Gastric Cancer: Epidemiology, Causes, Diagnosis, and Modern Treatment

From Helicobacter pylori and chronic inflammation to molecular classification, precision oncology, immunotherapy, and integrative care



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Introduction

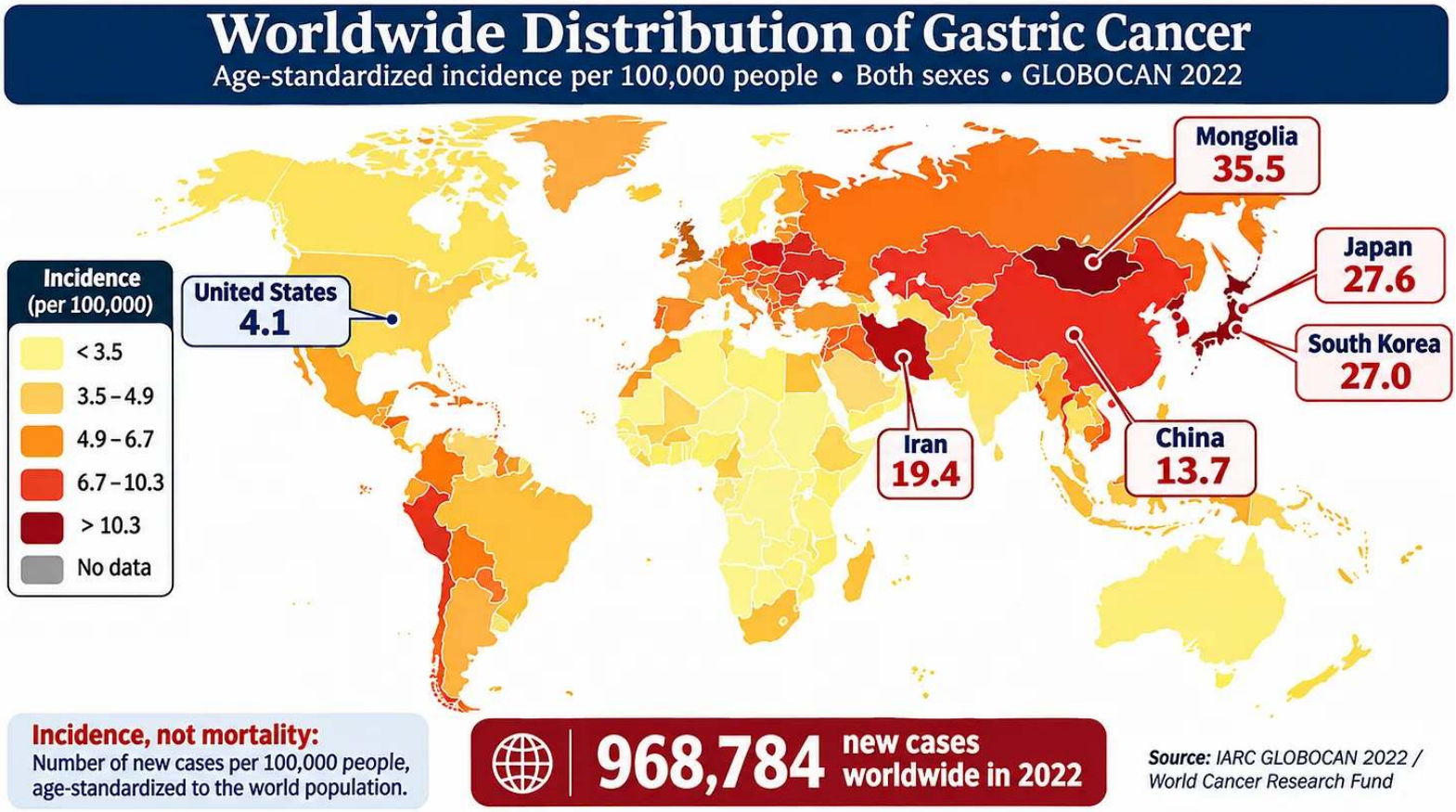
Gastric cancer remains a major cause of cancer death worldwide. Although incidence has declined in many Western countries, the burden remains high in East Asia, Eastern Europe, and parts of Latin America.[1] Historically, prognosis was poor because many patients presented with advanced disease, surgery was the only curative option, and chemotherapy offered modest benefit. Over the past decade management has been transformed by molecular characterization, perioperative chemotherapy, immunotherapy, and agents directed against human epidermal growth factor receptor 2 (HER2), claudin 18.2 (CLDN18.2), and defective DNA mismatch repair.[1]

The central principle of contemporary management is that gastric cancer is not one disease but a group of biologically distinct malignancies that differ in cause, molecular profile, behavior, and response to treatment.[2] This review focuses on gastric adenocarcinoma, which accounts for 90% to 95% of malignant gastric tumors; lymphomas, gastrointestinal stromal tumors (GISTs), and neuroendocrine neoplasms are distinct diseases.

1. Epidemiology

According to Global Cancer Observatory (GLOBOCAN) 2022 estimates, gastric cancer is the fifth most frequently diagnosed cancer and the fifth leading cause of cancer death worldwide, with approximately 969,000 new cases and 660,000 deaths.[3] Rates

are highest in Mongolia, Japan, South Korea, China, Iran, and parts of Eastern Europe. Non-cardia gastric cancer has declined substantially in North America and Western Europe, reflecting better sanitation and food preservation and declining Helicobacter pylori (H. pylori) infection, whereas cancers of the cardia and gastroesophageal junction are more closely linked to obesity and reflux.[1, 3]



The median age at diagnosis in the United States (US) is approximately 68 years, and men are affected about twice as often as women; younger patients may develop hereditary diffuse or signet-ring cell cancers.[4, 5] Survival depends strongly on stage: early cancers confined to the mucosa or superficial submucosa are often curable, whereas metastatic disease rarely is. Five-year relative survival in the US is approximately 36% overall.[4]

2. Causes and risk factors

Helicobacter pylori: the dominant preventable cause

H. pylori is classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC) and accounts for a large proportion of non-cardia gastric

adenocarcinomas worldwide.[6] Carcinogenesis follows the Correa cascade, from chronic gastritis through atrophy, intestinal metaplasia, and dysplasia to adenocarcinoma (Figure 2).[7] Progression depends on bacterial virulence, host genetics, immune responses, and environment. Eradication reduces subsequent cancer risk, particularly before advanced precancerous changes develop, and treating infected first-degree relatives of patients with gastric cancer also lowers their risk.[8, 9]

