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REVIEW ARTICLE

TTD expert consensus on emesis in gastric cancer: evidence-based strategies for prevention and treatment

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Abstract

Emesis is a common complication in patients with gastric cancer (GC), resulting from both tumor progression and adverse events of antineoplastic therapies. It is estimated that up to 70% of patients experience nausea and vomiting, significantly impacting quality of life. Symptoms can lead to dehydration, malnutrition, fatigue, and emotional disturbances like anxiety and depression. Etiological factors fall into two categories. First, neoplasia-related mechanisms—gastric outlet obstruction, gastroparesis due to autonomic dysfunction, invasion of adjacent structures, presence of peritoneal carcinomatosis or malignant ascites—contribute to gastric emptying disorders. Second, antineoplastic treatments—including chemotherapy, radiotherapy, and targeted therapies—carry an inherent inducing emesis risk and can cause acute, delayed, anticipatory, breakthrough, or refractory emesis. International guidelines from the American Society of Clinical Oncology (ASCO), European Society for Medical Oncology (ESMO), Multinational Association of Supportive Care in Cancer (MASCC), and National Comprehensive Cancer Network (NCCN) recommend combination regimens that include 5-hydroxytryptamine type 3 receptor antagonist (anti-5-HT3), dexamethasone, neurokinin-1 receptor antagonists (anti-NK1), and, occasionally, olanzapine. Prophylaxis should be tailored to the emetic risk of the treatment and the individual characteristics of the patient, like age, sex, and pharmacogenomic factors. Pharmacological strategies should be complemented with non-pharmacological interventions to enhance therapeutic efficacy and alleviate symptoms. This consensus guide from the Spanish Cooperative Group for the Treatment of Digestive Tumours (TTD) highlights the importance of multidisciplinary and individualized approaches to managing emesis associated with GC. Evidence-based therapeutic algorithms are essential to achieving these goals.

Keywords Antiemetics · Quality of life · Gastric cancer · Emesis · Nausea · Chemotherapy

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Abbreviations

5-HT3	5-Hydroxytryptamine type 3 receptor
Anti-5-HT3	5-Hydroxytryptamine type 3 receptor antagonist
Anti-NK1	Neurokinin-1 receptor antagonist
ASCO	American Society of Clinical Oncology
AUC	Area under the curve
CINV	Chemotherapy-induced nausea and vomiting
CTCAE	Common Terminology Criteria for Adverse Events
ESMO	European Society of Medical Oncology
GC	Gastric cancer
IL-1β	Interleukin-1 beta
IL-6	Interleukin 6
IL-33	Interleukin 33
MASCC	Multinational Association of Supportive Care in Cancer
NCCN	National Comprehensive Cancer Network
NEPA	Netupitant Palonosetron
NK1	Neurokinin-1 receptor
TNF-α	Tumor necrosis factor alpha
TTD	Spanish Cooperative Group for the Treatment of Digestive Tumours

Introduction

Gastric cancer (GC) is the fifth most common tumor worldwide, with almost one million new cases reported in 2022 (4.9% of all cancers). It is also the fifth leading cause of cancer-related mortality, accounting for 659,853 deaths (6.8% of the total), as reported in the GLOBOCAN 2022 estimates [1]. Data from the 2023 population-based SURVCAN-3 study show that 3-year net survival for GC ranged from 13 to 44%, showing disparities across countries based on development level and healthcare system performance [2]. Nausea and vomiting are common in gastric cancer patients and arise from the disease itself, its treatment, or other factors, which can significantly impact their quality of life.

Emesis is a frequent yet often inadequately managed symptom in GC patients. Approximately 30% of patients experience emesis, a figure that can rise to 70% in advanced stages. Beyond physical complications such as dehydration, malnutrition, and fatigue, emesis also contributes to emotional distress (anxiety, fear, depression) and social withdrawal, ultimately reducing treatment adherence and overall quality of life. These episodes may result from both cancer progression and the side effects of chemotherapy, radiotherapy, or emetogenic medications [3, 4]. Additionally, persistent emesis can compromise oncological treatment, leading to poor adherence and decreased therapeutic efficacy [5–7].

