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The Pathogenesis, Diagnosis, Treatment, and Etiology of Acute and Chronic Gastritis

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Annotation: These days, inflammation of the stomach mucosa is one of the symptoms of the disease known as gastritis. The author of this essay focuses particularly on the fact that inflammation in gastritis is frequently caused by infection with the same bacterium that causes the majority of stomach ulcers. The causes of this illness are examined in detail in this text, with the most prevalent being excessive use of painkillers, alcohol, and an unbalanced diet. A more thorough examination of the diseased process's clinical expression holds the promise for future research.

Keywords: gastritis, gastrointestinal tract, mucous membrane, extravasation.

Gastritis is characterized by a wide range of inflammatory, regenerative and degenerative lesions, dependent and correlating with each other. Researchers of this pathology classify gastritis as A, B, AB, C. Gastritis includes several groups of nosological conditions that can be divided into three main types - acute, chronic and atypical forms [1].

Etiology. The disease can be triggered by exogenous factors (alcohol, salicylates, corticosteroids, chemicals), as well as endogenous ones (burns, injuries, stress, bacterial toxins). Sometimes the cause of the disease cannot be determined, and sometimes complaints may be associated with the early phase of Helicobacter pylori (H.p.) infection.

Acute gastritis is a short-term inflammatory process with a sudden onset and subsidence after a short time without any traces, the symptoms of which, for example, epigastric pain, nausea and vomiting, are transient [2].

Thus, only a few patients suffering from this form of the disease undergo endoscopic examination. Signs of gastritis are also rarely found in conventional biopsy specimens.

The types of acute gastritis can be distinguished:

- Mild acute gastritis, characterized by generalized swelling of the gastric mucosa, stenosis of the prepyloric area without visible erosions and other lesions, often associated with H.p. infection [3].
- Acute hemorrhagic gastritis, characterized by numerous extravasations, hemorrhagic spots and erosion.
- Acute ulcerative gastritis with extensive erosions or ulcerations and bleeding.

Chronic gastritis is the most common chronic inflammatory disease affecting the stomach. Factors influencing the occurrence and development of chronic gastritis can be divided into exogenous (environmental), constitutional and immune. The first group includes repeated exposure to factors that cause acute gastritis, such as alcohol, smoking, spices, drinks, medications, frequent thermal injuries, bacterial (Helicobacter pylori, Gastrospirilium hominis) and fungal pathogens [4]. Constitutional factors include age, hereditary characteristics, hormonal factors, and systemic diseases. Immune phenomena include tissue hypersensitivity and humoral mechanisms.

Pathogenesis. As a result of the influence of endogenous and exogenous damaging factors, aggression factors, such as leukotrienes, free oxide radicals and lysosomal enzymes are activated and released by the gastric mucosa. Some authors suggest that immune reactions in the gastric mucosa precede the appearance of gastric lesions detected by histological examination [5]. In allergy sufferers, an anaphylactic reaction of the gastric mucosa occurs due to contact with allergens. The amount of penetrating allergen entering the gastrointestinal tract determines the intensity of the local allergic reaction and its organic consequences, for example, such as periodically formed erosions and ulcers of the gastric mucosa. Chronic exposure to allergens reaching the stomach may initiate or maintain chronic inflammatory condition of the mucous membrane and cause persistent painful and dyspeptic symptoms [6]. The transition from the acute, most often clinically asymptomatic phase, to the chronic phase may be the result of ineffective defense mechanisms of the human body, and also depends on the production of numerous enzymes and cytotoxins Helicobacter pylori.

Diagnostics. The use of endoscopy in gastroenterological diagnostics was a real breakthrough, which made it possible to more accurately diagnose many diseases of the gastrointestinal tract. Endoscopic studies in combination with the collection of biopsies provide information about the condition of the gastric mucosa [7]. Currently, biopsy is considered important for diagnosis because, based on the literature, 27% of patients have a suspected gastritis had normal histological results, while about 63% of patients with histological results, indicating gastritis, did not have signs of the disease detected by endoscopy [8].

Treatment. The goal of treatment is to reduce symptoms and accelerate the healing of mucosal lesions, as well as to prevent possible complications such as peptic ulcer disease, ulceration and its consequences [9]. In pharmacological therapy, 3 groups of drugs are used (similar to the treatment of peptic ulcers).

1. Inhibitors of gastric secretion of hydrochloric acid, including:

- ✓ H2 receptor blockers (ranitidine, cimetidine, famotidine).
- ✓ Proton pump inhibitors (omeprazole, pantoprazole, lansoprazole).

2. Agents that neutralize hydrochloric acid, alkaline preparations containing mainly aluminum or magnesium (Alumag, Aflomag, Malox).

3. Colloidal compounds (Ventrisol, DeNol), forming a protective film and causing an increase in the level of endogenous prostaglandin. Used in combination with antibiotics and metronidazole: sucralfate (Venter, Ulcogant, Ulgastran, Ankrusal, Gastorem) exhibits cytoprotective activity due to the formation of an aluminum-protein complex that adheres to the surface of the mucous membrane affected by inflammation or to an ulcer, protecting it from the access of hydrochloric

acid. The principle of treatment with these drugs is to take them at a time when the acidity of the gastric contents increases, i.e. an hour after eating and before bed.

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