WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 3, Issue 2, Feb., 2022

THE GASTRIC CANCER RISK FACTOR, DIAGNOSIS OF CANCER AND PRECURSOR LESIONS

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Summary

Literature review is devoted to the issues of prophylaxis and early diagnostics of one of the most wide-spread malignant neoplasms – carcinoma of the stomach. Precancerous conditions are considered to increase the risk of gastric cancer development. In the literature review significance of Hpylori infection, genetic factors and harmful factors of our environment in pathogenesis of gastric cancer are discussed. The article presents comparison of precursor lesions and gastric cancer diagnostic methods, their merits and shortcomings. Assessment of different methods of H.pylori detection in stomach is given. High morbidity and mortality of gastric cancer, long asymptomatic course require serach for diagnostic algorithm in the diagnosis of this pathology.

Key words: gastric cancer, precursor lesions, risk factors, diagnostics.

Introduction

According to experts' forecasts, oncological pathology in the XXI century will become the leading cause of death [6, 15], and cancer of the digestive organs in morbidity and mortality by 2010 will come out on top [4, 15]. According to IARS (International Agency for Research on Cancer), stomach cancer (SC) ranks fourth in morbidity among all oncological diseases, and second in mortality [4, 13, 45], second in the structure of morbidity among the digestive organs, and first in the structure of mortality [4, 15]. Japan has achieved the best success in detecting early stages of breast cancer: up to 60% of cases are diagnosed at the stages of early cancer, the detection of borderline changes is high [19, 28, 32, 45]. In Russia, with a high incidence rate of only 5-10%, cancer is detected in the early stages [2, 4, 6, 19].

In the development of SC, the interaction of three factors is distinguished: genetic, the presence of H.pylori, damaging environmental factors [7, 10, 11, 13, 32, 46, 49]. Most patients had a history of stomach pains in their closest relatives [17]. In 1994, the International Agency for Research on Cancer (IARC) registered H.pylori infection as a first-order carcinogen. The risk of developing stomach cancer in people infected with H.pylori is 2-6 times greater than in uninfected [6, 10, 13, 24, 39, 41, 48]. The CagA-positive strain is considered particularly virulent, which increases the risk of atrophy and metaplasia with subsequent malignant transformation [2, 10, 11, 41].

Approximately 80% of all neoplasms occur under the influence of environmental factors [38, 46]. At the same time, the most common and posing a real danger to humans are nitroso compounds [6, 32]. Foci of intestinal metaplasia and dysplasia are particularly sensitive to their effects. An imbalance in the diet, a lack of antioxidants contribute to the development of SC; smoking increases the risk of developing stomach cancer by 1.5-3 times [3, 15]. An increase in the consumption of milk and dairy products, fresh fruits and vegetables correlates with a decrease in mortality from SC (approximately 1.4 times in men and 1.3 times in women) [7, 11, 28, 56].

Most authors point to the relationship of background diseases and SC [7, 17, 22, 28, 34, 39, 49, 53]. Various pathological processes that precede the development of a malignant tumor, but do not necessarily go into it, are considered to be a precancerous (precancerous) condition.

Precancerous in the clinical aspect is a condition or disease against which cancer can develop, biologically and morphologically regarded as the beginning of cancer, characterized by the appearance of cellular atypia and/or structural rearrangements and changes in metabolism [11, 17, 25, 33, 46, 49]. According to the latest WHS classification (2000), the following conditions are distinguished among precancerous lesions and tumors of the stomach: previous lesions, early rye and SC [46]. Previous lesions include chronic atrophic gastritis, intestinal metaplasia, intraepithelial neoplasia (dysplasia), adenomas, familial adenomatous polyposis, foveolar hyperplasia, Peitz-Jaegers syndrome.

Many authors note the connection between atrophic gastritis and stomach cancer [3, 6, 12, 19, 28, 40, 46, 50]. In accordance with the model of gastric cancer development proposed by R. Soggea, a series of pathological processes in the gastric mucosa leads to SC: normal mucous membrane - atrophic gastritis - intestinal metaplasia - dysplasia - intestinal carcinoma [6, 15, 22]. In diffuse gastric cancer, there is no such sequence. The previous lesions for such a tumor are unknown, presumably genetic factors play a role [7, 32]. With severe epithelial atrophy, the risk of developing

stomach cancer is 5 times higher than with non-atrophic gastritis [13, 28, 50]. In autoimmune atrophic gastritis, stomach cancer occurs in 16% [6, 40].

The group at increased risk of malignant transformation of polyps includes patients with adenomatous gastric polyps [4, 19, 46]. Gardner syndrome (familial adenomatous polyposis) is characterized by a 100% risk of malignancy [4, 11, 19, 46].

A number of experts indicate that there is an increased risk of stomach cancer against the background of foveolar hyperplasia in chronic gastritis and ulcers [3, 21, 27]. According to others, patients diagnosed with foveolar hyperplasia do not require either "oncological alertness or further observation" [21, 27].

