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A REVIEW ON GASTRIC CANCER TREATMENT

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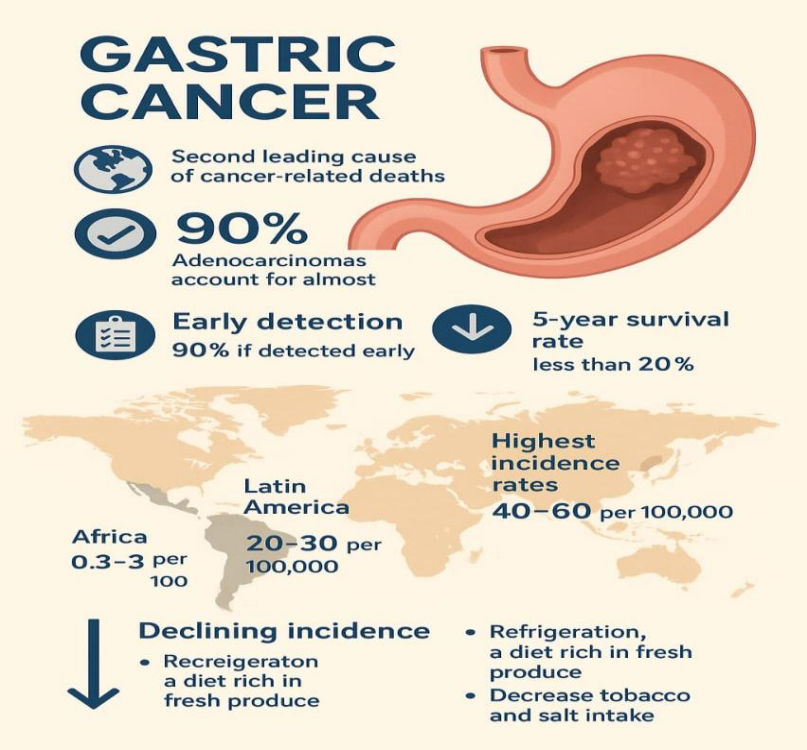
ABSTRACT

Gastric cancer is the second most common cause of cancer-related death globally and continues to be a significant global health burden. The majority of cases are adenocarcinomas, which are frequently brought on by a confluence of environmental exposures, dietary practices, genetic vulnerability, and Helicobacter pylori infection. In many areas, early-stage stomach cancer is often asymptomatic, leading to a poor prognosis and delayed diagnosis. Higher incidence is found in portions of South America, Eastern Europe, and East Asia, according to epidemiological variables. Chronic H. pylori infection, a high-salt diet, processed and smoked foods, smoking, obesity, and a low fruit and vegetable intake are all significant risk factors. Promising outcomes have been demonstrated by preventative measures such H. pylori eradication, dietary changes, quitting smoking, and routine endoscopic screening in high-risk groups. Depending on the stage of the tumor, treatment options include endoscopic resection, gastrectomy withradiation, chemotherapy, lymphadenectomy, and more recent multimodal techniques. Programs for early detection greatly increase 5-year survival rates. The epidemiology, risk factors, preventative measures, clinical characteristics, and contemporary management techniques for stomach cancer are all presented in this review.

