

Review Article

Journal of Clinical & Experimental Immunology

Nutritional Management of Celiac Disease

Obianuju A. Orjiekwe*

Dietitian Nutritionist (RDN), Nsukka, Enugu State, Nigeria

*Corresponding Author

Obianuju A. Orjiekwe, Dietitian Nutritionist (RDN), Nsukka, Enugu State, Nigeria

Submitted: 2023, May 22; Accepted: 2023, June 12; Published: 2023, June 19

Citation: Orjiekwe, O. A., (2023). Nutritional Management of Celiac Disease. J Clin Exp Immunol, 8(2), 561-572.

Abstract

Celiac disease (CD) is a chronic disease causing inflammation of the proximal small intestine that occurs in genetically predisposed individuals when they eat gluten which is a binding protein common in grains wheat, barley, and rye. The disease injury usually resolves when gluten is excluded from the diet. Although the injury will heal, the reaction to gluten is permanent and will recur with the reintroduction of gluten. The condition is surprisingly common, affecting as many as 1% of white populations. The consequences of the disease are predominantly those of malnutrition due to maldigestion and malabsorption, such as diarrhea, weight loss, and anemia. Symptoms caused by inflammation of the small intestine are also common. CD, although it is common and its pathology is well understood, frequently goes undiagnosed, probably because of the nonspecific or vague nature of many of the symptoms that occur. The cornerstone of treatment for CD is elimination of gluten from the diet. In most patients diagnosed with CD, a strict gluten-free diet (GFD) alone should result in complete symptomatic and histologic resolution of the disease and reduce risk of complications. Noncompliance with diet is the leading cause of failure to respond in patients with CD. For these reasons, thorough assessment and counseling and management at the time of diagnosis and ongoing care are crucial. This article addresses the overview and diagnosis of CD and addresses its nutritional management in detail.

1. Introduction

Over the past 2 decades, Celiac disease (CD) has emerged as a major public health problem estimated to affect approximately 1% of the European population. Until a few decades ago, CD was now considered to be an uncommon disease affecting mainly children and limited individuals of European ancestry (Lionetti and Catassi, 2011). Celiac disease is an autoimmune enteropathy triggered by dietary gluten in genetically susceptible individuals. This disease is triggered by eating foods containing gluten which damages the small intestine. It is a chronic disorder of the digestive tract that results in an inability to tolerate gliadin, the alcohol-soluble fraction of gluten. Gluten is a protein found naturally in wheat, barley, and rye, and is common in foods such as bread, pasta, cookies, and cakes. (Pietzak, 2012). Celiac disease is also different from a wheat allergy. In both cases, the body's immune system reacts to wheat. However, some symptoms in wheat allergies, such as having itchy eyes or a hard time breathing, are different from celiac disease. Wheat allergies also do not cause long-term damage to the small intestine.

This disease can be very serious and can cause long-lasting digestive problems. It keeps the body from getting all the nutrients it needs and also affects the body outside the intestine (Pietzak, 2012). When patients with celiac disease ingest gliadin, an immunologically mediated inflammatory response occurs that damages the mucosa of their intestines, resulting in maldigestion and malabsorption of food nutrients. Those diagnosed with the

condition will need to make changes in their diet to eliminate gluten. Classically, signs and symptoms initially appear upon the introduction of wheat into a child's diet (usually at age 6-12 months). However, the disease may not present until later in life, typically between the ages of 10 and 40. In children, the presentation may include failure to thrive, delayed growth, irritability, vomiting, constipation, large stools, peripheral edema, clubbing, and frequent respiratory infections. In adults, many cases are asymptomatic, but some patients may have diarrhea, weight loss, abdominal swelling, and bloating. Patients may also have non intestinal symptoms such as amenorrhea, infertility, dermatitis, herpetiformis, and neurologic symptoms (e.g., peripheral neuropathy, ataxia and seizures). Malabsorption of vitamin D and calcium may result in rickets, osteoporosis, and bone fractures. Iron malabsorption can result in anemia. Nutrition which is the science that interprets the interaction of nutrients and other substances in food in relation to maintenance, growth, reproduction, health and disease of an organism is necessary in celiac disease World Health Organisation (WHO) (2015). The nutritional management of patients with anorexia nervosa forms an essential part of treatment. Good nutritional management of patients with anorexia nervosa requires attention to a number of areas. Careful assessment of the patient's diet and supervision of the feeding within the context of the patient's cultural, ethnic and religious background are essential. Medical monitoring is also important.

This paper highlights the overview and nutritional management of celiac disease.

Overview of Celiac Disease What is Celiac Disease?

Celiac disease can be defined as a chronic autoimmune disease in which the gliadin protein fraction of gluten causes a mucosal lesion of the small intestinal that impairs nutrient absorption. This long-term autoimmune disorder affects the small intestine and the digestive tract resulting from inability to tolerate gliadin, the alcohol-soluble fraction of gluten. Gluten is a protein found naturally in wheat, barley, and rye, and is common in foods such as bread, pasta, cookies, and cakes. (Pietzak, 2012). When people diagnosed with celiac are exposed to gluten a binding protein common in grains but also in makeup products, the immune system malfunctions and attacks the walls of the small intestine, which is responsible for absorbing nutrients from food (Anderson, van Heel, Barnardo, Salio and Jewell, 2005). People with celiac disease have a swollen and irritated small intestine, which can interfere with this absorption, leading to nutrient deficiencies.

The term celiac is derived from the Greek (koiliakos, abdominal) and was introduced in the 19th century in a translation of what is generally regarded as an ancient Greek description of the disease by Aretaeus of Cappadocia. Celiac disease is also termed gluten sensitive enteropathy. In children, it is termed celiac disease and celiac sprue in adult. Other names include nontropical sprue and endemic sprue. The disease damages primarily the mucosa of the small intestine especially the duodenum and proximal jejunum.

Celiac disease is one of the most common intestinal diseases to cause malabsorption in childhood. Symptoms often begin during the second six months of life after wheat containing foods are added to the diet. The affected child gradually becomes irritable and unwell, experience loss of appetite and begins to pass frequent foul bulky stools. Vomitting is common; weight gain slows down during the time. Diminished body weight is seen in almost all patients especially in those with prolonged active disease; short stature is also common in children. The occurrence of problems may depend on the variety of oat. It occurs in people who are genetically predisposed. Upon exposure to gluten, an abnormal immune response may lead to the production of several different auto antibodies that can affect a number of different organs. In the small bowel, this causes an inflammatory reaction and may produce shortening of the villi lining the small intestine (villous atrophy). This affects the absorption of nutrients, frequently leading to anaemia (Sabatino and Corazza, 2009).

