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Celiac Disease- An Overview

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ABSTRACT

Celiac disease is a rare disease which occurs in about 1% of the population. It is induced by the ingestion of gluten. Diagnosis is on basis of serological testing, duodenal biopsy and observation of the response to gluten free diet. It requires extensive evaluation to rule out intestinal lymphoma and refractory sprue. Increasing the awareness of epidemiology and diverse manifestation of disease among primary care physicians can lead to wide spread screening and diagnosis which in turn can result in reduction of complication of disease. This paper reviews the clinical manifestation, the causes and detection and the treatment of celiac disease.

Keywords: Celiac disease, duodenal biopsy, gluten. serological testing.

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INTRODUCTION

Celiac disease is a unique autoimmune disorder. It is unique because the environmental precipitant is known. The disorder was previously called celiac sprue derives from the Dutch spruw, which means mouth blisters characterized by diarrhea, emaciation and malabsorption [1]. Celiac disease originally considered a rare malabsorption syndrome of childhood, is now recognized as a condition that may be diagnosed at any age and that effect many organ systems. Celiac disease is a digestive disease that damage the small intestine and interferes with absorption of nutrients from food. When people with celiac disease eat food or product containing gluten their immune system responds by damaging or destroying villi of small intestine [2].

CLINICAL MANIFESTATION

It varies according to age group. Infants and young children generally present with diarrhoea, abdominal distention and failure to thrive. Besides, vomiting, irritability, anorexia and even constipation are common. Older children and adolescents often present with extra intestinal manipulation such as short stature, neurological symptoms or anemia. In adults the prevalence in women is 2 to 3 times more than men due to unknown reason. The classic presentation in adult is diarrhoea accompanied by abdominal pain or discomfort [3]. May also present with iron deficiency anemia, osteoporosis, abdominal pain, constipation, weight loss, dermatitis herpetiformis, hypoproteinema, hypocalcaemia and elevated liver enzyme level. Persons with celiac disease have an increased risk of autoimmune disorders as compared to general population [4].

CAUSE AND DETECTION

Celiac disease results from the interaction of gluten and immune, genetic and environmental factors. Celiac disease is induced by the ingestion of gluten which is derived from wheat, barley and rye. The term gluten refers to the entire protein components of wheat. Gliadin is the alcohol soluble fraction of gluten that contains a bulk of the toxic component, undigested molecule of gliadin such as peptide from an α gliadin fraction is made up of 33 amino acid which are resistant to degradation by gastric, pancreatic and intestinal proteins. After gluten ingestion these gliadin remains in the intestinal lumen. These peptides pass through intestine possibly during intestinal infections or where there is an increase in intestinal permeability and interact with antigen presenting cells in the lamina propria which in turn promote an inflammatory reaction primarily in the upper small intestine [5]. This results in infiltrations of lamina propria and epithelium with chronic inflammatory cells and villous atropy. This response is mediated by both adaptive and innate immune response. The adaptive immune response is mediated by gliadin reactive CD4+T cells in lamina propria that recognize gliadin peptides which are bound to HLA class II molecules DQ2 or DQ8 or antigen present in cells. The T cells subsequently produce proinflammatory cytokines particularly interferon γ tissue transglutaminase which is an enzyme in the intestine that deaminates gliadin peptides, increasing the immune genicity.

The ensuring inflammatory cascades release metallo-proteinases and other tissue damaging mediator that induce apt hyperplasia and villous injury. Gliadin peptides also activates an innate immune response in the intestinal epithelium that is characterized by

increased expression of interleukins 15 by erythrocytes resulting in the activation of receptor N.K-G2D a natural killer cell marker. These activated cells become cytotoxic and kills erythrocytes with surface expressions of major histo compatibility complex class 1 chain related A, a cell surface antigen induced by stress such as infection[6].

The mechanism of interaction between the processes in epithelium and lamina propria has not been elucidated. The genetic influence in the pathogenesis of celiac disease is indicated by it familial occurrence. Celiac disease does not develop unless a person has alleles that encode for HLADQ2 or HLADQ8 protiens, product of HLA genes. However many people, most of whom do not have Celiac disease carry these alleles. Then their presence is necessary but not sufficient for the development of the disease. Studies in siblings and identical twins suggests that the contribution of the HLA genes to the genetic components of celiac disease is less than 50%. Environmental factors that have an important role in the development of celiac disease have been suggested by epidemiologic studies. These include protective effect of breast feeding and introduction of gluten in relation to weaning. The initial administration of gluten before four months of age is associated with increased risk of disease development and introduction of gluten after 7 months associated with marginal risk [7].Over lap of gluten with breast feeding may be more important protective factor in minimizing the risk of Celiac disease. The occurance of certain gastro intestinal infection such as rota viral infection also increases the risk of celiac disease.

DIAGNOSIS

Diagnosis is by duodenal biopsy that shows characteristic findings of Intra epithelial lymphocytosis, crypt hyperplasia and villous atrophy. The most sensitive antibody test for the diagnosis of celiac disease are of IgA class. The available test include those for antigliadin anti bodies which is sensitive only in children younger than 18 months of age. The diagnostic standard in celiac serology remains the endomysial IgA antibodies they are highly specific marker for celiac disease approaching 100% accuracy. The enzyme antitissue transglutaminase is an auto antigen for the development of endomysial antibody. Automated enzyme linked immune assay that are less expensive is available for the detection of anti tissue glutaminase. This test is recommended as single test for celiac disease [8].

TREATMENT

Treatment involves nutritional therapy together with lifelong elimination of wheat, rye and barley from diet. Oats can be substituted which is well tolerated and improves the overall quality of life after the diagnosis of celiac disease. If the disease have been established, the patient should be assessed for deficiencies of vitamins including folic acid, vitamin$B_{12,}$fat soluble vitamins and minerals like iron and calcium and should be replenished accordingly. The elimination of gluten usually induces clinical improvement within days or weeks though histologic recovery takes months or even years especially in adults in whom mucosal recovery is incomplete. In most cases corticosteroids induce clinical improvement [9].

DISCUSSION

The person who suffers from celiac disease have a sensitivity to gluten, a protein found in wheat. The patient develops gastro intestinal distress and other serious symptoms when they eat gluten containing products. University of Maryland School of Medicine researchers discovered that zonulin a mysterious human protein played a critical role in celiac disease and auto immune disorders such as multiple sclerosis and diabetes. Scientists lead by Alessio Fasano.MD has identified zonulin as a precursor molecule of hepatoglobulins. In celiac patients gluten generates an exaggerated release of zonulin that makes the gut more permeable to large molecules such as gluten to the rest of the body [10].This triggers an auto immune response in which a celiac patients immune system identifies gluten as an intruder and response with an attack targeting the intestine instead of gluten. There is no single satisfactory explanation for celiac disease but it does seem likely that wheat and other products containing gluten coupled with genetic factors may be responsible [11].

CONCLUSION

In cases reported in medical literature, the strong factors seem to contribute to the appearance of celiac disease is the presence of gluten, a protein in wheat with immune, genetic and environmental factors. In many cases patient shows dramatic recovery when on gluten free diet along with correction of nutritional deficiencies.

As Celiac disease is a rare, disease, diagnosis is difficult since the facilities are not routinely available. Although the rate of diagnosis is increasing, in most affected people celiac disease remains undiagnosed.

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