Gastrointestinal tract pathology 2023-2024

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Stomach

Learning objectives

- Causes of gastritis.
- Types and pathogenesis of chronic gastritis.
- Definition and types of peptic ulcer disease.
- Gross and histological features of chronic peptic ulcer.
- Pathogenesis of CPU

Gastritis

Inflammation of the gastric mucosa.

1) Acute gastritis and gastropathy
2) Chronic gastritis

Acute Gastritis & gastropathy:

- Acute gastritis results from transient mucosal injury
- When neutrophils are present--- **acute gastritis**
- When cell injury and regeneration are present but inflammatory cells are rare or absent--- **gastropathy**

Both acute gastritis and gastropathy may be asymptomatic, or cause nausea, vomiting, epigastric pain & bleeding in severe cases.

Causes of acute gastritis & gastropathy

- Heavy use of NSAIDs especially aspirin
- Excessive alcohol consumption & heavy smoking
- Chemotherapy, uremia, systemic infections
- Severe stress (trauma, burn, surgery)
- Irradiation , suicidal (acid and alkali)

Microscopically: Edema, vascular congestion & neutrophilic infiltration.

- In more sever mucosal damage --- erosion, and purulent exudate.

Chronic Gastritis:

Is the presence of chronic mucosal inflammatory changes leading eventually to mucosal atrophy and intestinal metaplasia.

A. *H. Pylori chronic gastritis* (Antral)

- Spiral-shaped or curved gram negative bacilli

- ❖ The mechanism of gastritis is unclear but may be stimulated by the production of pro-inflammatory cytokines as well as by directly damaging epithelial cells by the liberation of toxins and degrading enzymes.
- ❖ H.Pylori causes Antral gastritis with high acid secretion and increase risk of duodenal& gastric ulcer.
- ❖ In advanced untreated conditions lead to pangastritis associated with multifocal mucosal atrophy, reduced acid secretion, intestinal metaplasia, and increased risk of gastric adenocarcinoma.

B. Autoimmune chronic gastritis (less than10%) (body &Fundus)

- ➢ Autoantibodies against Parietal cell and intrinsic factor result in:

- ✓ Gland destruction and mucosal atrophy
- ✓ Reduced serum pepsinogen I levels
- ✓ Antral endocrine cell hyperplasia and hypergastrinemia
- ✓ Decrease intrinsic factor -- malabsorption of vitamin b12 -- Pernicious anemia.
- ✓ Impaired gastric acid secretion (achlorhydria)
- ✓ In contrast to H. Pylori chronic gastritis, autoimmune chronic gastritis Spares the antrum and induces gastric atrophy

Gross feature are similar in both types:

The mucosa of affected regions is usually hyperemic and has coarse rugae than normal

In long standing cases, the mucosa becomes **thin** and **flat**.

Histological features:

- Infiltration of mucosa by lymphocytes and plasma cells.
- Frequently lymphocytes are aggregate (lymphoid follicle) --- H.pylori.
- Neutrophils (mainly with H.pylori).

- ▪ **Other features:** intestinal metaplasia, atrophy, and dysplasia

Sequelae of chronic gastritis:

- ➢ **H. Pylori chronic gastritis** ---peptic ulcer, adenocarcinoma and lymphoma.
- ➢ **Autoimmune chronic gastritis**--- Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor.

PEPTIC ULCERS

An ulcer is defined as a breach in the mucosa of the alimentary tract that extends into the submucosa or deeper.

Peptic ulcer: ulceration which develops in sites exposed to the action of acid-peptic juices. Although it can occur anywhere in the alimentary tract, it is most common in the duodenum and stomach.

According to the

* Duration * Degree of penetration * Healing

Peptic ulcer is divided into;

1. Acute peptic ulcer. 2. Chronic peptic ulcer

Acute peptic ulceration (Stress-Related Mucosal Disease)

- **Stress ulcers:** critically ill patients with shock, sepsis, & sever trauma.
- Chronic exposure to Aspirin and NSAID.
- Extensive burns **(Curling ulcer)** that usually occur in the proximal duodenum.
- Traumatic or surgical injury to CNS or intracranial hemorrhage **(Cushing ulcer).**

Gross features:

- Usually small (<1cm), round, superficial, single or most often multiple, predominantly gastric but sometimes also duodenal.
- Sharply demarcated with essentially normal adjacent mucosa
- Healing is with complete re-epithelization.

Microscopically:

- There is focal loss of the mucosa and at least part of the submucosa.
- There is no chronic gastritis or scarring.

Chronic peptic ulcer

- Most often associated with H-pylori infection (70% of cases) or NSAID use.

Gross features:

- The gastric antrum and first portion of the duodenum are the commonest sites.
- Ulcer penetrates the submucosa and even muscle
- Most are solitary, round to oval, sharply punched out craters 2 to 4 cm in diameter
- The base of the crater appears clean and smooth.

Histological features 4 layers

1. Necrotic debris
2. Nonspecific acute inflammatory cells
3. Granulation tissue
4. Fibrosis

Pathogenesis

The imbalances between mucosal surface defenses and damaging forces that cause chronic gastritis are also responsible for PUD.

Defense forces

- Mucous layer and bicarbonate secreted by epithelium.
- Regenerative capacity of epithelial cells.
- Elaboration of Prostaglandin
- Mucosal blood flow

Damaging forces

- ↑ amount of HCl secretion (gastric hyperacidity)
- Infection by H. Pylori.
- Use of NSAIDs.
- Smoking &alcohol.
  - **Zollinger Ellison syndrome**: **Gastrinoma**

    Is a tumor of the pancreas or other part of the small intestine associated with excessive secretion of gastrin and hence excess acid secretion causing multiple ulcers in the stomach, duodenum, and even jejunum.

Complications of chronic peptic ulcers:

1- Bleeding

✓ Most frequent complication occurs in 15% to 20% of patients

✓ May be life-threatening and the first indication of an ulcer.

If it involves small blood vessels → cause loss of small amount of occult blood →Iron deficiency anemia.

If it involves large blood vessels → cause Hematemesis or melena

2- Perforation (5%)

- Cause escape of gut contents into the peritoneal cavity → acute peritonitis.

3- Healing by scarring

Contraction of fibrous scar. E.g. Pyloric obstruction

4- Malignant transformation: occurs in less than 1% in gastric ulcer and Never occurs in duodenal ulcer.

THANK YOU