Gastritis: Causes

- *Helicobacter pylori* infection
- NSAID use
- Excessive alcohol consumption
- Heavy smoking
- Radiotherapy
- Cancer chemotherapy
- Systemic infections (*Salmonella*, CMV)
- Severe stress
- Ischemia and shock
- Suicide attempts with acids or alkali
- Mechanical trauma
- Distal gastrectomy
GASTRITIS

Morphologic (descriptive) classification

1. Acute gastritis (neutrophils)
2. Hemorrhagic gastritis (fresh blood)
3. Erosive gastritis (destruction of parts of the mucosa)
4. Granulomatous gastritis
5. Eosinophilic gastritis
6. Chronic gastritis (most common)
Types of Chronic Gastritis

- **Autoimmune gastritis** (type A gastritis): diffuse gastritis of corpus; antibodies to parietal cells and intrinsic factor; low acid, pernicious anemia; associated with other autoimmune disorders; uncommon.

- **Helicobacter pylori gastritis** (type B gastritis): may affect all parts of the stomach, mostly antrum; 3 subtypes: antrum-predominant, corpus-predominant, pangastritis; very common.

- **Chemical gastritis** (type C gastritis): due to repeated chemical or toxic injury (bile acids, duodenal contents, NSAIDs); common.
Prevalence of Biopsy-proven *H. pylori* Gastritis

Asymptomatic adults: 30%
Non-ulcer dyspepsia: 67%
Gastric ulcer: 65%
Duodenal ulcer: 86%


**Helicobacter Pylori Infection**

Cofactor: Time of life when infection was acquired

Childhood: Multifocal atrophic gastritis
Gastric ulcer
Gastric cancer

Adulthood: Chronic active gastritis
Duodenal ulcer

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**H. Pylori Gastritis**

Topographic Types

Chronic gastritis of antrum and corpus
Chronic gastritis, antrum-predominant
Chronic gastritis, corpus-predominant
H. PYLORI
UNUSUAL FEATURES

First cultured in 1982 (April 14)
"Unidentified curved bacilli" 1983
Campylobacter pyloridis 1984
Campylobacter pylori 1987
HELCIOBACTER pylori 1989

Natural habitat: human stomach
Figure 1. The eradication rate of triple therapy decreased as the density of *H. pylori* increased. *, Significant difference
### Some Characteristics and Outcomes of the Chronic Gastritides

#### Topography of the Atrophy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Severe antral atrophy</th>
<th>None</th>
<th>Severe panatrophy</th>
<th>Severe corpus atrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td>HP related</td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Yes</td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>No</td>
</tr>
<tr>
<td>Gastrin output</td>
<td>Impaired</td>
<td>Mild increase</td>
<td>Low</td>
<td>Autoimmune pathogenesis</td>
</tr>
<tr>
<td>Acid output</td>
<td>Normal</td>
<td>Normal Increase</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Peptic ulcer</td>
<td>Increased</td>
<td>Increased</td>
<td>Slight</td>
<td>Achlorhydria</td>
</tr>
<tr>
<td>Relative risk</td>
<td>30–40</td>
<td>10–40</td>
<td>1–2</td>
<td>No</td>
</tr>
<tr>
<td>Gastric carcinoma</td>
<td>Markedly increased</td>
<td>Slightly increased</td>
<td>Greatly increased</td>
<td>0</td>
</tr>
<tr>
<td>Relative risk</td>
<td>18</td>
<td>2</td>
<td>Up to 90</td>
<td>Increased</td>
</tr>
<tr>
<td>Other features</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>HP-related gastritis.
Complications of *H. pylori* gastritis: Frequency

- Lymphoma: 0.1%
- Duodenal ulcer: 13%
- Carcinoma: 1%
Figure 1: Multistage development of gastric MALT lymphoma. H. pylori infection stimulates the production of lymphoid infiltrates, which leads to the formation of acquired mucosa-associated lymphoid tissue (MALT) in the gastric mucosa. As a result of both direct and indirect immunological stimuli (by anti-antigens and Helicobacter pylori-specific T cells, respectively), infiltrating B-cells actively proliferate and occasionally undergo malignant transformation because of the acquisition of genetic abnormalities. Lymphomas with t(11:18) (q21; p15) and t(11:14) (q21; q32) gain autocrine growth ability and do not respond to H. pylori eradication, but may evolve into high-grade tumors. Lymphomas with t(11:14) (q21; q32) or t(11;18) are probably H. pylori-independent and might undergo high-grade transformation. MALT lymphomas without these chromosomal translocations, sometimes carrying translocations of chromosomes 3, 12, and 16, are H. pylori-dependent at early stages and can be effectively treated by H. pylori eradication. However, they can progress to become H. pylori-independent, and transformation into high-grade tumors following the activation of the tumour suppressor genes TP53 and CDH1.
Fig. 2.03 Pathogenetic pathways operative in the evolution of oesophageal and gastric carcinoma. Intestinal metaplasia is a common precursor lesion that may result from gastro-oesophageal reflux disease (GERD) or chronic H. pylori infection.
**Helicobacter Heilmannii Gastritis**

