**ZAPOROZHZHIAN STATE MEDICAL UNIVERSITY**
The department of pathological anatomy and forensic medicine with basis of law

# GASTRITIS
# ULCER OF STOMACH AND DUODENUM
# GASTRIC TUMORS

Lecture on pathological anatomy for the 3-rd year students

# GASTRITIS

Classification of gastritis:

1. Acute (erosive/hemorrhagic) gastritis
2. Chronic (non-erosive)
3. Specific forms of gastritis
4. Others (eosinophilic, allergic, granulomatous)

Diagnosis can be established only by:

- at endoscopic observations
- histological evaluation of biopsy specimens

## ACUTE (EROSIVE OR HEMORRHAGIC) GASTRITIS

It is acute reversible disease of gastric mucous.

Reasons of development:

1. Non-steroidal anti-inflammatory drugs (NSAID's)
2. Alcohol abuse
3. Low-flow states (shock)
4. Stress, including illness, trauma, emotional problems
5. Cigarette smoking
6. Uremia
7. Toxic substances
8. Radiation

# Morphological forms of acute gastritis:

1. *Catarrhal (simple)* - edema, hyperemia, lympho- and leucocytes infiltration of mucus shell
2. *Erosive-hemorrhagic* - edema, hyperemia, inflammatory infiltration and formation of erosions (superficial necrosis of mucus shell)
3. *Fibrinous-necrotic* - destruction of mucus shell with formation of fibrinous exudates:
   - croupous (superficial damage)
   - diphteric (deep connection)
4. *Purulent (phlegmonous) pan-gastritis* - at deep immune insufficiency

# MORPHOLOGICAL FEATURES

Types of gastric epithelium damages:

1. localized - involving the acid-secreting mucosa of the fundus and body of the stomach
2. diffuse - all parts of the stomach
3. superficial inflammation not associated with significant hemorrhage or erosions
4. deep - accompanied by focal erosions and hemorrhages.

All variants are marked by:

1. mucosal and sub-mucosal hyperemia
2. edema
3. inflammatory infiltration by lymphocytes, macrophages and neutrophils

Acute gastritis under appropriate circumstances may disappear within days with complete restitution of the normal mucosa.

# Clinical Course

Depending on the severity of the anatomic changes, acute gastritis may be:

- asymptomatic
- may cause epigastric pain, nausea, vomiting
- overt hemorrhagic erosive changes
- may be responsible for massive hematemesis and potentially fatal blood loss

# CHRONIC (NON-EROSIVE) GASTRITIS

Chronic gastritis is characterized by the absence of visible mucosal erosions and by chronic inflammatory changes leading to mucosal (gastric) atrophy and atypical metaplasia.

The epithelial changes may become dysplastic and possibly be transformed into carcinoma.

Asymptomatic, chronic gastritis associated with:

1. Aging
2. Helicobacter (formerly *Campylobacter) pylori*
3. Auto-antibodies (pernicious anemia)
4. Idiopathic
5. Peptic ulcer (gastric and duodenal ulcer)
6. Cigarette smoking
7. Alcohol abuse
8. Gastric carcinoma

# Main variants of chronic gastritis:

*I. Autoimmune chronic gastritis (Type A)* it is related to pernicious anemia, involves mainly fundus and body of the stomach, characterized by high level of stomach cancer development.

*II. Chronic superficial gastritis (Type B)* - more common than type A, it is of non-immune origin, associated with H.pylori, and has been further subdivided:

1. "Hypersecretory" antral gastritis, with its elevated levels of gastric acid and pepsin, is related to duodenal ulcer disease.

2. "Environmental" gastritis, which is multi-focal (type AB) and often involves multiple regions of the stomach, is associated with gastric ulcer, atypical metaplasia, and carcinoma.

## Main variants of chronic gastritis:

*III. Chronic atrophic pan-gastritis (Type B)* – it is characterised by:

- atrophy of mucus,
- intestine metaplasia of stomach mucus – replacement of secretory type of stomach mucus epithelium into adsorption type of intestine epithelium

*IV. Reflux gastritis (Type C)* it is the inflammation of mucus shell of gastric antrum at duodenum-stomach reflux because of violation stomach and/or duodenum peristalsis

# MORPHOLOGY

1. The inflammatory changes may be limited to the superficial zone or may extend throughout the mucosa

2. The inflammation is accompanied by variable gland loss and mucosal atrophy. In the fundic autoimmune variant (type A), there is particularly prominent loss of parietal cells, owing to antibodies targeted on these cells and intrinsic factor.

3.There are no erosions in any form of chronic gastritis, but the surface of epithelium may undergo intestinal metaplasia and in some instances atypical metaplasia, accounting presumably for the increased incidence of gastric carcinoma.

**Clinical Course**:

- nausea, vomiting
- upper abdominal discomfort are uncommon.