In infants and young children, CD classically manifests as diarrhoea, steatorrhoea, failure to thrive and abdominal distension that occurs a few weeks to months after the introduction of gluten into the diet, usually between the ages of 6 months and 3 years. Vomiting, irritability, anorexia and constipation are also common. Some patients have more subtle symptoms such as oedema, anaemia, growth retardation and recurrent dental caries (Devlin, 2004). In older children and adults, diarrhoea and other gastrointestinal symptoms are less prominent. In older children, short stature, retarded puberty, iron deficiency anaemia and personality problems may predominate. Adult patients often

have symptoms of irritable bowel syndrome (Devlin, 2004) and may also present with recurrent aphthous stomatitis, iron deficiency without obvious cause, osteoporosis or osteopenia, and short stature. Other non-intestinal manifestations of CD include dermatitis herpetiformis and hepatitis. Certain malignant diseases are also more frequent in patients with CD. These include small bowel adenocarcinoma, oesophageal and oropharyngeal squamous carcinoma, and non-Hodgkins lymphoma (Green and Jabri, 2003). Lifelong avoidance of gluten ingestion is the cornerstone treatment for CD (Fasano and Catassi, 2001). This involves a diet free of wheat, rye and barley. There is controversy regarding the safety of oats for people with CD. In a recent systematic review, Haboubi found that although oats can be symptomatically tolerated by most people with CD, the long term effects of a diet containing oats remain unclear (Haboubi, 2006). Treatment of CD is important not only to improve the immediate quality of life of the patient but also to decrease the long-term risks of untreated CD such as growth failure in children, osteopenia and malignancies. Whilst it is generally accepted that avoidance of gluten is necessary for people with CD, the relationship between the quantity of gluten ingested and the development of symptoms and histological abnormalities is not clearly defined (Collin, Thorell and Kaukinen, 2004) and the exact amount of gluten that people with CD can tolerate on a daily basis without suffering any deleterious effects has not been established. Total avoidance is also extremely difficult, if not impossible to achieve as gluten contamination in 'gluten-free' products cannot be avoided completely (Hischenhuber, 2006; Collin, Thorell amd Kaukine, 2004).

Epidemiology of Celiac Disease

Globally celiac diseases affects between 1 in 100 and 1 in 170 people, however, vary between different regions of the world from as few as 1 in 300 to as many as 1 in 40 (Fasano and Catassi, 2012) Due to variable signs and symptoms it is believed that about 85% of people affected are undiagnosed. The percentage of people with clinically diagnosed disease (symptoms prompting diagnostic testing) is 0.05-0.27% in various studies. However, population studies from parts of Europe, India, South America, Australasia and the United States of America (USA) (using serology and biopsy) indicate that the percentage of people with the disease may be between 0.33 and 1.06% in children but 5.66% in one study of children of the predisposed Sahrawi people and 0.18-1.2% in adults an (Heel and West, 2006). People of African, Japanese and Chinese descent are rarely diagnosed this reflects a much lower prevalence of the genetic risk factors. People of Indian ancestry seem to have a similar risk to those of Western Caucasian ancestry (Hischenhube, Crevel, Jarry, Mäki, Moneret-Vautrin, Romano, Troncone and Ward, 2006). In Nigeria, more than 100 thousand cases of gluten intolerance are seen every year. It is fast becoming a common condition in a lot of people, apparently without regard to age or sex (Punch Newspaper, 2018). Populations at increased risk for celiac disease, with prevalence rates ranging from 5% to 10%, include individuals with Down and Turner syndromes, type 1 diabetes, and autoimmune thyroid disease, including both hyperthyroidism (overactive thyroid) and hypothyroidism (underactive thyroid) (Barker and Liu, 2008). Historically, celiac disease was thought to be rare, with a prevalence of about 0.02% (Barker and Liu, 2008). The reason for the recent

increases in the number of reported cases is unclear. It may be at least in part due to changes in diagnostic practice. There also appears to be an approximately 4.5 fold true increase that may be due to less exposure to bacteria and other pathogens in Western environments. In the United States, the median age at diagnosis is 38 years. Roughly 20 percent of individuals with celiac disease are diagnosed after 60 years of age (Oxentenko and Rubio-Tapia, 2019).

Causes of Celiac Disease

Celiac disease is caused by a reaction to gliadins and glutenins (gluten proteins) found in wheat, and similar proteins found in the crops of the tribe Triticeae (which includes other common grains such as barley and rye) and the tribe Aveneae (oats). Wheat subspecies (such as spelt, durum and Kamut) and wheat hybrids (such as triticale) also induce symptoms of celiac disease (Biesiekierski and Jessica, 2017; Kupper, 2005). Celiac disease is also a genetic disease that runs in families. A person can have the disease and not know it until it is triggered.

Factors that trigger symptoms includes:

- 1. Surgery
- 2. Pregnancy
- 3. Infection and
- 4. Emotional stress

Signs and Symptoms of celiac disease

Celiac disease affects people in different ways. Some persons may develop symptoms as children, whereas others do not experience symptoms until adulthood. Some may have diarrhea and abdominal pains, while others have irritability or depression with the onset of the disease.

While the following are common symptoms of celiac disease. However, each individual may experience symptoms differently. Symptoms may include:

Gastrointestinal symptoms

- Gastrointestinal symptoms may include the following:
- Diarrhea 45-85% of patients
- Flatulence 28% of patients
- Borborygmus 35-72% of patients
- Weight loss 45% of patients; in infants and young children with untreated celiac disease, failure to thrive and growth retardation are common
- Weakness and fatigue 78-80% of patients; usually related to general poor nutrition
- Severe abdominal pain 34-64% of patients

Extra intestinal symptoms

Extra intestinal symptoms may include the following:

- Anemia 10-15% of patients
- Osteopenia and osteoporosis 1-34% of patients
- Neurologic symptoms 8-14% of patients; include motor weakness, paresthesias with sensory loss, and ataxia; seizures may develop
- Skin disorders 10-20% of patients; including dermatitis herpetiformis, a condition with pruritic, papulovesicular skin lesions involving the extensor surfaces of the extremities,

- trunk, buttocks, scalp, and neck
- Hormonal disorders Including amenorrhea, delayed menarche, and infertility in women and impotence and infertility in men A bleeding diathesis is usually caused by prothrombin deficiency, due to impaired absorption of fatsoluble vitamin K.

A physical exam may reveal the following:

- A protuberant and tympanic abdomen
- Evidence of weight loss
- recurring abdominal pain and bloating
- pale, foul-smelling stool
- muscle cramps and/or bone pain
- pain in the joints
- tingling numbness in the legs
- delayed growth
- fatigue
- painful skin rash
- missed menstrual periods (which is linked to excessive weight loss)
- tooth discoloration or loss of enamel

Sometimes, persons with celiac disease are asymptomatic, as the undamaged part of the small intestine is still able to absorb enough nutrients. However, these persons are still at risk for complications of the disease. The symptoms of celiac disease may resemble other medical conditions or problems.

Medical Diagnosis of Celiac Disease

Diagnosis is typically made by a combination of blood antibody tests and intestinal biopsies, helped by specific genetic testing. Making the diagnosis is not always straightforward. Frequently, the autoantibodies in the blood are negative and many people have only minor intestinal changes with normal villi. People may have severe symptoms and they may be investigated for years before a diagnosis is achieved. Increasingly, the diagnosis is being made in people without symptoms, as a result of screening (Heel and West, 2006). In addition to a complete medical history and physical examination, diagnostic procedures for celiac disease may include the following:

• Blood work

Blood work is to (measure the level of antibodies to gluten). The American College of Gastroenterology (ACG) recommends that antibody testing, especially immunoglobulin, A anti-tissue transglutaminase antibody (IgA TTG), is the best first test for suspected celiac disease, although biopsies are needed for confirmation; in children younger than 2 years, the IgA TTG test should be combined with testing for IgG-deamidated gliadin peptides. Researchers have found that persons with celiac disease have higher than normal levels of certain antibodies in their blood. These antibodies by the immune system in response to substances (such as gluten) that the body perceives as threatening.

