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Review Article

Advancements in technology for the diagnosis and treatment of gastric cancer: A narrative review.

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Abstract

Gastric cancer is a major global health concern, ranking as the fifth most commonly diagnosed cancer and a leading cause of mortality, often detected at advanced stages. It's a multifactorial disease influenced by genetics, H. pylori infection, environmental factors, and diet, predominantly affecting older men. Approximately 90% are adenocarcinomas, characterized by specific molecular biomarkers. Diagnosis primarily relies on EGD with biopsy. Historically, treatment involved surgery and chemotherapy, but the field is rapidly evolving towards targeted and immunotherapies. Crucially, the CRISPR-Cas9 gene editing system has emerged as a promising tool with the potential to precisely modify genes involved in gastric carcinogenesis, offering novel therapeutic avenues.

This narrative review highlights that gastric cancer remains a major global health challenge, driven by multifactorial risk factors including H. pylori infection, dietary habits, and genetic predispositions, often presenting with non-specific symptoms that lead to late diagnosis. While diagnostic tools have advanced from EGD to AI-assisted endoscopy, and TNM staging has been refined, molecular heterogeneity necessitates the integration of biomarkers for personalized approaches. Therapeutically, management has evolved significantly, encompassing perioperative chemotherapy and surgical techniques (from endoscopic resections to D2 lymphadenectomy), increasingly integrating targeted therapies (like HER2 inhibitors) and immunotherapies for advanced disease. Crucially, emerging CRISPR-Cas9 gene editing technology shows immense promise for future therapeutic interventions, aiming to address genetic drivers of carcinogenesis and further revolutionize patient outcomes.

Hence, gastric cancer remains a significant global health challenge, with its complex nature (risk factors, symptoms, and diagnosis) necessitating a continuous evolution in its management. Advancements in diagnostic tools, refined staging, and the integration of molecular insights are driving personalized treatments, while CRISPR-Cas9 gene editing holds transformative potential for future therapies.

Keywords: Gastric cancer, epidemiology, diagnosis, treatment, CRISPR-Cas9, gene editing, precision medicine.
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Introduction

Cancer is a major global health concern. Gastric cancer is the 5th most commonly diagnosed and is known to be amongst the leading causes of mortality [1,2]. Gastric cancer is a multifactorial disease involving risk factors such as genetics, *H.pylori* infection, Epstein Barr Virus infections, environmental factors, dietary factors (such as very high salt intake, less intake of fruits and vegetables, increased smoked food consumption), social environment and advanced age [2,3,4]. Gastric cancer is mainly seen in

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middle aged and elderly people (50-70 years old), with a relatively higher incidence amongst men as opposed to women [4]. The overall 5-year survival rate of gastric cancer in the US is 32.4% [5].

Approximately 90% of gastric cancers are adenocarcinomas, which arise from glands in the superficial mucosa of the stomach [6]. Molecular biomarkers often associated with gastric cancer include HER2, p53, PD1, p73, mdm2, Bcl2, pRb-CCND1, p16, MUC, MRP2, MDR1, GST-P, MSI. The most common symptoms patients will exhibit include weight loss, persistent abdominal pain, occult gastrointestinal bleeding and iron deficiency anemia. Esophagogastroduodenoscopy (EGD) is the diagnostic test of choice. Since the procedure provides a tissue diagnosis and direct tumor visualization/localization, it is highly sensitive and specific.[5]. Therapeutically, techniques such as endoscopic mucosal resection (EMR) and endoscopic submucosal resection (ESD) are useful in cases of early gastric cancer with lower lymph node metastasis. Therapeutic treatment options for advanced gastric cancer include surgery and chemotherapy [7].

CRISPR and CRISPR associated proteins (Cas) are important parts of the adaptive immune system [8]. The CRISPR-Cas 9 system can be used for gene edition, expression, site directed mutation and functional studies [8]. In cancer treatment, they have been used in immunotherapy, manipulation of cancer genome sequencing, epigenome mapping along with inactivation and elimination of carcinogenic viral infections [9]. Development of gastric cancer is typically preceded by the occurrence of chronic gastritis, atrophic gastritis and intestinal metaplasia; these are associated with mutations in oncogenes such as KRAS, PIK3CA, and tumor suppressor genes (such as TP53, AT-rich interactive domain-containing protein 1A (ARID1A), cadherin-1 (CDH1), etc.) [10]. CRISPR has proven to be a promising tool to modify genes specific to gastric carcinogenesis and also has the potential to reverse these processes [11].

This review aims to illustrate the evolution of therapeutic treatment for gastric cancer.

Epidemiology and Risk factors of Gastric cancer:

Understanding the epidemiological landscape of gastric cancer is essential, as its global burden remains significant despite declining incidence rates in many regions due to improvements in food preservation and Helicobacter pylori eradication strategies [12]. In 2022, GLOBOCAN estimates indicated approximately 968,350 new cases of diagnosed gastric cancer, contributing to 4.9% of the global cancer burden while causing roughly 659,853 deaths which is close to 6.8% of global cancer mortality [13,14]. The global age-standardized incidence rate (ASIR) is estimated at 15.6 per 100,000 for men and 7.7 per 100,000 for women, indicating a clear male predominance [15,16]. There have been many hypotheses put forward to explain this gender disparity. These encompass the protective benefits of estrogen alongside lifestyle-related hazards like smoking, alcohol consumption, dietary habits and so on [17,18].

The incidence of gastric cancer varies markedly across different geographic regions. The highest rates are observed in East Asia, especially in Japan, South Korea, and China, which together account for more than 50% of global cases. Intermediate rates are seen in Eastern Europe and parts of South America. In contrast, North America, Northern Europe, and most parts of Africa have lower incidence rates [19, 20].

Anatomically, gastric cancer is divided into cardia and non-cardia types. Cardia cancers, more common in Western countries, are associated with GERD, obesity, and smoking, whereas non-cardia cancers, prevalent in Asia and Latin America, are strongly linked to Helicobacter pylori infection [21,22].

The regional distribution of gastric cancer and occurrence of Helicobacter pylori infection can be explained by an interplay between bacterial strain diversity, environmental conditions, and host factors. Distinct phylogeographic strains with varying virulence and resistance—such as Europe in Western countries and East Asia in China, Korea, and Japan—migrate and evolve with human populations. Countries like Nigeria, India, and China show high prevalence rates exceeding 50–70%, driven by poor sanitation, overcrowding, and early-life exposure, whereas lower rates are observed in developed regions like the United States (~35%) and Australia (~24%). These regional strains interact with local socioeconomic factors and host-specific genetics and diet, shaping the global burden and clinical outcomes of H. pylori infection, particularly gastric cancer incidence [23].

