Gastroenterology and Hepatology From Bed to Bench. ***2010;3(3):142-145***

CASE REPORT

A 27 year-old female patient with chronic watery diarrhea, weight loss, ascites, arthropathy and evidence of vitamin K deficiency

Mirhadi Mousavi, Mohammad Rostami Nejad

Research Institute for Gastroenterology and Liver Disease, Shahid Beheshti University, M.C., Tehran, Iran

ABSTRACT

Celiac disease (also known as gluten-sensitive enteropathy or nontropical disease) is a common autoimmune disorder that caused by sensitivity to dietary gluten and related proteins in genetically sensitive individuals. The disorder may be diagnosed at any age and that affects many organ systems. Celiac disease occurs in adults and children at rates approaching 0.5 to 1% of the population. We report a case of celiac disease (CD) in a young adult female patient with watery diarrhea, weight loss, ascites, arthropathy and evidence of vitamin K deficiency.

Keywords: Celiac disease, diarrhea, Vitamin K deficiency

(Gastroenterology and Hepatology From Bed to Bench 2010;3(3):142-145).

INTRODUCTION

Celiac disease (also known as gluten-sensitive enteropathy or nontropical disease) is a common autoimmune disorder that caused by sensitivity to dietary gluten and related proteins in genetically sensitive individuals. The disorder may be diagnosed at any age and affects many organ systems. Celiac disease occurs in adults and children at rates approaching 0.5 to 1% of the population (1).

We report a case of celiac disease (CD) in a young adult female patient with watery diarrhea, weight loss, ascites, arthropathy and evidence of vitamin K deficiency.

CASE REPORT

The patient was a 27-year-old female who was admitted to a teaching hospital because of diarrhea described as 3-4 watery stools per day accompanied by abdominal bloating and weight loss (approximately 15 kg) since five months ago. Two months before admission, bilateral ankle Arthritis developed and gradually lower extremity edema and ascites appeared.

On examination, the patient appeared ill and cachectic without paler and jaundice. Vital signs were normal. The detected abnormalities were mild ascites and lower extremity pitting edema without hepatomegaly and splenomegaly. Evidence of the left knee and bilateral ankles arthritis was obvious.

Biochemical tests at admission showed a prolongation of prothrombin time. Patient was not iron deficient and the serum levels of Na, K, and Ca were within the normal range. Thyroid-stimulating hormone (TSH) serum level was in normal range (table 1). Treatment with 30 mg vitamin K returned the levels of PT and INR to the normal range. The ascitic fluid had a low serum ascites albumin gradient (SAAG= 0.9) and serum levels of carcinoembryonic antigen and CA 19-9 and CA 125 were within the reference range.

Received: 15 February 2010 *Accepted*: 18 April 2010
Reprint or Correspondence: Mirhadi Mousavi, MD.
Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University, M.C., Iran
E-mail: Mousavi@yahoo.com

Table 1. Serology screening for patient at admission

At admission	
PT (Second)	21.7
INR (Ratio)	3.1
AST/ALT (IU/L)	32/29
ALP (IU/L)	246
Bilirubin (Total/Direct) (mg/dl)	1/ 0.3
Total protein (g/dl)	6.2
Serum albumin (g/dl)	2.1
White blood Cell (x10^3/UL)	8700
Hemoglobin (g/dL)	12.6
Platelet (x10^3/UL)	265000
BUN/Creatinin (mg/dl)	14 / 0.8
Calcium (mg/dl)	9.1
Phosphor (mg/dl)	4
Sodium (mEq/L)	140
Potassium (mEq/L)	4.7
Serum iron (micgr/dl)	65
TIBC (micgr/dl)	260
TSH (mIU/L)	1.4
T4 (ng/mL)	7.8
CRP	+ +
ESR (mm/hour)	5

An abdominal ultrasound study revealed marked ascites but no features of cirrhosis. Small bowel series study demonstrated dilation of the small intestine and replacement of the normal delicate feathery mucosal pattern with either marked thickening (figure 1). An abdominal computed tomography scan showed a marked ascites, minimal left side pleural effusion and distention of small bowel loop dominantly jejunaileal (40 mm) with normal wall thickness and normal wall thickness of colon without retroperitoneal and pelvic lymphopathy (figure 2).

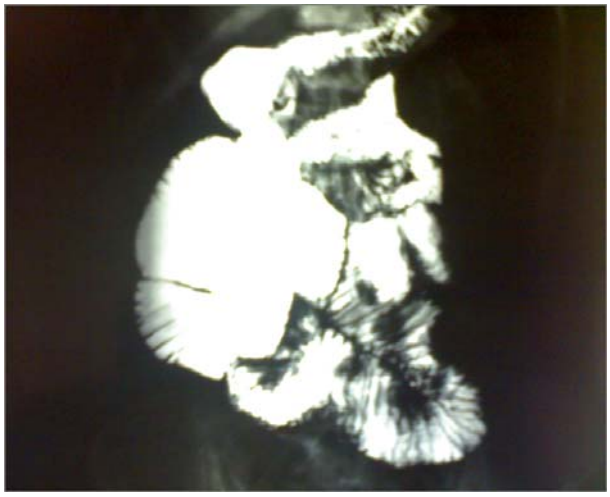

Figure 1. Small bowel series study. It showed dilation of the small intestine and replacement of the normal delicate feathery mucosal pattern with either marked thickening.