# STRESS ULCERS

## Reasons of development:

- Severe trauma including major surgical procedures, serious sepsis, or grave illness of any type
- Extensive burns (referred to as Curling's ulcers)
- Traumatic or surgical injury to the central nervous system or an intra-cerebral hemorrhage (called Cushing's ulcers)
- Long-term use of gastric irritant drugs (aspirin, NSAIDs and corticosteroids).

# MORPHOLOGY

Macroscopically, stress ulcers are small, round to ovoid, sometimes irregular, mucosal defects that initially involve only the proximal portion of the gastric mucosa then may extend throughout the entire stomach. They rarely exceed 2.5cm in diameter, usually have a brown base of digested blood, and only exceptionally penetrate more deeply than the muscularis mucosa.

Histologically, the lesions appear to result from enzymatic digestion with only a mild to moderate inflammatory infiltration. In some cases, particularly in association with burns and head lesions, stress ulcers may extend into the duodenum and in this location are more likely to be deep or penetrating.

Clinical Course. More often stress ulcers are totally silent and come to clinical attention only when they cause bleeding, which is sometimes massive and then carries more than a 50% risk of death.

# PEPTIC ULCER

- A peptic ulcer can be defined as *"a hole in the mucosa"* of any portion of the gastrointestinal tract exposed to acid-pepsin secretion.
- More often peptic ulcers are located in the first portion of the duodenum or in the stomach in a ratio of about 4:1.
- The great majority of individuals have a single ulcer, only in certain families and in the Zollinger-Ellison syndrome - ulcers in the stomach and duodenum.
- Despite a remarkably uniform morphology, gastric ulcers and duodenal ulcers may well constitute different diseases, as will become apparent.

# Predisposition factors of ulcer formation

1. There are hints of genetic susceptibility to duodenal ulcers (DU) but not to gastric ulcers (GU).
2. A positive family history of DU
3. In monozygotic twins (not in di-zygotic twins).
4. The familial syndromes associated with DU, such as the autosomal dominant hyper-pepsinogenemia-I.
5. Persons with blood group O have a greater risk for DU than those who have other blood groups.
6. Acquired disease may also predispose to DU:
   - alcoholic cirrhosis
   - chronic obstructive pulmonary disease
   - chronic renal failure
   - hyper-parathyroidism

# MORPHOLOGY

All peptic ulcers are usually:

- round form - 2 to 4cm in diameter
- sharply punched out defects in the mucosa that can penetrate into the submucosa, usually into the muscularis and sometimes more deeply

Favored sites - are the anterior and posterior walls of the first portion of the duodenum and the lesser curvature of the stomach.

Usually it involves the entire antrum with the ulcer crater in the margin of the affected area close to the adjacent acid-secreting fundic mucosa, but it may be more proximal or distal.

The histological appearance varies according to activity, chronicity and degree of healing.

During the active phase, four zones (layers) of changes can be distinguished in epithelium:

1) the base and margins have a thin layer of necrotic fibrinoid debris - N
2) a zone of active nonspecific inflammatory infiltration with neutrophils - I
3) active granulation tissue - G
4) fibrous, collagenous scar that fans out widely from the margins of the ulcer - S

# COMPLICATIONS OF PEPTIC ULCER DISEASE

*I. Ulcer-destructive:*

1. Bleeding - Occurs in 25 > 33% of patients
   - Most frequent complication; may be massive
   - Accounts for about 25% of ulcer deaths
   - May be first indication of presence of ulcer
2. Perforation - Occurs in only 5% of patients
   - Accounts for $^{2}/_{3}$ of all ulcer deaths
   - Rarely, is first indication of ulcer. Leads to peritonitis
3. Penetration – ulcer bottom is in the underlying organs (pancreas, fat tissue) with zones of destruction

# COMPLICATIONS OF PEPTIC ULCER DISEASE

*II. Inflammatory:*

1. Perigastritis and/or periduodenitis
2. Phlegmon of stomach

*III. Ulcer-scarring complications with stenosis*

1. Stenosis or obstruction from edema or scarring of pyloric canal or duodenum

   Causes incapacitating abdominal pain

   Rarely, may lead to total obstruction with intractable vomiting, intractable pain
2. Deformations

*IV. Malignisation of ulcer* - transformation into malignant tumor

# TUMORS

I Mesenchymal neoplasms:

1. Bening - stromal cell tumors, leiomyomas, leiomyoblastomas, neurofibromas, and lipomas
2. Malignant - sarcomas

II Epitelial neoplasms:

1. Bening – polyps, adenomas
2. Malignant - gastric carcinoma

III Carcinoids

IV Gastrointestinal lymphomas

# GASTRIC POLYPS

It is nodule or mass that projects above the level of the surrounding mucosa.