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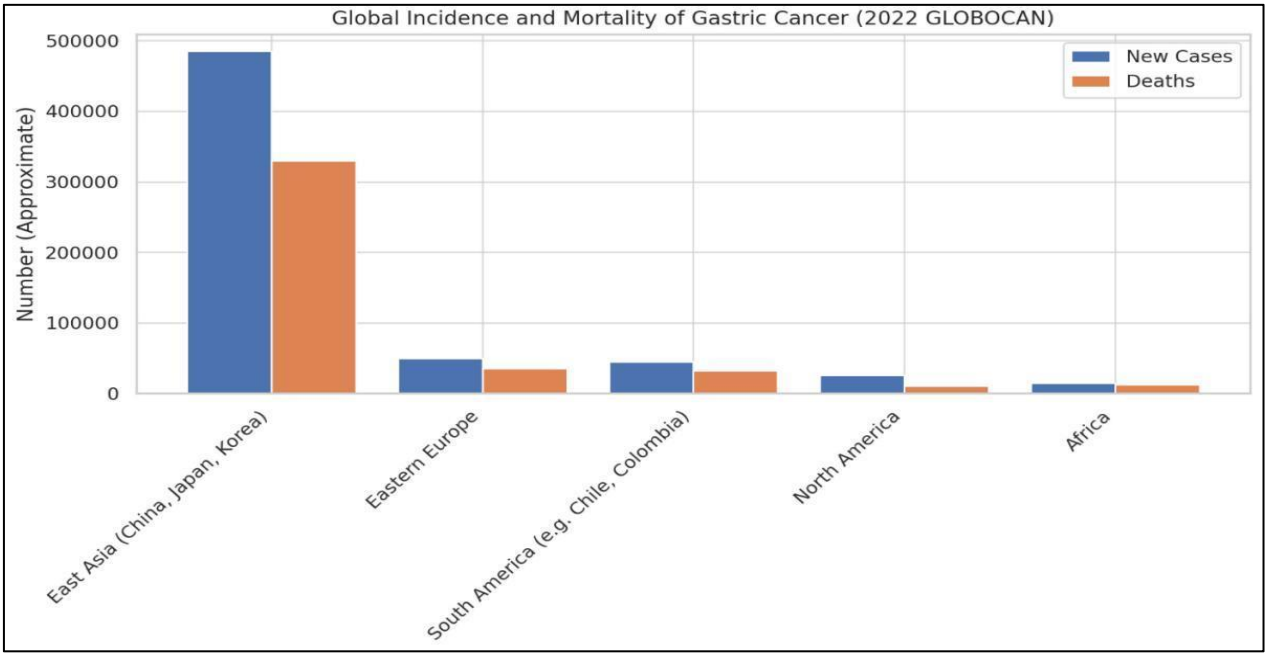
Socioeconomic status and related determinants such as poor sanitation, crowded living conditions, and limited access to healthcare influence both the acquisition of H. pylori infection and delays in diagnosis. Individuals from lower socioeconomic backgrounds are often diagnosed at advanced stages, contributing to higher mortality rates [24]. Dietary habits play a substantial role in shaping regional differences in gastric cancer risk. In East Asia, for instance, frequent consumption of salted, smoked, and pickled foods—which are high in nitrates and nitrites—has been strongly associated with an increased incidence of gastric cancer. Conversely, diets rich in fresh fruits and vegetables appear to offer a protective effect. The antioxidants and vitamin C found in these foods help counteract oxidative DNA damage and can inhibit the formation of carcinogenic nitrosamines [25,26]. Genetic predisposition, while responsible for only a minority of cases, remains significant in particular familial syndromes. For example, hereditary diffuse gastric cancer, linked to germline mutations in the CDH1 gene, substantially raises lifetime risk and often manifests at a younger age. Other inherited conditions, such as Lynch syndrome and familial adenomatous polyposis (FAP), also contribute to heightened susceptibility to gastric cancer [27,28]

Figure 1: Global Incidence and mortality of Gastric Cancer (2022 GLOBOCAN)



Clinical features

Gastric cancer generally presents with indefinite symptoms, chiefly in early stages. This vagueness imparts diagnostic delays and frequently results in detection at advanced stages or metastasis spread.

Early signs may comprise epigastric pain/discomfort, early satiety, anorexia, nausea, dysphagia and dyspepsia, fewer than 20% of cases present overt gastrointestinal bleeding, such as melena or hematemesis [29] while advanced stages cover alarm features which include systemic manifestations like unintentional weight loss, cachexia, anemia due to GI blood loss may be iron deficiency or normocytic if chronic and palpable nodes [30]. Symptoms also differ based on severity of disease and tumor location.

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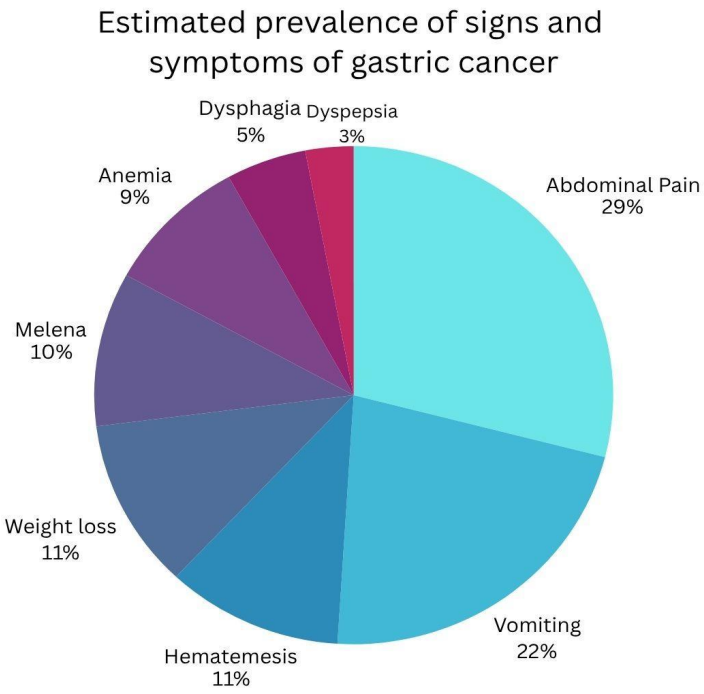
Recent evidence highlights sarcopenia as a common comorbidity in gastric cancer, affecting nearly 27% of patients, and showing strong associations with advanced age, larger tumor size, and poor nutritional status [31, (Figure 2)32].

Figure 2: Prevalence rates of signs and symptoms of gastric cancer



In its advanced stages, gastric cancer frequently spreads to regional lymph nodes and distant organs, leading to a variety of clinical signs that reflect metastatic involvement. Enlargement of the left supraclavicular lymph node, known as Virchow's node, is a well-recognized indicator of retrograde lymphatic spread from the abdomen through the thoracic duct. Similarly, a Sister Mary Joseph nodule, a firm periumbilical lesion, often signals peritoneal metastasis and is typically associated with a poor prognosis. Irish node, representing metastasis to the left axillary lymph nodes, is another less common but notable finding. During digital rectal examination, a firm, shelf-like mass—referred to as Blumer's shelf—may be palpable in the rectouterine or rectovesical pouch, suggesting peritoneal carcinomatosis. In female patients, Krukenberg tumors, which are metastatic lesions to the ovaries, can present with nonspecific abdominal discomfort or distension [33,34]. Additional signs such as ascites, due to peritoneal seeding, and jaundice, typically arising from liver metastases or biliary obstruction, may also be present in disseminated disease [34]. Recognition of these features is crucial for staging and guiding further management.

Though rare, paraneoplastic syndromes can occur in gastric cancer and might show as polyarteritis nodosa, Trousseau's syndrome, disseminated intravascular coagulation, microangiopathic hemolytic anemia, and membranous nephropathy [35,36]. Dermatologic signs such as Leser–Trélat sign—a sudden eruption of seborrheic keratoses—and acanthosis nigricans, marked by velvety hyperpigmented plaques in skin folds, are also notable

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indicators [37,38] These features may precede cancer diagnosis and often resolve with treatment of the underlying tumor.

