes of nutrients in Italian children with celiac disease and the role of commercially available gluten-free products. J Hum Nutr Diet 2013;26:436-44.

15. Mager DR, Qiao J, Turner J. Vitamin D and K status influences bone mineral density and bone accrual in children and adolescents with celiac disease. Eur J Clin Nutr 2012;66:488-95.

16. Kautto E, Ivarsson A, Norström F, Högberg L, Carlsson A, Hörnell A. Nutrient intake in adolescent girls and boys diagnosed with coeliac disease at an early age is mostly comparable to their non-coeliac contemporaries. J Hum Nutr Diet 2014;27:41-53.

17. García-Manzanares A, Lucendo AJ. Nutritional and dietary aspects of celiac disease. Nutr Clin Pract 2011;26:163-73.

18. Laparra JM, Olivares M, Sanz Y. Oral administration of Bifidobacterium longum CECT 7347 ameliorates gliadin-induced alterations in liver iron mobilisation. Br J Nutr 2013;110:1828-36.

19. Dekking L, Koning F, Hosek D et al. Intolerance of celiac disease patients to bovine milk is not due to the presence of T-cell stimulatory epitopes of gluten. Nutrition 2009;25:122-23.

20. Cabrera-Chávez F, de la Barca A M. Bovine milk intolerance in celiac disease is related to IgA reactivity to alpha- and beta-caseins. Nutrition 2009;25:715-16.

21. Basso MS, Luciano R, Ferretti F et al. Association between celiac disease and primary lactase deficiency. Eur J Clin Nutr 2012;66:1364-65.

22. Hörnell A, Lagström H, Lande B, Thorsdottir I. Breastfeeding, introduction of other foods and effects on health: a systematic literature review for the 5th Nordic Nutrition Recommendations. Food Nutr Res 2013; 57.

23. Nova E, Pozo T, Sanz Y, Marcos A. Dietary strategies of immunomodulation in infants at risk for celiac disease. Proc Nutr Soc 2010;69:347-53.

24. Fasano A, Catassi C. Early feeding practices and their impact on development of celiac disease. Nestle Nutr Workshop Ser Pediatr Program 2011;68:201-209.

25. Ludvigsson JF, Fasano A. Timing of introduction of gluten and celiac disease risk. Ann Nutr Metab 2012;60:22-29.

26. Soares FL, de Oliveira Matoso R, Teixeira LG et al. Gluten-free diet reduces adiposity, inflammation and insulin resistance associated with the induction of PPAR-alpha and PPAR-gamma expression. J Nutr Biochem 2013;24:1105-11.

27. Rose C, Howard R. Living with coeliac disease: a grounded theory study. J Hum Nutr Diet 2014;27:30-40.

28. Roma E, Roubani A, Kolia E, Panayiotou J, Zellos A, Syriopoulou VP. Dietary compliance and life style of children with celiac disease. J Hum Nutr Diet 2010;23:176-82.

29. Black J L, Orfila C. Impact of celiac disease on dietary habits and quality of life. J Hum Nutr Diet 2011;24:582-87.

30. Lee AR, Ng DL, Diamond B, Ciaccio EJ, Green PH. Living with celiac disease: survey results from the U.S.A. J Hum Nutr Diet 2012;25:233-8.

31. Zarkadas M, Dubois S, Macisaac K et al. Living with celiac disease and a gluten-free diet: a Canadian perspective. J Hum Nutr Diet 2013;26:10-23.

32. Pelegrí Calvo C, Soriano del Castillo JM, Mañes Vinuesa J. Quality of life and diagnosis process in adult celiacs from the Valencian Community. Nutr Hosp 2012;27:1293-97.

33. Verrill L, Zhang Y, Kane R. Food label usage and reported difficulty with following a gluten-free diet among individuals in the USA with coeliac disease and those with noncoeliac gluten sensitivity. J Hum Nutr Diet 2013;26:479-87.

34. Aziz I, Sanders DS. Are we diagnosing too many people with coeliac disease? Proc Nutr Soc 2012;71:538-44.