• Biopsy

To diagnose celiac disease, the physician may remove a tiny piece of tissue from the small intestine to check for damage to the villi. During the procedure, the physician eases a long, thin tube, called an endoscope, through the mouth and stomach into the small intestine. A sample of tissue is then taken using instruments passed through the endoscope. This procedure is considered the "gold standard" for diagnosis of celiac disease.

Other laboratory tests include the following:

- Electrolytes and chemistries Electrolyte imbalances; evidence of malnutrition
- Hematologic tests Aneamia, low serum iron level, prolonged prothrombin time (PT), international normalized ratio (INR)
- Stool examination Fat malabsorption
- Oral tolerance tests Lactose intolerance

Serology - Immunoglobulin A (IgA) antibodies

Treatment of Celiac Disease

The only known effective treatment is a strict lifelong gluten-free diet, which leads to recovery of the intestinal mucosa, improves symptoms and reduces risk of developing complications in most people (Kaukinen, Makharia, Gibson and Murray, 2015). Adhering to a gluten-free diet is a lifetime requirement, as eating any gluten will further damage the intestine.

For most persons, eliminating gluten from their diet will stop symptoms, heal intestinal damage that has already occurred, and prevent further damage. Usually, a person will see an improvement in symptoms within days of starting the diet and, within three to six months, the small intestine is usually completely healed, with villi intact and working. For older persons, complete healing may take up to two years. If untreated, it may result in cancers such as intestinal lymphoma and a slightly increased risk of early death (Lebwohl, Ludvigsson and Green, 2015).

Specific treatment for celiac disease will be determined by your physician based on:

- your age, overall health, and medical history
- extent of the disease
- your tolerance of specific medicines, procedures, or therapies
- expectations for the course of the disease
- your opinion or preference

Dietitian input is generally requested to ensure the person is aware which foods contain gluten, which foods are safe, and how to have a balanced diet despite the limitations. In many countries, gluten-free products are available on prescription and may be reimbursed by health insurance plans. Gluten-free products are usually more expensive and harder to find than common gluten-containing foods. Since ready-made products often contain traces of gluten, some celiacs may find it necessary to cook from scratch. A systematic review tentatively concluded that consumption of less than 10 mg of gluten per day is unlikely to cause histological abnormalities, although it noted that few reliable studies had been done (Akobeng and Thomas, 2008). Regulation of the label "gluten-free" varies. Gluten-free diet improves healthcare-related quality of life, and strict adherence to the diet gives more benefit than incomplete adherence. Nevertheless, gluten-free diet doesn't completely normalise the quality of life (Burger, de Brouwe, IntHout, Wahab, Tummers

and Drenth, 2017).

Codex Alimentarius Commission Standards for gluten free foods In CD, it is generally accepted that the term 'gluten-free' refers to a level of gluten that is supposed to be harmless, when ingested indefinitely, rather than to total absence of gluten. Considerable controversy exists among authorities as to what constitutes a 'gluten-free diet'. In 1981, the World Health Organisation (WHO) and Food and Agricultural Organisation (FAO) sets International Standards for foods, Codex Alimentarius, suggested that foods labeled as 'gluten-free' should contain less than 0.05g nitrogen per 100 g of food products on a dry matter basis (Codex Alimentarius- Commission, 1981). At the time this standard was set, methods for directly measuring the gluten content of grain were not available; therefore the nitrogen content of food was used as an indirect measure. It has since been approximated that wheat starch-based 'gluten-free' products meeting the 1981 Codex standard may contain up to 40-60 mg of gluten per 100 g or 400-600 ppm {mg per kilogram} (Thompson 2001; Peraaho, 2003 Kaukinen; 1999). This amount of gluten is equivalent to 200 to 300 ppm gliadin {mg per kilogram} (Thompson, 2001). In 1998, a draft revised standard for Codex gluten-free foods was proposed (Codex Alimentarius- Commission, 1998). In the revised standard, it was suggested that naturally 'gluten-free' foods (i.e. food consisting of or made only from ingredients which do not contain any prolamins from wheat or all Triticum species such as spelt, kamut or durum wheat, rye, barley, [oats] or their crossbred varieties) should not contain more than 20 ppm of gluten but that foods consisting of ingredients from wheat, rye, barley, oats, spelt or their crossbred varieties, which have been rendered 'gluten-free' should not contain more than 200 ppm gluten. 'Oats' was put in brackets because the committee could not decide whether it was toxic to people with CD. Although the revised Codex standard of not more than 200 ppm gluten in wheat starch-based gluten free products has been adopted in a number of countries including the United Kingdom (UK) and some European countries, it is not universally accepted. In some countries such as the United States of America (USA), food made from wheat starch is not recommended, and a naturally gluten-free diet is prescribed (Ciclitira 2005). These different practices reflect the fact that the exact amount of gluten that can be tolerated long term without harmful effects by patients with CD is unclear.

Nutritional Management of Celiac Disease

Medical nutrition therapy (MNT) is the only accepted treatment for celiac disease, the gluten- free (GF) diet (GFD). (Kupper, 2006). Good nutritional management of patients with celiac disease requires attention to a number of areas. Careful assessment and supervision of the patient's diet within the context of the patient's cultural, ethnic and religious background are essential. Medical monitoring is also important. However nutritional interventions that do not take the food preferences into account are unlikely to be successful. Close collaboration is necessary between the professionals providing medical care. Inadequate food consumption leads to wasting and functional changes in all tissues. Second, the general metabolic response to infection, trauma or other stress results in further specific nutrient losses and cellular damage.

Nutritional management primary goals for celiac disease

- The primary goals of MNT for people with celiac disease are to promote optimal health.
- To prevent and treat malabsorption/malnutrition and other comorbidities, and improve quality of life.

Medical Nutrition Therapy for celiac disease based on Nutrition care process (NCP) by Academy of Nutrition and Dietetics (AND) (2015).

A. Nutrition Assessment

- 1. Client history this comprises of the following
- Medical/health history
- Medication and supplement history
- Social history
- Personal history

Biochemical data

A detailed laboratory assessment is recommended at the time of initial assessment and again on admission to hospital. Recommended initial screening investigations are given below, further investigations may be required, depending on the patient's condition. If significant abnormalities are detected, expert advice may be needed from an appropriate specialist. Patients with previous biochemical abnormalities and those who purge are at particular risk. A low haemoglobin concentration may indicate iron deficiency. Furthermore as lean body mass decreases there is an associated fall in red cell mass. The iron derived from the reduced red cell mass cannot be excreted and is therefore stored, bound to ferritin. There is an increased risk of unbound iron, which acts as a centre for free radical generation and hence cell damage. Thus, in severely undernourished individuals there is an effective block on iron utilization, and any dietary supplementation cannot be used to good effect in this situation.

