Case Report

A Case of Celiac Disease Admitted to Our Clinic with Symptoms of Malabsorption

Burcin Atak Tel[*,1], Tuba Duman[1], Gulali Aktas[1], Satilmis Bilgin[1], Mustafa Ramiz Tel[2]

[1]*Abant Izzet Baysal University Hospital, Department of Internal Medicine, Bolu, Turkey.*
[2]*Abant Izzet Baysal University Hospital, Department of Emergency Medicine, Bolu, Turkey.*

Abstract: Celiac disease is an immune-mediated chronic intestinal disease that causes atrophy of the intestinal mucosa by ingestion of gluten and related proteins in genetically susceptible individuals. Presentation of the illness can differ from a severe malabsorption to mild symptomatic and even asymptomatic. The only treatment for celiac disease is to follow a strict gluten-free diet for life and it is not a cure.

A 37-year-old female patient was followed up with the diagnoses of irritable bowel syndrome and dyspepsia applied to our outpatient clinic with severe malabsorption symptoms. She was diagnosed as celiac disease.

Celiac disease should be questioned in all patients presenting with dyspeptic complaints. With a simple treatment, the quality of life increases significantly.

Keywords: Celiac disease, Chronic intestinal disease, Dyspepsia, Gluten-free diet, Malabsorption, Atrophy.

INTRODUCTION

Celiac disease is an immune-mediated chronic intestinal disease that causes atrophy of the intestinal mucosa by ingestion of gluten and related proteins in genetically susceptible individuals. According to data obtained from different regions, the prevalence of celiac disease is about 1%, and it has been increasing recently [1].

Presentation of the illness can differ from a severe malabsorption to mild symptomatic and even asymptomatic [2].

Genetic, environmental and immunological factors play a role in the pathogenesis of celiac. Genes responsible for the development of celiac disease have a strong link with human leukocyte antigens (HLA DQ2 and HLA DQ8). It has been reported that approximately 98% of patients carry one of these alleles [3].

In the pathogenesis of celiac disease, gliadin protein deamination by tissue transglutaminase in the lamina propria with the consumption of gluten-containing foods. The negatively charged molecules formed are presented as antigens and the release of T cell-mediated cytokines is increased. As a result of tissue damage, crypt hyperplasia, villus atrophy and lymphocyte infiltration are seen in the small intestines. Gliadin, tissue-transglutaminase and endomysial antibodies are released from B-cells [4].

The diagnosis of celiac disease is based on a combination of clinical, serological and histopathological data. In a group of children the diagnosis may be made without biopsy if strict criteria are met (classic small bowel symptoms, positive HLA DQ2 or DQ8 and immunoglobulin A [IgA], and levels 10 times higher of the normal rage for tissue transglutaminase [tTG]) . However, small intestine biopsy must be performed for a good and accurate histological evaluation [5].

The only treatment for celiac disease is to follow a strict gluten-free diet for life and it is not a cure. In this article, we present an adult celiac case with severe malabsorption.

CASE REPORT

A 37-year-old female patient; who, over the years, was followed up with the diagnoses of irritable bowel syndrome and dyspepsia by other clinics, was admitted to the internal medicine outpatient clinic because she was disturbed by the appearance of a swollen abdomen compared to her body. The patient have been seeking help from general surgery and internal medicine outpatient clinics for more than 10 years with the complaints of intermittent diarrhea and bloating. Reflux and irritable bowel syndrome treatments was given to the patient. There has been increased weight loss recently. There are no additional chronic diseases. His parents have no known history of genetic disorders. She is married and has two children.

In the physical examination of the patient, blood pressure was found to be 120/70 mmHg, fever: 36.5 ^{o}C, respiratory rate: 14 per minute, pulse: 90 beats/min. Her general appearance was cachectic (height:162 cm, weight:40kg bmi:.15), and exhausted. On abdominal examination, the abdomen is distended, increased bowel sounds and increased tympanic sound with percussion. There was no palpable organomegaly or mass. Other system examinations were normal.