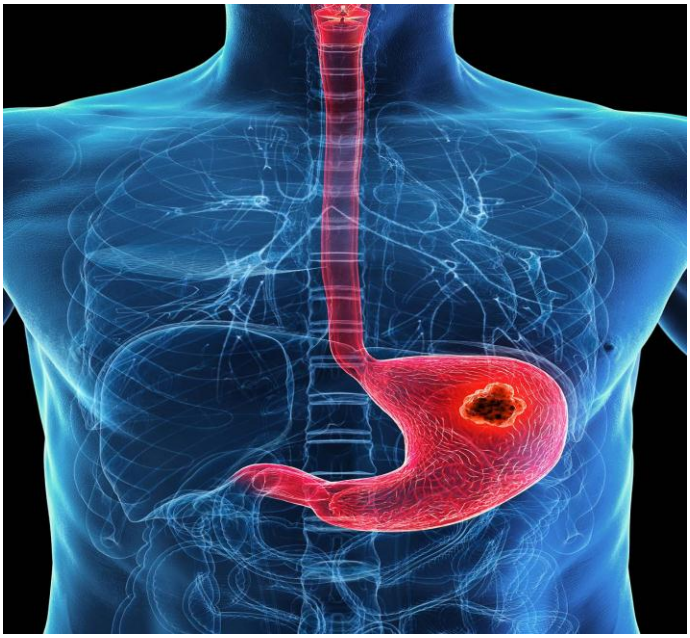
KEYWORDS: Higher incidence is found in portions of South America, Eastern Europe, and East Asia, according to epidemiological variables.



INTRODUCTION

Globally, gastric cancer is a significant health burden. After lung cancer, it is the second leading cause of cancer-related deaths.[1,2] The primary emphasis of this review is adenocarcinomas, which account for almost 90% of the tumors. Because the early stages are clinically quiet, the prognosis is poor, with an average 5-year survival rate of less than 20%. This is primarily due to late detection. Only a few nations have established comprehensive early detection programs, particularly Japan. The 5-year survival rate can reach 90% if the tumor is found and treated before it spreads to the stomach's muscle layer.[3] East Asia (Korea, Mongolia, Japan, and China) has the highest incidence rates, with yearly China, Japan, and Mongolia) with yearly incidence rates ranging from 40 to 60 per 100,000 people. In contrast to the far lower rates recorded for the coastal and river valley regions of Latin America, pockets of high risk are found in the Andes Mountains, with rates between 20 and 30 per 100,000.[4] Africa (~0.3 to 3 per 100,000) and wealthy North American people have lower rates. African Americans have an incidence rate that is around twice as high as that of white Americans. Men typically have an incidence rate that is twice as high as that of women. One In many communities, the incidence of stomach cancer has gradually declined over the past few decades. Some have suggested that this decline is a reflection of food handling trends.

Particularly refrigeration, a diet rich in fresh produce, and a reduction in tobacco and salt intake. Tumors of the cardia and esophagogastric junction are increasing in frequency, although not all forms of gastric cancer are decreasing. There have been reports of an unexplained rise in the incidence of stomach cancer in younger people, primarily those under 40.[5]



ETIOLOGY

Although Helicobacter pylori infection is thought to be the main cause of stomach cancer, its effects are influenced by host, environmental, and microbial variables. One of the few neoplasms that is directly connected to an infectious agent is gastric cancer. The International Agency for Research on Cancer in 1994 H pylori infection is a class I human carcinogen for stomach cancer, according to the International Agency for Research on Cancer (IARC). Six Gastric mucosa-associated lymphoid tissue (MALT) lymphoma is known to be mostly caused by the same infectious pathogen. The gram-negative bacterium H pylori can colonize the stomach mucosa and trigger an immunological reaction in the host. If antibiotics are not administered, the infection, which is primarily contracted in early infancy, persists for life. The precancerous process may be connected to multifocal atrophic gastritis, one kind of gastritis linked to the infection. Nonatrophicantral gastritis Is not associated with the precancerous process but may be linked to duodenal ulcer. Reactive oxygen species (ROS) may be generated by the Infection and could result in alterations in DNA. Additionally, H pylori can cause DNA hypermethylation, particularly in the CpG islands, which silences

