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Spectrum of Clinical Presentation Associated with Celiac Disease in a 55-Year-Old Woman

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Abstract

Introduction: Celiac disease is a chronic inflammatory autoimmune pathology of the intestine where its cornerstone is the immune reaction mediated to gluten. The disease has a prevalence of 1%-2% with an increasing trend in recent years. The inflammatory cascade that develops in the small intestine leads to progressive mucosal damage, villous atrophy and crypt hyperplasia in patients with genetic predisposition. It manifests with intraintestinal and extra-intestinal involvement, which often delays the diagnosis by exposing the patient to frequent and long-term complications. The basis of treatment is a gluten-free diet.

Methods: We describe a case with gastrointestinal and extraintestinal symptoms, which resulted in celiac disease. The diagnosis was based on laboratory, serological, and histopathological examinations, as well as the response to a gluten-free diet.

Results: A 55-year-old woman, with watery/frequent diarrhea, abdominal pain and bloating, headache for 9 days, while she had years of gastrointestinal problems, significant weight loss (35 kg in the last 2-3 years), emotional disorders. Laboratory, serological, imaging and histopathological examinations established the diagnosis of Celiac. The Anti tissue transglutaminase antibodies IgA test resulted positive, 392.9 U/mL (<20). The patient appears more stabilized and the recommended gluten-free diet enabled weight gain (46.6 kg five weeks after

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discharge, 50.7 kg eleven weeks, 55 kg ten months).

Conclusion: Celiac disease has numerous intra/extraintestinal manifestations, so clinicians must be vigilant in its timely identification. Proper dietary advice improves quality of life and avoids complications.

Keywords: Celiac disease, diarrhea, gluten-free diet, Transaminases, histopathological examinations

INTRODUCTION

Celiac disease is a chronic, multisystemic, autoimmune inflammatory bowel disease whose cornerstone is the immune reaction mediated by gluten. The disease has a prevalence of 1%-2% in the general population and an incidence with an increasing trend in the last 20 years (1). Early dating based on archaeological data from the Gobleki Tepe region of the Fertile Crescent, shows that this pathology was made possible when people moved from hunter-gatherer groups to societies dependent on agriculture to secure daily food (2). The first clear description was given by Samuel Gee in 1888 (3). The autoimmune process is the basis of the pathogenesis of this pathology, which is based on the deamidation of prolamin fragments, the formation of specific antibodies, and the activation of cytotoxic T cells (4). This inflammatory cascade that develops in the small intestine leads to progressive mucosal damage, villous atrophy, and crypt hyperplasia in patients with genetic predisposition (5). The presence of the human leukocyte antigens (HLA)-DQ2 and HLA-DQ8, described earlier, is a necessary condition for the development of the disease (6). Celiac disease manifests itself with both intraintestinal and extraintestinal effects, which delay diagnosis, causing the patient to suffer from various complications, ranging from severe malabsorption syndrome, celiac crisis, serious electrolyte disorders, tetanic crisis, cardiac arrhythmia, coagulopathy, etc. We must not forget its long-term complications such as bone damage, severe nutritional deficiencies, reproductive anomalies, neurological/neuropsychiatric disorders, etc (7). A large proportion of patients exhibit autoimmune disorders such as autoimmune thyroiditis, dermatitis herpetiformis, and type 1 diabetes mellitus (8). Kollcaku et al, in their study of 152 children under 15 years of age, newly diagnosed with Diabetes mellitus, Type 1, showed a prevalence of celiac disease of 2.6% at the time of diagnosis, and 5.2% after diagnosis (9). The basis of treatment is a gluten-free diet, meaning the elimination of foods based on wheat, rye, barley, and oats from the diet, although the toxicity of oats is reported to be lower (10).

METHODS

We describe the case of a woman admitted to our department in the setting of a celiac crisis, which would later result in celiac disease. She manifested gastrointestinal and extraintestinal signs and symptoms. The diagnosis was based on serology, histopathological examination of biopsy material, exclusion of other pathologies as possible causes of the clinical picture, and the rapid response to a gluten-free diet.

