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A Current View of an Old Problem: Risk Factors for Gastric Cancer

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ABSTRACT

Several risk factors are described for gastric cancer. *Helicobacter pylori infection* and a family history of gastric cancer are the two main risk factors for gastric cancer. Gastric cancer has significant geographic, ethnic, and socioeconomic differences in distribution. This review article will discuss risk factors for gastric cancer. The molecular cascade of events underlying colonic-type gastric cancer and its distinct precursor lesions is not fully understood. In contrast, much more is known about the molecular pathogenesis of diffuse gastric cancer, which has prominent molecular abnormalities in the cell adhesion protein E-cadherin. Today, gastric cancer remains a major clinical challenge because of its prevalence, poor prognosis, and limited treatment options. Therefore, one of the main goals of the World Health Organization and researchers is to organize a GC prevention programme.

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INTRODUCTION

There are two main histological variants of gastric adenocarcinoma. The “intestinal type” is the most frequent, named for its morphological similarity with adenocarcinomas that originate in the intestinal tract. Less common "diffuse type" gastric cancers are characterized by a lack of intercellular adhesions, leaving them unable to form glandular structures. In patients with the inherited form of diffuse gastric cancer, the absence of intercellular adhesions is caused by a germline mutation in the cell adhesion protein E-cadherin (CDH1). Although several risk factors are described, *Helicobacter pylori infection* and a family history of gastric cancer are the two main risk factors for gastric cancer. Gastric cancer has significant geographic, ethnic, and socioeconomic differences in distribution[1,2]. This review article will discuss risk factors for gastric cancer.

THEORETICAL FRAMEWORK

The molecular cascade of events underlying colonic-type gastric cancer and its distinct precursor lesions is not fully understood. In contrast, much more is known about the molecular pathogenesis of diffuse gastric cancer, which has prominent molecular abnormalities in the cell adhesion protein E-cadherin[3].

There are geographic and ethnic differences in the incidence of gastric cancer worldwide, as well as trends over time in each population. Immigrants from high-incidence countries to low-access countries often have a lower risk of developing stomach cancer. Such results strongly suggest that environmental factors play an important role in the pathogenesis of gastric cancer and that exposure to risk factors occurs early in life [3,4].

The International Agency for Research on Cancer (IARC) of the World Health Organization classifies Helicobacter pylori as Group 1 or a definitive carcinogen. Helicobacter pylori infection is thought to cause inflammation in the inner layers of the body, which leads to intestinal atrophy and metaplasia. H. Helicobacter pylori infection is associated with an approximately 6-fold increased risk of adenocarcinoma distal to the cardia, including intestinal and diffuse types[3].

Substantial evidence suggests that some traditional salted foods, such as salted fish, salted meats, and salted vegetables, and high salt intake increase the risk of stomach cancer. 2007 In the year, salt and salty/salty foods were identified as likely to be risk factors for stomach cancer. Although not all studies have reported possible synergistic effects of salt and H. pylori. High salt intake damages the gastric mucosa and

increases susceptibility to carcinogenesis in rodents. The proliferative changes induced may facilitate the action of food-borne carcinogens. Humans are exposed to N-nitroso compounds (compounds containing -NO groups) from the diet, cigarette smoke, other environmental sources, and endogenous synthesis, accounting for 40-75% of total exposure. I'm here. N-nitroso compounds are formed after consuming nitrates, which are natural constituents of foods such as vegetables and potatoes, and are used as food additives in some cheeses and cured meats. It is absorbed in the stomach, excreted in a concentrated form in saliva, and reduced to nitrite by oral bacteria. Nitrite can also react with nitroso compounds such as amines, amides and amino acids to form N-nitroso compounds. A meta-analysis of epidemiological studies found inconsistent associations between low folic acid levels and gastric cancer risk. Being overweight is associated with an increased risk of stomach cancer. In a meta-analysis of cohort studies that identified 9492 cases of gastric cancer, overweight (defined as body mass index (BMI) ≥25 kg/m2) was associated with an increased risk of gastric cancer (odds ratio [OR] 1.22; 95% CI 1.06-1.41). The strength of association increased with increasing BMI. It is unclear if this risk extends to all parts of the stomach[5–7].