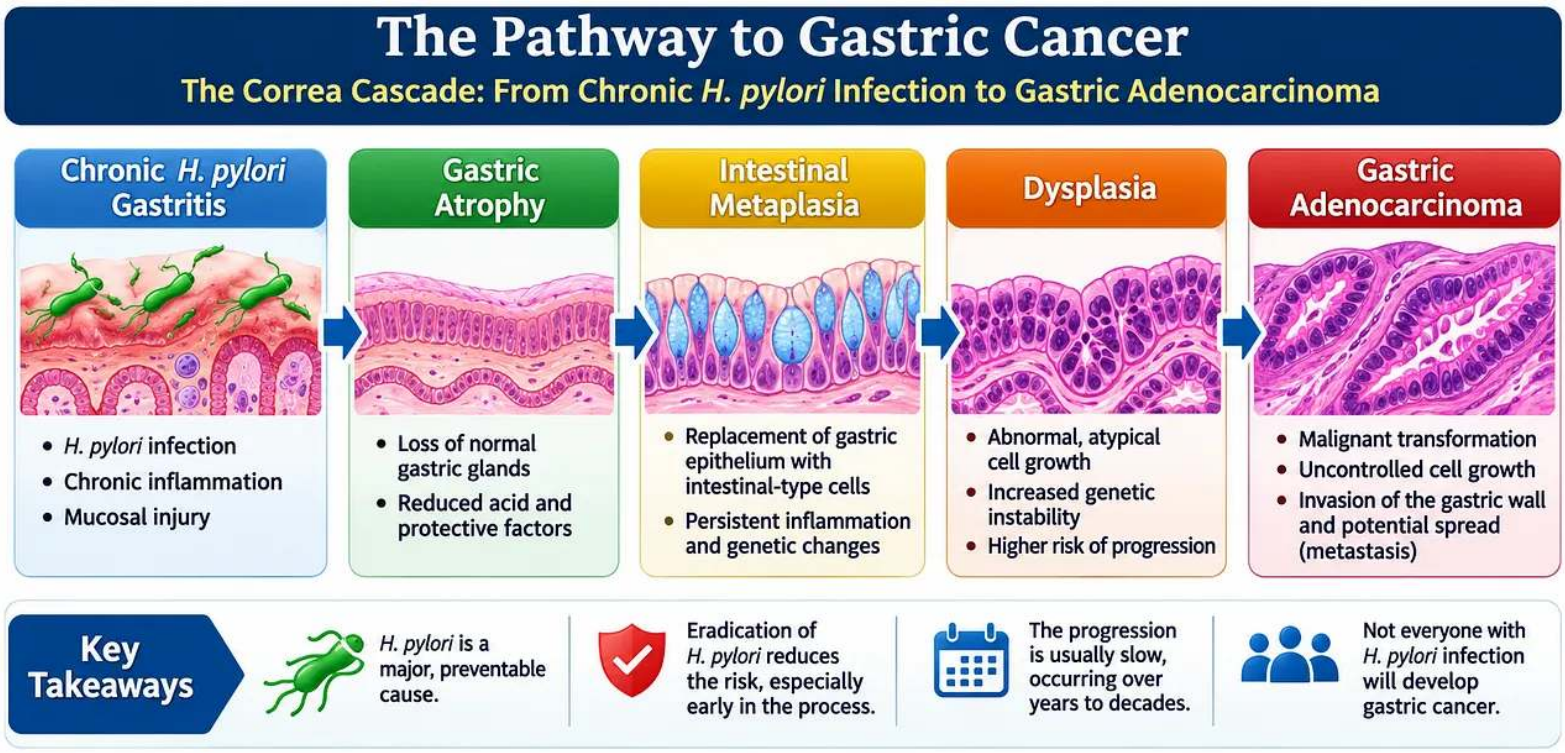
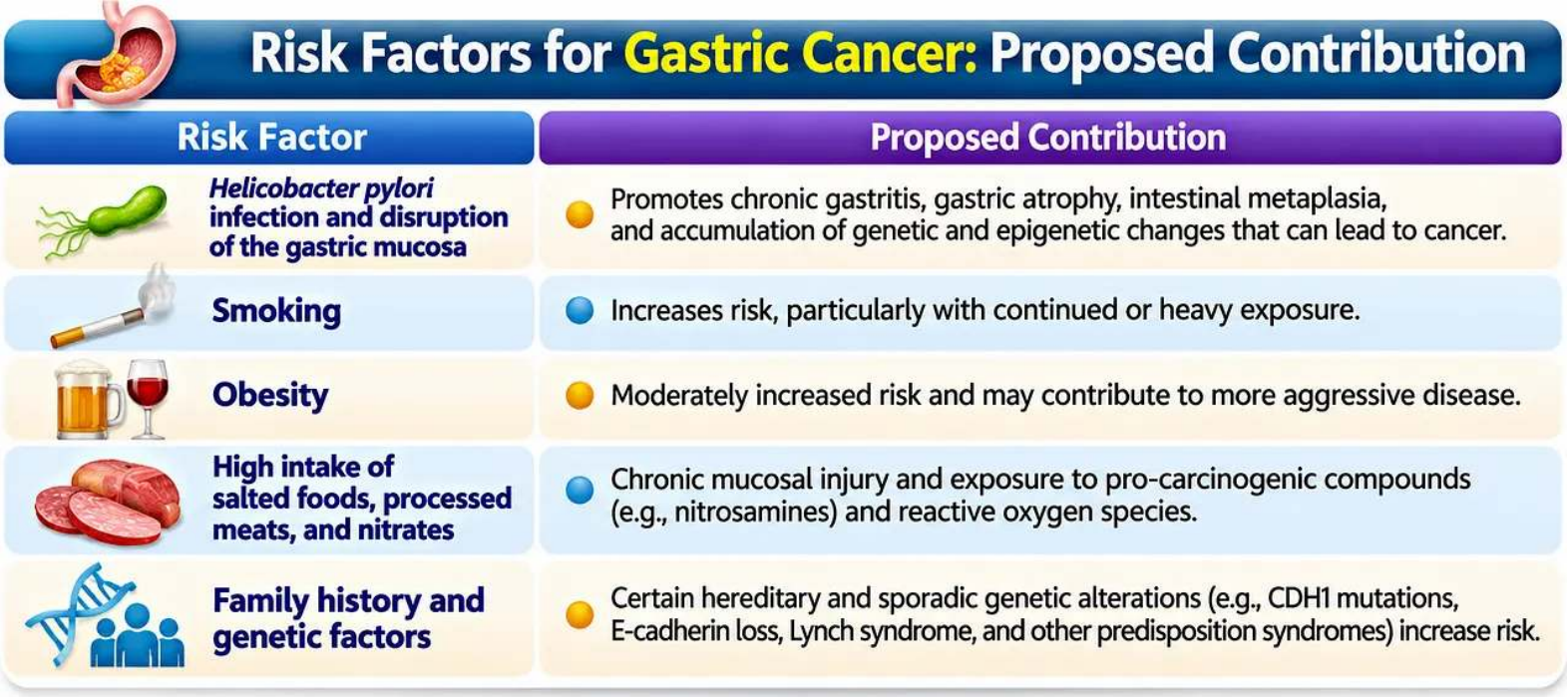


Figure 2. The Correa cascade: from chronic H. pylori infection to gastric adenocarcinoma.