*Address correspondence to this author at the Abant Izzet Baysal University Hospital, Department of Internal Medicine, Bolu, Turkey.
Email: burcinatak@hotmail.com

In laboratory examinations; erythrocyte sedimentation rate: 25mm/h Crp: 0.1 mg/l, thyroid function tests, liver enzymes and glomerular filtration rate were normal, serum albumin: 49 g/l. In whole blood count; hemoglobin: 10 g/dl, Mcv: 69 fL, Rdw was 17.8%. Vitamin B12:80 pg/ml, folate: 3 ng/ml, vitamin D: 8 ng/ml and ferritin:1ml/ng. The abdominal ultrasound was normal. Afterwards antiendomysium, antigliadin IgA was tested resulting highly positive and antitissue transglutaminase Ig A was 63 U/ml (0-12 U/ml) was also very high. Although the patient's laboratory supported the diagnosis of celiac disease, endoscopy and duodenal biopsy was performed with the gastroenterology consultation to make histological diagnosis. Scalloped duodenal folds, grooves and fissurations detected on endoscopy and crypt hyperplasia, villus atrophy and lymphocyte infiltration were observed in the pathology. After the diagnosis of celiac disease, the patient's dyspeptic complaints regressed with diet and abdominal distension quickly resolved.

DISCUSSION

Celiac disease is an immunologic disease triggered by gluten that is found in certain grains that causes widespread damage to the proximal small intestinal mucosa and malabsorption of nutrients [6]. In our case, diarrhea and cachexia were present due to severe malabsorption. It mostly occurs in childhood and adolescence usually after weaning from breast milk [3]. Our case was diagnosed at the age of 38, probably because it was overlooked due to crowding in the outpatient clinics. Another reason for late diagnosis of course the disease's own nature of mimicing other gastrointestinal symptoms [7]. Genes responsible for the development of celiac disease have a strong link with human leukocyte antigens (HLA DQ2 and HLA DQ8). It has been reported that approximately 98% of patients carry one of these alleles [5]. We could not find the opportunity to study genetic testing in our case. In mild cases, physical examination is normal. In cases with malabsorption, subcutaneous fat tissue and muscle loss, pallor due to anemia, easy bruising due to vitamin K deficiency, hyperkeratosis due to vitamin A deficiency, neurological findings due to vitamin B or vitamin E deficiency can be detected. Abdominal examination reveals hyperactive bowel sounds and abdominal distension. In some cases, dermatitis herpetiformis, the cutaneous variant of celiac disease, is seen [8]. When our case was evaluated, there were symptoms of severe celiac disease and physical examination findings.

Depending on the severity of the disease, malabsorption and related anemia and vitamin and calcium deficiencies may occur. Osteomalacia and osteoporosis should be investigated in hypocalcemia and vitamin D deficiency [9]. Our case was referred to be screened for osteoporosis.

The treatment is based on the elimination of all gluten in the diet. All wheat, barley and rye should be excluded from the diet. Patients should be given dietary education. Most patients with celiac disease may have permanent or temporary lactose intolerances. In these patients, dairy products should be avoided until symptoms improve with a gluten-free diet. Vitamin supplements may be required in the early stages of treatment. The biggest reason for failure in treatment is non-compliance with diet [10]. In our case, diet education was given and a gluten-free diet was started, and his symptoms began to regress. She is still in early follow-up stage.

As a result, celiac disease should be questioned in all patients presenting with dyspeptic complaints. With a simple treatment, the quality of life increases significantly.

CONFLICT OF INTEREST

Declared none.

ACKNOWLEDGEMENTS

Declared none.

REFERENCES

[[1] 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.

[2] Lebwohl B, Sanders DS, Green PHR. Coeliac disease. Lancet 2018; 391(10115): 70-81.

[3] Glissen Brown JR, Singh P. Coeliac disease. Paediatr Int Child Health 2019; 39(1): 23-31.

[4] Periolo N, Chernavsky AC. Coeliac disease. Autoimmun Rev 2006; 5(3): 202-8.

[5] Lundin KE, Wijmenga C. Coeliac disease and autoimmune disease-genetic overlap and screening. Nat Rev Gastroenterol Hepatol 2015; 12(9): 507-15.

[6] Chetcuti Zammit S, Sanders DS, Sidhu R. Refractory coeliac disease: what should we be doing different? Curr Opin Gastroenterol 2020; 36(3): 215-22.

[7] Rampertab SD, Pooran N, Brar P, Singh P, Green PH. Trends in the presentation of celiac disease. Am J Med 2006; 119(4): 355.e9-14.

[8] Malamut G, Cellier C. Refractory coeliac disease. Curr Opin Oncol 2013; 25(5): 445-51.

[9] Escudero-Hernandez C. Epithelial cell dysfunction in coeliac disease. Int Rev Cell Mol Biol 2021; 358: 133-64.

[10] Reilly NR, Aguilar K, Hassid BG, *et al*. Celiac disease in normal-weight and overweight children: clinical features and growth outcomes following a gluten-free diet. J Pediatr Gastroenterol Nutr 2011; 53(5): 528-31.

Received: January 01, 2023 Revised: March 08, 2023 Accepted: March 10, 2023