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Review

Gastric Cancer: A Brief Review

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Abstract

Gastric cancer (GC) is a major global health problem and ranks among the most frequently diagnosed and most deadly cancers, causing over 770,000 deaths in 2025. Gastric cancer is a common and highly lethal malignancy worldwide. Its incidence is higher in many Asian and less developed regions and is associated with both modifiable and non-modifiable risk factors, including Helicobacter pylori infection, poor diet, smoking, older age, and low socioeconomic status. Prevention strategies focus on H. pylori eradication, lifestyle modification, and appropriate use of screening programs where incidence is high. Several classification systems, such as those from the World Health Organization and Lauren, guide GC staging and incorporate anatomical, histological, and increasingly molecular characteristics. This review provides an overview of Gastric Cancer including its Introduction, Epidemiology, Risk factors, Prevention, Symptoms, Diagnosis, Treatment. Symptoms are often nonspecific such as weight loss, anemia, abdominal pain, dysphagia, and gastrointestinal bleeding so diagnosis typically relies on upper gastrointestinal endoscopy with biopsy, supported by imaging techniques like computed tomography and endoscopic ultrasound. Surgery with adequate lymph node dissection remains the cornerstone of curative treatment, while chemotherapy, chemoradiotherapy, and targeted or immunotherapies are used in a multimodal approach to improve survival in appropriate patients.

Keywords: Gastric Cancer, Risk Factors, Prevention, Symptoms, Diagnosis, Treatment.

Introduction

Gastric cancer (GC), also known as stomach cancer, constitutes a significant global health burden, originating anatomically from the gastroesophageal junction to the distal stomach regions (1,2). Worldwide, across both sexes, GC ranks as the 5th most prevalent neoplasm (accounting for 4.7% of cases) and the 3rd leading cause of cancer mortality, responsible for approximately 770,000 deaths in 2025 (3). Geographic incidence varies markedly, with roughly 40% of new diagnoses in developed countries, yet the highest burdens persist in Eastern Asia, Latin America, and Central/Eastern Europe due to entrenched risk factors like H. pylori prevalence and dietary patterns(4). Histological Classification include Lauren's classification remains the most clinically utilized system, delineating two primary histological subtypes intestinal and diffuse—with profound etiological (e.g., H. pylori-linked inflammation for intestinal vs. genetic factors like CDH1 mutations for diffuse), pathological (e.g., glandular vs. infiltrative growth), and epidemiological (e.g., declining intestinal rates vs. stable diffuse) distinctions that directly inform prognosis, screening, and therapeutic strategies(5,6). Declining Incidence Trends include Despite persistent high incidence, epidemiological data reveal a gradual global decline in new cases, attributable to multifaceted interventions: widespread H. pylori eradication programs, enhanced access to and preservation of fresh produce via cold-chain logistics, and population-level lifestyle modifications including increased physical activity, reduced dietary sodium (linked to atrophic gastritis), curtailed alcohol consumption (promoting mucosal damage), and declining smoking rates (elevating nitrosamine exposure)(7,8,9).

Risk Factors

The risk factors for gastric cancer are numerous and encompass both modifiable and non-modifiable elements (Fig. 1). Modifiable risk factors include lifestyle choices such as diet, smoking, and alcohol consumption, which can be altered to reduce risk. Non-modifiable risk factors include genetic predispositions, age, and certain medical conditions that are inherent and cannot be changed. Helicobacter pylori, classified as a Group 1 carcinogen, stands as the primary risk factor for gastric cancer,(11,12) particularly non-cardia subtypes, which it causes in nearly 90% of cases worldwide. This bacterium promotes carcinogenesis directly—via protein and gene modifications in gastric epithelial cells—and indirectly, through chronic mucosal inflammation that fosters precancerous changes like atrophic gastritis and intestinal metaplasia.(13,14) Virulence factors such as the cagPAI gene cluster (including CagA), VacA, and outer membrane proteins (OMPs) exacerbate this by inducing persistent infection, DNA damage, oxidative stress, and disrupted cell signaling, elevating risks for gastric adenocarcinoma and MALT lymphoma.(14) Challenges in Eradication and Prevention include Antibiotic-based eradication therapy remains central to prevention, yet antibiotic resistance and treatment failures hinder success.(15) A Korean cohort study noted a non-significant initial trend toward reduced incidence post-treatment, with benefits strengthening over time and with age. Strain variability further complicates risks, as CagA-positive strains confer higher malignancy potential.(16) Critical Role of Treatment Timing include Eradication timing profoundly affects cancer risk reduction, especially before precancerous lesions emerge.(17) Japanese research showed early intervention yielded markedly lower incidence than delayed therapy.(19) A meta-analysis of RCTs confirmed greater efficacy pre-atrophic gastritis or metaplasia, echoed by Taiwanese data indicating lower rates when treated within one year of diagnosis. Thus, prompt eradication in high-risk populations offers the strongest preventive benefit.(18)