Other important risk factors

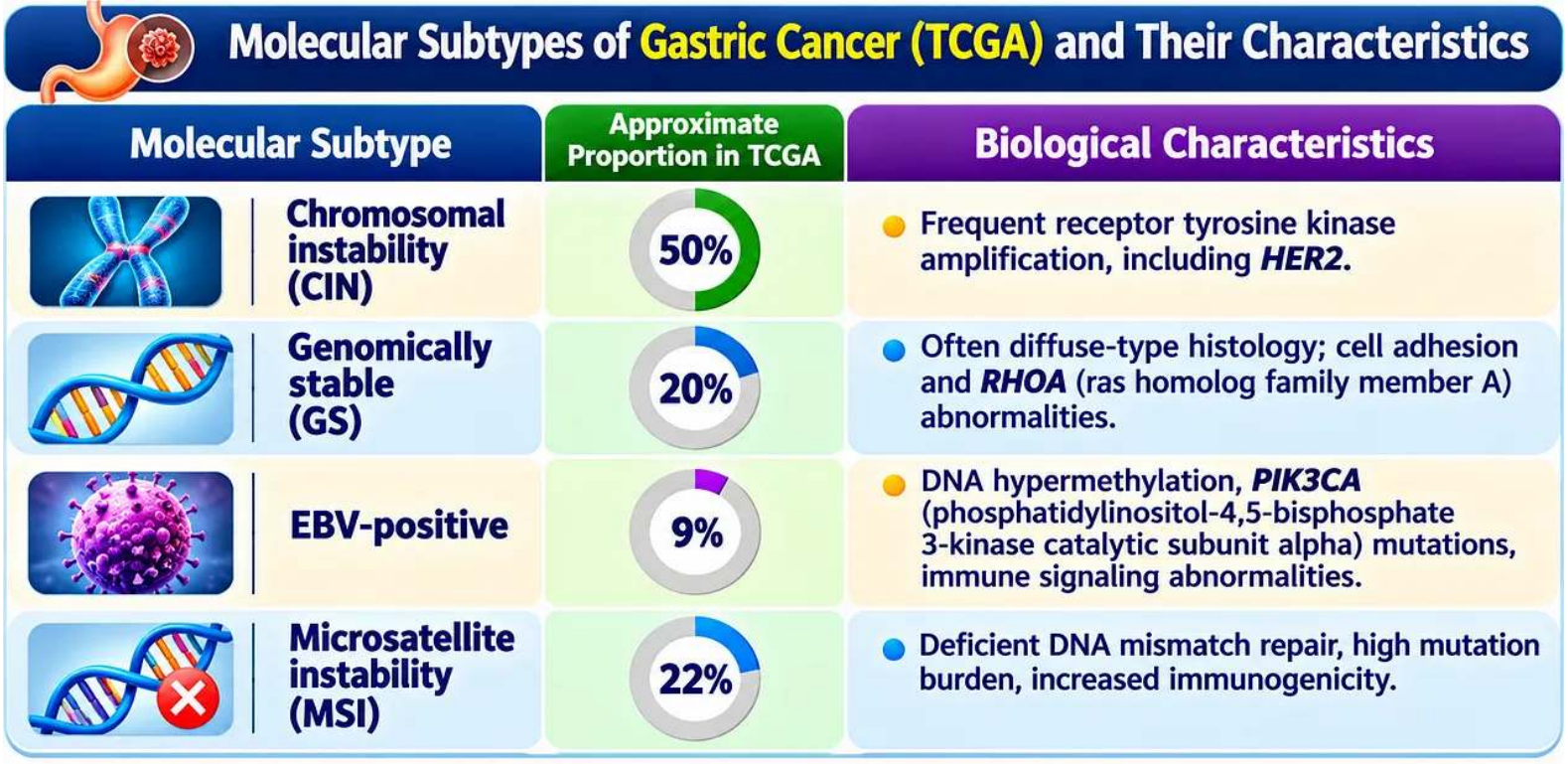


Adapted from Smyth et al.[1]

Germline CDH1 variants, which impair the adhesion protein E-cadherin, cause hereditary diffuse gastric cancer,[13] characterized by microscopic foci of signet-ring cell carcinoma that are difficult to detect endoscopically. Prophylactic total gastrectomy may be recommended for selected carriers after genetic counseling, with expert endoscopic surveillance as an alternative in some circumstances.[5]

3. Pathology and molecular classification

Laurén described two main histological types: intestinal-type adenocarcinoma, usually gland-forming and linked to the H. pylori-related cascade, and diffuse-type adenocarcinoma, composed of poorly cohesive cells, sometimes with signet-ring morphology, which may diffusely thicken the gastric wall (linitis plastica).[14] The Cancer Genome Atlas (TCGA) later identified four molecular subtypes.[2]