The clinical presentation of gastric cancer largely depends on the tumor's anatomical location. Tumors located in the cardia and gastroesophageal junction often mimic esophageal cancer, presenting with dysphagia, retrosternal discomfort, and reflux-like symptoms. In contrast, non-cardia tumors—including those in the body, antrum, and pylorus—are more likely to cause epigastric pain, early satiety, postprandial fullness, and vomiting, often due to partial obstruction. [39] Tumors in the pyloric region may progress to gastric outlet obstruction, leading to persistent vomiting and metabolic alkalosis. The diffuse infiltrative subtype, known as linitis plastica, typically causes abdominal distension, rapid weight loss, and minimal dyspeptic symptoms, reflecting widespread submucosal involvement and significant thickening of the stomach wall. [40]

Diagnosis

Gastric cancer is often diagnosed at a later stage due to its non-specific presenting symptoms, which can lead to a poorer prognosis [41]. Physical exam findings are typically apparent only in advanced stages of the disease. The primary diagnostic tool for patients with suspected gastric cancer is esophagogastroduodenoscopy (EGD), which is highly sensitive and specific, especially when combined with endoscopic biopsy for tissue diagnosis [5].

Endoscopic ultrasound (EUS) plays a significant role in staging via assessment of perigastric lymph nodes, which can be biopsied by EUS-guided fine-needle aspiration [41]. EUS allows accurate evaluation and staging of tumors along with the extent of tumor infiltration into the layers of the stomach [42]. Computed tomography (CT) scans of the chest and abdomen help evaluate local and distant lymphadenopathy, metastasis, and ascites, which aids in tumor staging [42]. Multi-row detector computed tomography (MDCT) allows multiplanar reconstructions, 3D image reconstruction, virtual endoscopy, contrast-enhanced examination, and faster imaging [42].

Recent advancements in diagnostic techniques include artificial intelligence (AI)-assisted endoscopic diagnosis, which can evaluate image features, detect early gastric cancer, and precancerous conditions [43]. Confocal laser endomicroscopy (CLE) provides high-resolution imaging and can help diagnose gastric cancer [44]. Texture and colour enhancement imaging (TXI) improves the texture, brightness, and color tone of lesion images, allowing for more accurate detection [44].

Several AI-based diagnostic systems have been developed, including WISENSE, a real-time assistance system that evaluates the entire stomach during endoscopy [45]. The Gastrointestinal Artificial Intelligence Diagnostic System (GRAIDS) is a real-time AI system that diagnoses upper gastrointestinal cancers with high accuracy [45]. ENDOANGEL LD is an AI-based system that detects early gastric cancer with high sensitivity and specificity [5]. AI-Scope is a Korean AI-based model that detects gastric mucosal lesions and estimates the depth of tumor invasion [5].

These advanced diagnostic techniques and AI-based systems have the potential to improve the accuracy and efficiency of gastric cancer diagnosis, particularly in early stages. Further research is needed to fully realize their potential and improve patient outcomes.

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TABLE 1 Diagnostic modalities and their recommended use as per grades of gastric cancer [46]

Diagnostic evaluation	Recommendations for Grade I gastric carcinoma	Recommendations for Grade II gastric carcinoma	Recommendations for Grade III gastric carcinoma
For definitive diagnosis of tumor	1. Gastroscopy should be done along with biopsy of the tumor.	1. Cytological examination of the tumor.	
Local examination	1. Gastroscopy should be done with or without Abdominal enhanced computed tomography.	2. Abdominal magnetic resonance imaging.	1. X ray barium double contrast radiography can be done in potential cases of Grade III gastric cancer.
Biopsy examination	1. Histopathological examination of the lesion	3. Immunohistochemical examination	1. Examination related to and for *H.Pylori* infection.
Evaluation of tumor staging	1. Abdominal and pelvic enhanced along with chest computed tomography 2. Endoscopic ultrasound scan(EUS)	4. Abdominal magnetic resonance imaging 5. PET-CT(Positron emission tomography- computed tomography) 6. Diagnostic laparoscopy along with examination of intraperitoneal washings	
Post-treatment evaluation	1. Abdominal and pelvic enhanced along with chest computed tomography	7. Abdominal magnetic resonance imaging 8. PET-CT(Positron emission tomography- computed tomography) 9. Gastroscopy	

Biomarkers

Gastric cancer is a complex disease that requires accurate diagnosis and treatment. Various biomarkers have been identified as potential diagnostic tools, including protein biomarkers, genetic and epigenetic biomarkers, RNA-based biomarkers, and emerging biomarkers. Protein biomarkers such as carcinoembryonic antigen (CEA), cancer antigen 19-9 (CA 19-9), and cancer antigen 72-4 (CA 72-4) are commonly used in gastric cancer diagnosis [47]. CEA is elevated in advanced disease and linked to liver metastasis relapse, while CA19-9 correlates with antral tumor location, differentiated histology, lymphovascular invasion, and advanced stage [47]. Combining CEA and CA19-9 improves diagnostic sensitivity to ~87% [47].

HER2 (ERBB2) is another established biomarker, with overexpression/amplification in 6–23% of GCs, especially intestinal subtype [47]. The pivotal phase III ToGA trial demonstrated trastuzumab plus chemotherapy improves survival in HER2-positive advanced GC, leading to its 2010 EMA approval and routine HER2 testing [47]. However, HER2 overexpression shows intratumoral

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Review Article

heterogeneity, requiring specialized assessment distinct from breast cancer protocols [48].

Genetic and epigenetic biomarkers, such as CDH1 promoter hypermethylation, microsatellite instability (MSI), and Epstein-Barr virus (EBV)-associated methylation patterns, offer additional precision in tumor subtyping and therapy selection [49,50,51]. MSI is a key biomarker in gastric cancer, with diagnostic, prognostic, and predictive significance [50]. EBV infection induces aberrant gene expression through promoter methylation, implicating virus-associated methylation patterns as potential biomarkers [51].

RNA-based biomarkers, such as microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), have emerged as pivotal regulators of gastric carcinogenesis. miR-21, miR-106b, and miR-421 were significantly upregulated in gastric cancer [47, 52]. HOTAIR and MALAT1 have been identified as prominent regulators of gastric cancer progression, influencing tumor invasion, metastasis, and epithelial-mesenchymal transition [53,54].

Emerging biomarkers, such as circulating tumor DNA (ctDNA), exosomal microRNAs, and serum or gastric fluid metabolite panels, offer potential for real-time monitoring of tumor dynamics, prognosis, and recurrence [55,56,57]. ctDNA levels were significantly associated with clinical outcomes in resectable gastric cancer [55]. Exosomal microRNAs and serum metabolite panels showed promise as non-invasive biomarkers [56,57].

Gastric juice provides a proximal, organ-specific medium for biomarker identification in gastric cancer, offering enhanced specificity over peripheral biofluids [58]. Studies highlight the presence of microRNAs, DNA methylation markers, and proteins in gastric juice with diagnostic relevance [59].

These biomarkers have the potential to improve gastric cancer diagnosis and treatment. Further research is needed to fully realize their potential and improve patient outcomes.

TNM Staging

The TNM staging system, jointly developed by the American Joint Committee on Cancer (AJCC) and the Union for International Cancer Control (UICC), is the most widely used framework for assessing the stage of gastric cancer. It provides a consistent method for describing the anatomical extent of the disease, which plays a crucial role in guiding treatment decisions, estimating prognosis, and facilitating clinical communication. The system evaluates three main components: the depth of tumor infiltration into the stomach wall (T), the extent of involvement of nearby lymph nodes (N), and the presence or absence of cancer spread to distant organs (M) [60, 61]. The AJCC 8th edition staging system, implemented in 2017, brought notable enhancements to gastric cancer classification, particularly through a refined nodal staging structure and redefined stage groupings, aimed at improving prognostic stratification and reducing stage migration. Effective application of this system depends on comprehensive histopathological assessment, high-quality imaging, and adequate lymph node dissection to ensure staging accuracy [62].