Emesis is commonly classified into grades based on its severity and impact on the patient's quality of life, following standardized systems, such as those provided by the Common Terminology Criteria for Adverse Events (CTCAE) (Table 1), which is widely used in oncology [8]. Other classification systems also exist, including the Multinational Association of Supportive Care in Cancer (MASCC) guidelines [9]. In GC, emesis arises not only from chemotherapy-induced nausea and vomiting (CINV), but also from tumor-related factors such as gastric outlet obstruction, gastroparesis, or peritoneal carcinomatosis. These mechanisms are often underrepresented in international guidelines. Furthermore, GC patients frequently present with altered gastric motility and may undergo surgical procedures or treatments that modify gastrointestinal physiology, all of which require tailored antiemetic approaches not specifically addressed in existing protocols.

This article explores the incidence and etiological factors of emesis in both gastric cancer itself and its primary treatment. It also provides a summary of prophylaxis and management strategies. Additionally, it proposes a therapeutic algorithm focused on prevention of nausea and vomiting associated with GC, since avoiding their occurrence is essential for improving clinical outcomes and patient quality of life. Effective anticipatory control of CINV is necessary to prevent the onset of difficult-to-manage symptoms, enhance treatment adherence, and reduce subsequent complications.

Table 1 Emesis Severity Grading According to CTCAE (v5.0) [8]

Grade	Frequency	Impact
1 (Mild)	Transient vomiting or a single episode within 24 h	No medical treatment required
2 (Moderate)	2–5 episodes within 24 h	Requires medical treatment Moderate impact on basic daily activities
3 (Severe)	≥6 episodes within 24 h	Requires immediate medical intervention (hospitalization or intravenous hydration)
4 (Life-threatening)	Intractable vomiting	Immediate life-threatening risk Requires intensive care
5 (Fatal outcome)	Death due to complications	Fatal outcome due to aspiration or organ failure

CTCAE common terminology criteria for adverse events

Etiological factors related to the neoplasm

Nausea and vomiting are common symptoms in GC patients, with an incidence ranging from 6 to 40% of cases. These symptoms have a multifactorial etiology, closely linked to tumor presence. Gastric outlet obstruction, often caused by tumor invasion of the pylorus, impairs gastric emptying and causes abdominal distension, early satiety, and postprandial vomiting. Beyond its significant impact on the patient's quality of life, these effects exacerbate malnutrition and weight loss, further complicating oncological treatment [10, 11].

In advanced stages, GC may infiltrate and compress adjacent structures such as the duodenum, pancreas, or transverse colon, which may lead to functional obstruction of the upper digestive tract and further impair gastric emptying. This complication is particularly common in patients with metastatic disease or locally advanced tumors. Likewise, peritoneal carcinomatosis and ascites are frequent manifestations in advanced stages and are associated with recurrent emesis. Malignant ascites can increase intra-abdominal pressure, induce gastric distension and trigger nausea and vomiting. Meanwhile, peritoneal carcinomatosis, which results from tumor dissemination within the peritoneum, may lead to partial or complete bowel obstruction and further aggravate persistent emesis. These challenging symptoms pose a significant hurdle in the palliative management of advanced GC [10, 11].

GC can also induce gastric motility disorders, such as gastroparesis, due to autonomic nervous system dysfunction. This dysfunction, often linked to tumor invasion of peripheral nerves, disrupts the coordination of peristaltic movements, which results in delayed gastric emptying and content retention. This process contributes significantly to the development of nausea and vomiting. Additionally, tumor progression further exacerbates these symptoms as it compromises the stomach's anatomical structure and reduces its functional capacity. The associated autonomic dysfunction caused by tumor infiltration further intensifies these clinical manifestations [12].

Chronic inflammation driven by tumor progression plays a critical role in the pathophysiology of vomiting in GC patients. Enterochromaffin cells in the gastrointestinal tract produce over 90% of the body's serotonin, along with substantial amounts of substance P. Serotonin activates 5-hydroxytryptamine type 3 (5-HT3) receptors in both the gastrointestinal tract and central nervous system, which are key regulators of gastrointestinal motility, nausea, and vomiting. Chemokines and cytokines such as interleukin-1 beta (IL-1β), tumor necrosis factor alpha (TNF-α) and interleukin 6 (IL-6) contribute to emesis by sensitizing vagal afferent pathways and acting as pro-inflammatory mediators that activate central and peripheral components of the emetic reflex [13]. Additionally interleukin 33 (IL-33), has been shown to stimulate serotonin release from enterochromaffin cells in the gastrointestinal tract [14]. Chronic inflammation can alter gastric mucosal responses, which leads to further functional deterioration and worsens digestive symptoms in these patients (Table 2) [15, 16].