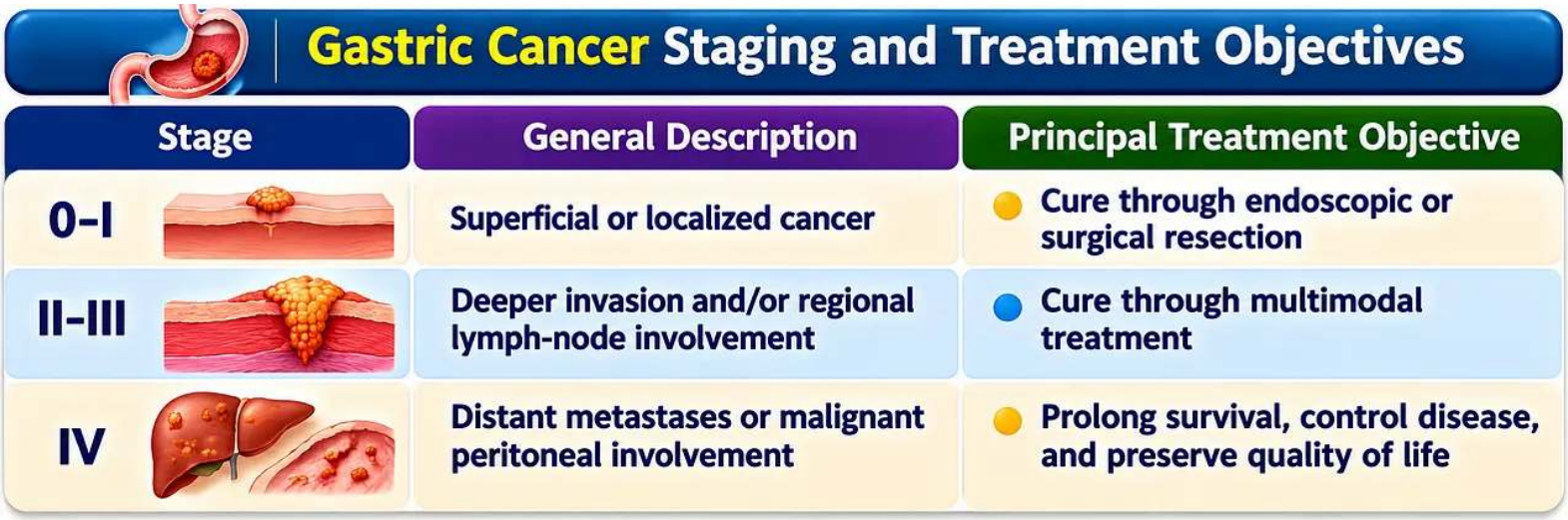
These proportions reflect a research cohort, but the classification matters clinically because some subtypes are especially responsive to immunotherapy and others carry druggable targets.

4. Clinical presentation and diagnosis

Early gastric cancer may be asymptomatic or cause nonspecific dyspepsia. Common features include epigastric discomfort, early satiety, anorexia, nausea, weight loss, and fatigue; advanced disease may cause iron-deficiency anemia, bleeding, vomiting from outlet obstruction, dysphagia, or ascites. Metastases commonly involve the liver, peritoneum, distant nodes, and occasionally the ovaries (Krukenberg tumors).[1] Alarm features such as unexplained weight loss, persistent vomiting, bleeding, anemia, or progressive dysphagia warrant prompt investigation.

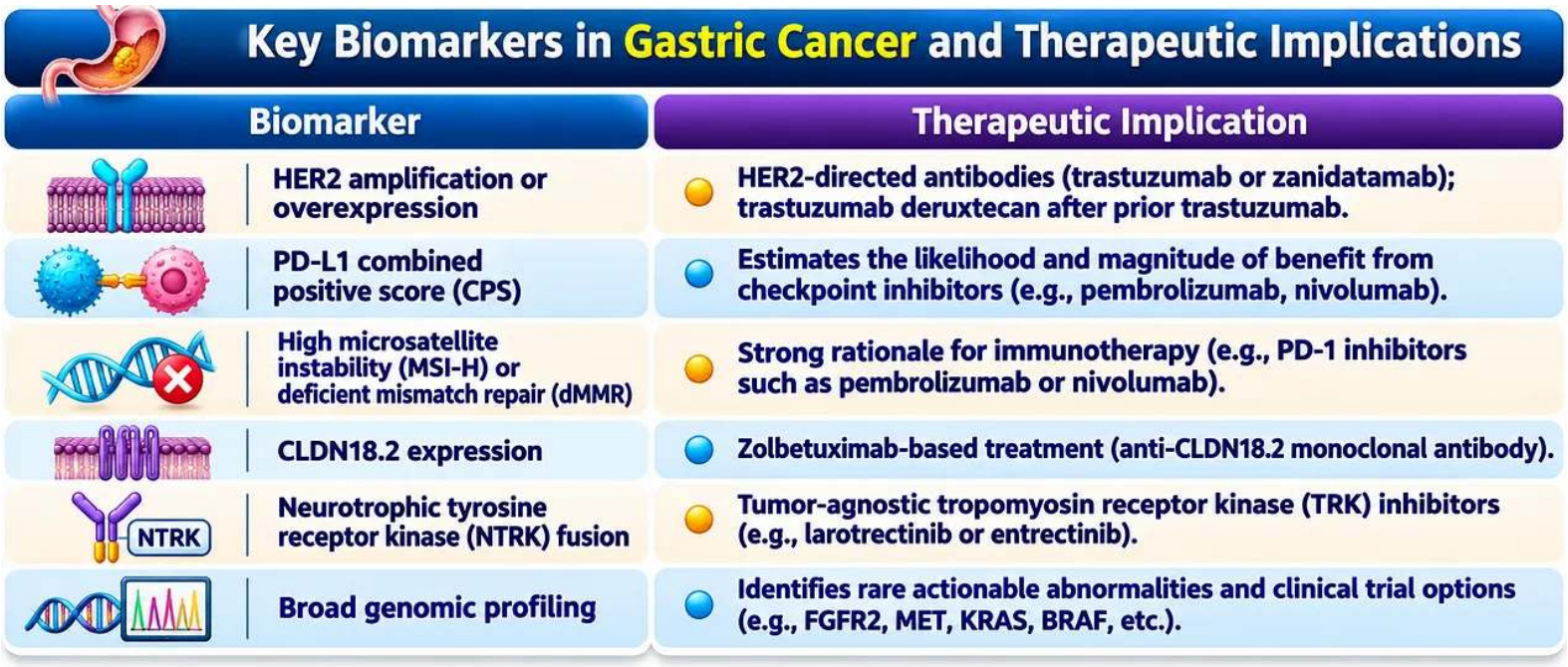
Upper gastrointestinal endoscopy with multiple biopsies establishes the diagnosis; diffuse infiltrative cancers may require deeper sampling. Staging includes contrast-enhanced computed tomography (CT) of the chest, abdomen, and pelvis, together with nutritional and functional assessment. Endoscopic ultrasound defines depth of invasion when endoscopic resection or curative surgery is being considered, and positron emission tomography (PET)/CT is useful in selected patients, although diffuse and mucinous tumors may show limited fluorodeoxyglucose (FDG) uptake. Staging

laparoscopy with peritoneal washings is important in locally advanced disease because CT can miss small peritoneal metastases.[15]