Human anti-tissue transglutaminase (tTG) antibody and antiendomysial antibodies (EMA) were positive. Endoscopic view of the duodenum showed scalloping of the folds (figure 3).

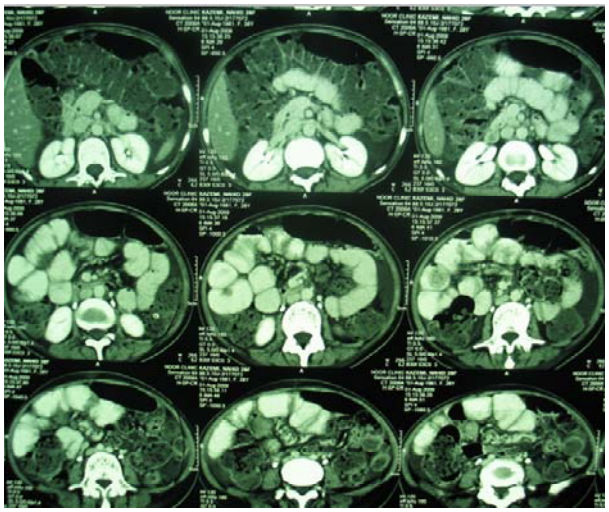

Figure 2. Abdominal computed tomography scan.
A marked ascites, minimal left side pleural effusion and distention of small bowel loop dominantly jejunaileal (40mm) with normal wall thickness and normal wall thickness of colon with out demonstrating retroperitoneal and pelvic lymphopathy.

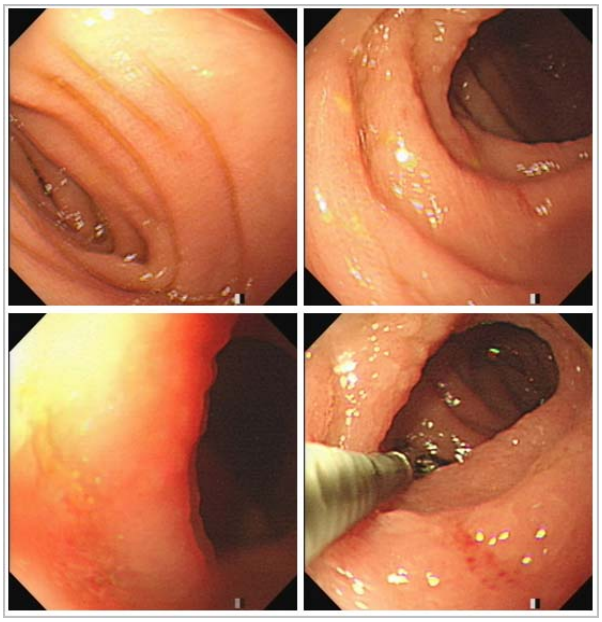

Figure 3. Endoscopic view of the duodenum.

Four biopsy specimens were taken for pathological investigations. Characteristic mucosal lesions on jejunal biopsy confirmed the diagnosis of CD.

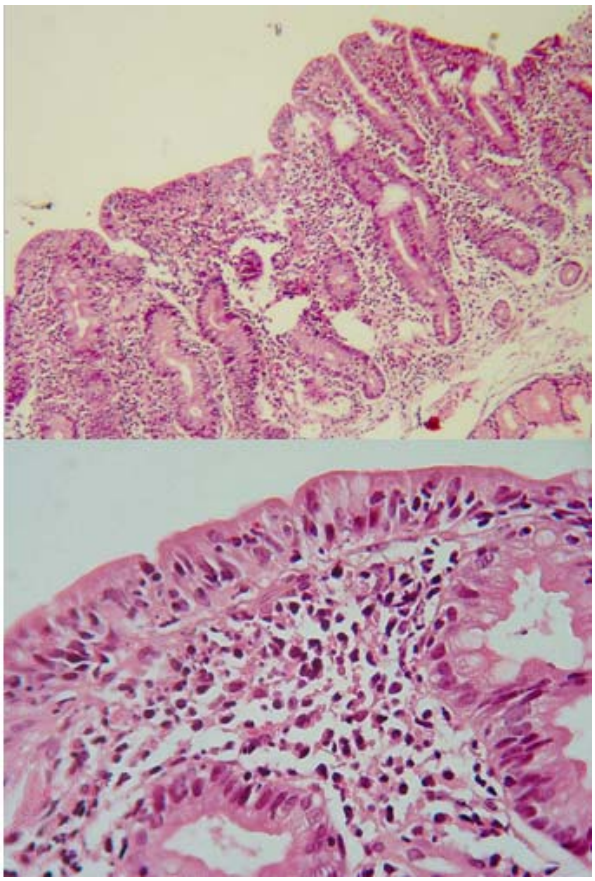

Figure 4. Duodenal biopsy.