Table 2 Anatomical and Functional Factors Associated with Emesis in GC [42]

Anatomical factors	Functional factors
Gastric outlet obstruction	Gastroparesis due to autonomic dysfunction
Invasion of adjacent structures (duodenum, pancreas, and transverse colon)	Chronic inflammation
Malignant ascites	Intestinal motility dysfunction

GC Gastric Cancer

Table 3 Emetic Risk of Antineoplastic Agents Used in GC [17]

Emetic risk	Emesis frequency (%)	Antineoplastic agents
High	> 90	Cisplatin
Moderate-High	61–90	Carboplatin Trastuzumab deruxtecan Zolbetuximab Oxaliplatin (in women ≤ 50 years of age)
Moderate-Low	31–60	Oxaliplatin Irinotecan
Low	10–30	Docetaxel Paclitaxel 5-Fluorouracil
Minimal	< 10	Capecitabine Nivolumab Pembrolizumab Ramucirumab Trastuzumab

Table based on the National Comprehensive Cancer Network (NCCN) Clinical Practice Guideline v2.2024

GC Gastric Cancer

Etiological factors related to primary treatment

One of the main causes of emesis in GC patients is antineoplastic treatment. These drugs inherently carry a potential risk of nausea and/or vomiting, which varies depending on the specific drug used (Table 3) [17]. Categorization of this

risk is complex because the data comes from heterogeneous studies that differ in tumor type and stage, therapeutic combinations, prophylaxis strategies and data collection methods. Additionally, factors such as dose, route of administration, and infusion rate influence the risk of emesis. Therefore, full awareness of these factors is essential to properly address the issue, improve patients'quality of life, and enhance their treatment adherence.

CINV is traditionally classified into acute, delayed, anticipatory, breakthrough, and refractory types [18]. Acute emesis occurs within the first 24 h after treatment administration and peaks around 5–6 h post-treatment. It is mediated by serotonin release in gastrointestinal tract cells, which stimulates the vagus nerve and afferent splanchnic pathways to the central nervous system. Delayed emesis appears between 24 and 72 h after treatment and is mediated by substance P, which acts on neurokinin 1 (NK1) receptors in the vagus nerve and central nervous system. Anticipatory emesis occurs before treatment, as a conditioned response. Breakthrough emesis arises beyond the fifth day of treatment, despite appropriate antiemetic prophylaxis. Finally, refractory emesis occurs during subsequent treatment cycles, even with adequate antiemetic management.

Targeted therapies such as monoclonal antibodies used in GC, have distinct mechanisms of action and toxicity compared to chemotherapy. In the case of emesis, it occurs through a complex process that involves immune mechanisms and effects on the central nervous system. The destruction of tumor cells and the release of pro-inflammatory cytokines activate chemoreceptors in the central nervous system, the vomiting center, and the autonomic nervous system. Among these agents, zolbetuximab stands out, as it is classified as a drug with a moderate-to-high emetogenic risk (Table 3). During the first infusion of zolbetuximab, the first episode of nausea and vomiting occurs within the first hour and is attributed to gastric damage caused by inflammation in the submucosa of the body of the stomach and pylorus [19, 20]. On the other hand, trastuzumab deruxtecan, a monoclonal antibody conjugated with a chemotherapy molecule, carries a high emetogenic risk, as stated in the National Comprehensive Cancer Network (NCCN) guidelines; it is at the upper end of the moderate-risk category, as defined by the American Society of Clinical Oncology (ASCO) and the European Society of Medical Oncology (ESMO) guidelines [21].

In addition to the intrinsic emetogenic risk of each drug, other factors can help estimate the risk of emesis, such as the combination of antineoplastic drugs, patient characteristics, pharmacogenomics, or the use of concurrent medications. In combination regimens, the agent with the highest emetogenic risk should be considered and prophylaxis should be adjusted to the duration of the emetogenic risk [22]. Among patient-related factors, female sex has been shown to be a risk factor for emesis [23], as well as age under 50 years old, a history of CINV, emesis during pregnancy, or motion sickness. Chronic alcohol consumption, paradoxically, is a protective factor against delayed CINV, possibly due to chronic damage to the brainstem associated with harmful alcohol consumption [24].