5. Molecular testing

For advanced disease, treatment selection should be based on testing for HER2, mismatch repair or MSI status, programmed death-ligand 1 (PD-L1), and CLDN18.2.[16]



6. Modern treatment

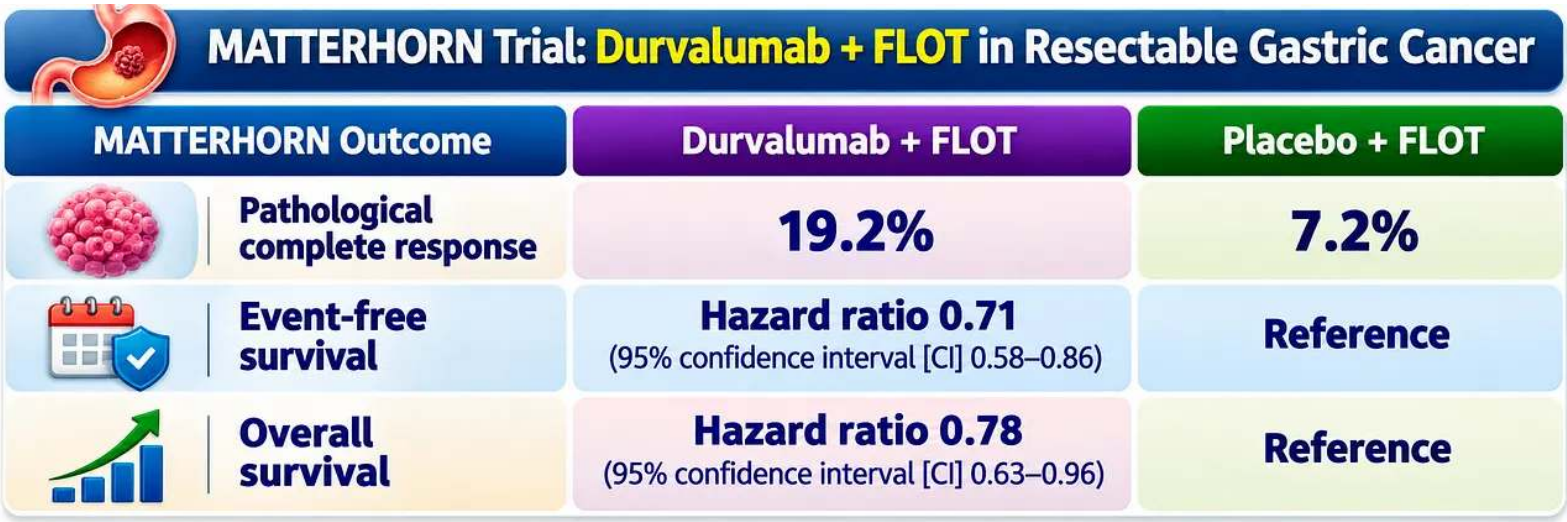
Stage I: early gastric cancer

Selected small, superficial, low-risk lesions can be removed by endoscopic submucosal dissection (ESD), preserving the stomach.[18] When endoscopic resection is inappropriate, gastrectomy with lymph-node assessment is the principal curative treatment, and surgery alone may suffice for appropriately selected early cancers.

Stages II–III: locally advanced disease

For fit patients with resectable stage II–III disease, perioperative chemotherapy is standard in Western practice. In the FLOT4 trial, the FLOT regimen (5-fluorouracil, leucovorin, oxaliplatin, and docetaxel) improved median overall survival from 35 to 50 months compared with older chemotherapy.[19] Surgery involves subtotal or total gastrectomy, commonly with D2 lymphadenectomy in experienced centers.[20] In East Asia, upfront surgery followed by adjuvant chemotherapy with S-1 or capecitabine plus oxaliplatin remains an established approach.[21, 22]

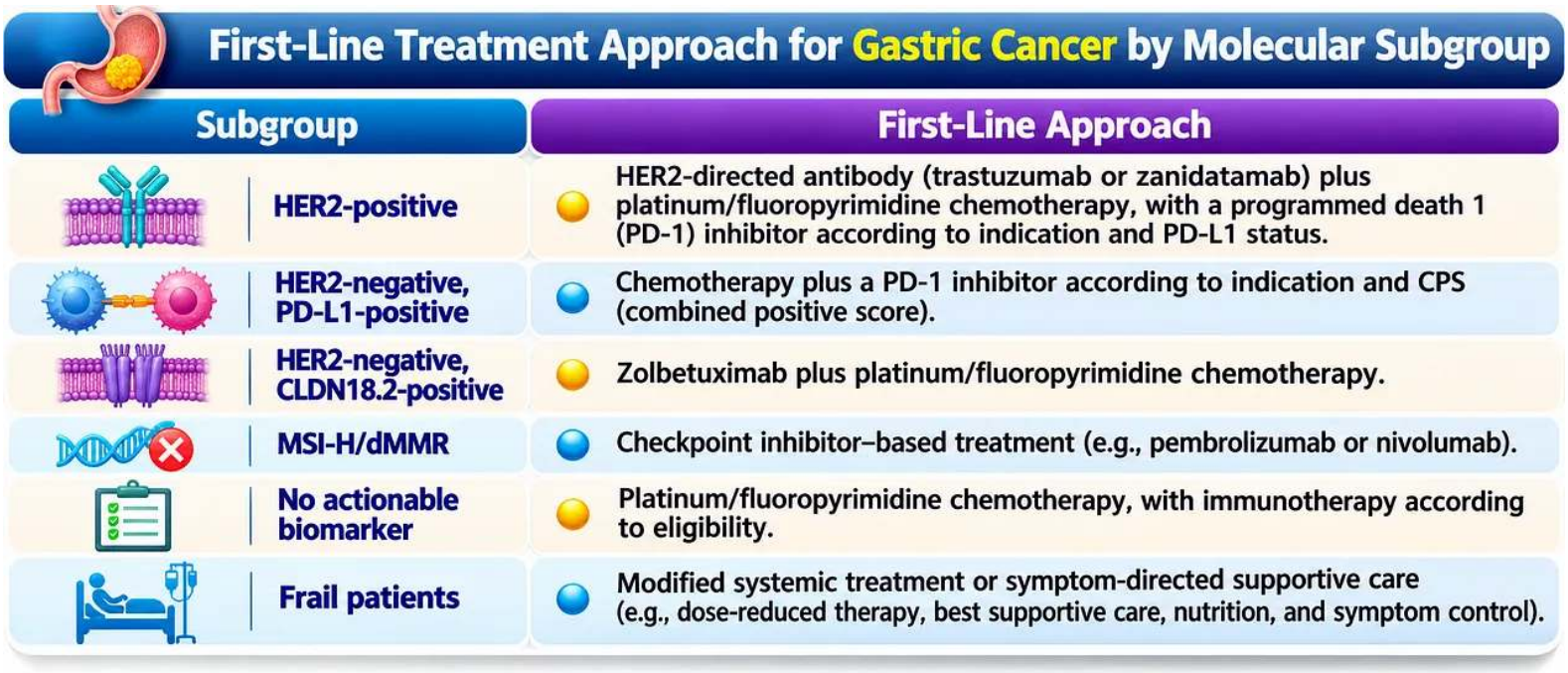
In November 2025, the US Food and Drug Administration (FDA) approved perioperative durvalumab plus FLOT on the basis of the phase III MATTERHORN trial (948 patients).[23]