According to the International Classification (WHS, 2000), there are two main types of intestinal metaplasia: small intestine, or type I, and incomplete (type II and III). In countries with a high incidence of stomach cancer, intestinal metaplasia is detected much more often than in countries where stomach cancer is relatively rare [6, 12, 28, 45, 50]. It is believed that the development of the tumor is associated with incomplete intestinal metaplasia, which is confirmed by molecular genetic studies [3, 6, 42, 57].

In the early stage of gastric cancer, intestinal metaplasia is detected in 65% of cases. The greater the volume of epithelial replacement by intestinal metaplasia, the more conditions for the development of adenocarcinoma [3, 19, 46, 53].

According to the International Classification of neoplasia of the digestive tract (WHS, 2000), there are 3 degrees of dysplasia (DI-DIII). Dysplasia is defined as a deviation of cells and the entire tissue complex from the normal structure in the direction of neoplastic development [3, 46]. The risk of developing cancer depends on the severity of dysplasia: with a low degree of severity — about 10%, with a high degree - up to 96% [12, 19, 20, 32].

Recently, WHS has excluded gastric ulcer from the list of previous lesions, however, due to the fact that intestinal metaplasia is often found in gastric ulcer, this exception seems impractical. Recent studies on this issue have shown that gastric ulcer malignancy is indeed rare, however, in 2% of patients with gastric ulcer, SC is detected [4, 13, 19, 24, 34].

For a long time, stomach resection was performed in Russia for gastric ulcer (Billrot II), but it was found that after this operation, the risk of developing gastric adenocarcinoma increases by 2 times and reaches 50-70%. Moreover, cancer develops in the anastomosis zone [6, 11, 15, 34].

Early stomach cancer. According to the WHS definition (Lion, 2000), the term "early gastric cancer" refers to a carcinoma limited to the mucosa or mucosa and submucosa, regardless of the status of the nodes. If the tumor is removed at an early stage, the 5-year survival rate is approximately 100% [6, 15]. The early stage of SC, even at the

maximum rate of tumor growth, takes a period of at least 5 years [19], so there are all conditions for the successful diagnosis of this stage of the disease. The frequency of relapses in early SC ranges from 0.3 to 7,5% [6, 21, 45].

In the same edition, in accordance with the Lauren classification, two main morphological forms of gastric adenocarcinoma are distinguished, which makes up the main part of malignant gastric tumors: intestinal and diffuse type.

During endoscopic examination of dispensary groups of patients suffering from chronic stomach diseases, cancer is detected in 5-10% of patients, and in 30% of them at the earliest stage of the disease [21, 54].

However, up to 10% of cases of early and up to 9% of advanced gastric cancer may not be noticed during endoscopic examination [6, 54, 55]. The sensitivity of the chromogastroscopic method in the detection of previous lesions and early SC is 86.9% [5, 54]. Fluorescent endoscopy makes it possible to identify foci of dysplasia and early cancer with a sensitivity of 71% and perform a targeted biopsy [21, 23]. The accuracy of ultrasound endoscopy (ultrasonography) is approaching 90% [15, 55]. The method allows for differential diagnosis between inflammatory and tumor changes, assess the depth of tumor invasion, accurately assess the extent and determine the boundaries of the lesion [8, 21].

By the routine X-ray method, early SC is "missed" in almost 25% [8, 19]. An additional method of diagnosing previous lesions of the gastric mucosa after preliminary X-ray and endoscopic methods can be X-ray computed tomography [6, 8, 21].

Diagnostic laparoscopy with biopsy makes it possible to correctly establish and histologically confirm the diagnosis in 96% of patients with abdominal cancer dissemination and thereby avoid trial laparotomy in 36% of patients. Laparoscopy helps to correctly assess the condition of regional lymph nodes in 72% of patients with an established diagnosis of SC [6, 21, 23, 54].

Noninvasive (immunological) methods are more often used to screen for H.pylori. In Europe, the most effective screening test is considered to be a breath test and a gastropanel (S-PG1, S-G-17, Ab H.pylori) [9, 10, 31, 51]. Invasive methods of H.pylori diagnosis are more widespread (cytological, histological, culture seeding, urease test (on fragments of the mucous membrane), PCR. The most sensitive of the diagnostic methods are N. pylori is a breath test (90-95%) and histological examination (93-98%), the most specific are culture seeding (100%) and morphological (histological and cytological examination (95-98%). A fairly large spread of data presented by different authors on the gastropanel (sensitivity - 86-100%, specificity — 82-100%) [6, 20, 37, 41, 47, 48, 51].

The sensitivity of the histological method in determining previous lesions and early gastric cancer is 85-90%, specificity is 93-100%. However, targeted biopsy does not always allow to establish precancerous pathology and early forms of cancer from a small biopsy, and in cases of early cancer to establish the degree of tumor invasion [3, 19, 35].