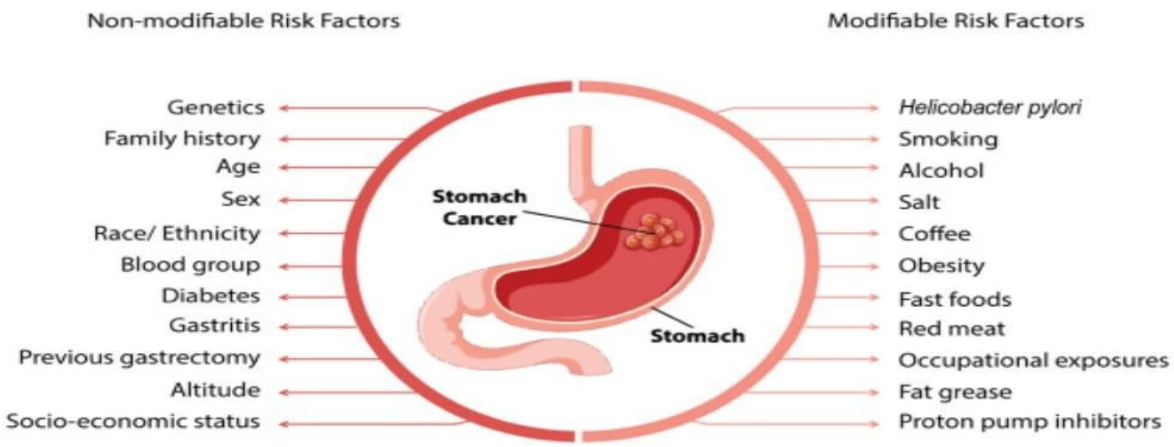

Fig:1. Modifiable and non-modifiable risk factors of gastric cancer.

Prevention

Gastric cancer (GC) prevention encompasses primary and secondary approaches. Primary strategies target modifiable risks through dietary shifts, such as reducing salty food intake while boosting fresh fruits and vegetables, alongside avoiding smoking and excessive alcohol (10).

Primary Prevention include Tactics H. pylori eradication via antibiotics, coupled with enhanced sanitation and hygiene, effectively curbs GC onset. These measures interrupt chronic inflammation pathways early (20,21).

Secondary Prevention include Focus Screening via endoscopy or photofluorography enables early detection and intervention in high-risk cases (22). H. pylori treatment further lowers GC risk and mortality rates (23).

Histopathological Classifications

WHO classifies gastric adenocarcinoma into papillary, tubular, mucinous, mixed, and poorly cohesive types (including signet ring cell carcinoma) (24,25). Lauren's widely used system divides adenocarcinomas into intestinal (arising from chronic gastritis-atrophy-metaplasia-dysplasia sequence), diffuse (infiltrating single cells/clusters), and indeterminate types (27,25). Ming's system distinguishes expansive (mass-forming nodules) from infiltrative (wall-invading cells) growths. Molecular Sub groups Recent molecular profiling identifies four GC subtypes (28,30): Epstein-Barr virus-associated (EBVaGC) about 10% of cases; features hypermethylation silencing tumor suppressors, atrophic gastritis); chromosomally unstable (copy-number gains like 8q, 17q, 20q in intestinal, 12q, 13q in diffuse) microsatellite unstable (31,32) (high mutation rates in oncogenic pathways); and genomically stable (linked to diffuse histology, rare mutations). EBV serological screening faces challenges from widespread prior exposure and marker limitations (33).