Immunotherapy is therefore now part of potentially curative treatment, although not every trial has been positive: perioperative pembrolizumab did not significantly improve event-free survival in KEYNOTE-585.[24] For MSI-H resectable cancer, neoadjuvant nivolumab plus ipilimumab produced a high pathological complete response rate in the phase II NEONIPIGA study, raising the possibility of chemotherapy-sparing strategies.[25]

Stage IV: advanced or metastatic disease

Systemic therapy rarely cures metastatic disease but can prolong survival, relieve symptoms, and sometimes produce durable control. Treatment choice depends on biomarkers, performance status, comorbidities, and the patient's goals.

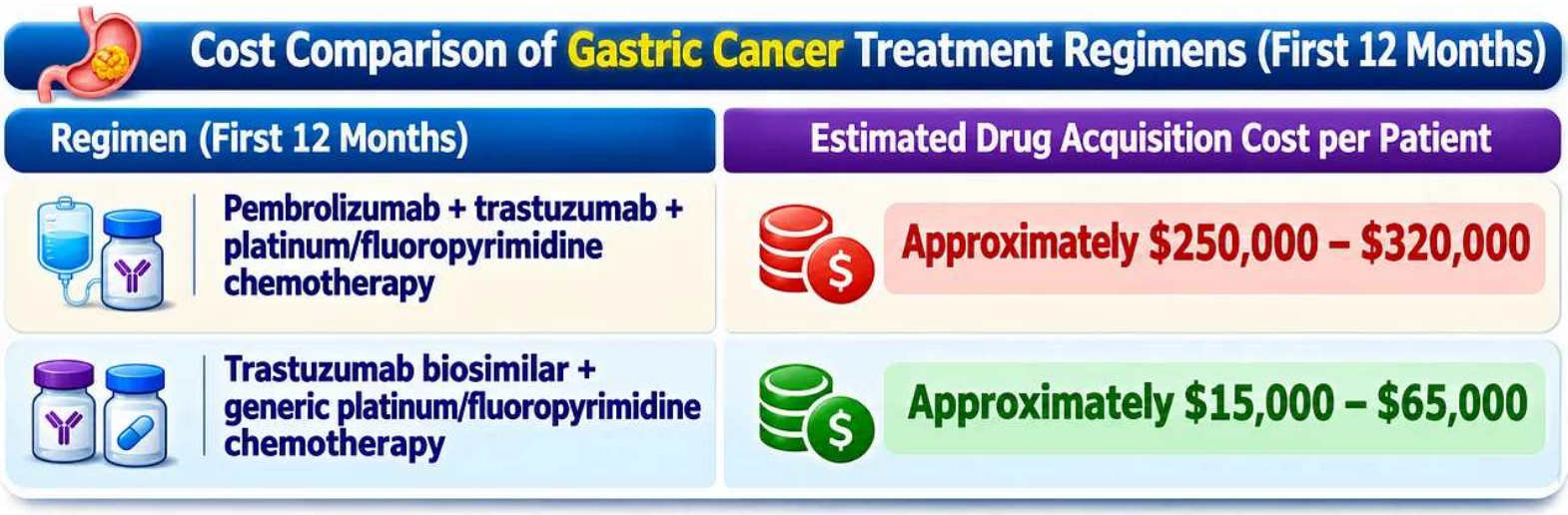


In HER2-positive disease, trastuzumab improved overall survival in the ToGA trial and remains foundational.[26] The FDA granted traditional approval to pembrolizumab with trastuzumab and chemotherapy for PD-L1 CPS ≥1 disease in March 2025, based on KEYNOTE-811.[27] In the phase III HERIZON-GEA-01 trial, the bispecific HER2 antibody zanidatamab improved progression-free survival compared with trastuzumab, and adding the PD-1 inhibitor tislelizumab also improved overall survival (median 26.4 versus 19.2 months); both regimens were approved in August 2026 and incorporated into the American Society of Clinical Oncology (ASCO) guideline.[16, 28]

In HER2-negative disease, PD-1 inhibitors added to chemotherapy improve outcomes, with greater benefit at higher PD-L1 expression.[29, 30] Zolbetuximab, an antibody targeting CLDN18.2, improved overall survival when added to modified FOLFOX6 (fluorouracil, leucovorin, and oxaliplatin) in SPOTLIGHT and to CAPOX (capecitabine and oxaliplatin) in GLOW, where median survival rose from 12.16 to 14.39 months.[31, 32]

After progression, options include paclitaxel plus ramucirumab, irinotecan-based chemotherapy, trastuzumab deruxtecan for previously treated HER2-positive disease, and trifluridine/tipiracil in later lines.[33-36] Progression does not mean that effective options are exhausted, and clinical trials are particularly important for patients with uncommon molecular alterations or resistance to established therapies. In DESTINY-Gastric04, second-line trastuzumab deruxtecan improved median overall survival compared with ramucirumab plus paclitaxel (14.7 versus 11.4 months).[37]

The cost of modern therapy



Author's estimates from US list prices, assuming 12 months of antibody therapy and about six months of chemotherapy; excludes administration, monitoring, and complications.