The 8th edition of the AJCC/UICC TNM staging system introduced significant updates to the pathological stage classification for gastric cancer, particularly within stage III. A major revision was the formal incorporation of the N3 subcategories—N3a (7–15 positive lymph nodes) and N3b (≥16 positive nodes)—into stage groupings, which was not the case in the 7th edition. This change resulted in the reallocation of several tumor categories to better reflect prognostic differences. For instance, T1N3bM0 and T2N3bM0 were upstaged from stage IIB and IIIA to IIIB, while T3N3bM0 was reclassified from IIIB to IIIC. Conversely, certain classifications such as T4bN0M0 and T4aN2M0 were down-staged to IIIA, and notably, T4aN3aM0 and T4bN2M0 were reassigned to stage IIIB[62,63], which outlines the key differences in stage groupings between the 7th and 8th editions.

The 8th AJCC edition also introduced clinical (cStage) and post-neoadjuvant (ypStage) classifications to reflect the increasing use of neoadjuvant therapy in resectable gastric cancer. These new stage groupings help assess prognosis and guide further treatment after preoperative therapy[67].

In the 8th edition of the AJCC/UICC TNM staging system, tumors with their epicenter located more than 2 cm distal to the esophagogastric junction (EGJ), or those within 2 cm of the EGJ that do not invade it, are classified as gastric cancers. Conversely, tumors centered within 2 cm of the EGJ that extend into the junction are staged as esophageal cancers. This revision leads to a reclassification of Siewert type III tumors—previously staged as esophageal cancers

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Review Article

in the 7th edition—as gastric cancers under the current system [68,69].

Treatment strategies in gastric cancer are closely tied to disease stage. In early-stage cases (T1a or T1b), minimally invasive options like endoscopic submucosal dissection (ESD) or limited surgery may be appropriate, depending on tumor features and patient health [70]. For stages II and III, a combined approach using neoadjuvant chemotherapy, surgery, and postoperative therapy is standard [71,72]. In metastatic (stage IV) disease, palliative systemic treatments—including HER2-targeted agents when applicable—are typically employed [73,74].

Surgical decisions are informed by TNM staging, determining the need for distal or total gastrectomy and whether to perform limited (D1) or extended (D2) lymph node dissection [75]. Although not officially part of TNM, positive peritoneal cytology (CY1) is often treated as an indicator of poor prognosis, influencing surgical intent.

Because survival rates can differ significantly even within the same stage, newer prognostic tools now incorporate factors such as age, tumor size and location, histologic subtype, lymph node status, margin status, and use of adjuvant therapy. Genomic and molecular characteristics—including microsatellite instability (MSI), Epstein–Barr virus (EBV) status, and molecular subtypes—are being studied for their potential to enhance personalized treatment and improve outcome prediction [76].

Survival statistics from datasets like the National Cancer Database (NCDB) and Shizuoka Cancer Center show marked differences across stages. Five-year survival in stage I disease exceeds 90% in some populations, whereas stage IV disease often carries a median survival under 7 months [77]. These patterns highlight the essential role of staging in guiding both prognosis and therapeutic decisions.

The TNM staging system, while widely adopted, has several notable limitations. One of its key drawbacks is its reliance solely on anatomical factors, without accounting for the molecular heterogeneity of tumors, which plays a crucial role in influencing prognosis and treatment response [77]. Another significant concern is the phenomenon of stage migration, particularly when lymph node dissection is inadequate [78]. This statistical artifact, often referred to as the Will Rogers phenomenon, can lead to inaccurate staging and misleading survival data [79]. Both the 7th and 8th editions of the TNM system base nodal classification on the absolute count of metastatic lymph nodes (MLNs), which makes them vulnerable to such biases [80]. To address this, alternative approaches such as the lymph node ratio (LNR)—the proportion of positive to examined lymph nodes—and the log odds of positive lymph nodes (LODDS) have been developed. These methods reduce the impact of variable lymphadenectomy and have demonstrated improved prognostic accuracy in multiple studies. Furthermore, emerging evidence suggests that incorporating the anatomical distribution of involved lymph nodes, in addition to their number, could enhance staging precision and better reflect disease burden in gastric cancer [81].

While the TNM staging system remains the foundation for clinical decision-making in gastric cancer, it does not encompass the full biological complexity of the disease. Recent advances have introduced supplementary approaches that offer greater precision in risk stratification. Molecular subtyping, such as the classification proposed by The Cancer Genome Atlas (TCGA), divides gastric cancer into subgroups including Epstein–Barr virus-associated (EBV+), microsatellite instability-high (MSI-H), genomically stable, and chromosomal instability types, each with distinct therapeutic and prognostic relevance [82-84]. Additionally, imaging-based analytics using radiomics and artificial intelligence (AI) are under investigation for their ability to predict disease stage and therapeutic response, though they are not yet part of routine practice [85,86]. Circulating tumor DNA (ctDNA) is another promising tool, potentially useful for detecting minimal residual disease and early dissemination, thereby complementing existing staging systems [84]. Moreover, positive peritoneal cytology (CY1), although not formally included in TNM classification, is often clinically regarded as metastatic due to its association with poor outcomes [85]. Immunological markers such as PD-L1 expression and MSI status are also gaining importance, particularly in selecting candidates for immunotherapy [86]. These evolving tools underscore the need for a more integrated staging model that goes beyond anatomical features to include molecular and functional tumor characteristics.

Treatment

Surgical treatment

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Surgical resection is the primary modality for curative treatment in gastric cancer, especially in patients with resectable tumors, while systemic therapies support or complement surgical outcomes. The goal of surgical treatment is a complete (R0) resection—defined as microscopically margin-negative excision of the primary tumor and associated lymphatic drainage—without leaving residual malignant tissue [87]. According to the Union for International Cancer Control (UICC), this entails en bloc removal of the tumor with an adequate lymphadenectomy, ensuring oncological safety.

Patient Selection

Optimal outcomes in gastric cancer surgery depend critically on appropriate patient selection and meticulous preoperative planning. The decision to proceed with surgery is typically guided by a multidisciplinary team (MDT) approach that integrates oncologic staging, surgical feasibility, nutritional assessment, anesthetic evaluation, and patient preferences [88]. Early-stage tumors without metastasis are typically treated with curative resection. In contrast, patients with locally advanced disease may benefit from neoadjuvant chemotherapy—such as the FLOT regimen—to increase the likelihood of complete (R0) resection and improve survival outcomes [89]. Assessment of functional status using tools like the ECOG or Karnofsky scales helps predict surgical risk; poor performance may contraindicate major surgery [90]. Nutritional optimization is essential, as malnutrition is common and correlates with increased postoperative complications and poorer prognosis [91]. Comorbidities such as cardiopulmonary or renal dysfunction also influence perioperative decisions and anesthesia planning. Patient preferences should guide choices between open, minimally invasive, or robotic approaches. In cases of peritoneal carcinomatosis, cytoreductive surgery (CRS) offers better outcomes when performed in the early stages, particularly when complete macroscopic tumor clearance can be achieved. The volume of peritoneal disease and tumor histology are key factors influencing prognosis and surgical outcomes. However, CRS combined with hyperthermic intraperitoneal chemotherapy (HIPEC) remains investigational and is typically considered only in highly selected patients—such as those with limited peritoneal spread and favorable biology—usually within the context of clinical trials [92]

Management of Early Gastric Cancer (EGC)

For early gastric cancer (EGC), particularly in lesions with a low risk of lymph node metastasis (LNM), endoscopic resection (ER) - comprising endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) - offers a curative, organ-preserving treatment option. EMR is generally indicated for mucosal tumors ≤2 cm in size, with differentiated histology and no ulceration. In contrast, ESD is preferred for larger lesions, those with ulceration, or those falling under expanded indications, due to its higher en bloc resection rates and lower recurrence risk. A resection is considered curative when it achieves en bloc removal with negative margins (R0), absence of lymphovascular invasion, and conforms to established criteria regarding tumor size and depth. Key factors that determine suitability for ER—including invasion depth, tumor size, ulcerative status, histological type, and lymphovascular involvement—are outlined in **Table 2** [93].