In addition, pharmacogenomics also influences the efficacy of antiemetic treatments. Certain genetic polymorphisms can affect the metabolism, transport and action of these drugs [25, 26]. For instance, patients with the CYP2D6 genotype are ultra-rapid metabolizers of 5-HT3 antagonists (anti-5-HT3) and are undertreated with standard dosing regimens [27].

Some commonly used drugs for the management of symptoms or complications of GC, such as opioids and certain nonsteroidal anti-inflammatory drugs, can increase the risk of emesis. Likewise, the emetogenic risk associated with radiotherapy differs based on the irradiation site, field volume, and dose, and ranges from minimal to high [28].

Therefore, it is essential to assess the emetogenic risk of the chosen regimen, individual patient characteristics, and the influence of other treatments to optimize antiemetic management and improve treatment tolerance.

Prophylaxis and treatment of nausea and vomiting associated with gastric cancer

Effective antiemetic prophylaxis is crucial, as inadequate control during early cycles increases the risk of subsequent treatment failure and complicates symptom management. The recommendations presented are based on current international guidelines (MASCC/ESMO, ASCO), and adapted to GC [22, 29–32].

For primary prophylaxis (Table 4 and Fig. 1) in highly emetogenic chemotherapy [22, 29–32], acute emesis (first 24 h) should be prevented by the administration of a four-drug regimen before chemotherapy, which includes a single dose of an anti-5-HT3 agent, dexamethasone, an NK1 receptor antagonist (anti-NK1), and olanzapine (I, A). Palonosetron is the preferred anti-5-HT3 agent due to its superior efficacy in delayed emesis control [18]. It is often combined with netupitant (anti-NK1) as a fixed-dose formulation (NEPA).

For delayed emesis (days 2–5), dexamethasone and olanzapine should be continued on days 2 to 4 to prevent delayed nausea and vomiting, regardless of nausea control on day 1 of the cycle (II, B). Regarding olanzapine, although the only investigated regimen is a once-daily dose for four days, individual assessment should determine whether to extend treatment beyond day 1 based on emesis risk, patient age, and drug tolerance. If sedation or drowsiness (the primary side effect) is problematic, particularly in elderly patients,

Table 4 Antiemetic Agents [10]

Pharmacological group	Drugs	Dosage	Indications
Anti-5-HT3 agents	Palonosetron	0.25 mg IV or 0.5 mg PO	High-, moderate- and low-risk chemotherapy (optional) High-, moderate- and low-risk radiotherapy (optional) Rescue medication (if not used in primary prophylaxis)
	Granisetron	1 mg IV or 2 mg PO	
	Ondansetron	8 mg IV or 16 mg PO	
Anti-NK1 agents	Netupitant*	300 mg PO	High- and moderate-risk chemotherapy: Rescue medication (if not used in primary prophylaxis)
	Fosaprepitant	150 mg IV	
	Aprepitant	D1: 125 mg PO; D2-3: 80 mg PO	
Corticosteroids	Dexamethasone	D1: 12 mg PO/IV; D2-3: 8 mg PO	High-risk chemotherapy
		4–8 mg PO or IV	Moderate- and low-risk chemotherapy Oral chemotherapy High- and moderate-risk radiotherapy Rescue medication
Atypical Antipsychotic with Antiemetic Action	Olanzapine**	5–10 mg/day PO	High-risk chemotherapy Rescue medication
Dopamine Antagonists	Metoclopramide	10 mg/8 h PO	Low-risk chemotherapy Rescue medication
	Domperidone	10 mg/8 h PO	
	Haloperidol	0.5–2 mg PO/IM 0.5 mg IV	Rescue medication
Benzodiazepines	Lorazepam	0.5–2.5 mg PO	Anticipatory emesis Rescue medication
	Alprazolam	0.5–2 mg (max. 3 mg) PO	

*Administer together with 0.5 mg palonosetron as part of the fixed-dose combination NEPA (Netupitant + Palonosetron)

