

H. pylori is a gram-negative, microaerophilic bacterium that can infect humans. It is often found in the stomach of affected individuals and causes inflammation and ulceration. Patients harboring the bacteria are asymptomatic with abdominal pain, nausea, vomiting, and dyspepsia developing only after gastritis and peptic ulcer disease have set in.

Helicobacter pylori (*H. pylori*) is a gram-negative spiral-shaped bacterium that affects up to 50% of the population worldwide, with a higher prevalence in developing countries. *H. pylori* is the most important cause for chronic or atrophic gastritis, peptic ulcer, gastric lymphoma, and gastric carcinoma; however, these complications are less often seen in children and adolescents compared to adults. *H. pylori* infection is usually acquired in early childhood and persists in the absence of treatment. A phase 3 clinical trial in children in China documented the efficacy and safety of an oral recombinant *H. pylori* vaccine, a future option to reduce the incidence of *H. pylori* infection.

Transmission of *H. pylori* can occur via the fecal-oral, gastric-oral, oral-oral, or sexual routes. A major risk factor for a higher prevalence of infection is lower socioeconomic status.

Symptoms

The majority of children with *H. pylori* infection are asymptomatic. Symptoms, if present, are usually of gastritis or peptic ulcer disease such as abdominal pain, nausea, vomiting or dyspepsia. Children with these gastrointestinal symptoms should be investigated to identify the underlying etiology of the symptoms. There are various extra-intestinal manifestations that are associated with *H. pylori* infection such as iron deficiency anemia and chronic immune thrombocytopenia (cITP).

Therefore, guidelines recommend that non-invasive testing for *H. pylori* can be considered in children with cITP or with refractory iron deficiency anemia without an identifiable cause. Children may also be anemic as it has been documented that those infected with *H. pylori* have lower iron stores. There seems to be a poor association with other extra-intestinal manifestations such as otitis media, upper respiratory symptoms, periodontal disease,

sudden infant death syndrome (SIDS), or short stature; therefore *H. pylori* testing is not recommended in such cases.

Diagnosis

H. pylori infection is diagnosed by both invasive and non-invasive methods.

Noninvasive tests include the detection of *H. pylori* antigens in the stool, detection of antibodies against *H. pylori* in serum, urine and oral samples, and a urea breath test (UBT). The stool antigen test and UBT have high sensitivity and specificity similar to the invasive methods. Serological assays detecting the presence of antibodies (IgG is used in clinical laboratory practice) to *H. pylori* have poor sensitivity and are not reliable for use in the clinical setting.

Invasive tests require gastric tissue for detecting the organism and include culture, rapid urease tests (RUT), histopathology, polymerase chain reaction, and fluorescent in situ hybridization (FISH). Culture is the only method with 100% specificity, and a positive culture is sufficient to diagnose *H. pylori* infection, but it has a lower sensitivity. For this reason, concordant results of at least two tests are needed to define the *H. pylori* status.

It is recommended that the initial diagnosis of *H. pylori* infection should be based on either positive histopathology and positive RUT or a positive culture from gastric biopsies. The current guidelines to diagnose *H. pylori* infection in children is an upper gastrointestinal endoscopy with gastric biopsies (antrum and corpus) for histology, culture, and RUT. To determine *H. pylori* eradication, both UBT and stool *H. pylori* antigen detection by ELISA are reliable non-invasive tests. Before a patient is tested for *H. pylori* eradication, it is recommended to wait at least 2 weeks after stopping proton pump inhibitors (PPIs) and 4 weeks after stopping antibiotics.

The differential diagnosis include: Celiac disease, Upper gastrointestinal Crohn disease, Peptic ulcer disease, NSAID-induced gastritis, Gastroesophageal reflux disease, Eosinophilic esophagitis, Lactose intolerance, Constipation.

Treatment

To achieve a higher eradication rate following *H. pylori* infection, the European Society for Pediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) and North American Society for Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN) have updated the treatment guidelines for children and adolescents.

Triple therapy including proton pump inhibitor (PPI) (1-2 mg/kg/day), amoxicillin (AMO) (50 mg/kg/day), and clarithromycin (CLA) (20 mg/kg/day) for 14 days is the treatment of choice if the strain is susceptible to CLA and metronidazole (MET). If this regimen fails, CLA can be replaced with MET (20 mg/kg/day) without further antibiotic susceptibility testing.

Another treatment option is sequential therapy for 10 days (PPI with AMO for 5 days followed by PPI with MET and CLA for 5 days) in pediatric patients infected with fully susceptible strains. Sequential therapy should not be given if the strain is resistant to MET or CLA; however, this treatment regimen exposes the child to three different antibiotics. Medication doses should be calculated based on the weight of the child, and a higher dose of PPI per kg body weight is recommended to suppress acid in younger children sufficiently compared to adolescents and adults. PPIs are preferred to be given at least 15 minutes before meals.

In the case of resistance to both CLA and MET or when antimicrobial susceptibility is not known, bismuth quadruple therapy consisting of bismuth salts (8 mg/kg/day), PPI, AMO (in children younger than 8 years) or tetracycline (in children older than 8 years) and MET is effective. Although there is limited evidence, the other regimen recommended, in this case, is the combination of high dose AMO triple therapy with MET.

Development of resistance to antibiotics is a serious problem that varies from region to region. Using the macrolide group of antibiotics for respiratory tract infection has led to the emergence of *H. pylori* CLA resistance. With treatment failure, rescue therapy should be individualized based on antibiotic susceptibility. In comparison to Asians, Caucasian populations metabolize PPI more rapidly due to CYP2C19 genetic polymorphism. Therefore PPIs like esomeprazole and rabeprazole that are less prone to degradation by rapid

metabolizers with CYP2C19 polymorphism, should be used when available. Also, probiotics have not shown to improve eradication rates or reduce side effects from current evidence.

Source:

Parikh NS, Ahlawat R. Helicobacter Pylori. [Updated 2022 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK534233/>

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