TABLE 2: Endoscopic Resection Criteria in Early Gastric Cancer

Clinical Factor	Standard/Absolute Indication	Expanded Indication	Not Suitable for ER (Consider Surgery)
Tumor Depth	Confined to mucosa (T1a)	Slight invasion into upper submucosa (T1b, ≤500 µm)	Deep submucosal invasion (>500 µm)
Tumor Size	≤2 cm (especially for EMR); ≤3 cm (ESD for well-differentiated tumors)	≤3 cm (with some high-risk features, treated with caution)	Larger tumors >3 cm or with irregular margins
Ulceration	No ulceration (UL0)	Mild ulceration allowed (UL1), if other features are	Ulcerated large or invasive lesions

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		favorable	
Tumor Type (Histology)	Well or moderately differentiated adenocarcinoma	Well or moderately differentiated adenocarcinoma	Poorly differentiated tumors with ulceration or size >2 cm
Lymphovascular Invasion	Must be absent	Must be absent	Presence of lymphatic or vascular invasion
Lymph Node Risk	Very low risk (<1%)	Low risk (<3%)	Moderate to high risk of nodal spread
Resection Margins	En bloc resection with clear margins (R0)	En bloc resection with clear margins (R0)	Piecemeal removal or positive margins
Curative Potential	Considered curative with very low recurrence risk	May still be curative, but needs close follow-up	Considered non-curative— surgery recommended
Suitability for ER	Ideal candidates	Consider if patient prefers non-surgical route and meets criteria	Surgery or multimodal treatment recommended

To guide post-endoscopic resection (ER) management, especially in cases falling under expanded indications or where curative status remains uncertain, the eCura scoring system serves as a validated risk stratification tool. It incorporates five pathological parameters: tumor size >30 mm, submucosal invasion ≥500 μm, lymphatic invasion, venous invasion, and a positive vertical resection margin. Based on the cumulative score, patients are categorized into three groups: eCura A (low risk), eCura B (intermediate risk), and eCura C (high risk). This classification assists in determining subsequent management strategies. Patients in the eCura A group can typically be managed with regular surveillance, given their minimal risk of lymph node metastasis (LNM). In contrast, those in the eCura C category are generally advised to undergo additional gastrectomy with lymphadenectomy due to a substantially increased risk of LNM. The eCura B group represents an intermediate-risk population, for whom management should be individualized. Decisions in this group often consider factors such as patient age, comorbidities, and surgical fitness. While gastrectomy may be recommended for younger, medically fit individuals, a strategy of close surveillance may be appropriate for older patients or those with significant operative risk [94].

Extent of gastrectomy and Reconstruction techniques

The extent of gastrectomy in gastric cancer surgery is primarily determined by tumor location, size, depth of invasion, histologic subtype, and the need for achieving oncologically safe margins [95]. Following resection, the choice of reconstruction technique plays a crucial role in restoring gastrointestinal continuity, maintaining nutritional function, and minimizing postoperative complications such as reflux and dumping syndrome.

Distal gastrectomy is the standard treatment for tumors located in the antrum or lower third of the stomach when a proximal margin of 4–6 cm is achievable. It includes resection of the distal two-thirds of the stomach along with regional lymphadenectomy [96]. For early-stage, node-negative cancers (cT1N0M0) in the middle third, pylorus-preserving gastrectomy (PPG) may be considered to reduce bile reflux, dumping syndrome, and gallstone formation while preserving postoperative nutrition and function [97]. However, its application is limited due to concerns over compromised lymph node dissection, particularly in the infrapyloric region.

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Review Article

Reconstruction after distal gastrectomy is critical to restore continuity and minimize complications. Billroth I (gastroduodenostomy) is preferred when feasible due to its simplicity and physiological route. Billroth II (gastrojejunostomy) is more flexible but has higher rates of bile reflux. Roux-en-Y gastrojejunostomy, though more technically demanding, is increasingly favored for its reduced reflux and better long-term outcomes [98].

Total gastrectomy is indicated for tumors involving the upper third of the stomach, diffuse-type histology, multiple synchronous tumors, or cases where adequate proximal margins cannot be ensured. This involves complete removal of the stomach and surrounding lymph nodes, leaving no gastric tissue behind [99]. Reconstruction is achieved via a Roux-en-Y esophagojejunostomy, which directly connects the esophagus to the jejunum, helping to prevent regurgitation of bile and pancreatic juices into the esophagus. Although some patients experience nutritional deficiencies and altered eating habits postoperatively, this approach remains oncologically robust. A jejunal pouch may be created in select patients to serve as a reservoir and improve postoperative nutrition and quality of life, though its benefit remains variable across studies.

Proximal gastrectomy is occasionally performed for early-stage tumors confined to the upper stomach (cardia or fundus) to preserve more gastric function. While this approach is less invasive than total gastrectomy, it presents technical challenges in reconstruction [100]. Options include esophagogastrostomy, which can lead to severe reflux due to disruption of the lower esophageal sphincter, and double-tract reconstruction, which diverts part of the food stream into the jejunum, reducing reflux and improving nutritional outcomes. Despite potential advantages, proximal gastrectomy is generally reserved for carefully selected patients due to its higher rates of anastomotic complications [101,102].

Lymphadenectomy

Lymphadenectomy is a fundamental component of curative gastric cancer surgery, serving both diagnostic and therapeutic roles. It allows for accurate pathological staging (especially the pN stage) [103], guiding appropriate adjuvant therapy, and facilitates removal of microscopic metastases not detectable on imaging or intraoperative inspection. The extent of lymph node dissection is categorized as D1, D1+, D2, and D3, based on the anatomical stations excised. D1 dissection includes only perigastric nodes (stations 1–6), while D2 dissection extends to nodes along major arteries (stations 7–11) such as the left gastric, common hepatic, and splenic arteries.

D2 lymphadenectomy is the globally accepted standard for stages IB–III, particularly in high-volume centers in Japan and South Korea, where it has shown improved survival with acceptable morbidity. While initial Western trials reported increased postoperative complications, enhanced surgical expertise and perioperative care have led to broader adoption of D2 dissection as the standard in appropriate settings [104]. D1+ dissection may be used for early-stage cancers or in patients with high surgical risk. D3 dissection, which includes para-aortic nodes (station 16), remains investigational, as its added morbidity often outweighs marginal oncologic benefit. Both JGCA and NCCN recommend retrieval of at least 16 lymph nodes for accurate TNM staging and optimal treatment planning [105,106].