** The standard dose is 10 mg. The 5 mg dose has shown superiority over placebo, but its efficacy compared to 10 mg remains uncertain due to a lack of direct comparative studies. If sedation or drowsiness (the primary side effects) are problematic, particularly in elderly patients, an initial daily dose of 5 mg and/or nighttime administration (before bedtime or with dinner) may be considered

Anti-5-HT3 5-Hydroxytryptamine Type 3 Receptor Antagonist, *Anti-NK1* Neurokinin-1 Receptor Antagonist, *D* day, *IV* intravenous route, *IM* intramuscular, PO oral route

an initial 5 mg daily dose (which has been shown to be superior to placebo but with unclear efficacy compared to the standard 10 mg dose) may be considered, and administration can be scheduled at bedtime or with dinner. In addition, if aprepitant (anti-NK1) is used at a dose of 125 mg on day 1 of the cycle, it may be administered again at 80 mg/day on days 2 and 3 to manage delayed emesis.

For moderately emetogenic chemotherapy regimens not based on carboplatin or oxaliplatin, primary prophylaxis for acute emesis includes a two-drug regimen with single doses of dexamethasone and anti-5-HT3 (II, C) agent, preferably palonosetron [18]. For delayed emesis, no steroid or other antiemetic should be routinely administered after day 1 of the cycle. In regimens that contain oxaliplatin in women ≤ 50 years old, a three-drug regimen with an anti-5-HT3 agent (preferably palonosetron), dexamethasone, and an anti-NK1 agent is recommended (III, B) [33, 34]. Trastuzumab deruxtecan has an emetic potential comparable to that of carboplatin at doses equal to or greater than an area under the curve (AUC) of 5. Although there are no prospective studies, the same three-drug prophylaxis (anti-5-HT3, anti-NK1 and dexamethasone) recommended for carboplatin is suggested. In this context, the use of the fixed-dose agent NEPA provides five-day coverage with a single dose, which eliminates the need for rescue medication. With regard to zolbetuximab (Fig. 2), the only guideline published so far recommends that olanzapine be added to the standard triple-drug prophylaxis before treatment [35]. Although immune checkpoint inhibitors are generally associated with a minimal emetogenic risk (Table 3), they may induce nausea and vomiting, particularly when gastrointestinal immune-related adverse events are present. These symptoms are believed to result from mucosal inflammation and the release of pro-inflammatory cytokines. Mild cases may be managed with a 5-HT3 receptor antagonist alone or combined with a short course of corticosteroids. In moderate or persistent cases, especially when immune-mediated toxicity is suspected, escalation to systemic corticosteroids such as dexamethasone (4–8 mg/day) or oral budesonide can be considered. Temporary suspension of immunotherapy may be considered in severe or clinically significant cases until symptom resolution is achieved [36]. Finally, prior to low-emetogenic chemotherapy, prophylaxis with a single antiemetic agent on day 1 of the cycle can be considered: dexamethasone, an anti-5-HT3 agent or a dopaminergic receptor antagonist (II, B).

If nausea and/or vomiting occur despite primary prophylaxis (refractory emesis), it is recommended to re-evaluate the emetogenic risk and adjust treatment accordingly, as well as to rule out other causes of emesis [37]. If olanzapine was