genes linked to tumor suppression. A population at high risk for gastric cancer from the Colombian Andes (Tuquerres) had significantly higher hypermethylation of the tumor suppressor gene RPRM in the gastric mucosa than those in a low-risk population on the Pacific coast (Tumaco), according to a study on subjects infected with H pylori. The virulence and carcinogenicity of seven H pylori strains differ significantly. The cytotoxic-associated gene cagA, which codes for an oncogenic protein that may be delivered straight into gastric epithelial cells via a type IV secretion system, is carried by more virulent strains.[8,9] The majority of strains in the high-risk region and East Asia The Andes of Colombia are cagA positive. Following its entry into the cytoplasm of the gastric epithelial cells, CagA undergoes phosphorylation in motifs containing EPIYA sequences, initiating a series of molecular processes associated with carcinogenesis. Based on the amino acids that surround them, the EPIYA sequences are categorized as A, B, C, or D. varying strains of H pylori have varying numbers and types of EPIYA motifs. EPIYA motifs A, B, and C are present in H pylori strains from western nations. The strains from East Asia have the D motif rather than the C motif. Intestinal metaplasia, gastric cancer, and gastric atrophy are considerably more common in strains with more than three EPIYA motifs. Studies conducted both in vivo and in vitro have demonstrated that CagA causes intercellular connection disruption, loss of epithelial polarity, and enhanced proliferation.[11] VacA is another gene linked to virulence that causes cytoplasmic vacuoles, cell membrane holes, and apoptosis. Twelve The vacA gene is present in all strains of H pylori, but its functional activity and cancer risk are determined by genetic variations. The s (signal) region of the vacA gene contains genetic variations that can be s1a, s1b, s1c, or s2. The middle region shows alleles that can be m1 or m2 and the intermediate region can be i1 or i2. Strains vacA s1/m1 or vacAs1/m1/i1 convey a higher risk of progression and cancer than strains vacA s2/m2 or vacA s2/m2/i2. Some adhesion proteins of the membrane have been linked to higher virulence. One of them is BabA (blood-group antigen-binding adhesin) encoded by the gene babA, not present in all strains.

H pylori infection The Contagious Agent is quite common; at least 50% of adults globally are thought to be infected. But less than 1% of people ever have stomach cancer. Since the beginning of time, H pylori has been a part of the human microbiome. About 60,000 years ago, both species—Homo sapiens and H. pylori—migrated together out of Africa and settled most of the world. Gradual changes to the bacterial genome over millennia have produced a

number of prototypes, such as hpEurope, hpAfrica1 (which includes hspWest Africa and hspSouth Africa), hpAfrica2, and hpSahul (Oceania).

Epstein-Barr virus

Between 5% and 16% of stomach malignancies have been found to contain the Epstein-Barr virus (EBV), suggesting that it may be a contributing factor. Tumors of the gastric body or cardia, as well as tumors found in gastrectomy specimens, are more common in men than in women. About 90% of gastric lymphoepitheliomas (carcinomas with lymphoid stroma) have it.[16]

Environmental Factors

Tobacco use has been found a risk factor for gastric cancer and precancerous lesions. 17 High dietary salt consumption increases cancer risk. 18 Consumption of processed meat has also been associated with a high cancer risk. 19 No clear association has been found with alcohol consumption. Consumption of fresh fruits and vegetables has been associated with reduced cancer risk.

Adenocarcinomas, which account for approximately 90% of stomach tumors, are classified into two primary histologic types: (1) intestinal or well-differentiated type and (2) diffuse or undifferentiated type. The diffuse type typically starts with pangastritis without atrophy, while the intestinal type is associated with corpus-dominant gastritis with gastric atrophy and intestinal metaplasia. Males, Black people, and older age groups are more likely to have the intestinal type, while younger people are more likely to have the diffuse form, which has a more equal male-to-female ratio.[7,8] A large portion of the global variation in gastric cancer is caused by intestinal type tumors, which are more common in high-risk regions including East Asia, Eastern Europe, Central America, and South America.[9] The geographic distribution of diffuse type stomach adenocarcinomas is more consistent. Molecular Numerous trials have compared radiation therapy with sensitizing 5-fluorouracil or capecitabine with chemotherapy or surgery alone due to the high likelihood of local recurrence in gastric cancer. The British Stomach Cancer Group[45], Moertel et al.[75], and Dent et al.[74] conducted the first prospective randomized trials to examine the addition of post-operative radiation to adjuvant chemotherapy. Although the data from these studies did not demonstrate a survival benefit for patients undergoing adjuvant therapy, it is challenging to make inferences from them due to their limited accrual, diverse cohort, unstandardized surgery and radiotherapy,

and 5-fluorouracil dosage. The Gastrointestinal Cancer Intergroup Phase III Trial (INT 0116) had a significant impact.

