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MORPHOLOGICAL FEATURES OF PRECANCER DISEASES OF THE GASTROINTESTINAL

Annotation: In the article, the authors analyzed the scientific and educational literature on precancerous diseases of the stomach. According to the latest WHO classification (2010), precancerous and early neoplastic lesions of the gastric mucosa include the following pathologies: chronic gastric ulcer, polyps that play a key role, as well as chronic gastritis associated with Helicobacter pylori infection, intestinal metaplasia, intraepithelial neoplasia (dysplasia), adenomas , familial adenomatous polyposis, foveolar hyperplasia, Peutz-Jeghers syndrome. The absence of clear morphological and immunohistochemical criteria for the differential diagnosis of various precancerous conditions indicates the need for further study of this issue.

Keywords: Stomach cancer, Helicobacter pylori, peptic ulcer, polyps.

Annotation: Maqolada muallifar oshqozonning saraton oldi kasalliklariga baghishlangan ilmiy va kuv adabiyotlarini tahlil qildilar. ЖССТнинг сўнгги таснифига кўра (2010), ошқозон шиллиқ қаватининг рак олди холатлар ва эрта неопластик шикастланишлари қуйидаги патологияларни ўз ичига олади: сурункали ошқозон яраси, полиплар, шунингдек Helicobacter pylori инфекцияси сурункали гастрит билан боғлиқ, ичак метаплазияси, интраэпителиал неоплазия (дисплази), аденомалар, oilavius adenomatosis polyposis, foveolar hyperplasia, Peitz-Eegers syndrome. Har hil cancer oldi holatlarni differential diagnosticssi uchun anik morphological va immunohistokimyovy mezonlarning yo'qligi ushbu masalani yanada ўrganish zaurligini kўrsatadi.

Kalit so'zlar: Oshkozon Saratoni, Helicobacter pylori, Oshkozon Yarasi, Polyplar.

According to the International Agency for Research on Cancer (IARC), published on the Globocan Internet resource, there were 4.8 million new cases of gastrointestinal (GI) cancer and 3.4 million associated deaths worldwide. Gastrointestinal cancer accounts for 26% of cancer incidence and 35% of all deaths. Stomach cancer (GC) remains one of the most important malignant diseases worldwide, ranking 5th in the ranking of the most frequently diagnosed and 3rd leading cause of death from cancer. Mortality from gastric cancer is estimated at a ratio of 1 case for every 12 cancer deaths. Despite the decline in the incidence of some gastrointestinal cancers, this group of malignancies continues to pose serious public health problems.

More than 70% of stomach cancer cases occur in developing countries, of which about 50% occur in East Asia. The highest incidence of gastric cancer worldwide is found in Mongolia, Japan and the Republic of Korea, while in North America and Northern Europe, as well as in some regions of Africa, incidence rates are usually low [3, 4, 12, 17].

Comparing the literature data, in men, GC is the most frequently diagnosed cancer and the leading cause of cancer mortality in Asian countries. In Uzbekistan, gastric cancer ranks second among malignant neoplasms; in 2019, the incidence was 5.7% per 100,000 population. The proportion among patients observed for 5 years or more patients with tumors of the stomach was 3.1%. The prevalence among men is 2 times higher than in women [3, 5, 6, 10].

Goal of the work. Based on the study of literature data, to determine the further prospect of research on the possibilities of morphological diagnosis of precancerous diseases of the stomach.

According to the latest WHO classification (2010), precancerous and early neoplastic lesions of the gastric mucosa (GM) include the following pathologies: chronic gastric ulcer, polyps that play a key role, as well as chronic gastritis associated with Helicobacter pylori infection, intestinal metaplasia, intraepithelial neoplasia (dysplasia), adenomas, familial adenomatous polyposis, foveolar hyperplasia, Peutz-Jeghers syndrome. These diseases are precancerous processes of the stomach. According to a number of authors, chronic gastric ulcers, polyps that play a key role, as well as chronic gastritis associated with Helicobacter

pylori infection, intestinal metaplasia, intraepithelial neoplasia (dysplasia) occupy a special place in the development of gastric cancer [2, 8, 13, 14].

The presence of precancerous pathology in the gastric mucosa affects the choice of management tactics for this group of patients, that is, the sequence of application of special diagnostic and treatment methods, followed by organ-preserving operations, which are the basis for assessing the prevalence of the tumor process [4, 6, 15], which determines the further prognosis.