The cytological method is characterized by significant diagnostic capabilities, including express diagnostics. Cellular material can be obtained from extensive surfaces that practically cannot be examined histologically [16, 21]. It is possible to correctly determine the degree of proliferative changes in cells when studying smears-prints from pieces of the gastric mucosa with previous stomach lesions in 76-83%, and to detect cancer in 78-98% [5, 16, 18]. The sensitivity of the cytological method in the diagnosis of intestinal metaplasia is 96.6%, specificity is 98.8% [5, 53]. The informativeness of the cytological method in the preoperative recognition of early SC is not inferior to the histological one, and, taking into account the presumed form of the conclusion, somewhat exceeds it. When examining patients with invasive gastric cancer, the malignant nature of the process based on cytograms was established or suspected in 93%, and according to gastrobiopsy material - in 73% [16, 18, 19].

According to L.B. Klyukina (1991), dysplasia detected by cytological method was confirmed histologically in 46%, but when performing subsequent morphological and endoscopic studies conducted on the recommendation of a cytologist, epithelial dysplasia was confirmed in 54% of patients.

Liquid cytology is a new promising technology of cytological diagnostics. When using this method, the number of false negative results of cytological examination decreases. The liquid material can be used for molecular biological studies. However, in the available literature, we have not found works on the use of liquid cytology in stomach diseases.

Simultaneous cytological and histological studies are essential for qualitative morphological diagnostics. This makes it possible not only to detect cancer in 81.3% of patients, but also to determine dysplasia; the effectiveness of simultaneous use of studies in detecting dysplasia is about 78,2% [12, 16, 32, 35].

Morphometric examination makes it possible to identify the quantitative characteristics of pathologically altered cells, to conduct an objective assessment of certain previous lesions of the gastric mucosa and SC. The types of distribution of epithelial cell nuclei in preparations by ploidy and the DNA accumulation index can serve as objective evidence of previous gastric lesions and the onset of malignant cell transformation [1, 3].

Molecular diagnostics. Stomach cancer is considered as the end result of a long multi-step and multifactorial process of accumulation of genetic and epigenetic changes, which lasts at least 5 years. These changes lead to disruption of the cell cycle, apoptosis, differentiation, morphogenetic reactions of the cell, as well as, probably, to the ineffective functioning of factors of specific and nonspecific antitumor immunity [15, 17, 20, 49].

The following epigenetic alterations were noted during the development of SC [7, 17, 29, 33, 42, 43, 46]: DNA hypermethylation; aberrant transcription of the CD44 gene; inactivation of the anti-oncogene pS2 of the stomach-specific gene. The most common genetic disorders are DNA aneuplodia, microsatellite instability [33, 42].

Mutation of the p53 gene may be an early sign of intestinal cancer [3, 17, 30, 49]. The level of p53 in true dysplasia is 100 times higher than in chronic gastritis, and in cancerous tissues its expression increases 400 times [3, 20, 30]. BCL-2 gene expression is detected in previous lesions: incomplete intestinal metaplasia in 91%, dysplasia in 81% [2, 17, 32].

In gastric cancer, the activity of telomerease and reverse transcriptase (hTERT) increases, and the level of both of these indicators correlates with the degree of H.pylori infection [17, 25, 33, 46, 52, 57]. In Japan, the achievements of molecular diagnostics have been introduced into clinical practice, which make it possible to identify genetic changes characteristic of malignant growth and formulate a molecular pathohistological diagnosis [33, 43].

Immunological diagnostics. The results of the determination of REA, CA-19-9, P-1MA in serum and biopsies showed insignificant diagnostic significance in the determination of early gastric cancer [3, 25, 49]. The association of CD25, CD71, CD95 and CD98 expression with the prognosis of the disease was established [37]. Herpesviruses (herpes simplex virus, cytomegalovirus, Epstein-Barr virus) affect the functioning of the local immunity systems of the mucous membranes and contribute to the formation and maintenance of local immune insufficiency, and later - malignant tissue transformation [7, 14, 33].

Biochemical research methods. The determination of the nm23 protein (non-metastatic cells protein), the production features of the main components of the plasminogen activation system, and the metabolic cancer marker Ti M2-PK are considered promising in the diagnosis of gastric cancer [17, 33]. The plasma concentration of the metalloproteinase I matrix inhibitor (TIMP-I) in patients with SC was a significant predictor of tumor aggressiveness and metastasis [29, 49].

A highly sensitive colorimetric chip-membrane method has been developed for detecting tumor cells in peripheral blood using a panel of markers including hTERT, cytokeratin-19, REA, MUC1 [26].

The category of diagnostically sensitive tests that provide timely recognition of malignant tumors includes those that give a minimum of false negative results. This criterion is most consistent with endoscopic and morphological (histological and cytological) methods of research, which complement each other (specificity 99%). Molecular studies play an important role in the problems of morphological diagnostics.

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