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Case Report

An Interesting Cause of Generalized Edema; a case report

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ABSTRACT

Celiac disease is a small intestine malabsorption syndrome triggered in genetically susceptible individuals by ingestion of gluten-containing diet. Celiac is often characterized by gastrointestinal symptoms such as diarrhea, abdominal pain, vomiting, bloating, constipation and steatorrhea, yet it may be associated with extraintestinal symptoms such as edema, weight loss, osteoporosis and anemia. The variety of clinical presentations of celiac often resultsing in delay diagnosis which may lead to serious complications.

A 47-year-old woman with generalized edema and exertional dyspnea was admitted. On physical examination, the conjunctiva was pale, the abdomen had significant ascites and she had bilateral symmetric 3+ pitting edema from ankle to thigh. Heart and lung examinations, CXR, ECG and echocardiography were normal. Renal function tests and sonography were normal. In laboratory tests, microcytic hypochromic anemia, prolonged PT, hypocalcemia, hypophosphatemia and AST ALT increased titer was observed. Reduction in Hemoglobin, serum albumin, vitamin D and ferritin was observed. Thus, was raised the possibility of cirrhosis for this patient. But ultrasound of liver and spleen was normal and thrombocytopenia not seen. The prolonged PT in this patient was recovered by administering vitamin K that also undermines the diagnosis of liver cirrhosis. Thus, was raised the possibility of malabsorption. So, performed celiac serological tests, Upper endoscopy and biopsy from D2 folds. Anti-TTG and EMA antibodies titer was increased. Atrophy in D2 folds and scalloping was seen in endoscopy. Pathologic findings were compatible with celiac disease, grade 3c according to Marsh criteria. Gluten-free diet started for patient. After 6 months follow-up, her clinical and paraclinical conditions were improved.

Celiac disease is a malabsorption syndrome which impairs the absorption of protein, leads to hypoalbuminemia and a decrease in plasma colloid pressure resulting in generalized edema. So, It is recommended, be raised the possibility of celiac disease in the treatment of generalized edema with unknown causes.

Keywords: Generalized Edema; Gluten-free diet; Celiac

INTRODUCTION

Celiac disease is a chronic autoimmune disease of the small intestine and is a kind of malabsorption syndrome that occurs in people who are genetically susceptible. These people are symptomatic when they come in contact with gluten-containing diet (1-4). Its prevalence in Iran is in average 1 in 166 patients (5-7). Genetic factors such as HLA alleles, especially HLA class II (HLA DQ8 and. HLA DQ2) have been identified as a certain risk factor (8,9). Gluten protein in wheat intake is as well an environmental factor involved in this disease

(10,11). Manifestations of celiac disease differ according to the patient's age, disease duration and extent. As it occurs with malnutrition and growth retardation in children or can be asymptomatic in adults or with gastrointestinal symptoms (including diarrhea, abdominal pain, vomiting, bloating, constipation, steatorrhea, etc.) or non-gastrointestinal (weight loss and anemia, osteoporosis, etc.) (12-15). Only half of patients with celiac disease have typical manifestations such as chronic diarrhea, abdominal pain and bloating. Approximately 30-50% of patients have extra-intestinal symptoms such as short stature, anemia, delayed puberty, ataxia, depression, infertility and osteoporosis (16-18). The risk of celiac disease in people with family history of celiac disease, underlying autoimmune diseases, like type 1 diabetes, and autoimmune thyroid, and liver disease is more than others. Also in patients with herpetiform dermatitis prevalence of celiac disease is very high (19-21). This disease leads to iron deficiency anemia, elevated liver enzymes, hypo-albuminemia, hypocalcemia, deficiency of vitamin D and other fat soluble vitamins (22,23). The diagnosis is based on clinical signs and symptoms, serological tests and duodenal biopsy findings and also improvement in symptoms and negative serology and pathology with gluten-free diet (24,25). Serologic tests include anti-gliadin, anti-endomysial and tissue transglutaminase antibodies with more than 98% sensitivity and about 100% specificity for celiac diagnosis (26). In high clinical suspicion, such as iron deficiency anemia and osteoporosis without justification, in case of a positive celiac disease serology test, endoscopic investigation should be performed to confirm the diagnosis. Upper endoscopy in patients with celiac disease can be normal or may present with loss of duodenal folds (atrophy) and scalloping or fissuring (indentation of folds); 6 biopsy specimens are taken from the second part of duodenum for histopathological examination (27-29). The main pathological findings of celiac disease include villous atrophy, crypt hyperplasia and intraepithelial lymphocytosis. The only accepted and effective treatment for celiac disease is gluten-free diet leading to stop in the immune response caused by gluten. The symptoms of celiac disease in most patients recover within a few weeks to several months and complications recover as well, like anemia and hypocalcemia, vitamin D deficiency and other disorders due to malabsorption also recover in a few months (30,31). Albumin is a water soluble protein. The main causes of hypo-albuminemia include reduction of protein synthesis and intake that (32,33).