These statistics indicate that the indicators currently not only remain at a high level, but also tend to increase, which necessitates maximum attention in the development of new effective methods for the early and timely detection of background and precancerous diseases of the stomach [1, 3, 6, 16, 18].

Despite the constant innovative progress in medicine, the growth of diagnostic capabilities, the equipping of medical institutions with high-resolution equipment, the development and implementation of new diagnostic and treatment methods into everyday practice, the percentage of detected cases with diseases diagnosed in the early stages of the tumor process remains quite low. [5].

The possibilities and objectives of the morphological diagnosis of precancerous diseases have not yet been finally determined and are largely debatable problems. Apparently, this is largely due to the fact that the concept of precancer is prognostic, and making such a diagnosis for a particular patient may entail therapeutic measures, the justification of which must be fully justified. Nevertheless, it is possible to prevent tumor progression only with early detection of a malignant neoplasm or timely treatment of precancerous diseases, therefore, a particularly important role should be given to the rational formation of cancer risk groups.

Chronic gastritis associated with Helicobacter pylori infection poses a high risk of developing gastric cancer. Many authors in their scientific articles suggest that Helicobacter pylori infection, as well as exposure to certain exogenous factors, may be associated with the initiation of cell cycle disorders and cell differentiation pathology, impaired DNA repair processes and apoptosis, which can lead to the development of gastric carcinoma [5, 8, 13,

16]. Recent studies have shown that Helicobacter pylori infection not only has a characteristic effect on its own GM, but is also a trigger for a number of other systemic lesions. Despite the fact that pathological changes in the gastric mucosa involved in carcinogenesis against the background of infection with various strains of Helicobacter pylori are described quite fully, a more detailed and in-depth study of local changes in this infection of the gastrointestinal tract is required. According to modern concepts, the starting point in the development of gastric cancer is the combination of chronic gastritis with infection with the most pathogenic strains of Helicobacter pylori. In most cases, chronic active gastritis develops against the background of this infection, and with the chronicity of the process and its progression, the development of background and precancerous processes is observed - atrophy of the gastric glands, intestinal metaplasia, dysplasia, and then gastric cancer. These stages of carcinogenesis and morphogenesis are called the "Correa cascade" [2, 7, 14, 15]. combination of chronic gastritis with infection with the most pathogenic strains of Helicobacter pylori. In most cases, chronic active gastritis develops against the background of this infection, and with the chronicity of the process and its progression, the development of background and precancerous processes is observed - atrophy of the gastric glands, intestinal metaplasia, dysplasia, and then gastric cancer. These stages of carcinogenesis and morphogenesis are called the "Correa cascade" [2, 7, 14, 15]. combination of chronic gastritis with infection with the most pathogenic strains of Helicobacter pylori. In most cases, chronic active gastritis develops against the background of this infection, and with the chronicity of the process and its progression, the development of background and precancerous processes is observed - atrophy of the gastric glands, intestinal metaplasia, dysplasia, and then gastric cancer. These stages of carcinogenesis and morphogenesis are called the "Correa cascade" [2, 7, 14, 15].

Helicobacter pylori has its own capsule, consisting of lipopolysaccharides and proteins, which are necessary for the adhesion of microorganisms on the surface of epitheliocytes [4, 5], which affects the duration and degree of the inflammatory process in the gastric mucosa. Persistence in the gastric mucosa of Helicobacter pylori, of course, determines the severity of the inflammatory process with the transition to its own muscle plate, which includes

lymphocytes, neutrophilic leukocytes, macrophages and plasma cells with the possibility of forming lymphoid follicles. Subsequently, damage to the lining epithelium is observed, followed by the development of atrophic changes, the formation of intestinal metaplasia, intraepithelial neoplasia, and, as a result, gastric cancer [7, 8, 16, 19].

Almost 75% of all polyps are inflammatory or hyperplastic. They are more common in people aged 50-60 years. These polyps usually develop in the setting of chronic gastritis, which initiates mucosal injury and reactive hyperplasia leading to polyp growth.