Relevant laboratory tests and values to check includes

• Hematologic tests - Anemia, low serum iron level, prolonged prothrombin time (PT), international normalized ratio (INR)

Table below shows the WHO (2005) classification of Hemoglobin thresholds

Age or group or gender	Hb (heamoglobin concentration) Threshold (g/dl)	Hb Threshold (mmol/l)
Children (0 to 5yrs)	11.0	6.8
Children (5 to 12 yrs)	11.5	7.1
Teens (11 to 15 yrs)	12.0	7.4
Women, nonpregnant (>15yrs)	12.0	7.4
Women, pregnant (<15yrs)	11.0	6.8
Men (>15yrs)	13.0	8.1

- Stool examination to dictate Fat malabsorption
- Oral tolerance tests to dictate Lactose intolerance
- Serology to find out Immunoglobulin A (IgA) antibodies
- Electrolytes and chemistries to find out Electrolyte imbalances; evidence of malnutrition Ranges according to medical e study (2020) for body electrolytes

Calcium Normal Values			
Age	Conventional Units	SI Units (Conventional Units × 0.25)	
Cord	8.2-11.2 mg/dL	2.1-2.8 mmol/L	
0-10 days	7.6-10.4 mg/dL	1.9-2.6 mmol/L	
11 days-2 yr	9-11 mg/dL	2.2-2.8 mmol/L	
3-12 yr	8.8-10.8 mg/dL	2.2-2.7 mmol/L	
13-18 yr	8.4-10.2 mg/dL	2.1-2.6 mmol/L	
Adult	8.2-10.2 mg/dL	2.1-2.6 mmol/L	
Adult older than 90 yr	8.2-9.6 mg/dL	2.1-2.4 mmol/L	

Ago	Conventional & SI Units
Age	Si Units
Premature	95-110 mEq/L or mmol/L
0-1 mo	98-113 mEq/L or mmol/L
2 mo-older adult	97-107 mEq/L or mmol/L

Magnesium Normal Values

Age	Conventional Units	SI Units (Conventional Units × 0.4114)
Newborn	1.7-2.5 mg/dL	0.7-1 mmol/L
Child	1.7-2.3 mg/dL	0.7-0.95 mmol/L
Adult	1.6-2.6 mg/dL	0.66-1.07 mmol/L

Phosphorus Normal Values

Age	Conventional Units	SI Units (Conventional Units × 0.323)
0-5 day 1-3 yr 4-6 yr	4.6-8 mg/dL	1.5-2.6 mmol/L
1-3 yr	3.9-6.5 mg/dL	1.3-2.1 mmol/L
4-6 yr	4-5.4 mg/dL	1.3-1.7 mmol/L
7-11 yr	3.7-5.6 mg/dL	1.2-1.8 mmol/L
7–11 yr 12–13 yr 14–15 yr	3.3-5.4 mg/dL	1.1-1.7 mmol/L
14-15 yr	2.9-5.4 mg/dL	0.9-1.7 mmol/L
16-19 yr	2.8-4.6 mg/dL	0.9-1.5 mmol/L
Adult	2.5-4.5 mg/dL	0.8-1.4 mmol/L

Potassium Normal Values

Serum	Conventional & SI Units
Cord	5.6-12 mEq/L or mmol/L
1-12 hr	5.3-7.3 mEq/L or mmol/L
12-24 hr	5.3-8.9 mEq/L or mmol/L
24-48 hr	5.2-7.3 mEg/L or mmol/L
48-72 hr	5-7.7 mEq/L or mmol/L
3-7 days	3.2-5.5 mEq/L or mmol/L
8 days-1 mo	3.4-6 mEq/L or mmol/L
1-5 mo	3.5-5.6 mEq/L or mmol/L
6 mo-1 yr	3.5-6.1 mEg/L or mmol/L
2-19 yr	3.8-5.1 mEq/L or mmol/L
Adult-older adult	3.5-5.3 mEq/L or mmol/L

Sodium Normal Values

Age	Conventional & SI Units	
Cord	126-166 mEg/L or mmol/L	
1-12 hr	124-156 mEq/L or mmol/L	
12-24 hr	132-159 mEq/L or mmol/L	
24-48 hr	134-160 mEq/L or mmol/L	
48-72 hr	139-162 mEq/L or mmol/L	
Newborn	135-145 mEq/L or mmol/L	
7 d-1 mo	134-144 mEq/L or mmol/L	
2 mo-5 mo	134-142 mEq/L or mmol/L	
6 mo-1 yr	133-142 mEq/L or mmol/L	
Child-Adult-older adult	135-145 mEq/L or mmol/L	

3. Anthropometric measurements

- Measurement of weight kilogram and height in centimeters
- Recumbent Length for infants and children that are 85cm below or above but cannot stand. (If a child is 85cm or more but cannot be measured standing, subtract 0.5 cm from the supine length).
- Weight for age and Height for age (for infants and children). Low weight for age (wasting) and length/height for age (stunting) are signs of malnutrition
- Z score (for infants and children) less than minus three standard deviation (<-3SD) is a sign of malnutrition.
- Mid arm circumference (MAC) for infants and children in

cm (MAC 0 to 11.5cm indicates severe acute malnutrition, MAC between 11.5 cm to 12.5 cm indicates moderate acute malnutrition, MAC from 12.5 cm and above indicates normal World Health Organisation, 2009).

• Body Mass Index (BMI) in kilogram per metre square kg/m (less than 18.5 kg/m is underweight and an indicator of the severity of starvation). Weighing should be as consistent as possible.

4.Food/nutrition history

The food and Nutrition history taken by a dietician is a reliable

tool for assessing habitual dietary intake in patients with celiac disease (Hadigan, 2000). It can be used to identify specific deficiencies of protein, fatty acids and micronutrients. Common Nutrient Deficiencies in Celiac Disease includes protein, Iron, Calcium, Vitamin D Magnesium, Zinc Folate, niacin, B12, riboflavin Folate, niacin (codex-Alimentarius Commission, 1983). Although patients characteristically overestimate their energy intake, body weight is an easily measured indicator of energy deficiency. Food and diet history comprises of the following.

- Food intakes
- Nutrition and health awareness
- · Physical activity and exercise
- Food availability
- Psychosocial and economic issues impacting nutrition therapy
- Consideration of co-morbid conditions and need for additional modifications in nutrition care plan

5. Physical examination findings

Sometimes, persons with celiac disease are asymptomatic, as the undamaged part of the small intestine is still able to absorb enough nutrients. However, these persons are still at risk for complications of the disease. The symptoms of celiac disease may resemble other medical conditions or problems. A physical exam may reveal the following:

- Skin disorders including dermatitis herpetiformis, a condition with pruritic, papulovesicular skin lesions involving the extensor surfaces of the extremities, trunk, buttocks, scalp, and neck
- A protuberant and tympanic abdomen
- Evidence of weight loss
- recurring abdominal pain and bloating
- pale, foul-smelling stool
- muscle cramps and/or bone pain
- pain in the joints
- tingling numbness in the legs
- · delayed growth
- fatigue
- · painful skin rash
- missed menstrual periods (which is linked to excessive weight loss)
- · tooth discoloration or loss of enamel