Surgical approaches

Open gastrectomy has long been the standard approach for gastric cancer surgery, offering direct visualization and tactile feedback. However, it is associated with greater postoperative pain, longer recovery times, and higher morbidity. Minimally invasive techniques—particularly laparoscopic gastrectomy—have become increasingly favored due to advantages such as reduced blood loss, shorter hospital stays, faster functional recovery, and less postoperative pain. These benefits make it especially suitable for early gastric cancer, and in high-volume centers with experienced surgeons, it is now being extended to locally advanced disease as well [107,108]. Robotic-assisted gastrectomy (RAG)is a more recent advancement that provides enhanced dexterity, three-dimensional visualization, and improved ergonomics. These features are particularly advantageous for complex procedures such as D2 lymphadenectomy and total gastrectomy, where precision is critical. Although RAG involves longer operative times and higher costs, studies have demonstrated its comparable oncologic outcomes and potentially lower complication rates in expert hands. Ultimately, the choice of surgical approach should be guided by tumor stage, patient factors, surgical expertise, and institutional resources [109].

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Review Article

Emerging innovations are steadily advancing the surgical management of gastric cancer toward greater individualization. Sentinel lymph node navigation surgery (SNNS) is under investigation for tailoring the extent of lymphadenectomy in early-stage disease. While it demonstrates high detection accuracy, limitations in real-time pathological assessment currently hinder its widespread clinical application, and D2 dissection remains the standard [110,111]. Simultaneously, molecular profiling—such as HER2 overexpression, microsatellite instability (MSI), and gene expression signatures—is increasingly informing perioperative strategies. These biomarkers aid in stratifying patients and optimizing adjuvant treatment decisions, marking a shift toward more personalized, biomarker-driven surgical approaches.

Enhanced Recovery After Surgery (ERAS) protocols—incorporating early feeding, mobilization, and optimized analgesia—are increasingly used in gastric cancer surgery to reduce complications and shorten hospital stays. Postoperatively, quality of life is a major concern, especially after total gastrectomy. Common issues include weight loss, nutritional deficiencies, and dumping syndrome. Function-preserving procedures, when feasible, help maintain nutritional status and improve long-term well-being. Ongoing nutritional monitoring and support are essential for optimal recovery [112].

Medical treatment

After surgical resection, systemic therapy is essential in both curative and palliative settings. In patients with potentially curable disease, neoadjuvant or adjuvant treatment helps eliminate microscopic tumor spread, improve surgical outcomes, and lower the risk of recurrence. For those with metastatic or unresectable cancer, systemic therapy focuses on prolonging survival, relieving symptoms, and maintaining quality of life [113].

Perioperative treatment of gastric cancer

Perioperative chemotherapy—comprising preoperative (neoadjuvant) and postoperative (adjuvant) phases—has emerged as a cornerstone in the management of resectable, locally advanced gastric and gastroesophageal junction (GEJ) cancers. The rationale is to reduce tumor burden, improve R0 resection rates, eliminate micrometastases early, and enhance long-term survival.

Neoadjuvant chemotherapy is recommended for patients with locally advanced gastric cancer (e.g., cT3–4a, N+, M0; Stage II–IVA) to improve tumor downstaging, facilitate curative (R0) resection, and enhance long-term survival. Common regimens include FOLFOX, PF, XELOX, SOX, and FLOT. For Siewert type II/III esophagogastric junction (EGJ) adenocarcinomas, neoadjuvant chemoradiotherapy followed by surgery is the preferred approach. Evidence from trials such as POET and RTOG 9904 supports its role in reducing local recurrence. Exploratory laparoscopy is advised before neoadjuvant therapy to identify occult metastases. If a pathologic complete response (pCR) is achieved, the same chemotherapy regimen may be continued postoperatively. In cases where R0 resection is not feasible after neoadjuvant treatment, a multidisciplinary team (MDT) discussion should determine further management, including the possibility of palliative care or participation in clinical trials. These strategies are summarized in **Table 3 [**114,115,116**].**

TABLE 3: Neoadjuvant Therapy Recommendations (Based on cTNM Staging – AJCC 8th Edition)

Clinical Stage Situation	Recommended Neoadjuvant Therapies	Grade & Evidence
cT3-4a, N+, M0 (Stage II)	FOLFOX, PF, XELOX, SOX, FLOT	Grade II, Evidence 2A
cT3-4a, N+, M0 (Stage III), EGJ carcinoma	ECF, mECF	Grade II, Evidence 2B
cT4b, N any, M0 (Stage IVA, no unresectable factors)	MDT discussion	Grade II
Disease progression after neoadjuvant therapy	Participation in clinical trials	Grade II

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Review Article

R1/R2 resection post-neoadjuvant therapy	Postoperative adjuvant therapy with same regimen (if response observed)	Grade II

Adjuvant therapy is primarily indicated for patients who did not receive neoadjuvant chemotherapy and underwent D2 resection. It is especially recommended in cases of pT3–4 or node-positive disease. XELOX and S-1 monotherapy are first-line options; FOLFOX and SOX may also be used. If D2 resection was not achieved or if resection margins are positive (R1/R2), fluoropyrimidine-based chemoradiotherapy is preferred. Alternative regimens like XP (capecitabine + cisplatin) may be considered in select cases. These recommendations are outlined in **Table 4 [**115,116,117**].**

TABLE 4: Adjuvant Therapy Recommendations (Based on pTNM Staging – AJCC 8th Edition)

Pathologic Stage / Resection Status	**Recommended Adjuvant Therapies**	**Grade & Evidence**
pT3–4, N any, M0, RO D2 resection	XELOX, S-1 alone	Grade I, Evidence 1A
pT any, N+, M0	FOLFOX, SOX	Grade II, Evidence 2A
pT2–4, Nany, M0, RO resection without D2	Chemoradiotherapy (45–50.4 Gy with concurrent fluoropyrimidine)	Grade I, Evidence 1A
pT2–4, Nany, M0, R1/R2 resection	Chemoradiotherapy (45–50.4 Gy with concurrent fluoropyrimidine)	Grade III, Evidence 3
Alternative (any of above)	XP (capecitabine + cisplatin)	Grade III, Evidence 2B

Targeted Therapy and Immunotherapy in Advanced and unresectable Gastric Cancer

Recent advances in the molecular understanding of gastric cancer (GC) have revolutionized the management of advanced and metastatic disease, shifting the therapeutic paradigm toward precision oncology. Biomarker-guided strategies now enable clinicians to personalize treatment based on the tumor's molecular and immunological profile, optimizing efficacy while minimizing unnecessary toxicity [118].

Among targeted therapies, HER2 remains the most validated biomarker. For patients with HER2-positive tumors, first-line therapy consists of trastuzumab combined with platinum-based chemotherapy, as established by the pivotal ToGA trial. In contrast, HER2-negative patients are typically treated with platinum-based regimens or enrolled in ongoing clinical trials such as FIGHT (evaluating FGFR2b-targeted bemarituzumab) and SPOTLIGHT (investigating Claudin 18.2-targeted zolbetuximab) [119]. To address primary and acquired resistance to trastuzumab, several next-generation HER2-targeted approaches are under development, including dual blockade with pertuzumab, HER2 antibody–drug conjugates like trastuzumab deruxtecan (T-DXd), and tyrosine kinase inhibitors such as lapatinib **[**119,120**]**.

Anti-angiogenic therapy targeting the VEGF/VEGFR signaling pathway constitutes a key strategy in advanced gastric cancer, particularly in later treatment lines. Ramucirumab, a VEGFR-2 monoclonal antibody, has demonstrated improved overall survival when combined with paclitaxel in the second-line setting. This benefit is especially relevant for patients with microsatellite-stable (MSS) tumors, for whom immunotherapy may be less effective. Apatinib, a VEGFR-2 tyrosine kinase inhibitor, is also being explored for its efficacy in refractory or third-line settings [121].