RISK FACTORS

Gastric cancer is a complex illness. The migratory effect, historical trends, and significant geographic variation in the incidence of stomach cancer indicate that environmental or lifestyle variables play a significant role in the disease's etiology.

Infection Helicobacter pylori

The most prevalent chronic bacterial infection in the world may be H pylori, a gram-negative bacillus that colonizes the stomach.[38] H pylori infection is generally more common in nations with high rates of gastric cancer, and the drop in H pylori prevalence in developed nations corresponds with the decline in gastric cancer incidence[39,40] (Figure 1B). The prevalence of H pylori infection in the United States is less than 20% at age 20 and 50% at age 50.[41][50–53]

H pylori was designated as a type I (definite) carcinogen in humans by the International Agency for Research on Cancer in 1994.[54] According to Correa's model of gastric carcinogenesis, chronic gastritis, gastric atrophy, intestinal metaplasia, dysplasia, and ultimately gastric adenocarcinoma are all caused by H pylori infection.[55] H pylori seropositivity has been linked to a 2.1- to 16.7-fold increased risk of gastric cancer compared to seronegative persons, according to several case-control studies.[56–62] The link between H pylori infection and the incidence of stomach cancer has also been confirmed by prospective studies.[50–52,63] A prospective analysis of 1,526 Japanese patients in which stomach malignancies occurred may provide the strongest evidence for the connection between H pylori and gastric cancer.

Dietary factors

Nutritional aspects Gastric cancer is not likely to be caused only by a H pylori infection. Instead, H pylori may combine with other environmental and lifestyle factors to create a milieu that is favorable to the development of cancer. There is proof that a low diet of fresh fruits and vegetables and a high intake of salty foods and N-nitroso compounds raise the risk of stomach cancer. The development of nitrosating bacteria, which catalyze the synthesis of carcinogenic N-nitroso compounds, is facilitated by H pylori gastritis.[92] Furthermore, ascorbic acid, a crucial scavenger of oxygen free radicals and N-nitroso compounds, is

known to be inhibited by H pylori infection.[93] Foods preserved with salt and dietary nitrite from preserved meats have the potential to cause cancer. Consumption of salted foods may rise.[93]

Foods preserved with salt and dietary nitrite from preserved meats have the potential to cause cancer. Consuming salted food may raise the chance of contracting H pylori and work in concert to accelerate the development of stomach cancer. Salt consumption has been shown to increase the effects of gastric carcinogens and induce gastritis in animal models.[9]

Tobacco

A substantial dose-dependent association between smoking and the incidence of stomach cancer has been shown by prospective studies.[126,127] With adjusted rate ratios of 2.0 (95% CI, 1.1-3.7) for former smokers and 2.1 (95% CI, 1.2-3.6) for current smokers, the impact of smoking was particularly noticeable for distal gastric cancer.[128] The link between alcohol consumption and stomach cancer is not well supported.[129] Being overweight One of the primary risk factors for gastric cardia adenocarcinoma is.[130,131]

Obesity

can exacerbate GE reflux disease, which increases the risk of Barrett's esophagus, a metaplastic precursor state for GE junction and esophageal cancer.[132,133] According to a Swedish study, the risk of gastric cardia adenocarcinoma was 2.3 times higher in the heaviest quarter of the population than in the lightest quartile.