B. Nutrition Diagnosis

Based on the rigorous assessments Nutrition Diagnosis is made

Below are some of the likely Nutrition diagnosis common in Celiac Disease

- Inadequate energy intake
- Excessive energy intake
- Inadequate oral food/beverage intake
- Excessive oral food/beverage intake
- Inadequate intake from enteral/Parenteral nutrition
- ❖ Excessive intake from enteral/Parenteral nutrition
- Inappropriate infusion of enteral/Parenteral nutrition
- Inadequate fluid intake
- Excessive fluid intake
- Excessive alcohol intake
- Evident protein-energy malnutrition
- Inadequate fiber intake
- Excessive fiber intake

- ❖ Altered GI function
- ❖ Altered nutrition-related laboratory values
- Underweight
- Involuntary weight loss
- Overweight/obesity
- ❖ Involuntary weight gain
- ❖ Food and nutrition-related knowledge deficit
- Swallowing difficulty
- Biting/Chewing (Masticatory) difficulty
- * Physical inactivity
- ❖ Inability or lack of desire to manage self-care
- Impaired ability to prepare foods/meals
- Self-feeding difficulty
- . Limited access to food

C. Nutrition Intervention (Planning and Implementation)

Nutrition interventions are based on Individualized assessments and diagnosis, they are prescribed based on the following

- 1. Food/Nutrition Intervention
- 2. Physical activity Interventions
- 3. Behavioral Interventions
- 4. Pharmacotherapy, when indicated

Nutrition Planning

Nutrition planning is targeted

- ❖ To Control gluten intake by giving gluten free diet.
- ❖ Provide all nutrients in quantities adequate to ensure that needs are met for growth, development and activity.
- ❖ Treat nutritional deficiencies and complications.
- ❖ Educate patients and care givers on the need for dietary modifications and lifestyle changes.
- ❖ For catch-up growth, energy and protein recommendation. When catch-up growth has occurred, a gluten-controlled diet that reflects normal energy and nutrient needs should be recommended.

Nutrition Implementations

Implementing the GFD once the diagnosis is made is imperative. Multivitamin or nutritional supplement are usually given, these help to correct deficiencies and build levels of essential nutrients needed for optimum growth should be taken. But some vitamins, minerals, and herbal supplements contain an ingredient called lecithin. Lecithin can be a hidden source of gluten. If gluten-free diet is nutritionally inadequate, prior to initiation of dietary treatment, nutrient deficiencies may be seen as a result of fat malabsorption and inadequate intake. Water miscible preparations of fat-soluble vitamins and a daily vitamin supplement may be indicated to convert these deficiencies in children. If the patient is also lactose intolerant and is not able to consume or tolerate adequate amounts of lactose treated dairy products, calcium supplementation to meet requirements may be indicated. Vitamin and supplementation may also be needed initially for patients with metabolic bone disease. Iron, folate or vitamin B12 supplementation maybe necessary for patients with anaemia. Increased absorption of nutrients occurs with dietary treatment, even if the recovery of intestinal mucosa may not be seen for a few months, so supplements may not be needed for more than a few months. If diarrhea has been severe treat with Oral rehydration soluction (ORS). For cases of electrolyte imbalances foods rich in minerals such as fruits, vegetables,

meat and fish should be given, electrolyte supplementation might be needed for the first few days of therapy. With severe malabsorption, calcium and magnesium blood levels may be low and thus may need to be corrected by supplementation.

For energy requirements, a high kilocaloric and high protein diet is recommended, especially if weight loss and specific deficiencies owing to malabsorption are pronounced. Encourage physical activities for physical inactive patients. Also check drug nutrient interactions and remodify diet where applicable.

Complete nutrition education in multiple visits, following the needs and learning ability of the patient since GFD diet is complex and can easily overwhelm patients. Follow-up education sessions should expand to include more detailed information and skills, as well as weight management and adjustments to improved nutritional balance of the diet. Long-term, patients with CD should receive a follow up session with the dietitian at least annually, possibly more frequently with children, pregnant and lactating women, and elderly patients.

Recommendations

- 1. Use of gluten free diet.
- 2. Proper monitoring for tolerance is necessary because temporary tolerance to lactose is common and temporary intolerance to fat is sometimes seen.
- 3. Some control of dietary fat should be considered because of unabsorbed long chain fatty acid which may produce diarrhoea. With control of symptoms, milk and milk products should again be added to the diet and the level of fat can be increased. Gluten-free diets are sometimes low in fiber, which can lead to constipation. Fibre rich foods such as fruits and vegetables should be given. A fiber supplement with psyllium seed husks can also help, as can fiber-rich foods, like fruits and veggies (Codex Alimentarius Commission, 1983). The widespread use of emulsifiers, thickners, or other additives derived from gluten containing grains in commercially processed foods further complicates strict adherence to gluten-free diet.
- 4. It is important for patients to read food labels carefully and to avoid products that lost ingredients that cannot be verified as gluten free by the manufacturer. The unintentional consumption of gluten is the most common cause of reoccurrence of symptoms. Other reasons patient may fail to maintain a strict gluten-free diet are boredom with the taste of alternatives to wheat, breads, crackers and pasta and the limited availability of appropriate foods when eating away from home.
- 5. Physical activities is also necessary for physical inactive patients

Foods to Avoid

Any foods made with wheat, barley, or rye contain some amount of gluten. That means that people with celiac disease shouldn't eat most breakfast cereal, bread, pasta, and processed food (Manual of clinical dietetics, 2000).

Processed foods that may contain gluten include:

- Bouillon cubes
- Brown rice syrup
- Candy
- Chewing gum
- Chips, including seasoned tortilla and potato chips

- · Cold cuts, hot dogs, salami, and sausage
- Communion wafers
- French fries
- Gravy
- Imitation fish
- Rice mixes
- Soy sauce
- · Beer and malt beverages

Soups and sauces are common sources of hidden gluten, as wheat is often used as a thickener. Pay special attention to sauces or soups that are cream based.

Gluten free foods includes

Cutting gluten out of your diet may seem like a difficult task, but many foods are naturally gluten free.

In general, the following food groups are naturally gluten-free:

- Fruits
- Vegetables
- Fresh cuts of meat and poultry
- Fish and seafood
- Dairy
- Beans, legumes, and nuts

Beware, though, that prepared or processed versions of any of the above foods, such as sausage or ice cream, may contain gluten. Here are many gluten-free grains and starches that you can substitute for wheat, barley, or rye products:

- Rice
- Corn
- Potato (but not potato chips)
- Tapioca
- Sorghum
- Soy
- Quinoa
- Millet
- Arrowroot
- $\bullet \ Amaranth$
- Teff
- Flax
- Chia
- · Gluten-free oats
- Buckwheat
- Nut flours
- · Bean flours

Tips for Dining Out on a Gluten Free Diet

The following strategies may help you stick to your gluten-free diet when eating out:

- Choose a restaurant with gluten-free options. This means picking a place that serves naturally gluten-free foods or has a special gluten-free menu.
- Inform your waiter. Let them know you have celiac disease and may get sick if you eat anything containing gluten, including flour, bread crumbs, or soy sauce. Also ask them to inform the chef or cook. This way, you'll have more confidence that nothing on your plate has touched gluten.
- Ask questions. Don't assume anything is gluten-free. Omelets, for instance, may have pancake batter added to the egg mixture to make them fluffier, and baked potatoes can be coated with flour to make the skins brown and crispy.