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Review Article

While EGFR inhibitors such as cetuximab, panitumumab, and erlotinib have not demonstrated meaningful clinical benefit in gastric cancer, attention has shifted toward other promising molecular targets, including MET, FGFR2b, and Claudin 18.2. Several agents targeting these pathways are currently under investigation in clinical trials, underscoring the broader evolution of gastric cancer treatment toward precision oncology and biomarker-driven therapy.

Immunotherapy, particularly through checkpoint inhibition, has revolutionized the treatment landscape of advanced gastric cancer. PD-1/PD-L1 inhibitors such as pembrolizumab and nivolumab have shown durable responses, particularly in tumors with microsatellite instability-high (MSI-H), deficient mismatch repair (dMMR), or elevated PD-L1 expression. In the first-line setting, the CheckMate 649 trial demonstrated a survival benefit with nivolumab combined with chemotherapy, irrespective of PD-L1 status.

As treatment progresses, biomarker selection becomes increasingly important. Third-line decisions are frequently guided by PD-L1 combined positive score (CPS): pembrolizumab has demonstrated efficacy in tumors with CPS ≥1%, based on the findings from KEYNOTE-059, -061, and -062. In contrast, for PD-L1–negative tumors, TAS-102 (trifluridine/tipiracil), a nucleoside analog, is utilized as a chemotherapy option [122,123].

To address resistance mechanisms and improve immunotherapy responses, several emerging strategies are under investigation, including inhibitors targeting MET, EGFR, FGFR2b, and Claudin 18.2. These reflect the ongoing evolution of precision immuno-oncology in gastric cancer. An overview of treatment stratification based on molecular profiling is presented in **Table 5** [124].

TABLE 5: Biomarker-Driven Treatment Algorithm for Advanced Gastric Cancer.

Treatment Line	Biomarker Status	Recommended Therapy	Remarks
First-line	HER2-positive	Trastuzumab + platinum-based chemotherapy	Standard for HER2-positive advanced GC
	HER2-negative	Platinum-based regimen or clinical trials (e.g., FIGHT, SPOTLIGHT)	For HER2-negative patients
Second-line	MSI-high	Pembrolizumab	Immune checkpoint inhibitor
	Microsatellite stable (MSS)	Ramucirumab ± paclitaxel	Anti-VEGFR2 therapy
Third-line	PD-L1 ≥ 1%	Pembrolizumab	Based on PD-L1 expression
	PD-L1 = 0%	TAS-102	Chemotherapy option in refractory settings

Beyond checkpoint inhibitors, several novel immunotherapeutic strategies—including adoptive immune cell therapy, tumor vaccines, oncolytic viruses, and bispecific antibodies—are under early-phase clinical evaluation. Combination strategies that integrate immunotherapy with chemotherapy or targeted agents are also being pursued to overcome resistance mechanisms and expand response rates [125].

Despite these advancements, major challenges remain. Therapeutic resistance, immune evasion, and adverse effects can compromise long-term efficacy. Hence, routine biomarker testing—including HER2, MSI/MMR, PD-L1, and other emerging targets—is now a cornerstone of advanced GC management. Additionally, the concept of conversion therapy, wherein systemic treatment is used to downstage initially unresectable tumors to resectable ones, is gaining traction, although criteria for optimal patient selection are still being refined [126].

Molecular Classification and Precision Oncology

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Review Article

Comprehensive molecular profiling has redefined gastric cancer (GC) as a biologically heterogeneous disease. Two landmark efforts—the Cancer Genome Atlas (TCGA) and the Asian Cancer Research Group (ACRG)—have established molecular classifications that guide the evolving paradigm of precision oncology. TCGA categorizes GC into four subtypes: Epstein–Barr virus (EBV)-positive (with PIK3CA mutations and PD-L1/PD-L2 overexpression), microsatellite instability-high (MSI-H), genomically stable (GS), and chromosomal instability (CIN) tumors. CIN tumors often feature TP53 mutations and receptor tyrosine kinase (RTK) amplifications such as HER2 or MET. ACRG further classifies GC into MSI-H, MSS/EMT, MSS/TP53-positive, and MSS/TP53-negative subtypes.

Although not yet standard in clinical practice, these classifications reveal distinct prognostic and therapeutic implications. MSI-H and EBV-positive tumors tend to respond well to immunotherapy, while CIN tumors may benefit from RTK-targeted agents. In contrast, GS and EMT-like tumors often exhibit chemoresistance, underscoring the need for novel approaches. As next-generation sequencing (NGS) becomes more accessible, molecular subtyping is expected to play a central role in biomarker-driven trials and personalized treatment strategies—enhancing prognostication, guiding therapeutic choices, and addressing resistance mechanisms in both curative and palliative settings [127].

Future Directions in Systemic Therapy

The systemic treatment landscape of gastric cancer is rapidly advancing, driven by precision medicine, immunotherapy, and novel targeted strategies. Antibody–drug conjugates (ADCs), such as trastuzumab deruxtecan (T-DXd), have shown efficacy even in HER2-low tumors, while agents targeting Claudin 18.2 are in early-phase development. Bispecific antibodies and CAR-T cell therapies targeting HER2 or Claudin 18.2 are also being explored, although their use in solid tumors remains challenging [128].

New checkpoint inhibitors, including dual PD-1/CTLA-4 blockade, are under investigation, especially for MSI-H and EBV-positive tumors. Combination regimens integrating immunotherapy with chemotherapy or anti-angiogenic agents are being tested to enhance efficacy and overcome resistance.

Targeted inhibitors of FGFR2b, MET, and other actionable mutations are entering clinical trials, with studies like FIGHT, SPOTLIGHT, and VIKTORY exemplifying biomarker-guided approaches. Emerging tools such as liquid biopsy and ctDNA analysis promise dynamic treatment monitoring and early resistance detection [129,130].

Conclusion

Gastric cancer continues to pose a significant global health burden, underscoring the critical need for continued advancements in its prevention, early detection, and treatment. This comprehensive review has illustrated the multifaceted nature of the disease, from its varied epidemiological patterns and intricate risk factors to its often-elusive clinical presentation. Significant strides have been made in diagnostic precision, with innovations like AI-assisted endoscopy and the evolving application of the TNM staging system, increasingly complemented by a deeper understanding of molecular biomarkers. These diagnostic and staging advancements are pivotal for accurate prognosis and guiding personalized treatment strategies.

The therapeutic landscape for gastric cancer has undergone a remarkable evolution. Surgical interventions, ranging from organ-preserving endoscopic resections for early-stage disease to sophisticated gastrectomies with meticulous lymphadenectomy, remain the cornerstone of curative treatment, now often enhanced by minimally invasive and robotic techniques. Concurrently, systemic therapies have progressed from conventional chemotherapy to a new era of precision oncology, driven by targeted agents against pathways like HER2 and VEGF, and the revolutionary impact of immunotherapy, particularly for MSI-H and PD-L1 positive tumors. The integration of molecular profiling, as highlighted by TCGA and ACRG classifications, is increasingly refining treatment selection, moving us closer to truly personalized medicine.

Perhaps the most exciting frontier lies in the integration of gene editing technologies. The CRISPR-Cas9 system represents a transformative tool, offering unparalleled potential to precisely modify genes implicated in gastric carcinogenesis, disrupt oncogenic pathways, and even combat underlying viral infections. While its application in

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Review Article

clinical practice is still nascent, CRISPR's ability to manipulate the cancer genome and epigenome holds immense promise for developing novel therapeutic modalities, including advanced immunotherapies and direct gene correction.