Most inflammatory and hyperplastic polyps are less than 1 cm in diameter and are often multiple, especially in individuals with atrophic gastritis. These polyps are ovoid in shape, with a smooth surface despite frequent erosions. Microscopically, cystically enlarged, uneven and elongated foveolar glands are determined. The lamina propria of the gastric mucosa is usually edematous, with acute or chronic inflammatory infiltration of varying degrees. Ulcerative defects of the integumentary epithelium are often determined. Fundic gland polyps can be sporadic or hereditary (hereditary adenomatous polyposis, HAP). Often, the discovery of polyps is an incidental finding during esophagogastroduodenoscopy, since clinically there may be no symptoms. Numerous studies have shown an association between Helicobacter pylori infection and the likelihood of developing polyps. [2, 7, 12]. Women are affected slightly more often than men. With a hyperplastic polyp, the risk of malignancy is quite high, especially if atrophic gastritis serves as a background change. A polyp from the fundic glands is perhaps the most common form of a polyp of the gastric mucosa [14].

Fundic gland polyps are localized in the body and fundus of the stomach and are clearly defined formations with a smooth surface. They may be single or multiple and are cystically enlarged irregular glands lined with flattened parietal and chief cells. Inflammatory changes are usually absent or mild.

In persons with Helicobacter-associated gastritis, after successful eradication of the pathogen, polyps may regress. The risk of dysplasia correlates with polyp size, so polyps larger than 1.5 cm should be resected and sent for histological examination.

Peptic ulcer ranks 4th in frequency among all diseases of the gastrointestinal tract. The lifetime risk of developing an ulcer is -10% for men and -4% for women, with women

typically experiencing the disease during or after menopause. Every year, more than 3 million people need treatment for this disease, 190 thousand of them are hospitalized, 5000 people die [4, 5].

Despite the use of modern diagnostic algorithms and therapeutic regimens for the treatment of gastroduodenal ulcers, the frequency and risk of developing precancerous changes and gastric cancer in our time remains high.

In practical medicine, the entire set of modern diagnostic methods is rarely used to determine the nature of a peptic ulcer. As a rule, a comprehensive examination of patients with gastric ulcer during the initial treatment is not carried out at all, but drug therapy is prescribed preventively [1,2]. The number of diagnostic errors in the differential diagnosis of malignant and benign gastric ulcers during endoscopic examination with biopsy can reach 15–25% [4]. The problem of timely diagnosis of malignancy of ulcers and detection of the primary form of gastric cancer in the early stages does not lose its relevance. Peptic ulcer is in most cases associated with hyperacid chronic Helicobacter-associated gastritis, which is observed in 65% of patients with gastric ulcer. Peptic ulcer can develop in any part of the gastrointestinal tract,

Peptic ulcers of the stomach are located mainly along the lesser curvature on the border of the body of the stomach and its antrum. More than 80% of patients develop solitary peptic ulcers. Lesions less than 0.3 cm in diameter are usually superficial, while lesions larger than 0.6 cm in diameter are deeper. A classic peptic ulcer has the appearance of a round or oval, well-defined mucosal defect. The mucous membrane of the edges of the defect may slightly hang over its bottom, especially from the cranial side, but is usually at the same level with the surrounding mucous membrane.

Raised edges are more characteristic of malignant tumors. An ulcer may penetrate the muscular layer of the stomach wall or spread to the pancreas, omentum, or liver. The bottom of peptic ulcers as a result of the digestion of exudate is smooth and clean, with well-defined blood vessels. In active ulcers, the bottom can be covered with a thin layer of tissue detritus mixed with fibrin, along which there is an inflammatory infiltrate, represented mainly by neutrophils. Below it is a layer of immature granulation tissue with numerous mononuclear

cells. The bottom of a necrotic ulcer forms a dense fibrous connective or granulation tissue. Vessels in the scar area usually have thickened walls and are sometimes thrombosed [1].

The size and location of benign and malignant ulcers are similar, but the macroscopic features of chronic peptic ulcers usually distinguish them from malignant ones.

Conclusion: Modern data on precancerous processes in the stomach indicate that many aspects of this problem have not been studied enough. This concerns both theoretical aspects of etiopathogenesis and practical morphological and immunohistochemical diagnostics, grading and interpretation of pre- and neoplastic changes in the gastric mucosa. The absence of clear morphological and immunohistochemical criteria for the differential diagnosis of various precancerous conditions indicates the need for further study of this issue. Perhaps these studies will open up new diagnostic possibilities and methods of influencing the tumor process, and will create prerequisites for changing the tactics of managing patients with precancerous diseases.

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