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Pathophysiology of Celiac Disease
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Introduction

- My future father-in-law was diagnosed with celiac disease (CD) approximately 2 years ago.
- He was 60 years old at the time of diagnosis and suffered with diarrhea and abdominal pain for a year prior to being told he had CD.
- My fiancé and daughter are at increased risk of developing CD due to the genetic/epidemiologic component of the disease.
- Those who have a first-degree relative with CD have a 4% to 7% increased risk of developing the disease compared to the general population.

About CD

- CD was first described by Samuel Gee in 1897 and it wasn't until 1941 that Seldin was hypothesized as the possible overflowing agent, noted by William Cole (Leonard, Sapos, Callow, & Pimplen, 2015).
- CD was once thought to almost exclusively affect young Caucasian children, but can actually occur at any age and can affect almost any race (Leonard et al., 2017).

Underlying Pathophysiology

CD is an autoimmune disorder occurring in genetically predisposed individuals. Individuals who are genetically predisposed to CD have the HLA-DQ2 or HLA-DQ8 haplotype (Lambert et al., 2015).

- The prevalence of CD is estimated to be 0.6% to 1% worldwide with approximately 0.7% to 0.8% of the United States (US) population testing affected (Cui et al., 2017).
- HLA-DQ2 or HLA-DQ8 genes are the primary genetic factors that contribute to the development of CD (Green et al., 2015).

Signs and Symptoms

- The classic presentation of CD manifests as gastrointestinal (GI) symptoms of diarrhea, abdominal pain, and malnutrition, resulting in poor growth/failure to thrive in children (Rodman, Davis, Veasey, & Lebwohl, 2015).
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Diagnostic Testing

- In addition to duodenal biopsy, serological and genetic testing can be performed for diagnosis of CD.
- Serological testing measures the levels of:
  - Anti-tissue transglutaminase (ITG)
  - Anti-endomysium (EMA)
  - Anti-deamidated gliadin peptide (DGP) (Green et al., 2015).
- First line testing to screen for CD includes measurement of serum IgA antibodies to ITG (Leonard et al., 2017).

Significance of Pathophysiology

- The inflammatory process described results in small histologic abnormalities including:
  - "Atrophy of the intestinal villi, hyperplasty of the crypts and lymphocytic infiltration of interstitial spaces and lamina propria" (Adelman, et al., 2018).
- Intestinal villi, pictured below, are finger-like projections that line the intestinal wall, allowing absorption and digestion of nutrients (Celiac Disease Foundation [CDF], 2017).
- Undiagnosed CD results in the continued ingestion of gluten, which results in continuous inflammation that erodes and flattens the villi, causing inefficient uptake of nutrients as they pass through the intestines (CDF, 2017).

Conclusion

- The reason for an increasing prevalence of CD is still unknown, as is the extent to which environmental factors other than gluten contribute to the development of CD (Green et al., 2015).
- The development of CD occurs among subjects with HLA-DQ2 or HLA-DQ8 genes who are exposed to gluten and other environmental factors (Green et al., 2015).
- Diagnosis of CD is based on characteristic autoantibodies seen in serological testing, as well as with small intestinal histological changes upon histologic examination (Green et al., 2015).
- Early diagnosis can prevent the extent of villous atrophy seen in CDF, allowing for better management of symptoms seen in those with CD (Green et al., 2015).
- Adherence to a strict GF diet is the best predictor of remission of the disease.