Monitoring and Evaluation

The monitoring is measuring and evaluating of outcomes against criteria to determine changes in specific

- Indicators of MNT outcomes.
- Food and nutrient intake outcomes
- Nutrition-related physical sign/symptoms outcomes
- Nutrition-related patient/client centered outcomes
- Physical activity
- Compliance to dietary regimen

2. Conclusion

Celiac disease is as a chronic disease in which the gliadin protein fraction of gluten causes a mucosal lesion of the small intestinal that impairs nutrient absorption. It is one of the most common intestinal diseases to cause malabsorption in childhood. An initial assessment of the patient's current nutritional status and potential risk factors for associated complications must be made and psychosocial, learning ability, and economic concerns addressed. Symptoms often begin during the second six months of life after wheat containing foods are added to the diet. Diminished body weight is seen in almost all patients especially in those with prolonged active disease; short stature is also common in children. Other symptoms include pain and discomfort in the digestive tract, chronic constipation and diarrhea, anaemia and fatigue. Medical nutrition therapy (MNT) is the only accepted treatment for celiac disease. The cornerstone of treatment for CD is elimination of gluten from the diet. In most patients diagnosed with CD, a strict gluten-free diet (GFD) alone should result in complete symptomatic and histologic resolution of the disease and reduce risk of complications. Nutritional supplements may be necessary, as well as referrals to social services for support of the person as he or she adjusts to this life-altering condition Noncompliance with diet is the leading cause of failure to respond in patients with CD. Good nutritional management of patients with celiac disease requires attention to a number of areas. Careful assessment and supervision of the patient's diet within the context of the patient's cultural, ethnic and religious background are essential. It is important for patients to read food labels carefully and to avoid products that lost ingredients that cannot be verified as gluten free by the manufacturer. The unintentional consumption of gluten is the most common cause of reoccurrence of symptoms. Other reasons patient may fail to maintain a strict gluten-free diet are boredom with the taste of alternatives to wheat, breads, crackers and pasta and the limited availability of appropriate foods when eating away from home. Medical monitoring, physical activities for physical inactive patients and follow-up education sessions are necessary for optimum management.

References

- 1. Abutaleb Y. (2013) FDA issues new rules for labeling 'gluten-free' foods. Reuters Health Information.
- 2. Akobeng, A. K., & Thomas, A. G. (2008). Systematic review: tolerable amount of gluten for people with coeliac disease. Alimentary pharmacology & therapeutics, 27(11), 1044-1052.
- 3. Alaedini, A., & Green, P. H. (2005). Narrative review: celiac disease: understanding a complex autoimmune disorder. Annals of internal medicine, 142(4), 289-298.
- 4. Alaedini, A., & Green, P. H. (2005). Narrative review: celiac

- disease: understanding a complex autoimmune disorder. Annals of internal medicine, 142(4), 289-298.
- Anderson, R. P., Henry, M. J., Taylor, R., Duncan, E. L., Danoy, P., et al. (2013). A novel serogenetic approach determines the community prevalence of celiac disease and informs improved diagnostic pathways. BMC medicine, 11, 1-13
- Askling, J., Linet, M., Gridley, G., Halstensen, T. S., Ekström, K., et al. (2002). Cancer incidence in a populationbased cohort of individuals hospitalized with celiac disease or dermatitis herpetiformis. Gastroenterology, 123(5), 1428-1435.
- 7. Benelli E, Zin A, Martelossi S. (2019) Celiac disease in children. Minerva Pediatr. 71(1):39-46.
- 8. Biesiekierski, Jessica R (2017). What is gluten?. Journal of Gastroenterology and Hepatology. 32:78-81.
- 9. Boggs W. (2013) Genetic testing might improve diagnosis of celiac disease. Medscape Medical News.
- 10. Caio, G., De Giorgio, R., Ursini, F., Fanaro, S., & Volta, U. (2018). Prevalence of celiac disease serological markers in a cohort of Italian rheumatological patients. Gastroenterology and Hepatology from bed to bench, 11(3), 244.
- 11. Catassi, C., Rätsch, I. M., Fabiani, E., Rossini, M., Coppa, G. V., et al. (1994). Coeliac disease in the year 2000: exploring the iceberg. The Lancet, 343(8891), 200-203.
- 12. Cavallaro, R., Iovino, P., Castiglione, F., Palumbo, A., Marino, M., et al. (2004). Prevalence and clinical associations of prolonged prothrombin time in adult untreated coeliac disease. European journal of gastroenterology & hepatology, 16(2), 219-223.
- 13. Ciclitira, P. J., Johnson, M. W., Dewar, D. H., & Ellis, H. J. (2005). The pathogenesis of coeliac disease. Molecular aspects of medicine, 26(6), 421-458.
- 14. Codex-Alimentarius-Commission. (1981). Codex standard for "Gluten Free Foods." Codex standard. Joint FAO. WHO Food Standards Programme. WHO, 118.
- 15. Collin, P., Kaukinen, K., Vogelsang, H., Korponay-Szabó, I., Sommer, R., et al. (2005). Antiendomysial and antihuman recombinant tissue transglutaminase antibodies in the diagnosis of coeliac disease: a biopsy-proven European multicentre study. European journal of gastroenterology & hepatology, 17(1), 85-91.
- 16. Collin, P., Thorell, L., Kaukinen, K., & Mäki, M. (2004). The safe threshold for gluten contamination in gluten-free products. Can trace amounts be accepted in the treatment of coeliac disease?. Alimentary pharmacology & therapeutics, 19(12), 1277-1283.
- Cristofori, F., Fontana, C., Magistà, A., Capriati, T., Indrio, F., et al. (2014). Increased prevalence of celiac disease among pediatric patients with irritable bowel syndrome: a 6-year prospective cohort study. JAMA pediatrics, 168(6), 555-560.
- Cummins, A. G., & Roberts-Thomson, I. C. (2009). Prevalence of celiac disease in the Asia–Pacific region. Journal of gastroenterology and hepatology, 24(8), 1347-1351.
- 19. Dahele, A., & Ghosh, S. (2001). Vitamin B12 deficiency in untreated celiac disease. The American journal of gastroenterology, 96(3), 745-750.
- 20. Delco, F., El-Serag, H. B., & Sonnenberg, A. (1999). Celiac