Symptoms

Gastric cancer (GC) symptoms often appear vague and mimic common digestive issues early on, delaying diagnosis until advanced stages.

Early Symptoms include Indigestion, heartburn, bloating after small meals, loss of appetite, mild nausea, and vague upper abdominal discomfort frequently signal initial GC, often persisting despite treatment. Early satiety (feeling full quickly) and burping also commonly occur (34,35).

Advanced Symptoms include Unexplained weight loss, persistent abdominal pain, vomiting (possibly bloody), difficulty swallowing, black/tarry stools from bleeding, fatigue, and anemia become prominent as the tumor grows or spreads. Swelling (ascites), jaundice, or weakness may indicate stage 4 involvement (36).

Diagnosis

Gastric cancer (GC) diagnosis begins with a thorough patient history and physical examination. Upper gastrointestinal endoscopy

with biopsy is essential for confirming suspicion, assessing lesion location, and extent (37).

Endoscopic Findings includes and reveals polypoid, ulcerative lesions, spontaneous bleeding, abnormal mucosal coloring, or gastritis-like changes (38). Superficial tumors classify into five types: (a) depressed (protruding into tissue with converging folds and abrupt edges) (b) flat (no projections, reddish/whitish hue) (c) elevated (slightly raised, red/pale, globular/lobular) (39) (d) protruding (polypoid) (e) excavated (deep tissue invasion) (40).

Imaging and Labs Computed tomography evaluates tumor site, metastases, and mucosal lesions; endoscopic ultrasound assesses wall invasion depth, though it's costlier and more invasive. Serum CA 72-4 levels aid diagnosis, correlating with advanced disease, but require validation studies (41,42).

Screening Programs Population includes screening via endoscopy/photofluorography boosts early detection in high-incidence Asian regions, reducing mortality and improving survival, as shown in Korean studies viable for high-risk groups elsewhere (43,44).

Staging Protocols includes Post-diagnosis, TNM staging by the American Joint Committee on Cancer (AJCC) and Union for International Cancer Control (UICC) guides therapy selection (45,46).

Treatment

Gastric cancer (GC) prognosis and treatment depend on tumor location, staging, lymph node involvement, and metastasis presence. No universal adjuvant therapy standard exists globally; regional phase III trials shape local protocols, with multidisciplinary teams weighing risks and benefits(47,48).

Early GC Management (Stages 0-IA)

ESMO guidelines endorse endoscopic resection for mucosal, well-differentiated tumors ≤2 cm without ulcers. Laparoscopic gastrectomy suits early GC, reducing trauma/complications despite D2 lymphadenectomy uncertainties versus open surgery(49).

Locally Advanced GC (Stages IB-III)

Total/subtotal gastrectomy with perioperative therapy is standard. Surgery alone yields poor survival; multimodal approaches excel—MAGIC trial showed perioperative chemotherapy (e.g., ECF) superior to surgery alone for overall survival (OS)(48). CRITICS confirmed perioperative chemo outperforms preoperative chemo + postoperative chemoradiotherapy. (50)

Regional variations include neoadjuvant chemotherapy (Europe), postoperative chemoradiation (USA), and adjuvant oral agents (Asia, e.g., S-1) outperforming surgery alone. Laparoscopic advantages (less trauma, shorter stays) favor it over open gastrectomy, except in obesity; D2 lymphadenectomy trumps D1 but risks high morbidity outside expert centers. (51&52)

Advanced/Metastatic GC (Stage IV)

Palliative therapy for unresectable/recurrent disease prioritizes ECOG PS ≤2 or Karnofsky ≥60, considering comorbidities/toxicity. (53) First-line platinum-fluoropyrimidine doublets add trastuzumab for HER2+ cases; conversion therapy may enable resection (54). Second-line (e.g., ramucirumab + paclitaxel) extends OS in fit patients, alongside continuous supportive care for physical/mental health and recurrence detection (55).

Conclusion

Gastric cancer causes high global morbidity and mortality. Advances in classification, diagnosis, and treatments, including endoscopic surgery for early resectable cases, have improved outcomes and reduced complications. New molecular markers and immunotherapies offer promise for metastatic disease management. Ongoing trials evaluate optimal combinations of chemotherapy, immunotherapy, and radiation for both surgical and non-surgical cases.

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