RESULTS

Case report

A 55-year-old woman presented to our emergency department with a 9-day history of watery diarrhea, 10-12 stools/day, abdominal pain and bloating, and headache. She reported having gastrointestinal problems for several

years, significant weight loss (35 kg lost in the last 2-3 years and the weight in the emergency department resulted in 42 kg), as well as anxiety and stress. On examination, the abdomen was soft, without tenderness and with bowel sounds on auscultation and severe weakness of the limbs, while the vital parameters were normal. Laboratory tests had the results presented in Table 1. Stool cultures and fecal occult blood test were negative. CT-thorax/abdomen revealed intestinal loops with slightly thick, edematous walls, multiple mesenteric lymph nodes measuring 11x10mm. The patient underwent endoscopy which showed atrophy of the duodenal villi and from there biopsies were taken at D2/3. Histopathological analysis showed the presence of dense chronic active inflammation and severe villous atrophy (Figure 1). Based on the anamnesis, lack of fever, clinical assessment, negativity of coproculture, lack of inflammatory tests, we requested the Anti tissue transglutaminase antibodies IgA test which resulted positive, 392.9 U/mL (<20). The patient was discharged from the hospital in better condition after the regulation of clinical and laboratory symptoms, fluid resuscitation with correction of the electrolyte imbalance, and with the advice of a gluten-free diet. The patient weighed 46.6 kg five weeks after discharge, 50.7 kg eleven weeks, 55 kg ten months after discharge.

Table 1. Laboratory data

Complete blood count	Hb 8.8 g/dL (12.1-15.8), RBC 3.62 x106 U/L (4-5.6), mean cell volume of 76.7 fL (80-100), HCT 27.8%, PLT 258 K.uL (150-400),WBC 5.6 K/uL (4-10.5), Limf 24% (27-45); Mono 12%
Biochemical balance	ALT 59 U/L (<55), AST 79 U/L (<34), LDH 293 U/L (125-230), GGT 10 U/L (9-36), Urea 9.5 mg/dL (21-43), Creatinine 0.51 mg/dL (0.57-1.11), Ferritinë 4.98 ng/mL (5-204), Amilazemi 76 U/L (25-125), Lipazemi 11 U/L (<60)
Serum electrolytes	Na 134 mmol/L (138-145), K 2.8 mmol/L (3.5-5.1),
Prothrombin Time Test and INR	PT (Quick time) 64% (70-110), INR 1.38 (0.85-1.15)
Inflammatory tests	C reactive protein <0.10 mg/dl (<0.5), Ddimer 0.84 (<0.5) ug/mL, Fibrinogen 328 mg/dL (200-400).
Viral markers	HIV negative, HBsAg negative, Anti HCV negative
Autoimune profile	ANA negative, ANCA negative
Thyroid function test	TSH 1.500 mU/L (0.35-4.94), FT3 2.16 ng/mL (1.58-3.91), FT4 0.92 ng/mL (0.7-1.48)
Tumor markers	CA 125, 8.4 U/mL (<35), CA 19.9, 7.7 U/mL (<37), CEA, 2.3 ng/mL (<5).

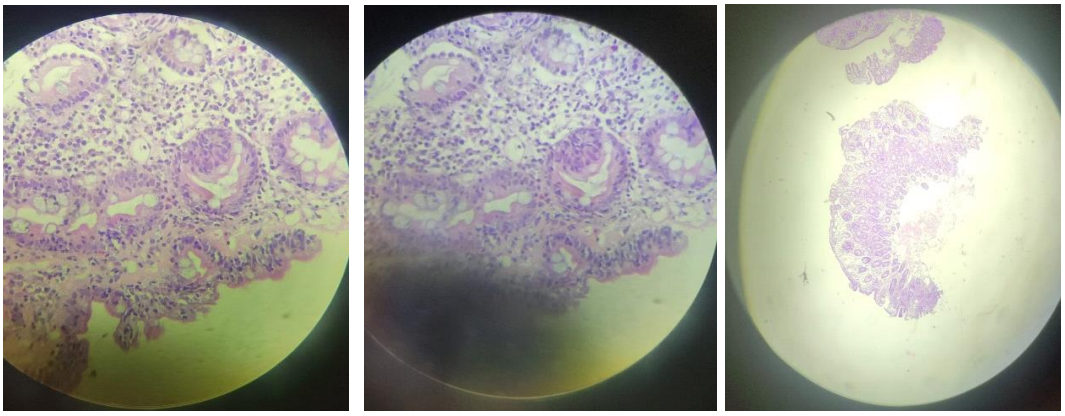

Figure 1. Duodenal biopsy, col Hex40, showing dense plasmocytic inflammatory infiltrate in lamina propria, eosiophyls and intraepithelial lymphocytes