Complications

- MALT lymphoma: 7/202
- Gastric cancer: 1/51 and 1/202
- Ulcers: 2/302 non *Hp* ulcers
- Coinfection with *H. pylori*: 1.6%
“Chemical” Gastritis
(type C gastritis, reactive gastropathy)

NSAIDS-related
Duodenal reflux-related

Foveolar hyperplasia
Mucosal edema and fibrosis
Mild chronic inflammation
Conditions which may demonstrate the changes of reactive gastropathy
Duodenogastric reflux
Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs)
Alcohol
Vascular disturbances
    shock, ischemia, stress,
Local trauma (nasogastric tubes)
Radiation and chemotherapy
Idiopathic
Gastric Polyps

- Hyperplastic polyp
- Fundic gland polyp
- Adenomatous polyp
- Inflammatory fibroid polyp
Epithelial tumours

Intraepithelial neoplasia – Adenoma

Carcinoma

Adenocarcinoma
  intestinal type
  diffuse type
Papillary adenocarcinoma
Tubular adenocarcinoma
Mucinous adenocarcinoma
Signet-ring cell carcinoma
Adenosquamous carcinoma
Squamous cell carcinoma
Small cell carcinoma
Undifferentiated carcinoma
Others

Carcinoid (well differentiated endocrine neoplasm)
**Fig. 3.01** Worldwide annual incidence (per 100,000) of stomach cancer in males. Numbers on the map indicate regional average values.

**Fig. 3.02** The mortality of stomach cancer is decreasing worldwide, including countries with a high disease burden.
Fig. 2.02 Incidence of adenocarcinoma of the stomach (left) compared to adenocarcinoma of the distal oesophagus and oesophago gastric junction (right). Rate per 10,000 hospitalisations from North America.
**Table 17-4. MAJOR FEATURES OF LAURENS’ CLASSIFICATION OF GASTRIC CARCINOMA**

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>Intestinal</th>
<th>Diffuse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most common gross configuration</td>
<td>Polypoid; fungating</td>
<td>Ulcerative; infiltrating</td>
</tr>
<tr>
<td>Microscopic features</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Differentiation</td>
<td>Well-differentiated; gland-forming</td>
<td>Poorly differentiated; signet-ring cells</td>
</tr>
<tr>
<td>Mucin production</td>
<td>Limited; confined to gland lumens</td>
<td>Extensive; may be prominent in stroma around glands (“colloid” carcinoma)</td>
</tr>
<tr>
<td>Growth pattern</td>
<td>Expansile; inflammation often prominent</td>
<td>Noncohesive; infiltrative</td>
</tr>
<tr>
<td>Association with intestinal metaplasia</td>
<td>Almost universal</td>
<td>Less frequent</td>
</tr>
<tr>
<td>Clinical features</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>55</td>
<td>48</td>
</tr>
<tr>
<td>Sex ratio (M:F)</td>
<td>2:1</td>
<td>approximately 1:1</td>
</tr>
<tr>
<td>Decreasing incidence in Western countries</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>


**Fig. 3.04** Growth features of early gastric carcinoma.
Table 3.02
Histological classification of endocrine neoplasms of the stomach

1. Carcinoid – well differentiated endocrine neoplasm
   1.1 ECL-cell carcinoid
   1.2 EC-cell, serotonin-producing carcinoid
   1.3 G-cell, gastrin-producing tumour
   1.4 Others

2. Small cell carcinoma – poorly differentiated endocrine neoplasm

3. Tumour-like lesions
   Hyperplasia
   Dysplasia

\[1\] Benign behaviour of ECL-cell carcinoid is associated with the following: tumour confined to mucosa-submucosa, nonangioinvasive, < 1cm in size, nonfunctioning, occurring in CAG or MEN-1/ ZES. Aggressive behaviour of ECL-cell carcinoid is associated with the following: tumour invades muscularis propria of beyond, > 1cm in size, angioinvasive, functioning, and sporadic occurrence.
### Non-epithelial tumours

<table>
<thead>
<tr>
<th>Tumour</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leiomyoma</td>
<td>8890/0</td>
</tr>
<tr>
<td>Schwannoma</td>
<td>9560/0</td>
</tr>
<tr>
<td>Granular cell tumour</td>
<td>9580/0</td>
</tr>
<tr>
<td>Glomus tumour</td>
<td>8711/0</td>
</tr>
<tr>
<td>Leiomyosarcoma</td>
<td>8890/3</td>
</tr>
<tr>
<td>GI stromal tumour</td>
<td>8936/1</td>
</tr>
<tr>
<td>benign</td>
<td>8936/0</td>
</tr>
<tr>
<td>uncertain malignant potential</td>
<td>8936/1</td>
</tr>
<tr>
<td>malignant</td>
<td>8936/3</td>
</tr>
<tr>
<td>Kaposi sarcoma</td>
<td>9140/3</td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
</tbody>
</table>

### Malignant lymphomas

<table>
<thead>
<tr>
<th>Type</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal zone B-cell lymphoma of MALT-type</td>
<td>9699/3</td>
</tr>
<tr>
<td>Mantle cell lymphoma</td>
<td>9673/4</td>
</tr>
<tr>
<td>Diffuse large B-cell lymphoma</td>
<td>9680/3</td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
</tbody>
</table>

### Secondary tumours