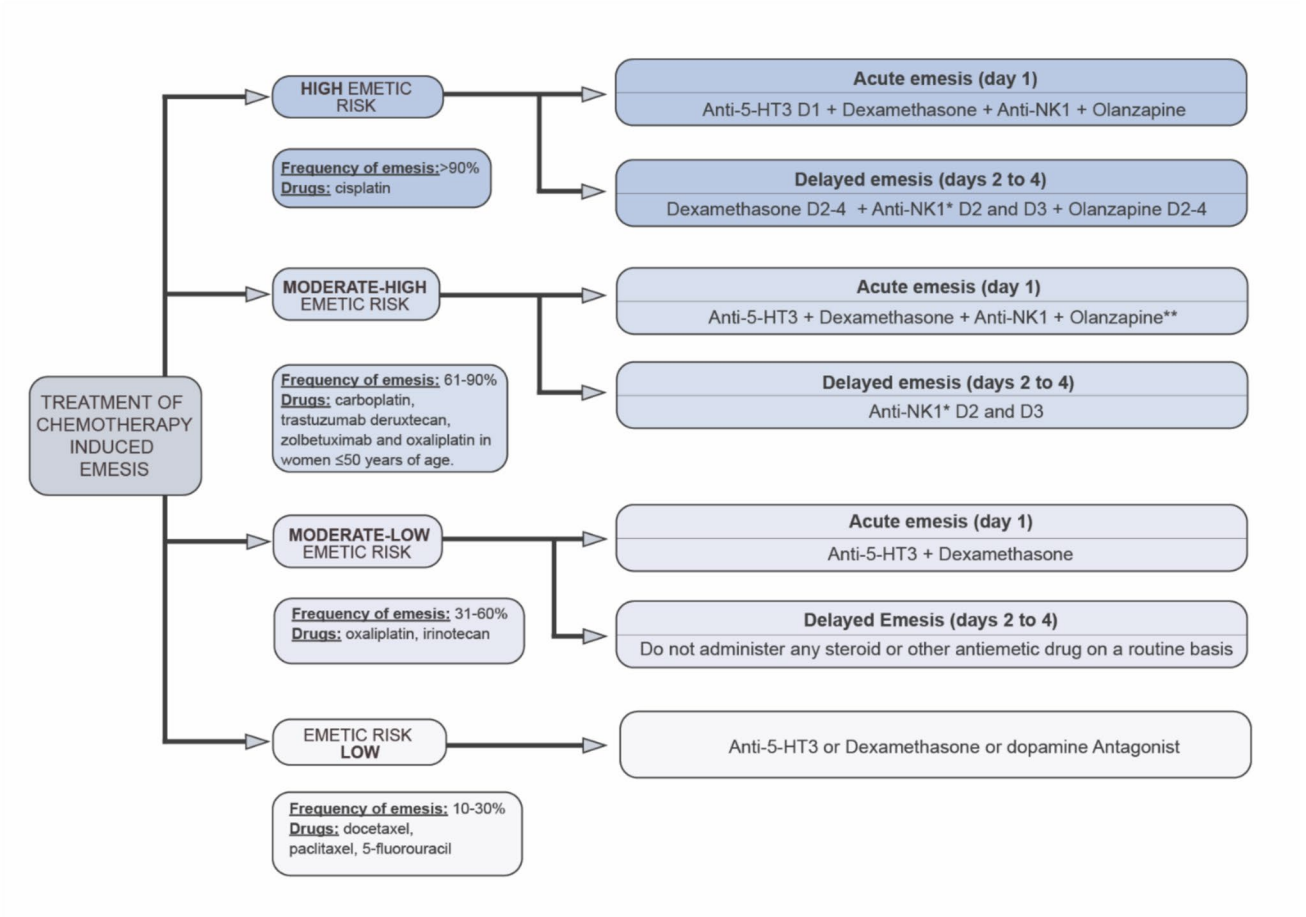
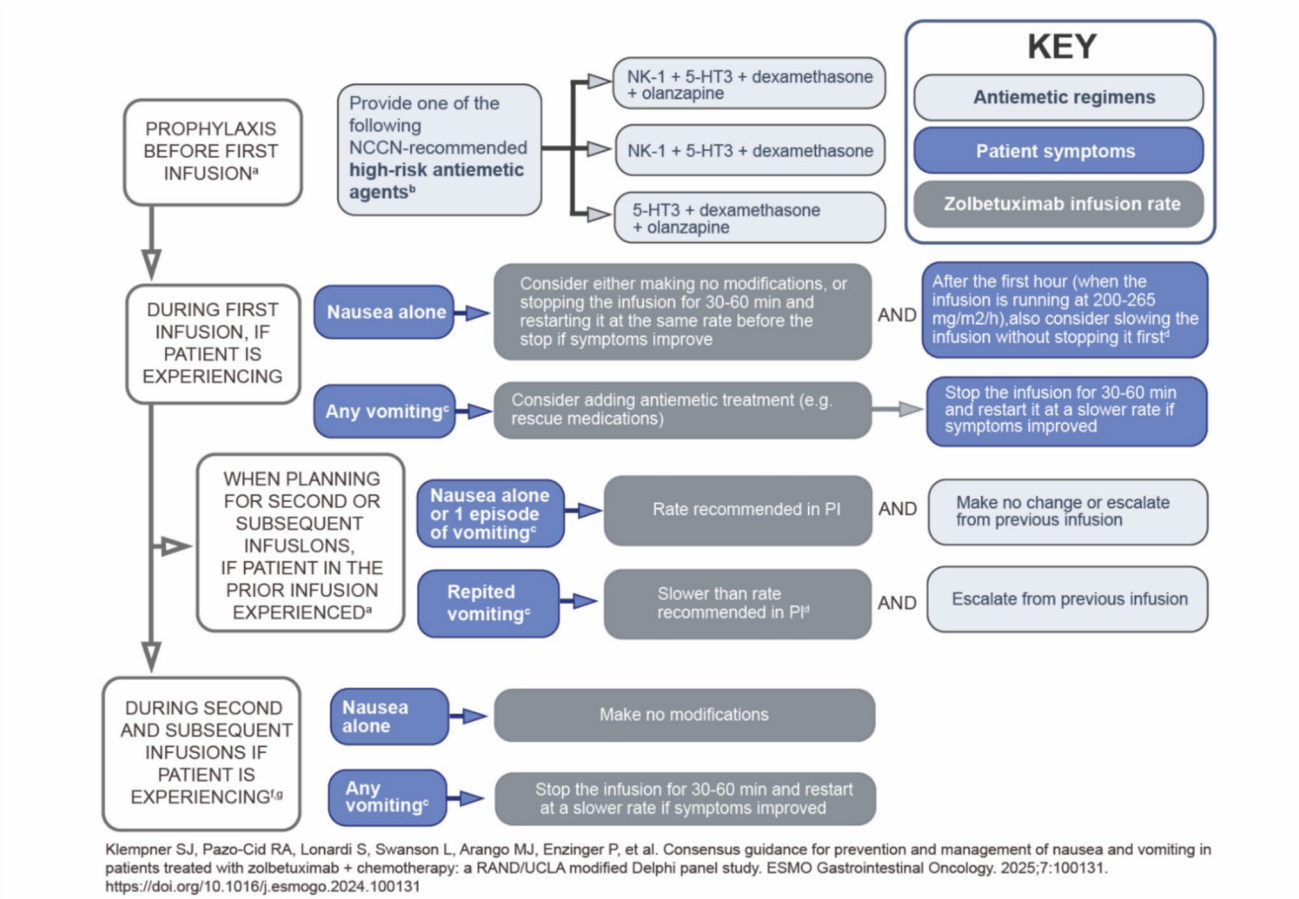


