

Case Report

Celiac disease masquerading as refractory iron deficiency anemia

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ABSTRACT

Iron deficiency is most common cause of anemia worldwide and more so in our country. Dietary factors and malabsorption are the major factors responsible for iron deficiency. Celiac disease is one of the most common causes of intestinal malabsorption during childhood. Here we present a case of celiac disease in a child presenting with iron deficiency anemia, refractory to iron therapy.

Keywords: Anemia, Celiac disease, Iron deficiency

INTRODUCTION

Celiac disease also known as gluten-sensitive enteropathy, is a chronic, autoimmune disorder affecting primarily the small intestine that interferes with the digestion and absorption of food nutrients and occurs in people who are genetically predisposed.¹ Classic symptoms include gastrointestinal problems such as chronic diarrhoea, abdominal distention, malabsorption, loss of appetite, and among children failure to thrive. It is associated with other autoimmune diseases, such as diabetes mellitus type 1 and thyroiditis, among others.² Celiac disease is caused by a reaction to gluten, which are various proteins found in wheat and in other grains such as barley, and rye. Upon exposure to gluten, an abnormal immune response may lead to the production of several different autoantibodies 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.¹ Diagnosis is typically made by a combination of blood antibody tests and intestinal biopsies, helped by specific genetic testing. The peculiarity of our case was

that the child presented with isolated iron deficiency anemia, refractory to iron therapy with no features of failure to thrive and was ultimately diagnosed as celiac disease after intestinal biopsy.

CASE REPORT

A 6 year old female child presented with complaints of progressive pallor for the last 15 days associated with generalized weakness. There was history of fever for 5 days about 2 weeks back. There was no history of blood loss or any abdominal pain or loose stools. Diet was appropriate for age. On examination there was severe pallor, no lymph nodes were enlarged and there was no organomegaly. Anthropometry was within normal limit with a weight of 18 kg and a height of 105 cm, both above the 3rd percentile. The child was evaluated for the cause of anemia. Investigations showed features of IDA with a low MCHC and MCV with a haemoglobin 5.1 gm% with ferritin 1.97 ng/ml, TIBC 420 µg/dl, transferrin saturation 3.1% and peripheral smear showing microcytic hypochromic anemia. The child was discharged with iron supplement. But on follow up after two months the haemoglobin did not increase in spite of good compliance and the child was admitted again for

further evaluation. Again reports showed a similar picture and a plan was made for an upper GI endoscopy to rule out celiac disease as well as *H. pylori* infection. A celiac screening i.e. Anti TTG IgA and total IgA was also sent which came out to be 606.9 IU/ml (normal <12) and 234.8 mg/dl respectively. UGI endoscopy revealed duodenal scalloping indicative of CD. Histopathological examination of the biopsy showed marked villous atrophy, flat mucosa, increased intraepithelial lymphocytes and hyperplastic crypts, all characteristic findings of CD (Figure 1). The caregivers were counselled and the child was put on a gluten free diet to which she responded. The child is now on follow up and is doing quite well.

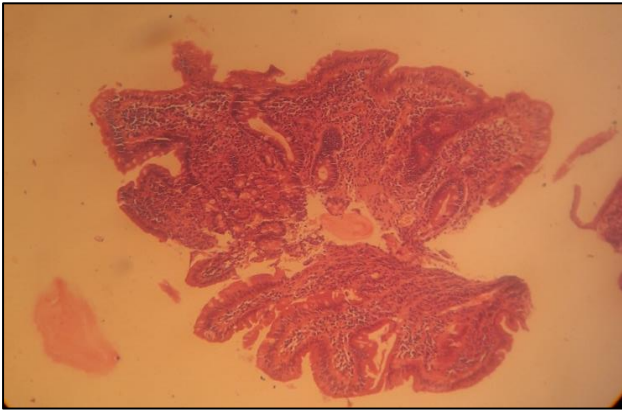


Figure 1: Histopathological study of small intestinal biopsy specimen showing crypt hyperplasia

DISCUSSION

IDA without overt clinical evidence of intestinal malabsorption is one of the most common extraintestinal manifestations of celiac disease.³ Conversely, among patients presenting with unexplained IDA, celiac disease is responsible for the anemia in 5% to 6% of cases.⁴ Celiac disease can present with many symptoms, including typical gastrointestinal symptoms (e.g., diarrhea, steatorrhea, weight loss, bloating, flatulence, abdominal pain) and also non-gastrointestinal abnormalities (e.g., abnormal liver function tests, iron deficiency anemia, bone disease, skin disorders, and many other protean manifestations). Indeed, many individuals with celiac disease may have no symptoms at all. Demonstration of histological changes in the small bowel mucosa is still regarded as the gold standard of diagnosis in celiac disease.⁵ However; the discovery of antiendomysial antibodies in patients with celiac disease had a major impact on the screening and diagnostic

algorithms for recognizing celiac disease. Its specificity and sensitivity are estimated at 99% and over 90%, respectively. More recently, an enzyme-linked immune absorbent assay has been developed to measure anti-TTG IgA antibody activity and is more convenient, is cheaper, and yields comparable sensitivity and specificity to the older antiendomysial indirect fluorescence test.⁵ Additional diagnostic criteria include identification of HLA-DQ2 or DQ8 genotypes, and response to the gluten-free diet, up to 5% of people have refractory disease, which means they do not improve on a gluten-free diet. If alternative causes have been eliminated, steroids or immunosuppressants (such as azathioprine) may be considered in this scenario.⁶

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REFERENCES

1. Celiac disease. NIDDKD. Available at <https://www.niddk.nih.gov/health-information/digestive-diseases/celiac-disease>. Accessed on 12 July 2016.
2. Ciccocioppo R, Kruzliak P, Cangemi GC, Pohanka M, Betti E, Lauret E, et al. The spectrum of differences between childhood and adulthood celiac disease. *Nutrients*. 2010;7(10):8733-51.
3. Halfdanarson TR, Litzow MR, Murray JA. Hematologic manifestations of celiac disease. *Blood*. 2007;109(2):412-21.
4. Orazza GR, Valentini RA, Andreani ML. Subclinical coeliac disease is a frequent cause of iron-deficiency anaemia. *Scand J Gastroenterol*. 1995;30(2):153-6.
5. Tapia RA, Hill ID, Kelly CP, Calderwood AH, Murray JA. ACG clinical guidelines: diagnosis and management of celiac disease. *Am J Gastroenterol*. 2013;108(5):656-76.
6. American Gastroenterological Association. Medical position statement: celiac sprue. *Gastroenterology*. 2001;120(6):1522-5.

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