- sprue among US military veterans (associated disorders and clinical manifestations). Digestive diseases and sciences, 44, 966-972.
- 21. Devlin, S. M., Andrews, C. N., & Beck, P. L. (2004). Celiac disease. CME update for family physicians. Canadian family physician, 50(5), 719-725.
- 22. Dickey, W. (2002). Low serum vitamin B12 is common in coeliac disease and is not due to autoimmune gastritis. European journal of gastroenterology & hepatology, 14(4), 425-427.
- 23. Dieterich, W., Ehnis, T., Bauer, M., Donner, P., Volta, U., et al. (1997). Identification of tissue transglutaminase as the autoantigen of celiac disease. Nature medicine, 3(7), 797-801.
- Duerksen, D., Pinto-Sanchez, M. I., Anca, A., Schnetzler, J., Case, S., et al. (2018). Management of bone health in patients with celiac disease: practical guide for clinicians. Canadian Family Physician, 64(6), 433-438.
- 25. Enns, R. A., Hookey, L., Armstrong, D., Bernstein, C. N., Heitman, S. J., et al. (2017). Clinical practice guidelines for the use of video capsule endoscopy. Gastroenterology, 152(3), 497-514.
- Errichiello, S., Esposito, O., Di Mase, R., Camarca, M. E., Natale, C., et al. (2010). Celiac disease: predictors of compliance with a gluten-free diet in adolescents and young adults. Journal of pediatric gastroenterology and nutrition, 50(1), 54-60.
- 27. Farrell, R. J., & Kelly, C. P. (2002). Celiac sprue. New England Journal of Medicine, 346(3), 180-188.
- Fasano, A., Berti, I., Gerarduzzi, T., Not, T., Colletti, R. B., et al. (2003). Prevalence of celiac disease in at-risk and not-at-risk groups in the United States: a large multicenter study. Archives of internal medicine, 163(3), 286-292.
- 29. Fasano, A., & Catassi, C. (2001). Current approaches to diagnosis and treatment of celiac disease: an evolving spectrum. Gastroenterology, 120(3), 636-651.
- Frellick M. Celiac disease screening should focus on kids with IBS. Medscape.
- Gale, L., Wimalaratna, H., Brotodiharjo, A., & Duggan, J. M. (1997). Down's syndrome is strongly associated with coeliac disease. Gut, 40(4), 492-496.
- 32. Green, P. H., & Jabri, B. (2003). Coeliac disease. The Lancet, 362(9381), 383-391.
- 33. Hallert, C., Grant, C., Grehn, S., Grännö, C., Hultén, S., et al. (2002). Evidence of poor vitamin status in coeliac patients on a gluten-free diet for 10 years. Alimentary pharmacology & therapeutics, 16(7), 1333-1339.
- 34. Harbour, R., & Miller, J. (2001). A new system for grading recommendations in evidence based guidelines. Bmj, 323(7308), 334-336.
- 35. Harding A. (2014) Endoscopy without biopsy may miss many with celiac disease. Reuters Health Information.
- Herman, M. L., Rubio-Tapia, A., Wu, T. T., & Murray, J. A. (2015). A case of severe sprue-like enteropathy associated with valsartan. ACG Case Reports Journal, 2(2), 92.
- 37. Hill, I. D., Dirks, M. H., Liptak, G. S., Colletti, R. B., Fasano, A., et al. (2005). Guideline for the diagnosis and treatment of celiac disease in children: recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. Journal of pediatric gastroenter-

- ology and nutrition, 40(1), 1-19.
- 38. Hoffenberg, E. J., Haas, J., Drescher, A., Barnhurst, R., Osberg, I., et al. (2000). A trial of oats in children with newly diagnosed celiac disease. The Journal of pediatrics, 137(3), 361-366.
- Howard, M. R., Turnbull, A. J., Morley, P., Hollier, P., Webb, R., et al. (2002). A prospective study of the prevalence of undiagnosed coeliac disease in laboratory defined iron and folate deficiency. Journal of clinical pathology, 55(10), 754-757.
- Husby, S., Koletzko, S., Korponay-Szabó, I., Kurppa, K., Mearin, M. L., et al. (2020). European society paediatric gastroenterology, hepatology and nutrition guidelines for diagnosing coeliac disease 2020. Journal of Pediatric Gastroenterology and Nutrition, 70(1), 141-156.
- 41. Johnson, T. C., Diamond, B., Memeo, L., Negulescu, H., Hovhanissyan, Z., et al. (2004). Relationship of HLA-DQ8 and severity of celiac disease: comparison of New York and Parisian cohorts. Clinical Gastroenterology and Hepatology, 2(10), 888-894.
- 42. Kagnoff, M. F. (2007). Celiac disease: pathogenesis of a model immunogenetic disease. The Journal of clinical investigation, 117(1), 41-49.
- 43. Kaur, N., Bhadada, S. K., Minz, R. W., Dayal, D., & Kochhar, R. (2018). Interplay between type 1 diabetes mellitus and celiac disease: implications in treatment. Digestive diseases, 36(6), 399-408.
- 44. Kelly, C. P., Bai, J. C., Liu, E., & Leffler, D. A. (2015). Advances in diagnosis and management of celiac disease. Gastroenterology, 148(6), 1175-1186.
- 45. Kleopa, K. A., Kyriacou, K., Zamba-Papanicolaou, E., & Kyriakides, T. (2005). Reversible inflammatory and vacuolar myopathy with vitamin E deficiency in celiac disease. Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine, 31(2), 260-265.
- 46. Kordonouri, O., Dieterich, W., Schuppan, D., Weber, G., Müller, C., et al. (2000). Autoantibodies to tissue transglutaminase are sensitive serological parameters for detecting silent coeliac disease in patients with type 1 diabetes mellitus. Diabetic Medicine, 17(6), 441-444.
- 47. Lewis R. Pediatric celiac disease more common with HLA haplotype. Medscape Medical News.
- 48. Lionetti, E., Castellaneta, S., Francavilla, R., Pulvirenti, A., Tonutti, E., et al. (2014). Introduction of gluten, HLA status, and the risk of celiac disease in children. New England Journal of Medicine, 371(14), 1295-1303.
- Lionetti, E., & Catassi, C. (2011). New clues in celiac disease epidemiology, pathogenesis, clinical manifestations, and treatment. International reviews of immunology, 30(4), 219-231.
- Liu, E., Lee, H. S., Aronsson, C. A., Hagopian, W. A., Koletzko, S., et al. (2014). Risk of pediatric celiac disease according to HLA haplotype and country. New England Journal of Medicine, 371(1), 42-49.
- 51. Lowry F. Current screening guidelines fail to identify celiac disease. Medscape Medical News from WebMD.
- Lundin, K. E. A., Nilsen, E. M., Scott, H. G., Løberg, E. M., Gjøen, A., et al. (2003). Oats induced villous atrophy in coeliac disease. Gut, 52(11), 1649-1652.
- 53. MacReady N. Celiac disease: new data may change