Actual costs depend on whether list, negotiated, or Medicare prices are used, and on patient weight, dosing schedule, and biosimilar use. A first-year drug cost approaching $300,000 is a formidable barrier, especially in low- and middle-income countries. Where checkpoint inhibitors are inaccessible, trastuzumab, preferably a biosimilar, plus platinum/fluoropyrimidine chemotherapy remains an evidence-based option, and the 2026 ASCO guideline retains trastuzumab when cost or access is a factor; biosimilars have shown equivalent efficacy to the reference product.[16, 26, 38] Wider access to biosimilars, generic chemotherapy, and reliable molecular testing may have a greater immediate impact worldwide than new therapies priced beyond the reach of most patients.

7. Radiotherapy, nutrition, and prevention

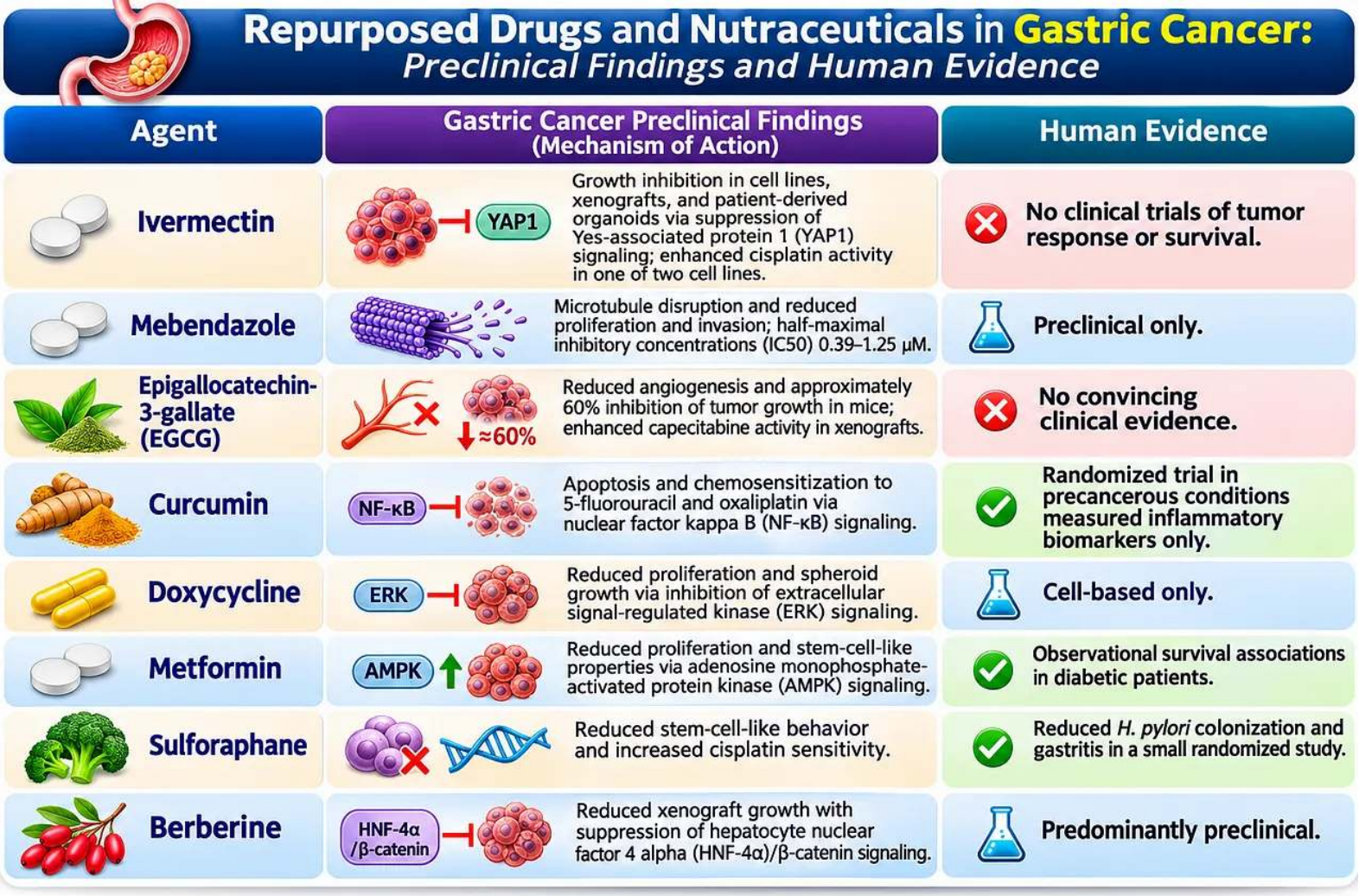
Radiotherapy is not routine but is used in selected postoperative situations, particularly after inadequate surgery or lymph-node dissection, and for palliation of bleeding, pain, or obstruction, where it can provide meaningful relief even when cure is no longer possible.[39, 40]

Nutritional care is central because the disease and its treatment impair intake and absorption. Weight loss, sarcopenia, and iron and vitamin B12 deficiency are common, and lifelong vitamin B12 replacement is generally required after total gastrectomy. Iron, calcium, vitamin D, and other nutritional parameters also require monitoring. Dietitian involvement, adequate protein, resistance exercise when feasible, and symptom management are essential, and severe dietary restriction or prolonged fasting may harm patients who are already losing weight or muscle.[41]

Because many cases have a treatable infectious cause, the key prevention measures are H. pylori eradication, smoking cessation, limiting salt and salted preserved foods, and surveillance of individuals with advanced atrophy or intestinal metaplasia.[8] Organized endoscopic screening in South Korea has been associated with reduced gastric cancer mortality.[42] Population screening is not recommended in the US, but targeted surveillance may be appropriate for high-risk individuals.

8. Metabolic and integrative care

Gastric cancers show metabolic abnormalities involving glycolysis, mitochondrial and lipid metabolism, hypoxia signaling, and the tumor microenvironment, which vary among molecular subtypes and even within a single tumor. This biology has prompted interest in repurposed drugs and nutraceuticals. Each of the agents most often discussed has published preclinical activity against gastric adenocarcinoma, but laboratory activity is far better established than benefit in patients, and the human evidence is largely observational or prevention-oriented.