CASE REPORT

A 47-year-old woman referred to cardiac clinic of the hospital with generalized edema and dyspnea. The patient had a fair health status a month ago until the edema, which has started since a year ago and was limited to the ankles, advanced from ankle to calf and upper thighs. She also had abdominal swelling as well. The patient also complained of dyspnea during exercise and weakness and fatigue. But did not have PND, orthopnea and weight loss. One year ago the patient was hospitalized due to lower extremity edema, but refused complementary diagnostic workup after a relative improvement of signs and symptoms and was discharged. The patient had a history of heart disease, type 2 diabetes mellitus from 30 years ago, hypertension, hypothyroidism from 15 years ago, and anemia. Patient’s medication history included 40 mg furosemide taken BD, ferrous sulfate 2 pills a day, NPH insulin 20 units mornings and 14 units evenings, 200µG levothyroxine daily, and 50000IU vitamin D weekly, but did not mention the use of NSAIDS. The patient was admitted due to the history of heart disease and exertional dyspnea with a diagnosis of congestive heart failure in the cardiac ward. Physical examination at admission demonstrated the following: T=36.5, PR=56, RR=14, BP=160/90. Her conjunctiva was pale. Her JVP was not bulging and lung and heart auscultation were normal. In abdomen, a significant ascites and in organs a bilateral symmetric pitting edema +++ from ankle to thigh without redness and warmth was observed. Other

examinations were normal. In preliminary laboratory tests, she had microcytosis anemia, but other tests including electrolytes, urinalysis and renal function tests were normal. Other tests are shown in the table 1.

Table 1-Laboratory results

	A	B	C	Normal range		A	B	C	Normal range
RBC	3.54	3.82	3.77	4.2-5.4 mil/ul	**Serum Alb**	2.8	2.45	-	3.5-5.2 g/dl
Hb	8.5	11.2	11.3	12.3-15.3 g/dl	**b-globulin**	-	0.37	-	0.5-1 g/dl
Hct	29.4	34.9	36.2	35.9-44.6%	**Urine Pr**	127	2	-	<150 mg/24h
MCV	83.1	91.4	96	80-96 fl	**Urine Alb**	-	18	-	<30 mg/24h
Fe	36	90	-	40-150 micg/ml	**TSH**	100<	76.8	25.88	0.4-6.7 mlu/ml
ferritin	8	70	6.59	10-125 micg/l	**T3**	0.57	1.2	-	1.25-3 nmol/l
Na	132	133.4	-	135-145 mEq/L	**T4**	15	57	7.24	71-165 2nmol/l
K	4.7	4.3	-	3.5-5.5 mEq/L	**PT**	13.4	24.6	-	11-15s
Ca	8.3	7.4	9.1	8.5-10.5 mg/dl	**PTT**	40.4	37	-	25-40s
Ph	4	2.3	5.3	2.5-4.5 mg/dl	**INR**	1.15	3.8	-	-
VitD	22	9		10-30 ng/ml	**ALT**	153	102	32	<31 IU/L
VitB12	-	173	-	160-970 pg/ml	**AST**	406	147	39	<31 IU/L
Folic acid	-	4.4	-	1.5-17 ng/ml	**ALP**	627	657	560	65-305 IU/L
BUN	22	37	29	17-43 mg/dl	**GGT**	-	57	-	
Cr	0.7	0.9	0.7	0.7-1.4 mg/dl	**Anti-TTG**	-	407	26.5	<20 u/ml
Serum Pr	3.5	3.54	-	6.6-8.8 g/dl	**Anti-EMA**	-	349	neg	<20 u/ml

