Japanese Journal of Gastroenterology and Hepatology

Research Article ISSN 2435-1210 |Volume 6

Nutritional Myths and Challanges in Celiac Disease

Malhotra P*, Malhotra V, Gupta U, Sanwariya Y, Pahuja I and Akshay

Department of Medical Gastroenterology and Obstetrics & Gynecology, PGIMS, Rohtak (Haryana) & Director Health Services, Haryana India

***Corresponding author:**

Parveen Malhotra,
Department of Medical Gastroenterology, Pt.B.D.S. Post Graduate Institute of Medical Sciences, 128/19, Civil Hospital Road, Rohtak – 124001, Haryana, India, Email: drparveenmalhotra@yahoo.com

Received: 23 Mar 2021
Accepted: 13 Apr 2021
Published: 21 Apr 2021

Keywords:

Celiac disease; Infertility; Gluten free diet Abbreviations: CD: Celiac disease; GFD: Gluten free diet; TG2: Transglutaminase 2

Citation:

Malhotra P. Nutritional Myths and Challanges in Celiac Disease. Japanese J Gstro Hepato. 2021; V6(9): 1-2

1. Abstract

1.1. Background: Celiac Disease (CD) is a common chronic immune-mediated, inflammatory disorder of the small intestine induced by a permanent intolerance to dietary wheat, barley, and rye.

1.2. Discussion: Celiac disease has a multifactorial pathogenesis and common symptoms may include bulky stool, constipation, anemia, delayed growth, failure to thrive and infertility. At the time of diagnosis of celiac disease, patient and his family members are in a state of mental shock that how the patient will survive life-long without gluten. It is very important to determine hidden source of gluten being taken in the diet, of which patient is unaware, thus there is unsatisfactory clinical, biochemical and histological recovery.

1.3. Conclusion: At the time of initial diagnosis of celiac disease, the patient and family members should be taken into confidence with proper counseling and clearly explained about prognosis and course of disease. The need of strict adherence to gluten free diet must be made understood to patient and family members.

2. Introduction

Celiac Disease (CD) is a common chronic immune-mediated, inflammatory disorder of the small intestine induced by a permanent intolerance to dietary wheat, barley, and rye [1]. Celiac Disease (CD) is a permanent intolerance to gluten, for which the only treatment currently available is a lifelong adherence to a Gluten-Free Diet (GFD). Once patients are diagnosed with celiac disease and begin the gluten-free diet, 70% report symptom relief within two weeks [2].

3. Discussion

Celiac disease is a unique autoimmune disorder in that the environmental precipitant is known. Until 2004, medical schools taught that celiac disease was a rare disease of childhood. However, as 95% of Celiac disease patients remain undiagnosed, it is the most common, and one of the most under diagnosed, hereditary autoimmune disease. Celiac disease (CD) has a multifactorial pathogenesis [3]. Common symptoms may include bulky stool, constipation, anemia, delayed growth, failure to thrive and infertility [4]. Celiac disease used to be perceived as presenting with gastrointestinal symptoms suggestive of malabsorption, such as edema secondary to hypoalbuminemia, hypocalcaemia, vitamin deficiency states and osteomalacia [5]. This manner of presentation is now described as the "classic" or "typical" form. Patients with celiac disease may have the "silent" or "atypical" form with no gastrointestinal symptoms and the condition may present outside the intestines and can affect any organ system [6]. The reproductive alterations most frequently found in women affected by CD include: infertility, spontaneous abortions, amenorrhea and shorter fertility period (delayed puberty, early menopause) [7].

The diagnosis of early developing celiac disease should be based on a combination of clinical features, histology, serology, and genetics. Historically, diagnosis was suggested by positive serology and confirmed with endoscopy. Serum immunoglobulin IgA-class endomysial (EmA) and transglutaminase 2 (TG2) anti- bodies are powerful tools in diagnosing celiac disease with overt villous atrophy [8]. The diagnosis of celiac disease requires the presence of small intestinal

mucosal villous atrophy and crypt hyperplasia.

