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Editorial

Celiac Disease: A Brief Review of the Literature

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Celiac disease is an autoimmune disease that is characterised by various type of intestinal villous damage, triggered by dietary gluten in genetically predisposed individuals [1].

The prevalence of celiac disease is estimated to be approximately 1% in the world. However, the risk of developing celiac disease is higher in the autoimmune diseases such as autoimmune thyroiditis and type 1 diabetes mellitus and in the first-degree relatives of celiac patients [2].

It has been suggested that a number of diseases such as cystic fibrosis, familial Mediterranean fever, functional abdominal pain and epilepsy are associated with celiac disease [3-6].

There are two peaks of celiac disease; 1 to 2 years and around 30 years old [7]. Celiac disease may present with gastrointestinal symptoms, extra-intestinal symptoms or without symptoms. Classical symptoms associated with gastrointestinal system are weight loss, steatorrhea and diarrhea due to malabsorption. Approximately 50% of celiac patients may present with extraintestinal or atypical findings such as anemia, elevated liver enzymes, short stature, delayed puberty, osteoporosis and epilepsy [8].

The reason why celiac disease presents with different clinical findings is that celiac disease has both the genetic and immunological pathogenesis, as well as the difference in the degree of mucosal injury, dietary habits and gender differences [2].

The prevalence of celiac disease has increased dramatically over the last two decades because of using highly sensitive and specific serological tests. Approximately 90% of celiac patients is asymptomatic. Although screening tests for celiac disease are recommended in high-risk groups such as autoimmune diseases, Down syndrome and type 1 diabetes mellitus, the majority of asymptomatic patients are still undiagnosed [7].

The European Society of Paediatric Gastroenterology, Hepatology and Nutiriton (ESPGHAN) latest guideline recommends tissue transglutaminase antibody IgA (tTG IgA) and total IgA tests for initial screening of celiac disease. If tTG IgA test is found positive, endomysium antibody IgA (EMA IgA) test, HLA-DQ genotyping and small intestinal biopsy are recommended [1]. If IgA deficiency was detected, tTG IgG and EMA IgG tests help to decide small intestinal biopsy. If those tests are found negative but celiac disease is considered clinically, esophago-gastroduodenoscoy should be performed and four biopsies from duodenum and at least two biopsies from bulbus were obtained [1,9]. When EMA and tTG IgA are used in combination, the sensitivity and specifity are above 95% [10].

90 - 95% of celiac patients have HLA-DQ2 and/or HLA-DQ8 haplotypes. However, the prevalence of HLA-DQ2 and/or HLA-DQ8 haplotypes in the healthy population is approximately 30 - 35% [1]. If HLA DQ2/DQ8 is found negative, the possibility of developing celiac disease is unlikely [1,11].

It has been suggested that the risk of celiac disease in various autoimmune diseases is approximately 5% and celiac disease should be considered in those diseases [12,13].

Currently, the only effective treatment is adherence to a gluten-free diet. The gluten-free diet usually provides rapid clinical recovery (several days or weeks), but the histological recovery may take several months or even years [14].

As a result, celiac disease is a life-long disorder. Timely diagnosis of celiac disease can prevent long-term complications such as osteoporosis, infertility and gastrointestinal cancer in affected individuals [15-17].

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