Three distinctions matter. First, metformin has a genuine human signal: a meta-analysis of seven retrospective cohorts (2,858 diabetic patients) associated its use with better overall survival (hazard ratio 0.72) and cancer-specific survival (hazard ratio 0.81), although heterogeneity was high and confounding and immortal time bias could not be excluded.[52] Metformin use has also been associated with a lower risk of gastric cancer after H. pylori eradication.[56] It is best regarded as a plausible repurposing candidate with supportive but inconsistent observational evidence. Second, the human studies of curcumin and sulforaphane address inflammation in precancerous or infected stomachs, not treatment of established cancer.[49, 54] Third, the 2026 ivermectin organoid study, in which ivermectin with cisplatin produced 66% to 82% dead-cell staining in samples from two patients, used high concentrations (100 μM), showed no response in one cell line, and found that YAP1 suppression faded over time; dead-cell percentages in culture are not clinical response rates.[44]

Translation to patients depends on whether tolerable doses achieve effective tumor concentrations, whether formulations match those studied (curcumin's poor oral

bioavailability is a recognized obstacle), whether effects hold across different gastric cancers, and whether controlled trials show improved outcomes.[57] Laboratory reports that individual agents enhance chemotherapy do not establish that a multi-agent combination is effective or safe; combinations require their own safety and efficacy testing, and doses should not be extrapolated from cell-culture concentrations or animal experiments. Similarly, ketogenic diets, caprylic acid (C8) medium-chain triglyceride (MCT) oil, and fasting have not been tested in adequately powered gastric cancer trials, and because malnutrition and cachexia are common, preserving nutritional status and lean body mass takes priority over achieving high ketone levels. [41, 58]

A pragmatic approach

These agents are inexpensive, generally well tolerated, and biologically plausible, and definitive randomized trials are unlikely ever to be conducted. For patients who choose this approach, integrative care can therefore begin alongside standard therapy from the outset, rather than being reserved for when standard options have failed. It should complement, never replace, surgery, chemotherapy, and targeted or immune therapies, and each agent should be reviewed by the oncology team and an oncology pharmacist for interactions with concurrent treatment, organ function, and nutritional risks. Diet, nutrition, and physical activity remain the central pillar of care. There is not yet clinical proof that a Five-Axis metabolic regimen improves gastric cancer survival, and patients should be counseled accordingly and offered relevant clinical trials.

9. A practical treatment framework

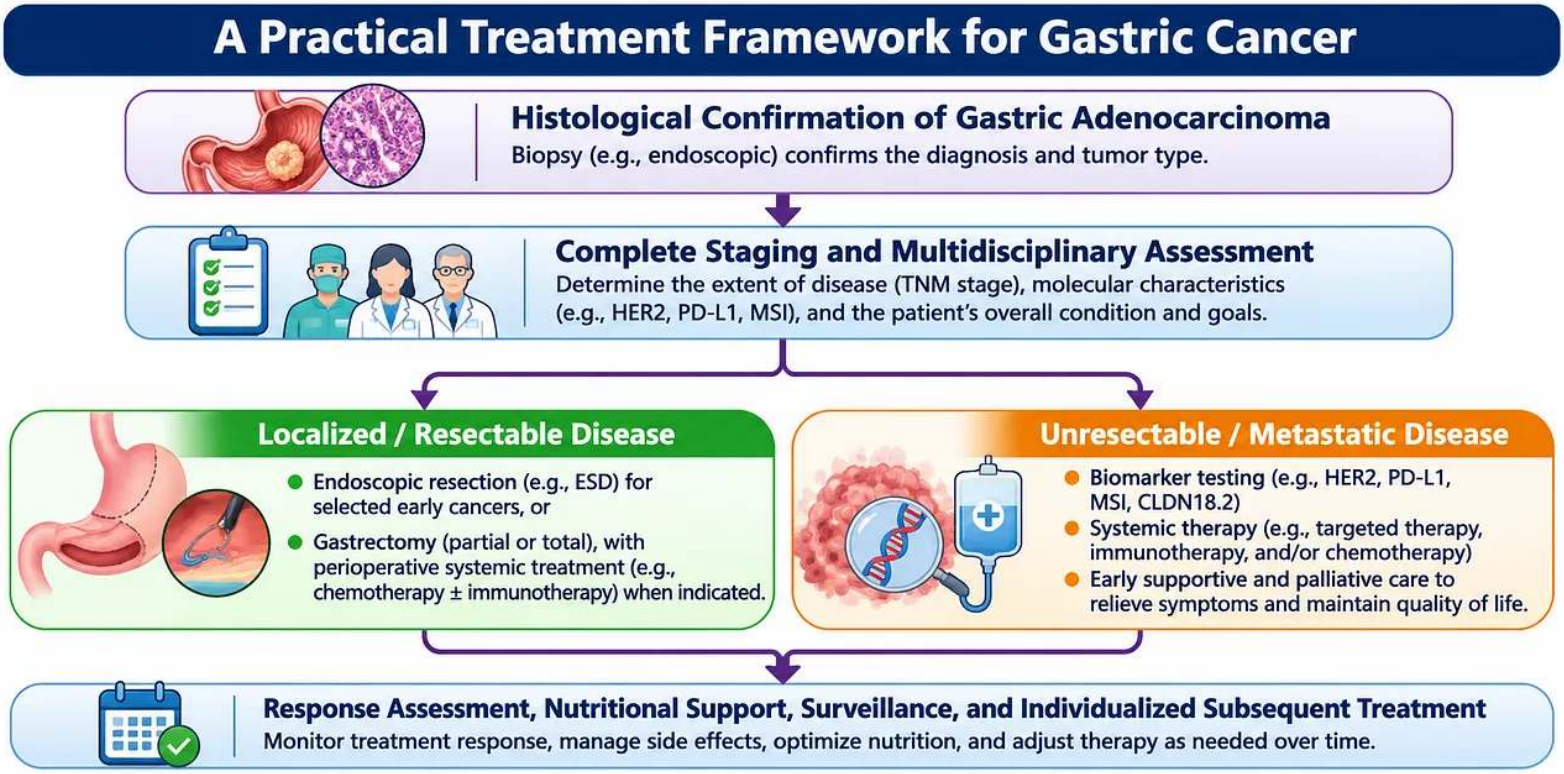


Figure 3. A practical treatment framework for gastric cancer, from histological confirmation through staging, stage-appropriate treatment, response assessment, nutritional support, surveillance, and individualized subsequent treatment.

Conclusion

Gastric cancer management is moving from treatment based on anatomical stage and histology to biomarker-informed care. Three developments stand out: H. pylori eradication as a major prevention strategy; integration of perioperative chemotherapy and immunotherapy into curative treatment; and molecular selection of targeted and immune therapies in advanced disease. Surgery and conventional chemotherapy nevertheless remain foundations of cure. The greatest opportunities lie in earlier detection, accurate staging, comprehensive molecular profiling, coordinated multidisciplinary care including nutritional and, where patients choose, integrative support, and affordable access to effective medicines worldwide.

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