According to modified Marsh classification (2), study of the duodenal biopsy specimen showed severe villous atrophy with Crypt hyperplasia and Infiltration of the lamina properia with lymphocytes (Marsh IIIc) (Figure 4).

A gluten-free diet was started. All her sign and symptoms had improved after 3 months.

DISCUSSION

In patients who suspected of having CD barium evaluation of the small bowel are seldom required. But by barium investigation of this patient we found abnormal roentgen findings included straightening of the valvulae conniventes, replacement of the normal delicate feathery mucosal pattern with either marked thickening or complete obliteration of the mucosal folds dilation of the small intestine.

One of the diagnostic clues to the presence of celiac disease or refractory disease is abdominal computed tomography (CT) which may provide by revealing lymphadenopathy, hyposplenism, the presence of cavitating mesenteric lymph nodes and ascites.

In our patient, abdominal CT- scan showed a marked ascites, minimal left-sided pleural effusion, distention of small bowel loop dominantly jejunaileal (40mm) with normal wall thickness and normal wall thickness of colon without retroperitoneal and pelvic lymphopathy.

Diagnosis criteria especially in patients with chronic diarrhea are different and complex, so the variety of tests applicable to these patients can be bewildering. Therefore, serological test for CD in this group has simplified the diagnostic approach in subjects with typical and atypical forms (3) and saves cost and time when being used in conjunction with small bowel biopsy.

Duodenal scalloping is not specific for celiac disease, and may present in other conditions such as amyloidosis, giardiasis, eosinophilic enteritis, tropical diseases, and human immunodeficiency

virus enteropathy (4). These mucosal features should alert the endoscopist to the need for small intestinal biopsy to evaluate for possible celiac disease (5).

Celiac disease and arthritis are associated in some patients and 25 percent of adults with celiac disease have arthritis (6). In many cases GI manifestations relevant to CD and Joint symptoms will respond to a gluten free diet.

Glucocorticoids are not indicated in the routine management of celiac disease but are reserved for severely ill patients who present with acute celiac crisis manifested by severe diarrhea, dehydration, weight loss, acidosis, hypocalcemia, and hypoproteinemia. These few patients may benefit from a short course of glucocorticoids until the gluten-free diet takes effect (7).

Patients with severe disease should receive appropriate supplemental therapy to help correcting nutritional deficiencies caused by malabsorption. Also patients with prolongation of prothrombin time require supplemental vitamin K.

Serologic tests (IgA EMA and anti-tTG) and small bowel biopsy were performed before dietary treatment.

After starting a gluten-free diet, most patients improve within a few weeks (8).

The rapidity of the response to a gluten-free diet is variable. Approximately 70 percent of patients have noticeable clinical improvement within two weeks (8). For our patient, a gluten-free diet was started. Three months later, all her signs and symptoms were improved. She gained weight and serum tTG and EMA were negative after 6 months.

REFERENCES

1. Shahbazkhani B, Malekzadeh R, Sotoudeh M, Moghadam KF, Farhadi M, Ansari R, et al. High prevalence of celiac disease in apparently healthy Iranian blood donors. Eur J Gastroenterol Hepatol 2003; 15: 475–78.

2. Rostami K, Kerckhaert J, Tiemessen R, von Blomberg BM, Meijer JW, Mulder CJ. Sensitivity of antiendomysium and antigliadin antibodies in untreated celiac disease: disappointing in clinical practice. Am J Gastroenterol 1999; 94: 888–94.

3. Rostami Nejad M, Rostami K, Pourhoseingholi MA, Nazemalhosseini Mojarad E, Habibi M, Dabiri H, et al. Atypical Presentation is Dominant and Typical for Coeliac Disease. J Gastrointestin Liver Dis 2009; 18: 285-91.

4. Jabbari M, Wild G, Goresky CA, Daly DS, Lough JO, Cleland DP, et al. Scalloped valvulae conniventes: an endoscopic marker of celiac disease. Gastroenterology 1988; 95: 1518-22.

5. Lubrano E, Ciacci C, Ames PR, Mazzacca G, Oriente P, Scarpa R. The arthritis of coeliac disease: Prevalence and pattern in 200 adult patients. Br J Rheumatol 1996; 35: 1314-18.

6. Lloyd-Still JD, Grand RJ, Khaw KL, Shwachman H: The use of corticosteroids in celiac crisis. J Pediatr 1972; 81: 1074-81.

7. Shah VH, Rotterdam H, Kotler DP, Fasano A, Green PH. All that scallops is not celiac disease. Gastrointest Endosc 2000; 51: 717-20.

8. Pink IJ, Creamer B. Response to a gluten-free diet of patients with the coeliac syndrome. Lancet 1967; 1: 300-304.