- recommendations for kids. Medscape Medical News.
- Mahadev, S., Laszkowska, M., Sundström, J., Björkholm, M., Lebwohl, B., et al. (2018). Prevalence of celiac disease in patients with iron deficiency anemia—a systematic review with meta-analysis. Gastroenterology, 155(2), 374-382.
- 55. Marthey, L., Cadiot, G., Seksik, P., Pouderoux, P., Lacroute, J., et al. (2014). Olmesartan-associated enteropathy: results of a national survey. Alimentary pharmacology & therapeutics, 40(9), 1103-1109.
- 56. Martucciello, S., Paolella, G., Esposito, C., Lepretti, M., & Caputo, I. (2018). Anti-type 2 transglutaminase antibodies as modulators of type 2 transglutaminase functions: a possible pathological role in celiac disease. Cellular and Molecular Life Sciences, 75, 4107-4124.
- 57. Mention, J. J., Ahmed, M. B., Bègue, B., Barbe, U., Verkarre, V., et al. (2003). Interleukin 15: a key to disrupted intraepithelial lymphocyte homeostasis and lymphomagenesis in celiac disease. Gastroenterology, 125(3), 730-745.
- 58. Mollazadegan, K., Kugelberg, M., Lindblad, B. E., & Ludvigsson, J. F. (2011). Increased risk of cataract among 28,000 patients with celiac disease. American journal of epidemiology, 174(2), 195-202.
- Murray, J.A., Van Dyke, C., Plevak, M. F., Dierkhising, R. A., Zinsmeister, A. R., et al. (2003). Trends in the identification and clinical features of celiac disease in a North American community, 1950–2001. Clinical Gastroenterology and Hepatology, 1(1), 19-27.
- Negro, A., Rossi, G. M., Santi, R., Iori, V., & De Marco, L. (2015). A case of severe sprue-like enteropathy associated with losartan. Journal of Clinical Gastroenterology, 49(9), 794.
- Pazianas, M., Butcher, G. P., Subhani, J. M., Finch, P. J., Ang, L., et al. (2005). Calcium absorption and bone mineral density in celiacs after long term treatment with gluten-free diet and adequate calcium intake. Osteoporosis International, 16, 56-63.
- 62. Peräaho, M., Kaukinen, K., Paasikivi, K., Sievänen, H., Lohiniemi, S., et al. (2003). Wheat-starch-based glutenfree products in the treatment of newly detected coeliac disease: prospective and randomized study. Alimentary pharmacology & therapeutics, 17(4), 587-594.
- Pietzak, M. M., Schofield, T. C., McGinniss, M. J., & Nakamura, R. M. (2009). Stratifying risk for celiac disease in a large at-risk United States population by using HLA alleles. Clinical Gastroenterology and Hepatology, 7(9), 966-971.
- 64. Pietzak, M., & Kerner Jr, J. A. (2012). Celiac disease, wheat allergy, and gluten sensitivity: when gluten free is not a fad. Journal of parenteral and enteral nutrition, 36, 68S-75S.
- 65. Rashtak, S., & Murray, J. A. (2009). Celiac disease in the elderly. Gastroenterology Clinics, 38(3), 433-446.
- Richter, J. C., Netzer, P., Cottagnoud, P., & Stucki, A. (2006). Testing strategies and follow-up for coeliac disease in a general internal medicine outpatient department from 2000 to 2005. Swiss medical weekly, 136(4546), 732-738.
- 67. Robson, K., Alizart, M., Martin, J., & Nagel, R. (2014). Coeliac patients are undiagnosed at routine upper endoscopy. PloS one, 9(3), e90552.

- 68. Rosén, A., Sandström, O., Carlsson, A., Högberg, L., Olén, O., et al. (2014). Usefulness of symptoms to screen for celiac disease. Pediatrics, 133(2), 211-218.
- Rostami, K., Malekzadeh, R., Shahbazkhani, B., Akbari, M. R., & Catassi, C. (2004). Coeliac disease in Middle Eastern countries: a challenge for the evolutionary history of this complex disorder?. Digestive and liver disease, 36(10), 694-697.
- Rostom, A., Murray, J. A., & Kagnoff, M. F. (2006). American Gastroenterological Association (AGA) Institute technical review on the diagnosis and management of celiac disease. Gastroenterology, 131(6), 1981-2002.
- Rubio-Tapia, A., Herman, M. L., Ludvigsson, J. F., Kelly, D. G., Mangan, T. F., et al. (2012, August). Severe spruelike enteropathy associated with olmesartan. In Mayo clinic proceedings (Vol. 87, No. 8, pp. 732-738). Elsevier.
- Rubio-Tapia, A., Hill, I. D., Kelly, C. P., Calderwood, A. H., & Murray, J. A. (2013). ACG clinical guidelines: diagnosis and management of celiac disease. Official journal of the American College of Gastroenterology ACG, 108(5), 656-676.
- Rubio-Tapia, A., Kyle, R. A., Kaplan, E. L., Johnson, D. R., Page, W., et al. (2009). Increased prevalence and mortality in undiagnosed celiac disease. Gastroenterology, 137(1), 88-93.
- Rubio-Tapia, A., Ludvigsson, J. F., Brantner, T. L., Murray, J. A., & Everhart, J. E. (2012). The prevalence of celiac disease in the United States. Official journal of the American College of Gastroenterology ACG, 107(10), 1538-1544.
- 75. Rubio-Tapia, A., & Murray, J. A. (2019). Updated guidelines by the European Society for the Study of Coeliac Disease. United European Gastroenterology Journal, 7(5), 581-582.
- 76. Salmi, T. T., Collin, P., Korponay-Szabo, I. R., Laurila, K., Partanen, J., et al. (2006). Endomysial antibody-negative coeliac disease: clinical characteristics and intestinal autoantibody deposits. Gut, 55(12), 1746-1753.
- 77. Sanders, D. S., Hurlstone, D. P., McAlindon, M. E., Hadjivassiliou, M., Cross, S. S., et al. (2005). Antibody negative coeliac disease presenting in elderly people—an easily missed diagnosis. Bmj, 330(7494), 775-776.
- 78. Singh, P., Arora, A., Strand, T. A., Leffler, D. A., Catassi, C., et al. (2018). Global prevalence of celiac disease: systematic review and meta-analysis. Clinical gastroenterology and hepatology, 16(6), 823-836.
- 79. SKERRITT, J. H., & HILL, A. (1992). How "Free" is "Gluten Free"? Relationship Between Kjeldahl Nitrogen Values. Cereal Chem, 69(1), 110-112.
- Smedby, K. E., Åkerman, M., Hildebrand, H., Glimelius, B., Ekbom, A., et al. (2005). Malignant lymphomas in coeliac disease: evidence of increased risks for lymphoma types other than enteropathy-type T cell lymphoma. Gut, 54(1), 54-59.
- 81. Squires, J. E., Fei, L., & Cohen, M. B. (2014). Role of celiac disease screening for children with functional gastrointestinal disorders. JAMA pediatrics, 168(6), 514-515.
- 82. Thompson, T. (2001). Wheat starch, gliadin, and the gluten-free diet. Journal of the American Dietetic Association, 101(12), 1456-1459.
- 83. Tursi, A., Brandimarte, G., & Giorgetti, G. M. (2003).

- Prevalence of antitissue transglutaminase antibodies in different degrees of intestinal damage in celiac disease. Journal of clinical gastroenterology, 36(3), 219-221.
- 84. US Food and Drug Administration. FDA defines "glutenfree" for food labeling [press release]. August 2, 2013.
- 85. van Heel, D. A., & West, J. (2006). Recent advances in coeliac disease. Gut, 55(7), 1037-1046.
- Vriezinga, S. L., Auricchio, R., Bravi, E., Castillejo, G., Chmielewska, A., et al. (2014). Randomized feeding intervention in infants at high risk for celiac disease. New England Journal of Medicine, 371(14), 1304-1315.
- 87. World Health Organization (2008). Geneva: World Health

- Organization. Associated disorders?. Eur J Gastroenterol Hepatol. 2001 Sep. 13(9):1119-21.
- Yachha, S. K., Misra, S., Malik, A. K., Nagi, B., & Mehta, S. (1993). Spectrum of malabsorption syndrome in north Indian children. Indian journal of gastroenterology: official journal of the Indian Society of Gastroenterology, 12(4), 120-125.
- 89. Ludvigsson, J. F., Zingone, F., Tomson, T., Ekbom, A., & Ciacci, C. (2012). Increased risk of epilepsy in biopsyverified celiac disease: a population-based cohort study. Neurology, 78(18), 1401-1407.

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