A: In last year admission, B: In recent admission, C: After GFT

To evaluate her heart, CXR, ECG, and echocardiography were performed for the patient. In echocardiography, MVP, LVH, and mild left ventricular dysfunction with EF=55% were observed. To reduce the symptoms of edema, she was treated with 40 mg furosemide three times a day for 5 days. The patient also complained of fatigue and swelling of the extremities, and her ascites was also intensified. On the fifth day of admission, her vital signs were stable, the conjunctiva was pale but JVP was not prominent and auscultation of heart and lungs were normal and had no crackle. Abdomen was distended and lower extremities edema was +++. Due to the lack of recovery, renal function tests were performed to check kidney failure. In 24-hour urine, there was 200mg/24h protein, but 24h hour urine volume was more than normal (3050 ml). For further investigation of kidney problems, ultrasonography was performed for the patient. The patient was treated with furosemide injection to reduce edema, but the swelling persisted, so that on the tenth day of hospitalization, gastrointestinal consultation was requested for detection of liver cirrhosis, due to exacerbation of edema and abdominal distention. For further investigation of ascites, liver enzymes were tested that showed an increase in ALT, AST and ALP, but bilirubin was within normal limits. Serum protein and albumin were significantly reduced, PT was prolonged and INR was 3. Although the patient had no jaundice or other symptoms associated with liver disease such as spider angioma or palmar erythema, abdominal ultrasound was performed due to low albumin and prolonged PT, suspected to chronic liver disease and cirrhosis. In ascite fluid analysis, albumin level was low and ascite fluid was transudates (SAAG>1.1). Coagulation tests, performed in the early days of hospitalization, showed a significant increase in anticoagulant PT that was corrected with vitamin K in the following days. PTT was within normal limits in all tests. CBC test showed that hemoglobin was constantly low, which was in line with low hemoglobin in the patient's previous tests. The patient was treated with ferrous sulfate from previous years, but still had anemia. Although iron and ferritin were in the lower normal limit in the new tests, it had a large fluctuation in previous tests and were less than normal in most of them. In previous hospitalization in the past year, patient’s hemoglobin was 8g/dl and ferritin was 8.5g/dl. Although the patient took ferrous sulfate two pills a day regularly, her anemia was resistant to

treatment. In the other tests, hypocalcemia, hypophosphatemia, and low serum protein was impressive and for further investigation, levels of vitamin D and serum Alb were measured that were both lower than normal, and had low calcium even after modifying the amount of calcium with serum albumin. Hypocalcemia, hypophosphatemia and hypoproteinemia and hypo-albuminemia were present in previous tests. Serum protein analysis was conducted due to low levels of serum protein. To further evaluation enteropathy, Anti-TTG and EMA was requested that elevated levels of these two antibodies lead to perform upper endoscopy to confirm the diagnosis. In endoscopy, the mucosa of body and D2 was atrophic and also scaloping was seen in D2 folds (figure 1). Multiple biopsies of these areas was prepared for histopathologic examination. The pathology reported chronic active gastritis and Helicobacter pylori in the stomach and pathological feature of chronic malabsorption (intraepithelial lymphocytosis, villus atrophy, crypt hyperplasia) suggestive of Marsh 3 and celiac disease (figure 2).

