JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS
VOLUME 5, ISSUE 02
MOPNTHLY JOURNALS
ISSN NUMBER: 2751-4390
IMPACT FACTOR: 9,08

HISTOMORPHOLOGICAL FEATURES OF PRECANCEROUS DISEASES OF THE STOMACH

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Abstract: Stomach cancer occupies one of the leading places in the structure of the incidence of malignant neoplasms and ranks 3rd in mortality. This pathology has geographical heterogeneity, among the leaders are East Asian countries, the regions with the lowest incidence include North America and Western Europe.

The frequency and distribution of histological types of gastric cancer vary depending on the region. Over the past 50 years, morbidity and mortality have decreased worldwide, especially in developed countries, primarily the number of intestinal adenocarcinomas has decreased.

Despite the high incidence in Asian countries, the 5-year survival rate is 64.2%, in North America and Europe — 2.7% and 8.5%, respectively.

Despite the fact that there is a downward trend in the incidence of stomach cancer, mortality remains at a consistently high level, with about 700,000 deaths recorded annually.

Keywords: sign histomorphological features of precancerous diseases of the stomach forms of histomorphological features of precancerous diseases of the stomach types of histomorphological features of precancerous diseases of the stomach

The precancerous cascade of Correa.

The so-called Correa cascade reflects the pathogenesis of intestinal adenocarcinoma against the background of chronic helicobacter gastritis followed by atrophic gastritis, intestinal metaplasia and dysplasia.

The oncoprotein CagA plays the most significant role in the pathogenesis of chylicobacter-associated gastritis with further transformation of the mucous membrane into adenocarcinoma. Penetrating into the cytoplasm of cells of the gastric epithelium, it leads to destabilization of intercellular connections, activates oncogenic signaling pathways, but the mechanism has not yet been fully studied.

In 2018, it was found that the CagA protein inhibits p14ARF, which is a suppressor oncogene, and its reduced expression eventually leads to the inhibition of p53-independent autophagy.

For clarity, the Correa cascade is shown in the following micrographs. The chronology of events in the cascade corresponds to the deepening of morphological changes during carcinogenesis.

1. The mucous membrane of the antrum of the stomach of the usual histological structure.

Disparate mononuclears present in their own plate surrounding glandular structures. Staining with hematoxylin and eosin, ×20.

2. Superficial non-reactive moderate gastritis.

The mucous membrane of the antrum of the stomach with well-preserved glands and abundant mononuclear leukocyte infiltration of its own plate. Staining with hematoxylin and eosin, ×20.

The morphologically unchanged gastric mucosa contains a small number of scattered mononuclear inflammatory cells. In gastritis, its own plasticity is infiltrated by mononuclear lymphocytes (responsible for chronic inflammation), as well as polymorphonuclear neurophils (acute inflammation), most often this process occurs due to a helicobacter infection.

Helicobacter gastritis through immune cells leads to the production of pro-inflammatory cytokines: tumor necrosis factor (TNF), interferon γ, interleukin (IL) 1b, in some cases IL-4 and IL-5 are expressed. Inflammation of the mucous membrane due to the combined interaction of factors associated with the trigger, as a rule, CagA-positive strain H. pylori, hereditary features of the macroorganism, environmental factors, dichotomously leads either to chronic non-atrophic gastritis, which can persist for a long time, or to the progression of the severity of the disease, leading to chronic atrophic gastritis. A decrease in the number and disappearance of glands are the first, earliest manifestations of the precancerous condition of the gastric mucosa. Atrophy fields are more often present in the body and antrum, with time the loss of glands becomes smaller, and the area of the affected area increases. Another variant of the development of atrophic gastritis is autoimmune gastritis type A, which is an integral part of the syndrome of pernicious anemia. In this pathology, the antrum does not undergo atrophy, while in non—autoimmune atrophic gastritis, this particular part of the stomach is most often affected.

3. Microscopic picture of chronic atrophic gastritis.

The mucous membrane of the antrum is characterized by leukocyte infiltration of its own plate and loss of glandular structures, which are replaced by fibrous tissue. Staining with hematoxylin and eosin, ×20.

The next stage is intestinal metaplasia, which is the earliest phenotypic expression of oncotransformation of the gastric mucosa (Fig. 4). In this condition, not only the nature of the mucus produced changes, but also its pH, as acidic mucins are secreted during intestinal metaplasia.

