PUD CAUSES: H. PYLORI

FLAME LECTURE: 74

BERMAN & BUCHOLTZ 3.2.20

LEARNING OBJECTIVES

- To understand H. Pylori and PUD
- To describe background, epidemiology, pathophysiology, diagnosis and consequences
- Prerequisites:
  - NONE
- See also – for closely related topics
  - FLAME LECTURE for other causes of peptic ulcer disease as well as for diagnosis, treatment, management of complications in PUD

BACKGROUND

- H. Pylori is a spiral urease-producing gram-negative organism
- Colonizes gastric antral mucosa in:
  - ~100% of pts with duodenal ulcers
  - ~80% of pts with gastric ulcers
- The risk for the development of an ulcer in the presence of H. pylori is determined by a combination of host- and bacteria-related factors
  - Host factors include immune response, smoking, and stress

EPIDEMIOLOGY

- H. pylori has been isolated in all parts of the world
- The global prevalence of *H. pylori* in 2015 was estimated to be 4.4 billion people and has been declining
- H. pylori associated with higher age and lower SES
- Prevalence is likely decreasing as a result of:
  - Smaller family sizes
  - Decreased crowding
  - Improved sanitation
  - Antibiotic use

PATHOPHYSIOLOGY

- Increased release of gastrin due to:
  - Stimulation of antral G cells by cytokines released by inflammatory cells
  - Diminished production of somatostatin by D cells
- An exaggerated acid response to gastrin due to an increased parietal cell mass resulting from gastrin stimulation

DIAGNOSIS

1. Serum Antibodies

- >80% sensitive, >90% specific
- Inexpensive, convenient; not useful for early follow up

2. Rapid Urease Test of Antral Biopsy

- 80-95% sensitive, 95-100% specific
- Simple but requires biopsy
- False negative with recurrent use of PPI, antibiotics, or bismuth

3. Urea Breath Test (generally used to confirm eradication)

- >90% sensitive and specific
- Simple, rapid, useful for early follow up

4. Fecal Antigen

- >90% sensitive and specific
- Inexpensive, convenient, not established for eradication

OVERVIEW OF ASSOC. GI LESIONS

LESION	ASSOCIATION WITH H. PYLORI
Chronic diffuse superficial gastritis	Nearly always associated
Type A (pernicious anemia) gastritis	Negative association
NSAID gastropathy	Negative or no association
Acute erosive gastritis (e.g., alcohol, aspirin)	No association
Gastric ulceration	Commonly observed in patients who are not ingesting NSAIDs or aspirin
Duodenal ulceration	Usually associated with idiopathic lesions (non–drug induced, non–Zollinger-Ellison syndrome)
Gastric adenocarcinoma	Positively associated with (non-cardia) cancers of the gastric body and antrum

OVERVIEW OF ASSOC. GI LESIONS

LESION	ASSOCIATION WITH H. PYLORI
Gastric lymphoma	Strongly associated with MALT-type B-cell lymphomas
Idiopathic thrombocytopenic purpura	Often associated
Non-ulcer dyspepsia	Little or no association
Gastroesophageal reflux disease	Presence of *cag* $^{+}$ strain has protective association
Barrett's esophagus	May colonize distal gastric epithelium in pts w/ gastric colonization[4]; presence of *cag*$^{+}$ strain has protective association
Adenocarcinoma of the esophagus	Presence of *cag* $^{+}$ strains has protective association
Childhood asthma and related allergic disorders (allergic rhinitis, eczema, and skin sensitization)	Presence of *cag* $^{+}$ strains has protective association

IMPORTANT LINKS / REFERENCES

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