Inflammation of the stomach

Inflammation of the stomach (gastritis) is defined as inflammation of the gastric mucosa. On the mucosal surface it appears as catarrhal inflammations, in the mucosal stroma it appears as lymphoplasmocellular inflammations. Gastritis can be **acute or chronic**, which are more common.

Acute gastritis

Acute gastritis is defined as an acute inflammatory process that manifests itself in various forms of gastric mucosal involvement, from hyperemia to **erosions and ulcers**. These alterations can be the cause of acute gastrointestinal bleeding. Acute inflammation of the stomach has a transient character and its incidence is considerably lower than that of chronic inflammation.

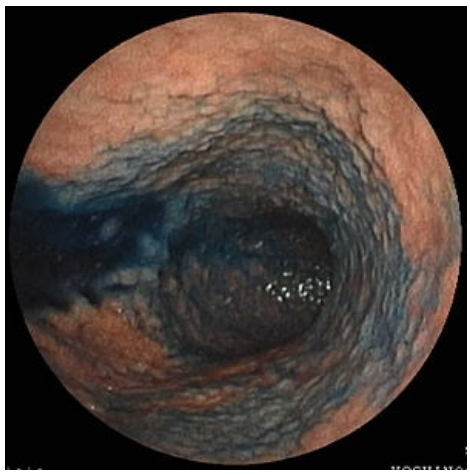
Pathogenesis

The mechanism of acute inflammation of the stomach is not completely clear, the following factors are used in the etiology:

- large doses of nonsteroidal anti-inflammatory drugs, such as aspirin;
- excessive alcohol consumption;
- smoking;
- cytostatics;
- systemic infections (eg salmonellosis);
- uremia;
- severe stress conditions (trauma, burns, conditions after surgical operations);
- ischemic injury and shock;
- ionizing radiation;
- mechanical trauma (eg, nasogastric intubation);
- objectionable food products;
- inappropriate diet (especially animal fats, spices).

Morfology

Acute gastritis can be localized or diffuse and macroscopically it appears from simple congestion of the mucous membrane to the formation of erosions. Hemorrhage and erosion can be observed with an endoscope, this condition is known as acute erosive gastritis. Microscopically, the mucosa is congested, edematous, the stroma is inflammatory infiltrated mainly by **neutrophils**, but there may also be lymphocytes. Epithelial regeneration is evident in the gastric pits. If the cause disappears, acute gastritis heals within a few days with complete restoration of the gastric epithelium. If the cause persists, the inflammation can become chronic.



Endoscopic image of nodular gastritis

Clinical picture

Acute gastritis can be completely **asymptomatic** or with **only mild symptoms**, such as nausea, pain, vomiting or hematemesis, melena, but also **fatal blood loss.** Acute gastritis is one of the main causes of hematemesis, especially in alcoholics.

Therapy

Therapy is symptomatic. It consists in dietary measures, rehydration, gradual realimentation. Analgesics with an antispasmodic effect can be given for colicky abdominal pain (for example, a mixture of Metamizole-pitofenone-fenpiverine, butylscopolamin); in case of nausea, antiemetic (metaclopramid); antidiarrheal for diarrhea (chloroxin), possibly probiotics.

Chronic gastritis

Chronic gastritis is defined as the presence of **chronic inflammatory changes of the mucosa**, which can result in mucosal atrophy and epithelial metaplasia. Erosions are usually not present, as in acute gastritis. It is noteworthy that different types of histological variants of chronic gastritis typically occur in different parts of the world.

Pathogenesis

The most important etiological association is Helicobacter pylori infection. This organism is a common pathogen with the highest prevalence in developing countries. In areas where the infection is endemic, persistent carriage is proven. Most carriers suffer from associated asymptomatic gastritis.