4. The mucous membrane of the fundus of the stomach with intestinal metaplasia.

Intestinal-type gastrocytes have been replaced, and eosinophilic absorbent enterocytes with a brush border are visible. Staining with hematoxylin and eosin, ×20.

As the changes progress, sulfated acidic mucins begin to be secreted in some areas of the morphologically altered mucosa, such as those produced by the glands of the large intestine, and the set of enzymes characteristic of gastrocytes partially or completely disappears. Transformed cells of the gastric mucosa do not have a brush border, as do enterocytes of the colon. The following types of intestinal metaplasia of the gastric mucosa are distinguished: complete (the presence of goblet cells with sialomucin, Pannet cells, absorbing cells) and incomplete (goblet cells and columnar cells with sialomucin or sulfomucin in type III intestinal metaplasia).

Dysplasia or noninvasive neoplasia is characterized by deep-seated changes, an enlarged hyperchromic nucleus, and an increased frequency of mitosis. Histoarchitectonics is no longer preserved: the glands are irregularly shaped and branching. The next stage of the cascade will be intestinal adenocarcinoma.

5. The mucous membrane of the fundus of the stomach with low-grade intraepithelial dysplasia.

Dysplastic epithelial cells have large hyperchromic nuclei combined with a loss of polarity with respect to the basement membrane. Staining with hematoxylin and eosin, ×20.

Current morphological classifications of gastric cancer

Currently, the Lauren classification is most widely used, with some additions and changes. In the classification proposed by the author, depending on the histological phenotype, the following morphological types are distinguished: intestinal, diffuse, intermediate. The intestinal type is characterized by distinct glandular structures consisting of highly differentiated cylindrical cellular elements with a developed brush border. Intestinal adenocarcinomas are the most common type with a frequency of about 55%. The diffuse type consists of poorly organized (discogesive) cell groups or scattered ring-shaped cells with a high content of mucin, occurs in 32% of cases.

With the mixed type, the tumor contains elements of both intestinal and diffuse histological structure.

Thus, adenocarcinomas of the intestinal type (fig. 6) are more associated with environmental factors, while the diffuse type (Fig. 7) is associated with a hereditary predisposition. An interesting fact is that gastric cancer associated with the Epstein—Barr virus (about 10% of cases of intestinal adenocarcinomas according to Lauren) has a high level of microsatellite instability and a more favorable prognosis compared with diffuse and mixed types.

6. Invasive gastric adenocarcinoma, low-grade, intestinal type according to Lauren with areas of vascular invasion, peritumoral lymphoid infiltration.

Tumor cells are tightly located in irregular glandular structures infiltrating the stroma. Staining with hematoxylin and eosin, ×20.

7. Gastric adenocarcinoma, high-grade, diffuse type according to Lauren.

Tumor cells are hypochromic, polymorphic, with a diffuse type of growth without signs of formation of any structures. Staining with hematoxylin and eosin, ×20.

However, other classifications are also used, for example, the WHO classification, 2010: papillary adenocarcinoma; tubular adenocarcinoma (highly differentiated; moderately differentiated; low-differentiated); mucinous adenocarcinoma; ring-shaped cell adenocarcinoma; glandular squamous cell carcinoma; squamous cell carcinoma; carcinosarcoma; choriocarcinoma; undifferentiated cancer.

The Carneiro classification does not take into account the histophenotype of tumor cells, but the immunohistophenotype, as the author divides gastric tumors into glandular, isolated cellular, solid and mixed. The fundamental criterion is the expression of specific mucins (MUC6, MUC5AC), intestinal markers (MUC2, CDX2, CD10), and the gastric peptide marker (TFF1).

Goseki identifies 4 groups of gastric cancer depending on the level of intracellular mucin production (high or low) and tubular differentiation (high or low).

Some molecular mechanisms and signaling pathways of gastric cancer carcinogenesis

Before the phenotypic expression of carcinogenesis, many events occur, which eventually lead to the morphological expression — gastric adenocarcinoma.

Currently, many signaling pathways have been discovered that play a role in the development of gastric cancer.

Y. Hu et al in their study established a decrease in RACK1 expression, which leads to carcinogenesis via the NF-kB signaling pathway. The study showed a decrease in RACK1 in the tumor tissue compared to the morphologically unchanged mucosa.

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