Despite these substantial advancements, challenges persist, notably the high rate of late-stage diagnosis, the development of therapeutic resistance, and the need for more effective strategies for genomically stable and EMT-like tumors. Future directions will undoubtedly focus on further refining molecular subtyping, developing next-generation targeted agents and immunotherapies, and harnessing the full potential of CRISPR-Cas9 to overcome current limitations. A collaborative, multidisciplinary approach, coupled with continued investment in research and clinical trials, will be paramount in translating these scientific breakthroughs into tangible improvements in survival and quality of life for gastric cancer patients worldwide. The journey toward conquering gastric cancer is ongoing, and the promise of precision medicine, spearheaded by innovative technologies like CRISPR-Cas9, offers a hopeful outlook for future patient care.

Abbreviations

- **H.pylori:** *Helicobacter pylori*
- **HER2:** Human Epidermal Growth Factor Receptor 2
- **p53:** Tumor protein 53
- **PD1:** Programmed cell death protein 1
- **p73:** Tumor protein 73
- **mdm2:** Murine double minute 2
- **Bcl2:** B-cell lymphoma 2
- **pRb-CCND1:** Retinoblastoma Protein-Cyclin D1
- **p16:** Cyclin-dependent kinase inhibitor 2A
- **MUC:** Mucin
- **MRP2:** Multidrug Resistance-Associated Protein 2
- **MDR1:** Multidrug resistance protein 1
- **GST-P:** Glutathione S-transferase P
- **MSI:** Microsatellite instability
- **EGD:** Esophagogastroduodenoscopy
- **EMR:** Endoscopic mucosal resection
- **ESD:** Endoscopic submucosal resection
- **CRISPR:** Clustered Regularly Interspaced Short Palindromic Repeats
- **Cas:** CRISPR-associated proteins
- **KRAS:** Kirsten rat sarcoma viral oncogene homolog
- **PIK3CA:** Phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha
- **TP53:** Tumor protein 53
- **ARID1A:** AT-rich interactive domain-containing protein 1A
- **CDH1:** Cadherin-1
- **GLOBOCAN:** Global Cancer Observatory
- **ASIR:** Age-standardized incidence rate
- **GERD:** Gastroesophageal reflux disease
- **GI:** Gastrointestinal
- **EUS:** Endoscopic ultrasound
- **CT:** Computed tomography
- **MDCT:** Multi-row detector computed tomography
- **AI:** Artificial intelligence
- **CLE:** Confocal laser endomicroscopy
- **TXI:** Texture and colour enhancement imaging
- **WISENSE:** Wide-angle endoscopy with AI-based real-time assistance system
- **GRAIDS:** Gastrointestinal Artificial Intelligence Diagnostic System
- **ENDOANGEL LD:** Endoscopic AI-based system for lesion detection
- **CEA:** Carcinoembryonic antigen
- **CA 19-9:** Cancer antigen 19-9
- **CA 72-4:** Cancer antigen 72-4
- **EMA:** European Medicines Agency
- **ctDNA:** Circulating tumor DNA
- **miRNAs:** microRNAs
- **lncRNAs:** long non-coding RNAs
- **HOTAIR:** HOX transcript antisense RNA
- **MALAT1:** Metastasis Associated Lung Adenocarcinoma Transcript 1
- **TNM:** Tumor, Node, Metastasis
- **AJCC:** American Joint Committee on Cancer
- **UICC:** Union for International Cancer Control
- **EGJ:** Esophagogastric junction
- **NCDB:** National Cancer Database
- **MLNs:** Metastatic lymph nodes
- **LNR:** Lymph node ratio
- **LODDS:** Log odds of positive lymph nodes
- **TCGA:** The Cancer Genome Atlas
- **EBV:** Epstein–Barr virus
- **MSI-H:** Microsatellite instability-high
- **GS:** Genomically stable
- **CIN:** Chromosomal instability
- **CY1:** Positive peritoneal cytology
- **PD-L1:** Programmed death-ligand 1
- **R0:** Microscopically margin-negative excision
- **MDT:** Multidisciplinary team

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Review Article

- **ECOG:** Eastern Cooperative Oncology Group
- **CRS:** Cytoreductive surgery
- **HIPEC:** Hyperthermic intraperitoneal chemotherapy
- **EGC:** Early gastric cancer
- **LNM:** Lymph node metastasis
- **ER:** Endoscopic resection
- **UL0:** No ulceration
- **UL1:** Mild ulceration
- **PPG:** Pylorus-preserving gastrectomy
- **JGCA:** Japanese Gastric Cancer Association
- **NCCN:** National Comprehensive Cancer Network
- **RAG:** Robotic-assisted gastrectomy
- **SNNS:** Sentinel lymph node navigation surgery
- **ERAS:** Enhanced Recovery After Surgery
- **GEJ:** Gastroesophageal junction
- **pCR:** Pathologic complete response
- **XP:** Capecitabine + Cisplatin
- **GC:** Gastric cancer
- **VEGF:** Vascular endothelial growth factor
- **VEGFR:** Vascular endothelial growth factor receptor
- **MSS:** Microsatellite stable
- **EGFR:** Epidermal growth factor receptor
- **FGFR2b:** Fibroblast growth factor receptor 2b
- **CPS:** Combined positive score
- **TAS-102:** Trifluridine/tipiracil
- **ADCs:** Antibody–drug conjugates
- **T-DXd:** Trastuzumab deruxtecan
- **CTLA-4:** Cytotoxic T-lymphocyte–associated protein 4
- **ACRG:** Asian Cancer Research Group
- **EMT:** Epithelial-mesenchymal transition
- **NGS:** Next-generation sequencing

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Biography

Dr. Nidhi Vadhavekar is a medical professional who earned her MBBS degree from Padmashree Dr. D.Y. Patil School of Medicine in India. She specializes in the fields of oncology and neuropsychiatry. She has demonstrated strong leadership skills through her extensive involvement in research, having published over nine articles. Her role as a first author on a publication is a clear indicator of her ability to guide a project from start to finish. As a first author, Dr. Vadhavekar was instrumental in the research process. Her leadership was evident from the early stages of conceptualization, where she helped shape the study's direction. She continued to lead the effort through the critical phases of revising and finalizing the drafts, ensuring the project's successful completion. This experience highlights her capacity to take initiative and drive a complex scientific undertaking.

Deepinder Kaur, a second-year MBBS student at AIIMS Bathinda, is an enthusiastic undergraduate medical student with a strong academic interest in oncology and pathology. For this article on Gastric Cancer, she played a significant role in the research and writing process. Her contributions included an in-depth review of current literature, identification of recent advances in diagnostic and therapeutic strategies, and synthesizing complex clinical data into a cohesive and comprehensible narrative. Her dedication to evidence-based writing and collaborative teamwork was instrumental in shaping the final version of the article.

Vanshika Pannu, is a second-year MBBS student at AIIMS, Bathinda, and an ICMR-STS fellow. She has a growing interest in oncology and clinical research. Her analytical skills, enthusiasm for academic learning, and commitment to evidence-based medicine contributed significantly to the preparation of this manuscript.

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Review Article

conferences, securing awards at intercollegiate competitions, and has authored several peer-reviewed publications. His academic interests include pharmacology, rational drug use, and clinical biomarkers. He has played a key role in the research process, specifically by taking the lead in writing and revising the manuscript.

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