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
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.
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

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
Complications of *H. pylori* gastritis: Frequency

- Lymphoma: 0.1%
- Duodenal ulcer: 13%
- Carcinoma: 1%
**Helicobacter Heilmannii Gastritis**

**Complications**

- MALT lymphoma: 7/202
- Gastric cancer: 1/51 and 1/202
- Ulcers: 2/302 non *H. pylori* 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
Gastric Polyps

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

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 (endoscopic tubes)
- Radiation and chemotherapy
- Idiopathic
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 neoplasms)

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. 3.42: Incidence of adenocarcinomas of the stomach (left) compared to adenocarcinomas of the distal esophagus and esophagogastric junction (right). Rate per 10,000 hospitalizations from North America.
Table 17.4. MAJOR FEATURES OF LAURENS' CLASSIFICATION OF GASTRIC CARCINOMA

<table>
<thead>
<tr>
<th>TYPE OF CARCINOMA</th>
<th>Infiltrative</th>
<th>Diffuse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most common gross configuration</td>
<td>Polypoid; fungating</td>
<td>Ulcerative; invading</td>
</tr>
<tr>
<td>Microscopic features</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</td>
<td>Extensive; may be prominent in stroma around glands (&quot;colloid&quot; carcinoma)</td>
</tr>
<tr>
<td>Growth pattern</td>
<td>Exophytic; inflammation often prominent</td>
<td>Nodular; noncohesive; infiltrative; less frequent</td>
</tr>
<tr>
<td>Association with intestinal metaplasia</td>
<td>Almost universal</td>
<td>Less frequent</td>
</tr>
<tr>
<td>Clinical features</td>
<td>Mean age (years)</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>Sex ratio (M:F)</td>
<td>2:1</td>
</tr>
<tr>
<td></td>
<td>Decalcifying incidence in Western countries</td>
<td>Yes</td>
</tr>
</tbody>
</table>


Table 3.1D. Histological classification of endocrine neoplasms of the stomach

1. Carcinoid - well-differentiated endocrine neoplasm
   11. EC-cell carcinoid
   12. EC-cell, serotonin-producing
   13. G-cell, gastrin-producing tumor
   14. Others

2. Small cell carcinoma - poorly differentiated endocrine neoplasm

3. Tumor-like lesions
   Hyperplasia
   Dysplasia

Benign behavior of ECL-cell carcinoma is associated with the following: early development of mesenchymal neoplasms, nonuniversal, but in some nonfamilial cases. Aggressive behavior in DAE or DCL is associated with the following: tumor-related neoplasms, in progress, in epithelial, in progress, in epithelial, in progress, in epithelial, in progress.
### Non-epithelial tumours

<table>
<thead>
<tr>
<th>Tumour Type</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leiomyoma</td>
<td>8995%</td>
</tr>
<tr>
<td>Schwannoma</td>
<td>9550%</td>
</tr>
<tr>
<td>Granular cell tumour</td>
<td>9685%</td>
</tr>
<tr>
<td>Glomus tumour</td>
<td>8712%</td>
</tr>
<tr>
<td>Leiomyosarcoma</td>
<td>8890%</td>
</tr>
<tr>
<td>GI stromal tumour</td>
<td>9636%</td>
</tr>
<tr>
<td>Benign</td>
<td></td>
</tr>
<tr>
<td>Uncertain malignant potential</td>
<td></td>
</tr>
<tr>
<td>Malignant</td>
<td></td>
</tr>
<tr>
<td>Kaposi sarcoma</td>
<td>9143%</td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
</tbody>
</table>

### Malignant lymphomas

<table>
<thead>
<tr>
<th>Type</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal zone B-cell lymphoma of MALT-type</td>
<td>9699%</td>
</tr>
<tr>
<td>Mantle cell lymphoma</td>
<td>9673%</td>
</tr>
<tr>
<td>Diffuse large B-cell lymphoma</td>
<td>9683%</td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
</tbody>
</table>

### Secondary tumours