

Obesity in adolescents with celiac disease: two adolescents and two different presentations

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Celiac disease (CD) usually presents with diarrhea and growth retardation in childhood. Obesity is one of the paradoxical conditions in children with CD. We present two adolescents with CD and obesity. One of these patients was diagnosed as CD with malnutrition. His body weight had returned to normal after a gluten-free diet, and after stopping the diet, he had become obese. The second patient was an obese adolescent presenting with dyspeptic symptoms who was diagnosed as CD. Although rare, pediatricians should remember that obesity might be seen in CD before or after the diagnosis.

Key words: celiac disease, obesity, adolescent.

Celiac disease (CD) is characterized by immune-mediated injury of the small bowel mucosa that is caused by ingested gluten. Common clinical findings are diarrhea, malabsorption and growth retardation; however, atypical cases have been reported in the literature^{1,2}. As an atypical case, CD and obesity in children is seen rarely.

Herein, two adolescents with CD and obesity are presented, and we discuss the possible relationship between CD and obesity.

Case Reports

Case 1

A 21-month-old girl presented with chronic diarrhea, failure to thrive and bloating for the last 10 months. Her previous medical history and family history were unremarkable.

On admission, her weight was 8.5 kg (3-10 percentile) and her height was 77 cm (25-50 percentile). Physical examination was otherwise normal. Hemoglobin level was 10.5 g/dl, anti-gliadin antibody (AGA) IgG was positive (320 mg/dl), and AGA IgA and anti-endomysium antibody (EMA) were negative. Serum IgA was 126 mg/dl. Upper gastrointestinal endoscopy was performed, and

small bowel biopsy revealed villous atrophy, increased intraepithelial lymphocytes and hyperplastic crypts. A gluten-free diet (GFD) was started. Her complaints regressed and AGA IgG became negative six months after treatment. Thereafter, she was followed every six months until 7 years of age. She did not present for follow-up visits between the ages of 7 and 10 years. At the age of 10, she had no complaint and she was on GFD. Physical examination and laboratory tests were completely normal. She quit GFD at the age of 13 and was seen at 14 years of age. On physical examination, her body weight was 81.8 kg (>97 percentile), height was 166 cm (75-90 percentile) and body mass index (BMI) was 29.6 kg/m². Physical examination was normal apart from exogenous obesity. She was anemic (hemoglobin 9.8 g/dl). AGA IgG was positive (23.7 mg/dl), AGA IgA was negative, and EMA and tissue transglutaminase antibodies (TTG) IgA were positive. The findings of small bowel biopsy were compatible with the diagnosis of CD. The patient received GFD, which included 2200 kcal/day, and was prescribed an exercise program for CD and obesity.

Case 2

A 17-year-old girl was referred to our center with the complaints of epigastric pain and

vomiting. Her complaints had been present for three years. She had been using metformin for obesity and polycystic ovary syndrome. Her parents reported that the weight gain had begun at the age of 9 years. They also described intermittent episodes of diarrhea since infancy. Her family history was normal except for autoimmune thyroiditis in the mother. Her body weight was 88.5 kg (>97 percentile), height was 164 cm (>97 percentile), and BMI was 32.9 kg/m². Her physical examination revealed epigastric tenderness and obesity.

Laboratory data were as follows: hemoglobin: 13.7 g/dl, AGA IgG: 116.9 U/ml (range: 0-50), AGA IgA: 200 U/ml, and TTG IgA: 200 U/ml (range 0-20), and EMA was positive. Serum IgA level was normal. After the upper gastrointestinal endoscopy, she was diagnosed as CD according to the findings of the small bowel biopsy. GFD was started and she was called for follow-up visits every six months. During the two-year follow-up, her complaints regressed and the antibody titers decreased; however, she continued to gain weight. She was referred to adult gastroenterology after the age of 19 years.

Discussion

It is well known that adult patients with CD may present with obesity^{2,3}, but in children, this is a very rare and atypical presentation. There are a limited number of studies on this subject; one is a retrospective original article and the others are case reports⁴⁻⁷.

Venkatasubramani et al.⁴ reported that 7 of 143 patients with CD had BMI over 95%. Obesity is an epidemic problem in the United States (US) due to the large consumption of fast food⁸. Thus, the high caloric intake may be related with the high rates of obesity in CD patients in the US population.

Semeraro et al.⁶ reported a one-year-old child with CD who presented with malabsorption, nutrient loss and growth failure. The child, who was initially cachectic, eventually developed obesity while on a gluten-containing diet in spite of the medical control. Czaja-Bulsa et al.⁷ reported the body weight changes of a CD patient from his newborn period until 18 years

of age. Their patient had completely atrophic duodenal villi and suffered from malnutrition during the newborn period. After the diagnosis of CD, obesity occurred in later childhood on a GFD. Conti et al.⁵ reported a five-year-old girl with CD who presented with recurrent abdominal pain and short stature. The diagnosis was suspected because of her positive family history of CD. Obesity was observed in the untreated period. They emphasized that mild malabsorption was present in the untreated state of the disease but was overwhelmed by excessive caloric intake, resulting in obesity. Oso and Fraser⁹ reported a 14-year-old boy who also had CD and obesity. In spite of GFD, obesity worsened in their patient. Aslan et al.¹⁰ reported a seven-year-old girl with CD and obesity. She also had Hashimoto's thyroiditis, and refractory iron unresponsive to anemia. Our first patient was diagnosed as CD when she was 21 months old and developed obesity at the age of 14 years. Our second patient had obesity and presented with non-specific gastrointestinal symptoms and was diagnosed as CD.

Some mechanisms have been put forward to explain the reasons for obesity in children with CD. It is well known that the intestinal mucosa heals after GFD in patients with CD. Accordingly, these patients may suffer from obesity later in their life due to excessive energy intake. Furthermore, due to the fact that the GFD is less palatable and not readily available, patients are likely to consume high-energy foods, especially when they are not with their families². On the other hand, the relevant mechanism in CD patients without GFD can be explained by the compensation of the malabsorption by the intact distal bowel. Likewise, the increased surface of the small bowel with aging also contributes to this¹¹. The absence of gastrointestinal findings and malnutrition in CD patients presenting in late childhood or the adolescent period supports this hypothesis.

In conclusion, although rare, obesity should also be kept in mind during the follow-up of children with CD, even if they present with malnutrition and are commenced on GFD. On the other hand, in obese adolescents with non-specific gastrointestinal complaints, CD should also be considered in the differential diagnosis.

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