

Publication of the Month

January 01/12: The new ESPGHAN guidelines for the diagnosis of celiac disease

Key messages:

- *In case of confirmed high tTG IgA antibody titers celiac disease can be diagnosed without performing a duodenal biopsy.*
- *Not only Marsh 3 but also Marsh 2 lesions are compatible with a diagnosis of celiac disease.*

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European Society for Pediatric Gastroenterology, Hepatology, and Nutrition Guidelines for the Diagnosis of Coeliac Disease

JPGN 2012;54:136-160

Background:

In the last 20 years the perception as well as the diagnosis of celiac disease (CD) has changed by discovering the genetic predisposition by HLA-DQ2 and -DQ8 and developing CD-specific anti-tissue transglutaminase (tTG) assays.

With regards to these developments 17 experts of the ESPGHAN developed new guidelines for the diagnosis of CD and present diagnostic algorithms which are designed to achieve a high diagnostic accuracy combined with a reduction of the burden for patients and their families.

Summary:

The definition of celiac disease has been updated. The disease is now seen as an immune-mediated systemic disorder elicited by gluten and related prolamines in genetically susceptible individuals. Different diagnostic algorithms were developed for two groups of subjects: children or adolescents with symptoms or signs suggestive of CD (group 1) and asymptomatic children or adolescents at increased risk for CD (group 2). The importance of serological markers (tTG IgA, deamidated gliadin peptide (DGP) IgG, total IgA) is substantially increased, while intestinal biopsy is not that essential any more. In certain cases serology is now sufficient for the diagnosis of CD.

In symptomatic subjects CD diagnosis can be made if tTG IgA antibody titers are high (>10 x the upper limit of normal). The introduction of this decision point is based on experiences with the Celikey IgA assay from Phadia, now Thermo Fisher Scientific.

In contrast to the old guidelines not only a Marsh 3 but also a Marsh 2 lesion has now been accepted compatible with CD. These alterations are not specific for CD and may be found in enteropathies other than CD. This weakens the significance of the biopsy, which is not regarded as the gold standard for CD diagnosis any more.

Conclusions:

The diagnosis of CD depends on gluten-dependent symptoms, CD-specific antibodies, the presence of HLA-DQ2 and/or HLA-DQ8, and characteristic histological changes in the duodenal biopsy. In case of high antibody levels the diagnosis of CD may be based on a combination of symptoms, antibodies, and HLA, thus omitting the duodenal biopsy.

Comment:

The new ESPGHAN guidelines are a large step forward in the diagnosis of CD. This is achieved by the combination of highly reliable antibody tests (tTG IgA and DGP IgG) and genetic testing, which makes the inconvenient and expensive procedure of duodenal biopsy obsolete in many cases.