In the stomach, these lesions can be subdivided into:

1) hyperplastic polyps
2) adenomatous polyps

# GASTRIC CARCINOMA

Morphologic types:

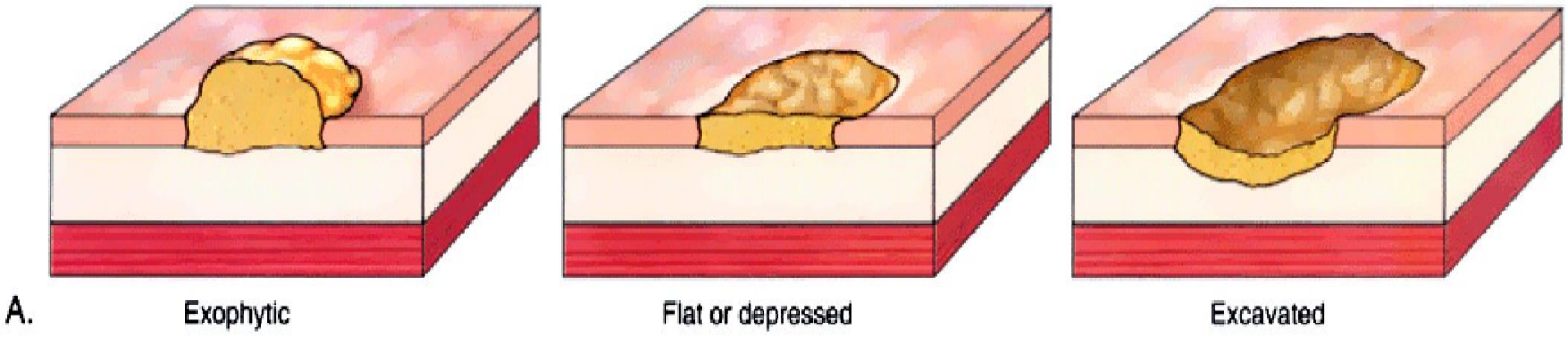
1. intestinal - arise from gastric mucous cells that have undergone metaplasia into intestinal type cells, occurs primarily after age 50 years in 2:1 male predominance

2. diffuse - arise de novo from native gastric mucous cells, occurs at an earlier age with no male predominance.

# MORPHOLOGY

*1. Early gastric carcinoma (EGC)* constitutes a lesion confined to the mucosa and sub-mucosa, regardless of the presence or absence of peri-gastric lymph node metastases. The tumor is confined to the mucosa and sub-mucosa and may exhibit an exophytic, flat or depressed, or excavated conformation

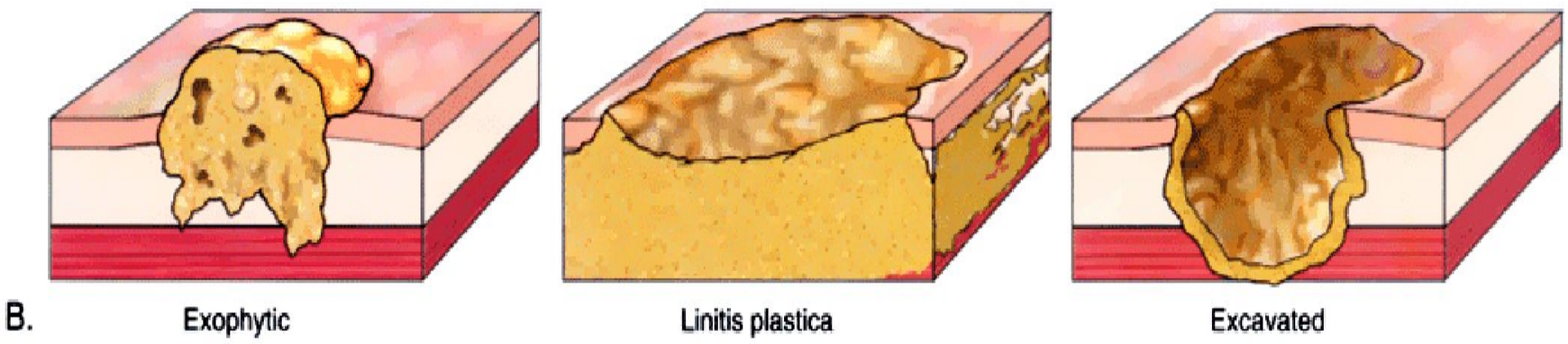
*Anatomic forms of EGC:*



# MORPHOLOGY

*2. Advanced gastric carcinoma (AGC)* is a neoplasm that has extended below the sub-mucosa, into the muscularis, and has spread more widely - extends into the muscularis propria and beyond. Linitis plastica is an extreme form of flat or depressed advanced gastric carcinoma. Most arise in the gastric antrum.

*Anatomic forms of AGC:*



# Features of tumor propagations

All gastric carcinomas eventually penetrate the wall to involve the serosa and may spread to regional and more distant lymph nodes and liver.

Two patterns of spread are particularly distinctive. Gastric carcinomas frequently metastasize to the *supraclavicular sentinel (Virchow's) node* as the first clinical manifestation of an occult neoplasm.

More uncommonly, these cancers metastasize to one or both ovaries to cause solid tumorous enlargements - *Krukenberg tumors*.