DISCUSSION

Our patient had been suffering from these complaints for years, for which she had been seeing doctors from time to time. There are other authors who have raised the issue of delay, and it is even mentioned that for every diagnosed case, 5-10 cases remain undiagnosed (11, 12). A significant increase in its incidence has been recorded in the Mediterranean, so to cope with this "epidemic" spread, a simple and accurate diagnosis is needed, states Andrea S et al in his article (1). Our patient was a 55-year-old woman, and the higher prevalence in women of similar age has been noted by other authors (13). Clinically, our patient manifested gastrointestinal symptoms. Roberta R et al in their study emphasize that 58.95% of patients reported gastrointestinal symptoms such as diarrhea, abdominal bloating, dyspepsia, etc. which helped in making the diagnosis (14). Our patient's laboratory data showed increased transaminases, anemia, decreased ferritin, hypoproteinemia, hypoalbuminemia. Liver involvement is widely described, so testing for this pathology is necessary in patients with autoimmune disorders, hepatic steatosis, non-cirrhotic intrahepatic portal hypertension, cryptogenic cirrhosis (15). Cryptogenic hypertransaminasemia was seen in 29% of patients (8). This pathology damages the small intestine and as a result there will be a decrease in the absorption of iron, folate and vitamin B12, leading to anemia as a consequence. Anemia may be the only presenting sign. Kochhar et al. reported it in 39% of patients (16). Chronic atrophy of intestinal villi leads to metabolic disorders. This pathology should be suspected in patients who manifest hypoproteinemia/hypoalbuminemia without other etiological causes. Our patient had long manifested pronounced emotional problems, as frequent diarrhea, abdominal discomfort penalized her at work, as well as underweight (42 kg). Julie et al stresses that articles on this pathology are numerous, but not so many that focus on the psychological sphere of patients (17). The diagnosis was also based on serological

examination, as Anti-tissue transglutaminase antibodies IgA resulted positive, 392.9 U/mL. Anti-gliadin IgA/IgG antibodies are detected in the sera of patients with gluten-sensitive enteropathy (celiac disease) where anti-gliadin IgA antibodies are less sensitive, but more specific, sensitivity 71%, specificity 97% (18). Histopathological examination of duodenal biopsies remains the gold standard not only for diagnosis but also for the dynamic follow-up of patients after their treatment (19). Histological abnormalities of the small intestine were demonstrated by Paulley in 1954, and biopsy techniques described by Royer, 1955, and Shiner, 1956 provided reliable diagnosis (3). Our patient's biopsy showed the presence of villous atrophy, a finding that has been confirmed by other authors to the extent of 87% (8). The basis of treatment is a gluten-free diet. As early as 1950, Wim Dicke, in his doctoral thesis, emphasized that eliminating wheat, rye, and oats from the diet leads to a significant improvement in the clinical picture (3). Our patient showed significant improvement on the gluten-free diet. The patient weighed 46.6 kg five weeks after discharge, 50.7 kg eleven weeks, and 55 kg ten months after discharge. As early as 1888, Samuel Gee, a London pediatrician, suggested that the diet could be a cure, as he saw improvement on a mussel diet, while in 1924, Haas, an American physician, published positive results on a banana diet (2). Gluten is a food ingredient in many products on the market, so it is quite difficult to avoid these products from the diet. Therefore, there is a need for a therapeutic innovation that will enable these patients to have a variety of food products. In addition to the diet, the initiation of supplements and the treatment of anemia are important. The latter can last longer than other signs and symptoms. Thus, Marco et al show in their work that the diagnosis of anemia persists in 17.8% of adults due to the delay in diagnosis and the presence of ultrastructural changes in enterocytes (20). In our country, there is no association for this disease, but there is "Gluten-Free Albania", a project created in 2019 with the aim of raising awareness in our society about the challenges of the disease, food safety, and the relief that restaurants should offer to these patients.

CONCLUSIONS

Celiac disease with its numerous manifestations, especially extraintestinal and often atypical, should make clinicians more vigilant in order to quickly and accurately identify these patients. All this will avoid complications that can often be life-threatening. It is enough to advise the right diet to improve the patient's quality of life, and consequently avoid morbidity and fatality. Since this pathology has a high cost of living, these patients should be included in the reimbursement scheme.

Acknowledgement: The study was conducted and approved according to the ethical standards of the National Ethics Committee. Informed consent was obtained from the parents of the patient.

Conflict of Interest Statement: The authors declare that they have no conflict of interest.

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