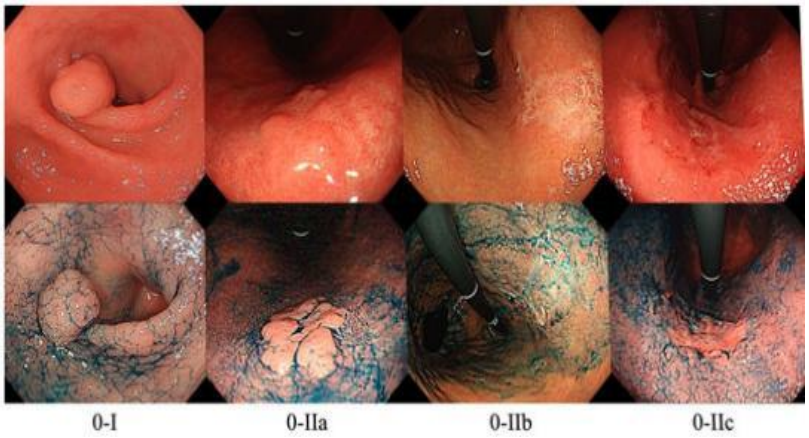
Other

Radiation[136], pernicious anemia[137], blood type A[138], previous stomach surgery for benign illnesses[139], and Epstein-Barr virus[140–142] are other less frequent risk factors for gastric cancer. Furthermore, a positive family history is a major risk factor, especially for genetic syndromes like Li-Fraumeni syndrome and hereditary nonpolyposis colon cancer.[143–145]

GASTRIC CANCER PREVENTION

Changes in lifestyle key prevention is the key technique for improving clinical outcomes because stomach cancer is frequently linked to a poor prognosis. Unplanned prevention is a major factor in the reduction of stomach cancer mortality. Fresh fruit and vegetable consumption has increased and chemically preserved food intake has decreased as a result of the widespread adoption of refrigeration.[98,146] Improvements in housing and sanitation,

along with the use of eradication therapy, may be responsible for a decrease in the prevalence of H pylori infection.[54] Additionally, a decrease in tobacco usage, at least among men, may have contributed to the decrease in the incidence of stomach cancer.[147] Thus, risk factors that may be changed, such smoking cigarettes, consuming a lot of salt and nitrite, and eating few fruits and vegetables, Clinical Features.



Early-stage stomach cancer is typically asymptomatic or accompanied by vague symptoms like dyspepsia. Anorexia, weight loss, and chronic stomach pain may accompany latter phases. Hematemesis may be linked to ulcerated tumors. Pyloric stenosis may be indicated by persistent vomiting. A delayed diagnosis could result from the absence of particular symptoms. In the majority of nations without early detection systems, almost 80% of patients are diagnosed at advanceddistal. When a tumor of the gastroesophageal junction has grown to a significant size, it is often challenging to determine whether the tumor originated in the stomach or the esophagus. Distal tumors have been less common in recent decades, but proximal tumors—which appear to be linked to gastric reflux—have become more common, particularly in developed nations. Adenocarcinomas of the cardia have aggressive aggressiveness, penetrating the walls of the stomach and esophagus and spreading to nearby lymph nodes; in the United States, the 5-year survival rate is approximately 14%. Tumors of the gastroesophageal junction and those affecting the first five centimeters of the stomach were categorized as esophageal carcinomas by the American Joint Commission on Cancer Classification (AJCC).[23]

Antioxidants Consuming a lot of antioxidants, like vitamins C and E and β-carotene, may reduce the risk of stomach cancer. In a Shanghai, China cohort, high serum levels of α-carotene, β-carotene, lycopene, and vitamin C were significantly linked to a lower risk of

stomach cancer.[153] Selenium, β-carotene, and α-tocopherol supplements were found to lower the risk of both cardia and noncardia stomach cancers in Linxian, China, according to a randomized experiment.[154] However, a Finnish randomized trial found no correlation between the occurrence of gastric cancer in older men with atrophic gastritis and supplementing with α-tocopherol or β-carotene.[155]

