HELICOBACTER PYLORI

By Clarisa E. Cuevas, MD, and Youhanna S. Al-Tawil, MD

Since 1982, when Helicobacter Pylori (H. pylori) was successfully cultured by Marshall and Warren, there have been multiple evolving studies about its pathogenesis, diagnostic tests and treatment. The infectious process in H. pylori occurs during childhood by intrafamilial transmission with a later prevalence greater than 60% in the adult population. In developed countries, the acquisition of this organism is felt to be around 1%. Eventually, more than 30% of the adult population will be colonized. Prolonged infection and inflammation leads to ultimate chronic gastritis. This chronic inflammation can lead to severe gastric pathologies such as peptic ulcer, gastric adenocarcinoma, and gastric mucosa-associated tissue lymphoma. Clinical presentation of H. pylori includes chronic gastritis, duodenal and gastric ulcer, GERD, extraintestional manifestation including iron deficiency anemia, chronic uriticaria, Idiopathic Thrombocytopenia Purpura (ITP), and short stature.

H. pylori can be found in a normal stomach and be asymptomatic. When there is a strong suspicion that H. pylori are causing clinical symptoms, testing cannot only detect the presence of H. pylori but can also confirm a diagnosis. Upper gastrointestinal endoscopy and biopsy remains the 'gold standard' in the diagnosis and identification of H. pylori infection and its consequences in childhood. It allows visualization of the upper gastrointestinal tract and facilitates the collection of mucosal biopsies. Various tests can detect H. pylori from endoscopic biopsy specimens, including

(1) A histological exam. Works by staining gastric mucosal biopsies to detect H. pylori bacterium and can be done using a variety of different stains. A superficial infiltrate is usually seen with substantial numbers of plasma cells and lymphocytes within the mucosa;

(2) A Rapid Urease Test (RUT). Urease catalyzes the hydrolysis of urea into ammonia and carbon dioxide. The production of ammonia leads to an increase in the local pH. Samples are placed within a gel containing urea and a pH indicator. A color change occurs as urea is broken down by the bacteria. Use of RUTs in pediatrics is limited by a significantly lower sensitivity compared to histology;

(3) H. pylori culture. A potential 'gold standard' for the diagnosis of suspected H. pylori infection. Its sensitivity has been reported to vary greatly between laboratories.

Several nonendoscopic tests are noninvasive and also used to diagnose H. pylori. One is a serological test. H. pylori infection induces both cellular and humoral immune responses, resulting in an early increase in specific IgM, and a later and persistent increase in specific IgA and IgG. In children, IgA-based tests detect only 20-50% of H. pylori infected patients. Serologic tests based on the detection of specific anti-H. pylori IgG antibodies in the serum offer a better sensitivity than IgA-based tests. Their most important limitation is the inability to distinguish active from past infection. Another is the Urea Breath Test (UBT). Urea is labeled with either 13 C (non-radioactive) or 14 C (radioactive) isotopes, and then ingested. 13 C is a naturally occurring nonradioactive isotope. It can be safely used in even very young infants, and can be repeated without risk to the child. Labeled urea comes into contact with the mucosa and diffuses through the mucus. Urea hydrolysis by H. pylori produces ammonia and labeled carbon dioxide. Urea rapidly passes down its concentration gradient into the epithelial blood supply, and within minutes appears in the breath. Breath samples are collected at variable times post ingestion. A third noninvasive test is a stool antigen. H. pylori antigen can be detected in the stool. Stool testing is a potentially inexpensive, noninvasive method for determining H. pylori infection. Overall sensitivity and specificity of the stool test are comparable to the UBT (94% and 97%, respectively). A rapid H. pylori stool antigen test is available that permits testing during a clinic visit but is slightly less accurate than a traditional laboratory based stool test. The sensitivity of stool testing is negatively affected by PPIs, bismuth, and antibodies, which can decrease bacterial load.

The current first line of treatment therapy recommended in the United States and in the Western world is clarithromycin, amoxicillin, or metronidazole, plus a PPI twice a day. Treatment regimens of seven, ten, or fourteen days depend on the population and resistance pattern in a specific area. More critical than the duration of therapy is the resistance of H. pylori to a specific antibiotic. Most treatment failures occur when the organism is resistant to clarithromycin. In that case, metronidazole, amoxicillin, and bismuth base therapy along with a twice-a-day PPI becomes the treatment of choice. Major stumbling blocks to eradication include patient compliance, H. pylori strain resistance to antibiotics, and asymptomatic patients that remain at-risk of developing atrophic gastritis and eventual cancer.

In the past few months GIforKids, PLLC, has seen an increase in the number of children diagnosed with infection from H. pylori. Our state of the art endoscopic suite, located in East Tennessee Children's Hospital, can provide upper/lower endoscopy and other appropriate testing to diagnose this infection. For more information, please contact our clinic at (865) 546-3998 or visit our website at www.giforkids.com.

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