Several studies have investigated the association between smoking and stomach cancer[2].

There is evidence that occupations in coal and tin mining, metalworking (particularly steel and iron), and rubber production increase the risk of stomach cancer. However, the data are mixed. Epstein-Barr virus (EBV) infection is associated with many malignancies, especially nasopharyngeal carcinoma[8,9].

The risk of distal gastric cancer is approximately doubled in populations with low socioeconomic status. In contrast, proximal gastric cancer was associated with a higher socioeconomic class. The risk of gastric cancer after gastric surgery is high, and there is a risk and time between the first gastric surgery and the development of residual gastric cancer, depending on the reason for the first surgery and the type of reconstruction [5,10].

Billroth II (gastrojejunostomy) has higher risks than Billroth I (gastroduodenal anastomosis). The exact cause of the increased risk is unknown, but it is thought to be due to reflux of alkaline bile and pancreatic juice (higher after Billroth II than after Billroth I)[3,4].

Hereditary diffuse gastric cancer, a hereditary form of diffuse gastric cancer, is a highly invasive tumour characterized by late onset and poor prognosis. Germline truncating mutations in the cadherin-1 gene (CDH1), which encodes the cell adhesion protein E-cadherin, have been identified in approximately 19% to 50% of affected relatives who meet clinical criteria for HDGC as described by the International Gastric Cancer Linking Consortium. I'm here. (IGCLC). These mutations are not concentrated in a single hotspot but are evenly distributed throughout the CDH1 gene in several different exons. The triggers and molecular mechanisms by which the second allele of E-cadherin is inactivated appear to be diverse and include promoter hypermethylation, mutation, and loss of heterozygosity. The result is a loss of expression of the cell adhesion molecule E-cadherin[1,10].

HDGC is inherited in a highly penetrating autosomal dominant inheritance pattern. Her cumulative risk of gastric cancer at age 80 for a CDH1 mutant carrier is up to 70% in men and up to 56% in women. Affected patients usually develop gastric cancer at a young age (mean age of 38 years)[10].

Gastric polyps are usually found by chance when performing upper gastrointestinal endoscopy for an unrelated indication. They rarely cause symptoms or other clinical signs. However, many polyps can be malignant, so their detection is important. The role of genetic factors was first suggested through studies of determinants of blood type and chronic gastritis[10].

It has been known for decades that people with type A blood have an approximately 20% higher incidence of stomach cancer than people with type O, B, or AB blood. Also, the incidence of pernicious anaemia is increasing as well. Some data suggest that group A may be specifically associated with diffuse gastric cancer. It is possible that the observed associations are not due to the blood group antigens themselves but to the influence of genes closely associated with them. Hypertrophic gastropathy (including Ménétrier's disease) and several immunodeficiency syndromes are associated with stomach cancer[5,10]. However, the strength of these partnerships remains undefined. The association between benign peptic ulcer disease and gastric cancer may reflect common risk factors (ie, predominantly H. pylori infection). Pernicious anaemia, a sequela of chronic autoimmune atrophic gastritis against potassium hydrogen ATPase in gastric parietal cells, is associated with an increased risk of colonic gastric cancer. Risk increases of 2- to 6-fold have been reported, but as with other predisposing conditions, the actual level of risk varies with disease duration and geographic location. Pernicious anaemia is also associated with an increased risk of gastric neuroendocrine tumors, possibly due to prolonged achlorhydria resulting from parietal cell loss, compensatory hypergastrinemia, and argyrophilic cell hyperplasia [1,6,10].

CONCLUSION

Today, gastric cancer remains a major clinical challenge because of its prevalence, poor prognosis, and limited treatment options. Therefore, one of the main goals of the World Health Organization and researchers is to organize a GC prevention programme, which means:

• Proper diet with increased consumption of vegetables and fruits from childhood.

• Reduce the prevalence of H. pylori by improving hygiene in families and communities.
• Testing for her E-cadherin germline mutation in a relative of a familial GC patient.
• Search and treatment of Helicobacter her pylori in offspring of GC patients.
• H. pylori search and treatment in patients with chronic atrophic gastritis and myalgia, and careful endoscopic and histologic follow-up unrelated to H. pylori should be planned if premalignant lesions persist. I have. Helicobacter pylori sterilization.

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