

## Case Report: Celiac Disease in a 14 Month Palestinian Child

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### Abstract

Celiac disease (CD) is a life-long systemic immune-mediated disorder induced in genetically susceptible individuals after ingestion of gluten proteins. An early diagnosis is of highest importance. The therapy for the disease is lifelong elimination of wheat, rye and barley from the diet.

A 14 month old child is presented with four months history of constipation, abdominal distension, loss of weight and foul-smelling stools, associated with irritability, poor feeding and decreased activity. Specific serological markers for CD were positive. CD was confirmed by histopathology where endoscopic examination of duodenal biopsy revealed chronic duodenitis with crypt hyperplasia, increased intraepithelial lymphocytes, epithelial nuclear stratification and near total villous atrophy; corresponding to modified Marsh stage 3 and after one month of gluten free diet, the child improved clinically. Early diagnosis and treatment and regular follow-up visits, are necessary to ensure nutritional adequacy and to prevent malnutrition while adhering to the gluten-free diet for life.

**Keywords:** Celiac Disease; Antibody to Tissue Transglutaminase (TTG); Antiendomysial Antibodies (EMA); Villous Atrophy

### Introduction

Celiac disease is a systemic chronic immune-mediated disorder triggered by dietary gluten in genetically susceptible individuals, it is a multifactorial disease, including genetic and environmental factors [1,2] and after ingestion of gluten, enteropathy with impairment of the mucosal lining of the small intestine will ensue [3]. The estimated prevalence of CD in Gaza strip, the already diagnosed cases of CD, has been estimated about 237 patients i.e. 0.02% of the total population in the Gaza strip [4]. The prevalence of CD among the western population is estimated to be 1%

[5,6]. The clinical manifestations of CD are abdominal pain, chronic diarrhea, failure to thrive, anorexia, abdominal distention, constipation, muscle wasting, idiopathic short stature, dermatitis herpetiformis, dental enamel defect, osteoporosis, pathological fractures, delayed menarche, unexplained anemia or treatment-resistant iron-deficiency anemia, recurrent aphthous stomatitis and unexplained hepatic disease [7]. The development of serologic markers specific to CD has revolutionized the ability both to diagnose and monitor patients with the disease [6].

## Case Report

A 14 month-old boy child presented with history of constipation, abdominal distension, weight loss, foul-smelling stool, irritability, poor feeding and decreased activity for four months. No similar cases in the family and family history were unremarkable. There is no Consanguinity. Normal spontaneous vaginal delivery and uncomplicated pregnancy.

His height is 76 cm (below the 3<sup>rd</sup> centile) and his weight is 8 kg (below the 3<sup>rd</sup> centile). Physical examination showed severe abdominal distension, proximal muscle wasting, loss of skin folds, wasting of the buttocks, (Figure 1). Investigations demonstrated CBC (hypochromic, microcytic anemia), ESR [5], antiendomysial antibodies (EMA) 300 U/ml. the normal range is below 20 u/ml), anti-tTG-IgA antibody more than 200 u/ml (normally up to 10 u/ml). Liver enzymes were slightly elevated. AST (71), ALT (64), kidney function test (N), serum glucose (94 mg/dl). Endoscopic examination of duodenal biopsy revealed chronic duodenitis with crypt hyperplasia, increased intraepithelial lymphocytes, epithelial nuclear stratification and near total villous atrophy; corresponding to modified Marsh stage 3, strongly supporting our diagnosis of CD. After one month of nutritional therapy, the child skinfolds, proximal and gluteal muscle improved, in association with disappearance of most other symptoms..



**Figure 1:** A 14 months old boy, with celiac disease before and after gluten free diet.

## Discussion

Celiac disease also known as gluten-sensitive enteropathy, is a systemic immune-mediated enteropathy, induced by dietary gluten in genetically susceptible individuals with class II human leukocyte antigen (HLA) DQ2 and DQ8 and is characterized by injury to the small intestinal mucosa. It affects 1% in many populations worldwide, making it one of the most common gastrointestinal disorders [8,9].

Gluten is currently most commonly introduced into the diet of infants 4-6 months old. Breastfeeding and timing of gluten introduction, may delay the onset of the disease or modify its symptoms and can protect against celiac disease [10-12].

Clinical manifestations vary greatly according to age group, the classic form refers to presentation in children aged 9 - 24 months with signs and symptoms of malabsorption including diarrhea, steatorrhea, weight loss, or growth failure or non-classical including atypical (mono or oligosymptomatic), silent and latent forms and symptomatic (with evident gastrointestinal and/or extra-intestinal symptoms) or asymptomatic [13]. This form is strongly associated with the genes in the HLA class II complex, though other genes are also involved [14]. Extra-intestinal manifestations include iron-deficiency anemia, other deficiency states (vitamin B12, vitamin D, folate, zinc, vitamin B6), fatigue, recurrent aphthous stomatitis, elevated hepatic transaminases, short stature, delayed puberty/menarche, amenorrhea, dermatitis herpetiformis, osteopenia/osteoporosis, dental enamel hypoplasia, peripheral neuropathy and hyposplenism [15].

The serological tests used for diagnosing CD include specific and sensitive serological markers, such as EMA antibodies with 91 - 100% specificity and 88 - 100% sensitivity and tTG antibodies with 92 - 100% sensitivity and 91 - 100% specificity [16,17].

The diagnosis of CD is confirmed by endoscopic small intestinal biopsy. The sensitivity and specificity of the endoscopic findings were reported between 75% and 100%. Our patient show significant changes in endoscopic duodenal features and the pathology proving fully developed CD Marsh 3 [18-19]. Differential diagnoses of CD: Bacterial gastroenteritis, inflammatory bowel disease, chronic *Helicobacter pylori* associated duodenitis, giardiasis, autoimmune enteropathy, malabsorption and protein-losing enteropathy [20,21]. In CD treatment a lifelong total exclusion of gluten is imposed [22].

### Conclusion

CD is complicated to diagnose, as it is associated with misleading symptoms, the diagnosis of CD can be suspected by positive serologic EMA or TGA antibodies and confirmed pathologically via endoscopy and duodenal biopsy, proving a fully developed CD Gluten free- diet can show improvement in treating CD.

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