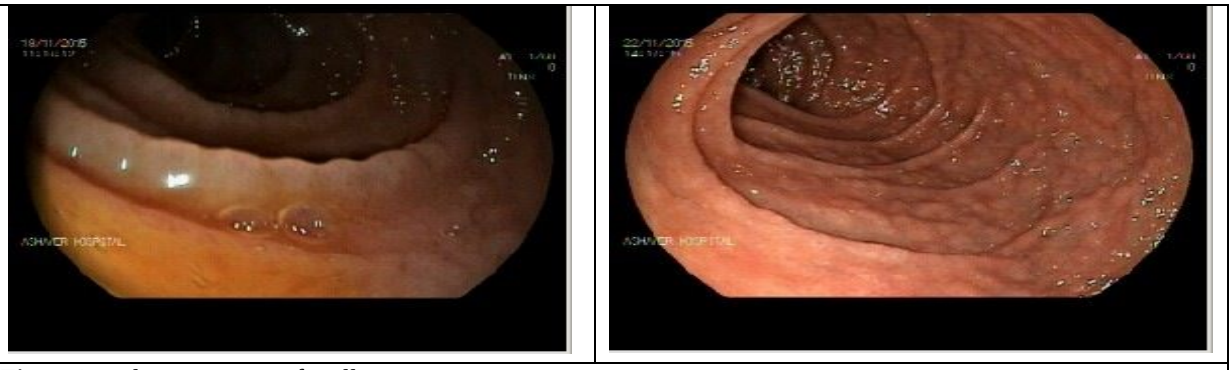

Figure1- endoscopic view of scalloping

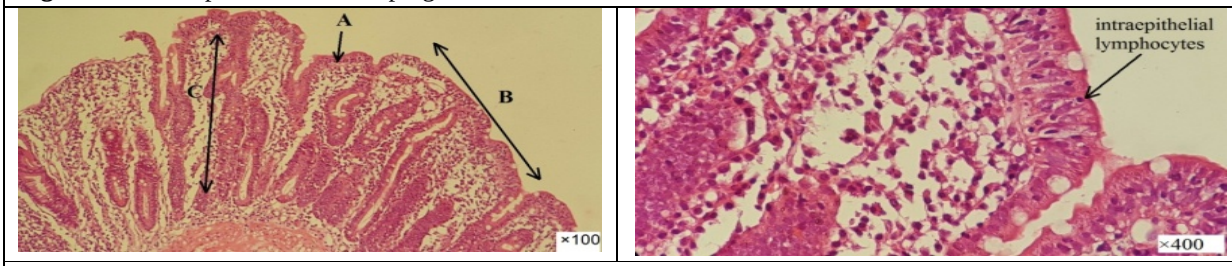

Figure2- pathologic view of celiac. A= Intraepithelial lymphocytosis, B= Villus atrophy, C= Crypt hyperplasia

After pathologic confirmation of celiac disease, gluten-free diet started and necessary training was given. After three months, the patient was visited again that edema and ascites of the patient were recovered. Her general condition was good and her anemia was corrected. Also the level of calcium and vitamin D has also been normal, but the level of Anti-TTG did not drop than before and was still higher than normal. At 6 months' follow-up, the patient's general condition was very good and there was even no need for diuretics and no evidence of edema. Hemoglobin, ferritin, calcium and vitamin D were all within normal limits and hypothyroidism of the patient was well controlled with 100µg of levothyroxine.

DISCUSSION

Differential diagnosis:

A 47-year-old female patient with lower limb edema and dyspnea was admitted to the hospital to determine the cause of edema. She suffered from exertional dyspnea, weakness, and fatigue. In her physical examination, laboratory tests and other measures, she had some points in favor of certain diseases, whereas other tests and paraclinic measurements rejected it.

Heart failure:

As one of the features of cardiac edema is starting from lower extremities with slow advancement. Left heart failure has symptoms such as PND, orthopnea, or crackles on lung auscultation; and in

right heart failure there are evidences of bulging JVP or hepatomegaly. But the patient did not have such symptoms and signs; also S3, found in auscultation, or cardiomegaly on CXR that are in favor of heart failure were not observed. ECG revealed no particular problem except bradycardia (rate=56). In CXR, the heart size was normal and had no pleural effusion or any other abnormality. Thus, heart failure was less suggested and did not justify the symptoms.

