*Helicobacter pylori*: How to decide when to treat

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**Why treat?**

- **Relieve symptoms**
  - Example: functional dyspepsia
- **Manage associated diseases**
  - Examples: peptic ulcer disease, idiopathic thrombocytopenic purpura,
- **Prevent disease complications**
  - Example: bleeding peptic ulcer
- **Prevent cancer**
  - Example: gastric adenocarcinoma
Peptic ulcer disease

- *Helicobacter pylori* infection predisposes to the development of peptic ulcer
- About 20% of infected individuals develop PUD
- Continued *H. pylori* infection predisposes to recurrence
- Gastric acid is a necessary co-factor
- Gastric metaplasia in bulb leads to DU

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Peptic ulcer disease

- Treatment promotes ulcer healing
- Treatment prevents ulcer recurrence
- Treatment reduces ulcer bleeding
Peptic ulcer disease

- **Uncomplicated** DU: no need to prolong PPI therapy once *H. pylori* is treated
- GU and **complicated** GU: continue PPI until GU has healed or *H. pylori* eradication is confirmed in DU
- In patients with bleeding ulcer, start eradication treatment when oral feeding is restarted


Dyspepsia

- Uninvestigated dyspepsia in primary care
  - Some patients will be infected with *H. pylori*
  - If prevalence of *H. pylori* is high (>20%), a test-and-treat strategy makes sense
    - Use non-endoscopic technique for diagnosis (urea breath test or fecal antigen)
    - Treat if positive
    - Must be young enough to have low risk of gastric cancer

**Functional dyspepsia**

- *H. pylori* eradication produces long-term relief of dyspepsia in 8% of patients with *H. pylori* and functional dyspepsia
  - More successful than any other single treatment tested


**NSAIDs**

- *H. pylori* + NSAIDs = increased ulcer risk
- Eradication reduces risk of ulcer and complications in NSAID naïve patients
- Less clear if eradication is helpful in long-term NSAID user → continue PPI
- Useful to treat before starting NSAID therapy; mandatory if history of ulcer

MALT lymphoma

- *H. pylori* eradication is first line treatment for low-grade gastric MALT lymphoma
  - Stage I/II lesions can be cured in 60—80%
  - If t(11,18) translocation is present, unlikely to be cured by *H. pylori* eradication and additional treatment may be needed


Extragastric diseases

- Unexplained iron deficiency anemia
  - Association in both adults and children
  - Eradication associated with increased hemoglobin level
- Idiopathic thrombocytopenic purpura
  - Eradication led to increased platelet counts in 50%
- Vitamin B₁₂ deficiency
- Poor drug absorption (thyroxine, l-dopa)

Cancer prevention

• *H. pylori* is the most common proven risk factor for non-cardia gastric adenocarcinoma

• Eradication of *H. pylori* is the most promising strategy to reduce the incidence of gastric cancer

• Chronic active gastritis, atrophy, and intestinal metaplasia are associated with the development of cancer


Cancer prevention

• *H. pylori* eradication abolishes the inflammatory response, and may slow, arrest, or reverse atrophy in the corpus (but not the antrum)

• Intestinal metaplasia is irreversible

• Eradication therapy has benefit in primary and secondary cancer prevention

• Eradication therapy is most effective if delivered before development of atrophy

Cancer prevention

• Screening may be cost-effective in populations with a high risk of gastric cancer
  – Early *H. pylori* eradication would be more cost-effective than life-time surveillance
  – Screening young adults in China might reduce one of every 4 to 6 gastric cancers
  – Not useful in U.S.


Cancer prevention

• Who should be considered for treatment?
  – 1st relatives of gastric cancer patients
    • 2—3-fold increase with one relative
    • 10-fold increase with >1 relative
  – Patients with severe pan-gastritis, corpus-predominant gastritis, severe atrophy
  – (Patients with previously resected gastric neoplastic lesions)

Who should NOT be treated?

- Patients with gastroesophageal reflux disease
- Patients on chronic PPI therapy
- Patients with intestinal metaplasia


Treatment tips

- Standard triple therapy
  - PPI + clarithromycin + metronidazole
  - PPI + clarithromycin + amoxicillin

- Sequential therapy
  - PPI + amoxicillin X 5 days, followed by
  - PPI + clarithromycin + metronidazole X 5 days

- Quadruple therapy
  - PPI + bismuth + tetracycline + metronidazole

Treatment tips

• Clarithromycin-containing triple therapy recommended if resistance rate is <20%
• Bismuth-containing quadruple therapy is alternative
• BID PPI therapy increases efficacy
• Extending therapy from 7 to 10—14 days improves success by ~5%


New treatment paradigm

• Because of frequency of clarithromycin and metronidazole resistance, traditional regimens may be obsolete in most places
• Preferred choices in U.S.:
  – 14-day concomitant therapy
  – 14-day bismuth quadruple therapy
  – 14-day hybrid sequential-concomitant therapy
• Knowing local resistance pattern is essential

Treatment tips

- Levofloxacin-containing triple therapy is a salvage regimen if levofloxacin resistance is not high in community
- Antibiotic susceptibility should be assessed if second-line therapy fails
- Prove eradication with urea breath test or fecal antigen test (not serology)