**Helicobacter pylori**: Diagnosis and Treatment

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**Helicobacter pylori** is a common pathogen with the potential to cause gastric cancer. Various treatment regimens are now recommended and can achieve cure rates approaching 90%, thus preventing long-term complications.

**Pathogenesis**

All patients with gastric H pylori will develop a cellular infiltrate in the gastric mucosa, called chronic gastritis. In most people this causes no symptoms, but H pylori carriers do have an increased risk of developing peptic ulcer disease and adenocarcinoma of the antrum and body of the stomach. The antrum plays an important role in the regulation of acid secretion through secretion of gastrin from antral G cells and somatostatin D cells. Host and environmental factors, such as gender, genetic factors, diet and whether the patient smokes, may modulate the clinical response to H pylori infection.

Bacterial factors are also important. Recently, different genetic loci have been identified: cagA, vac A and ice A. The presence of these genes is associated with a more severe clinical outcome. Recent work suggests that the diversity of H pylori is clinically important: the presence of particular alleles incurs different risks for disease. Persons harbouring H pylori strains that possess the cagA gene and have in vitro production of vacuolating cytotoxin (cagA + tox +) develop a more severe mucosal inflammatory response that may increase the risk of progression to ulceration.

The presence of H pylori in the stomach is responsible for a variety of clinical conditions, including peptic ulceration, adenocarcinoma of the lower stomach, MALT lymphoma, oesophageal disease, and non-ulcer dyspepsia.

**Diagnosis**

Diagnosis may be confirmed by invasive or non-invasive tests. Invasive tests include performing a biopsy/rapid urease test followed by histology and culture. The gold standard for diagnosis remains a positive culture.

Non-invasive tests include the urea breath test, IgG-H pylori (serology) serum test and faecal H pylori antigen test.

When endoscopy is clinically indicated, infection can be confirmed by urease testing of a biopsy specimen or by histology. The sensitivity and specificity of histological examination are claimed to be >90%. If endoscopy is not indicated, diagnosis...
can be made by serological testing or by the urea breath test. The latter is based on the detection of labelled carbon dioxide (labelled with $^{13}$C or $^{14}$C) in expired air as a result of H. pylori urease activity. In the biopsy urease test, a change in colour of the phenol red indicator can confirm infection within an hour, so that diagnosis can be made with the patient still present. The test is interpreted after insertion of the gastric biopsy sample into the well containing the agar gel. H. pylori urease hydrolyses the urea contained in the agar gel of the test packet leading to a colour change of the phenol red indicator. These urease tests are inexpensive and a rapid (usually <30 minutes) means of obtaining a specific diagnosis.7,12

Serological tests are based on the detection of a specific anti-H. pylori immune response. Such tests are widely available, relatively inexpensive, and more convenient than the urea breath test. The latter however, is a useful means of evaluating treatment response a few weeks later.

Both non-invasive assays can be used to screen young patients with dyspepsia, so that endoscopy services are not overloaded and resources are saved. The tests are also safe and acceptable to patients.

Treatment

Eradication of H. pylori in patients with gastric ulceration is both cost effective and beneficial. Pharmacological regimens comprise an agent to suppress gastric acid secretion and one or two antibiotics. Eradication aims to relieve symptoms and cure the disease.

The European Helicobacter pylori study group13 has recommended formal diagnostic evaluation for patients >45 years old with dyspepsia not previously investigated or for patients with "alarm" symptoms such as weight loss, bleeding, anaemia or dysphagia, regardless of age.

For those aged <45 years, the clinician must decide among initial testing for H. pylori, empirical treatment with an anti-secretory agent, prokinetic or anti-H. pylori therapy or immediate endoscopy.13

In populations with a high incidence of ulcer disease it might be cheaper to prescribe antibiotics to all dyspeptic patients who are seropositive for H. pylori than to screen all dyspeptic patients for gastric ulceration.12,13

Treatment Regimens

New drugs are continually being developed and evaluated. The popularity of dual treatment that combines...
a proton pump inhibitor with either amoxicillin or clarithromycin has waned due to its lack of efficacy. Dual treatment with ranitidine bismuth citrate and clarithromycin for 14 days is effective and has few side effects. It is easy to administer, acceptable to patients, and thus attractive for general practice.14,15

The Food and Drug Administration currently approves six combination regimens for the treatment of H pylori infection in patients with active duodenal ulcer.16 (Table 1)

### Triple Therapy

Therapy with three agents is currently favoured, and achieves eradication rates of around 90%.15 (Table 2)

### Who Should be Treated?

Current indications for treatment include patients with H pylori-related duodenal ulcer, gastric ulcer or active chronic gastritis.14 Treatment is generally not indicated for H pylori in the absence of ulceration, such as in non-ulcer dyspepsia. It may however be reasonable to treat infected patients who have a family history of gastric cancer.

### Conclusion

H pylori is the most common chronic bacterial infection in humans and has been linked to the development of peptic ulcer, gastric adenocarcinoma and gastric lymphoma. Curing H pylori infection cures ulcer disease; since reinfection in adults is rare, adequate treatment permanently cures this former chronic condition. Although becoming simpler, treatment regimens are still complex and their side effects and cost do not allow mass treatment. In addition, antibiotic resistance would likely develop and be a major problem if universal eradication were adopted.

### References


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