4. Myths Associated with Celiac Disease

We are running a special dedicated celiac clinic under Medical Gastroenterology department for last nine years and suspected cases of celiac disease are referred from various specialties/ super specialties available at PGIMS, Rohtak and private practitioners also. So these myths in mind of patients of celiac disease have been understood from the day of diagnosis to their long term follow up.

At the time of diagnosis of celiac disease, patient and his family members are in a state of mental shock that how the patient will survive life-long without gluten, as in our area gluten is staple diet. The normal perception and myth is that gluten is required for normal physical and mental growth but corollary is that gluten is detrimental for celiac patient. Hence, at time of diagnosis at least fifteen minutes counseling is done of patient and family members, to make them understand the disease aspects and how to mould their lives regarding the same. The patient and family members are explained about the places in India where rice is staple diet like South India and their excellent performance in every field of life, thus breaking the myth of requirement of gluten for normal physical and mental development.

The second most common myth is that gluten can be started after initial resolution of symptoms and for this main culprit are certain medical professionals with half cooked knowledge of celiac disease and alternative medicine practitioners who under cover of steroids restart gluten in celiac patients. Thus steroids/ immunosuppressant's temporarily mask the development of symptoms due to re-entry of gluten in body but truth is that damage of gluten on intestine goes unabated and even side effects of immunosuppressant's like hypertension, diabetes mellitus, osteoporosis etc. develop. The corollary is that even some allopathic practitioners are also allowing 25 %- 50 % of gluten in diet to celiac patient after initial resolution of symptoms. In our long term follow up, we found that the compliance rate for gluten restriction is around 70%.

The first challenge in celiac disease is to make patient and his family understand that he/she is suffering from celiac disease and it requires life-long restriction of gluten in diet.

The next challenge is to pin-point hidden source of gluten being taken in the diet, of which patient is unaware, thus there is unsatisfactory clinical, biochemical and histological recovery. The reasons for it are adulteration of gluten in diet due to common kitchen practices for preparing food for whole of family, using same utensils or consumption of market products thinking to be gluten free but in reality containing minute amounts of gluten in diet and even frequent and heavy use of lip-stick, as it contains gluten as a base. In school going children, the reason came out to be sharing of food with other children.

5. Conclusions

At the time of initial diagnosis of celiac disease, the patient and family members should be taken into confidence with proper counseling and clearly explained about prognosis and course of disease. The need of strict adherence to gluten free diet must be made understood to patient and family members. The hidden, unknown and inadvertent source of gluten in diet should be pin-pointed and removed. The use of alternative medications should be discouraged. The screening of mother and other siblings in family for celiac disease should be done.

References

1. Murray J. The widening spectrum of Celiac Disease. Am J Clin Nutr. 1999; 69: 354-365.
2. Taranta A, Fortunati D, Longo M, Rucci N, Iacomino E. Imbalance of osteoclastogenesis-regulating factors in patients with celiac disease. J Bone Miner Res. 2004; 19: 1112-1.
3. Alaedini A, Green PH. Narrative review: celiac disease: under- standing a complex autoimmune disorder. Ann Intern Med. 2005; 142: 289-98.
4. Stazi A, Montovani A. A risk factor for female fertility and pregnancy: celiac disease. Gynocol Endocrinol. 2000; 14: 454-63.
5. Green PH, Alaedini A, Sander HW, Brannagan TH, Latov N. Mechanisms underlying Celiac Disease and its Neurologic Manifestations. Cell Molecul Life Sci. 2005; 62: 791-9.
6. Fasano A. Celiac Disease - How to Handle a Clinical Chameleon. NEJM. 2003; 348: 2568-70.7.
7. Collin P, Vilska S, Heinonen PK, Hallstrom O, Pikkarinen P. 1996.
8. Salmi TT, Collin P, Jarvinen O, Haimila K, Partanen J. Immunoglobulin A auto antibodies against transglutaminase 2 in the small intestinal mucosa predict forthcoming celiac disease. Aliment Pharmacol Ther. 2000; 24: 541-52.