COX-2 inhibitors Cyclooxygenase-2 (COX-2) may contribute to the development of gastric cancer and is implicated in angiogenesis, apoptosis, and cell proliferation.[157, 158] As atrophic gastritis progresses to intestinal metaplasia and stomach cancer, COX-2 levels rise.[159] COX-2 expression is induced by acidic environments, cigarette smoke exposure, and H pylori infection.[160–162] Additionally, McCarthy et al. demonstrated that following H pylori eradication, COX-2 expression in the antral mucosa decreased in the epithelium.[163] It is believed that aspirin and other nonsteroidal anti-inflammatory medications (NSAIDs) mainly prevent the proliferation of cancer cells by inhibiting COX-2, and there is growing evidence that COX-2 inhibitors may be helpful in.

Endoscopic screening and surveillance

Because of the high risk of gastric cancer in Japan, there has been a national endoscopic surveillance program within the commercial workforce. Annual screening with a double-contrast barium technique and endoscopy is recommended for persons over the age of 40 years.[168] With mass screening, about half of gastric tumors are being detected at an early stage in asymptomatic individuals and the mortality rate from gastric cancer has more than halved since the early 1970s.[33] An intervention study in China is underway which involves a comprehensive approach to gastric cancer prevention, including *H pylori* eradication, nutritional supplements, and aggressive screening with double contrast X-ray and.

Classification of Histology

The Lauren classification, which distinguishes between two types of nosologic entities—intestinal (with intercellular connections) and diffuse (without intercellular junctions)—is the most often used classification. H pylori infection is linked to both kinds. The Japanese Endoscopic Society and the World Health Organization have proposed other classifications that are more complicated and have limited applications. 25, 26 The intestinal type adenocarcinoma gets its name from the formation of glands or tubules that are bordered with intestinal mucosa-like epithelium. In all high incidence populations, it is the most common form, and in recent decades, its incidence has declined. It shows that tumor cells are cohesive.

Lacking cohesiveness, diffuse carcinoma cells penetrate tissues either singly or in little groups. Diffuse signet ring cell carcinomas are categorized as.

PREVENTION AND EARLY DETECTION

In order to prevent cancer, patients with extensive atrophic or metaplastic changes in the gastric mucosa should undergo periodic endoscopic surveillance; if incomplete metaplasia or dysplasia is diagnosed, such surveillance is required; if the lesions are clearly identified topographically, endoscopic resection is a valid strategy; in Japan, endoscopic resection of such lesions leads to 5-year survival rates of up to 90%.

ADVANCED THERAPY

A significant rate of locoregional and distant recurrences has been documented, despite the fact that the only curative therapies for gastric cancer are total resection of the malignancy (R0) and prolonged lymph node dissection (D2). Locoregional recurrence occurs in 19%–42% of patients, peritoneal recurrence occurs in 21%–72%, and distant recurrence occurs in 18%–49% of cases. Chemotherapy or chemoradiotherapy added to surgery alone has been shown to improve survival, however adjuvant radiation alone has not shown any effect.[28,45–47] Chemotherapy adjuvant The effect of adjuvantChemotherapy adjuvant The effect of adjuvant chemotherapy vs surgery alone has been examined in a number of phase III trials over the past few decades, but the findings have been inconsistent. The wide range of patient enrollment, the limited number of series, the varying surgical precision, and the various chemotherapy regimens employed can all account for these discrepancies.[48–54] The effectiveness and safety of the combination of epirubicin, leucovorin, 5-fluorouracil, and etoposide (ELFE regimen) as adjuvant therapy for patients with radically resected gastric cancer were also examined in a randomized, multicenter, phase III trial. When compared to surgery alone, the ELFE regimen did not improve overall survival after a 5-year follow-up.[55]