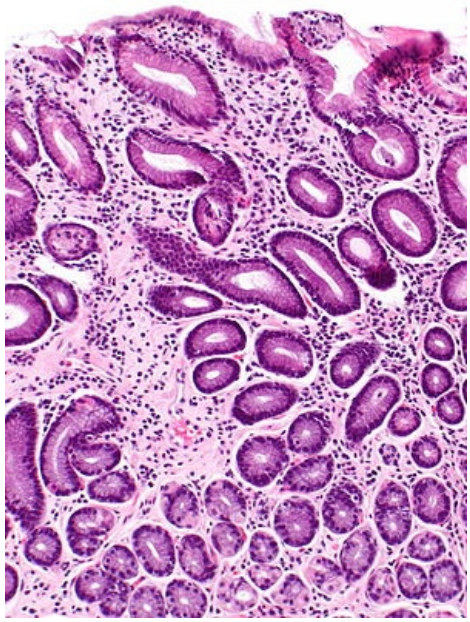
Types of chronic gastritis

From a morphological point of view, it is possible to distinguish between: **superficial or deep gastritis** - the distinction is made according to the level of the mucosal tissue to which the lymphoplasmocellular infiltrate extends, superficial gastritis is the mildest form of chronic gastritis, in which small alterations of the mucosa occur; **atrophic gastritis with loss of gastric glands** - partial or complete - the most severe form of atrophic gastritis, in which the cells of the gastric glands are replaced by mucus cells; **atrophic gastritis with intestinal metaplasia** - replacement of the gastric epithelium with intestinal mucosa with goblet, Paneth cells and enterocytes - can be complete or incomplete (precancer).

From the etiological point of view, it is possible to distinguish:

- **type A - autoimmune** (antibodies against parietal cells and intrinsic factor) - affects the body

A rarer form of chronic gastritis is autoimmune gastritis, the cause of which is the **immunotoxic effect of lymphocytes** damaging the parietal cells, and the production of **antibodies against the parietal cells** of the gastric mucosa. Autoimmune damage leads to destruction and atrophy of the gastric epithelium with concomitant **loss of hydrochloric acid and intrinsic factor** produced by parietal cells. Intrinsic factor deficiency can then result in pernicious anemia and achlorhydria. We find hypergastrinemia. This type of gastritis typically affects the body of the stomach, where the main and parietal cells are located. The autoimmune type of gastritis is found in areas of Scandinavia and is associated with other diseases of an autoimmune nature such as Hashimoto's thyroiditis and Addison's disease.



Intestinal metaplasia

- **type B – bacterial** (Helicobacter pylori infection) – affects the antrum

This type of chronic gastritis arises as a result of the action of the enzymes of the colonizing bacteria H. pylori and the release of harmful chemical compounds by neutrophil granulocytes. The mucosal damage itself is of two types. On the one hand, it is a high production of hydrochloric acid, which poses a risk of developing a duodenal ulcer, on the other hand, it affects the body and antrum (pangastritis) with mucosal atrophy of the epithelium, low production of hydrochloric acid and a high risk of developing adenocarcinoma.

Morphology

Regardless of the cause and type of chronic gastritis, microscopic inflammatory changes of the mucosa represent a lymphoplasmacytic infiltrate in the lamina propria, occasionally accompanied by the presence of neutrophils. Inflammation can be accompanied by **loss of glands and mucosal atrophy**, when the mucosa has a smooth surface. In these cases, we detect the H. pylori bacteria nestled in the gastric mucus. In the autoimmune type of gastritis, the most prominent feature is the **loss of parietal cells. Intestinal metaplasia** means the replacement of gastric epithelium with intestinal mucosa with goblet, Paneth cells and enterocytes. Later, dysplasia of the epithelium may occur, until the transition to malignant adenocarcinoma.

Clinical picture

Chronic gastritis usually appears **asymptomatic** or with only **mild symptoms** such as pain in the stomach area, nausea and vomiting. If there is a large loss of parietal cells in the autoimmune type of chronic gastritis, hypochlorhydria or achlorhydria occurs, as well as hypergastrinemia. Patients with chronic gastritis due to other causes may be hypochlorhydric, but because not all of their parietal cells have been destroyed, they may not develop achlorhydria or pernicious anemia. Chronic gastritis can be associated with the development of peptic ulcer and adenocarcinoma of the stomach. H. pylori infection has been demonstrated in most patients who have a peptic ulcer. For these patients, the risk of gastric adenocarcinoma is up to five times greater than in the normal population. In the autoimmune type of chronic gastritis, the risk of adenocarcinoma is between 2 and 4%, which is slightly above the average for the normal population.

Therapy

When Helicobacter pylori eradication is proven, especially in the presence of an ulcer. Further therapy is mainly symptomatic. There is no known cure for autoimmune gastritis, vitamin B12 replacement is recommended.

Links

related articles

- Chronic gastritis (preparation)
- Gastroduodenal ulcer disease
- Crohn's disease

- Ulcerative colitis
- Sudden abdominal events

sources

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