Renal failure:

Although the patient had a 15-year history of hypertension, serum BUN, Cr, and urine protein were within normal limits and ultrasound did not reveal any evidence of renal failure, such as reduced size of the kidneys or corticomedullary border disorder. Moreover, the development of edema due to renal failure is rapid and starts from upper parts of the body like face and periorbits. In this test, BUN, Cr and urinalysis were normal. In the last year`s tests, urine protein and albumin were within normal limits. In ultrasonography, parenchyma and kidneys were in normal size and there was no hydronephrosis or stone. Thus, renal failure is so unlikely in this case.

Cirrhosis and chronic liver disease:

Another differential diagnosis of generalized edema is cirrhosis. Although fatigue, ascites, increased ALT and AST, reduced albumin was present in this patient, the absence of symptoms such as jaundice, palmar erythema and spider angioma, and normal platelet was a negative point for diagnosis of cirrhosis. Besides, the increase in PT in this patient was recovered by administering vitamin K that also undermines the diagnosis of liver cirrhosis. Another evidence of liver cirrhosis is liver size reduction, coarseness of the liver echo and splenomegaly in ultrasonography; liver and spleen sonogram was normal in this patient and there was no point in favor of cirrhosis and chronic liver disease. Only an 8-mm stone was found in the gallbladder, but the patient had no complaints in this regard. Previous tests revealed lower than normal levels of albumin and globulin. Therefore, ascites and edema caused by cirrhosis and chronic liver disease was rejected in this patient.

Medications:

Drugs that cause edema include NSAIDS, steroids, cyclosporine, growth hormone, anti-hypertensive medication such as calcium channel blockers and alpha-adrenergic antagonists. But the patient reported no history of taking these medications. Also, the patient had no history of allergies to certain substances and did not mention symptoms of mucosal edema and airway involvement. Thus drug-induced edema and Angioedema was not supported in this patient.

Malnutrition:

Nutritional edema of Kwashiorkor, cancer and total gastrectomy are another cause of generalized edema. The patient reported no previous history of known cancer or gastrectomy. Another nutritional edema is malnutrition. Hypo-albuminemia, hypophosphatemia, vitamin D deficiency, and low serum ferritin were all in favor of the diagnosis of malnutrition, but patient`s anemia did not diminish, despite the use of ferrous sulfate over the years and despite taking vitamin D, the serum level of vitamin D, calcium and phosphorus remained low; the resistance to treatment reflects that the impairment was not due to the nutrients deficiency, since it would have then resolved with treatment.

Hypothyroidism:

One of the most complications of severe and untreated hypothyroidism is generalized edema. In this patient, despite daily intake of 200µg of levothyroxine, TSH was still high. This patient used regular levothyroxine, and also used ferrous sulfate with interval from levothyroxine. So resistant-to-treatment hypothyroidism in this patient could be caused by the absorption of the drug. Although hypothyroidism alone cannot justify generalized edema in this patient.

Malabsorption:

In our patient, malabsorption justify hypoproteinemia, hypo-albuminemia, hypocalcemia, hypophosphatemia, deficiency of iron, ferritin, vitamin D, resistant-to-treatment hypothyroidism due to malabsorption of

levothyroxine and prolonged PT due to malabsorption of vitamin K. Malnutrition, liver diseases and nephropathy were rejected in this patient, so protein- losing enteropathy is possible. Hypoproteinemia, hypo-albuminemia and edema, hypocalcemia, hypophosphatemia and refractory anemia due to iron malabsorption justified with celiac disease; on the other hand, other autoimmune disease such as diabetes mellitus and hypothyroidism and the sex of the patient was in favor of the diagnosis of autoimmune malabsorption disorder such as celiac.

Since celiac disease is a malabsorption syndrome with problem in the absorption of protein leading to hypo-albuminemia. Hypo-albuminemiacan leads to edema with reduction in plasma colloid pressure. According to the severity and amount of hypo-albuminemia and hypoproteinemia, edema can range from mild edema in the lower extremities to generalized edema. Thus, the celiac disease justifies generalized edema in this patient because of malabsorption of protein and albumin and decreased plasma colloid pressure.

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