chemotherapy vs surgery alone has been examined in a number of phase III trials over the past few decades, but the findings have been inconsistent. The wide range of patient characteristics can account for these variations. Adjuvant for chemotherapy Over the past few decades, several phase III trials have compared the effects of adjuvant chemotherapy to surgery alone; nonetheless, the results have been mixed. These disparities can be explained by a variety of factors, including the wide range of patient enrollment, the small number of series, the varied surgical precision, and the different chemotherapy regimens used.[48–54] A randomized, multicenter, phase III trial also looked at the safety and efficacy of the ELFE

regimen—epirubicin, leucovorin, 5-fluorouracil, and etoposide—as adjuvant therapy for patients with radically resected gastric cancer. After a 5-year follow-up, the ELFE treatment did not increase overall survival when compared to surgery alone.[55] A recent meta-analysis performed by the GASTRIC group[62], including 3838 patients from 17 different trials of adjuvant chemotherapy, concluded for a modest but statistically significant benefit with the use of adjuvant post-operative chemotherapy with respect to surgery alone (HR = 0.82; 95%CI: 0.76-0.90, *P* = 0.001). The estimated median survival was 4.9 years (95%CI: 4.4-5.5) in the surgery-only group *vs* 7.8 years (95%CI: 6.5-8.7) in the group of treated patients. However, no standard CT regimen has been defined in this setting. Chemoradiation adjuvant Numerous trials have compared radiation therapy with sensitizing 5-fluorouracil or capecitabine with chemotherapy or surgery alone due to the high likelihood of local recurrence in gastric cancer. The British Stomach Cancer Group[45], Moertel et al.[75], and Dent et al.[74] conducted the first prospective randomized trials to examine the addition of post-operative radiation to adjuvant chemotherapy. Although the data from these studies did not demonstrate a survival benefit for patients undergoing adjuvant therapy, it is challenging to make inferences from them due to their limited accrual, diverse cohort, unstandardized surgery and radiotherapy, and 5-fluorouracil dosage.

Neoadjuvant chemotherapy

Over the past ten years, the role of neoadjuvant chemotherapy in lower esophageal adenocarcinoma, gastric cancer, and gastro-esophageal junction has changed from regrettably negative studies to favorable ones.[61] In fact, 56 patients with seemingly treatable gastric cancer were randomly assigned to undergo either surgery alone or four cycles of 5-fluorouracil, doxorubicin, and methotrexate (FAMTX) prior to surgery in the first Dutch randomized controlled trial of neoadjuvant chemotherapy. In the most recent update, the median survival since randomization was 18 months for the FAMTX group and 30 months for the surgery alone group (P = 0.17); additionally, preoperative chemotherapy was linked to a negative effect.[84] The rate of curative resection favored the surgery alone group.

Stages. Diagnosis

Symptoms and indicators Regretfully, the majority of individuals with early-stage stomach cancer exhibit few or no symptoms. Patients usually come with nonspecific and non-specific symptoms, such as mild upper gastrointestinal distress (heartburn), flatulence, abdominal fullness early after meals, excessive belching, and at this time only infrequently

nausea/vomiting and pain. This is the primary cause of delayed diagnosis. A lengthy history of dyspepsia, which is identical to chronic peptic ulcer discomfort, is present in about 30% of all patients with EGC. Dysphagia may occur in patients with tumors of the proximal or cardioesophageal junctions. Intestinal bleeding is rarely severe and is typically occult. A palpable tumor in the abdomen usually suggests that the disease has spread to other areas. Anorexia, inexplicable weight loss, and the growth of the tumor Vomiting, anemia, haematemesis, and a general decrease in health are signs of an advanced stage of the illness. Abdominal pain, liver enlargement, ascites, jaundice, or palpable lymph nodes, such as those in the left axillary nodes or the left side of the neck (Virchow's node), can all be signs of metastatic disease. Drop metastases into the peritoneal reflection in the prerectal and postvesical region can cause peritoneal metastatic dissemination, which can manifest as a palpable ovary on pelvic examination (Krukenbergtumor) or Blumer's retinal shelf. Paraneoplastic diseases, such as cutaneous syndromes (dermatomiositis or acantosisnigricans), microangiopathichemolyticanemia, and chronic intravascular coagulation resulting in arterial and venous thrombi (Trousseau's syndrome), are rarely seen in patients with advanced stomach cancer.[54]

This discrepancy can be attributed to the extensive population screening for stomach cancer in Japan. 3.2.

The MAGIC[85] and FFCD9703[86] randomized studies have been used throughout Europe to promote perioperative chemotherapy. In the former, which took place in the UK, 503 patients with resectable adenocarcinoma of the stomach, esophagogastric junction, or lower esophagus cancer (of which 25% had lower esophageal or gastro-esophageal junction cancer) were assigned to either surgery alone (253 patients) or perioperative chemotherapy and surgery (250 patients).

SURGERY

Since then, stomach cancer treatment has relied mostly on surgery.[1] Endoscopic resection and minimally invasive access are two significant technological developments that have transformed therapeutic approaches in recent decades.[2, 3] Due to widespread screening programs, early gastric cancer—that is, tumors limited to the mucosa (T1a) or submucosa (T1b) with a low rate of nodal metastasis—has become more frequently detected in Eastern countries. As a result, early gastric cancer currently accounts for a significant portion of newly diagnosed tumors in South Korea and Japan. A few years ago, endoscopic resection

was thought to be a radical treatment for early gastric cancer, requiring no significant abdominal manipulation. However, invasion of the horizontal and vertical margins, as well as the possibility of nodal involvement, had to be taken into consideration right away toearly-stage stomach cancer.[2,7,8] Laparoscopic stomach surgery, a minimally invasive procedure, was first developed for benign esophago-gastric disorders and is now a common choice for achalasia and hiatal hernia repair.[9] At first, laparoscopic access was limited to treating distal-sided early gastric cancer that did not require a whole gastrectomy or an expanded lymphadenectomy due mostly to technical challenges and oncological concerns.[10,11] The laparoscopic method has been progressively expanded to include advanced gastric cancer needing total gastrectomy with radical lymphadenectomy following reports of acceptable oncological adequacy for laparoscopic surgical treatment of colorectal cancer. Several studies have demonstrated the viability, safety, and oncological sufficiency of the laparoscopic technique in the treatment of advanced stomach cancer, despite the fact that the results are still debatable.and repair of the digestive system.[13] Surgery is unquestionably the only possibly curative treatment for all T1b to T4 gastric tumors, regardless of the method (open or laparoscopic), and after endoscopic resection has failed.[14] The extent of resection and the role and expansion of lymphadenectomy are the most significant and ongoing concerns. Since proximal gastric resection is faulty and has a high rate of postoperative complications, the majority of surgeons undertake total gastrectomy in cases of gastric cancer involving the fundus and/or the body of the stomach.

Chemoradiation during surgery A phase III experiment was recently conducted to examine if preoperative chemoradiotherapy, as opposed to chemotherapy alone, could improve survival in patients with locally advanced gastric and gastroesophageal cancer. 119 patients were randomly assigned to receive either 5-fluorouracil, leucovorin, and cisplatin (PLF) followed by surgery or PLF followed by chemoradiation with cisplatin and etoposide and then surgery in the German study PreOperative Chemotherapy or Radiochemotherapy in Esophagogastric Adenocarcinoma Trial.[92] Regretfully, the trial was.

CONCLUSION

Because of its aggressive nature and delayed appearance, gastric cancer remains a major health concern. Dietary variables, lifestyle choices, and H. pylori infection continue to be the main causes of illness development. Effective methods for lowering the burden of disease include early detection through screening and timely H. pylori eradication. Although patient

results have improved because to advancements in surgical techniques, chemotherapy, and endoscopic procedures, survival is still low in advanced stages. Reducing world mortality requires increased awareness, prompt diagnosis, and preventive actions. To provide more effective treatment and diagnostic methods, more research is required.

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