Fig. 1 Evidence-based therapeutic algorithm for the treatment of chemotherapy-induced emesis. *If aprepitant (anti-NK1) is used at a dose of 125 mg on day 1 of the cycle, it may be administered again at 80 mg/day on days 2 and 3 to manage delayed emesis.**It is recommended to consider the addition of olanzapine to the three-drug regimen before the first infusion of zolbetuximab. *Anti-5-HT3* 5-Hydroxytryptamine Type 3 Receptor Antagonist, *Anti-NK1* Neurokinin-1 Receptor Antagonist, *D* day

not used in primary prophylaxis, it should be prescribed as rescue medication. If it was already included, an antiemetic from a different class (e.g., anti-NK1, dopamine receptor antagonists, or benzodiazepines) should be offered, along with continuing the standard antiemetic regimen (II, B). Cannabis derivatives such as dronabinol and nabilone are not considered first-line options for the management of CINV. Their use is not supported by solid clinical evidence, as no randomized trials have demonstrated clear efficacy in this setting. Moreover, their toxicity profile—which includes frequent adverse events such as dizziness, somnolence, and cognitive impairment—further limits their clinical utility. For these reasons, international guidelines recommend caution and restrict their use to selected cases of refractory emesis, when standard antiemetic therapies have failed (II, B) [17, 29, 38].

In elderly patients, careful consideration is needed due to the increased risk of adverse events and drug interactions with antiemetics. Beyond the sedative effects of olanzapine, 5-HT3 receptor antagonists—particularly ondansetron—may prolong the QT interval, especially in those with cardiac comorbidities or concurrent QT-prolonging drugs. NK1 receptor antagonists can interact with CYP3A4-metabolized agents, such as corticosteroids and anticoagulants, raising toxicity risk. Dopamine antagonists (e.g., metoclopramide, haloperidol) may worsen extrapyramidal symptoms or trigger delirium in frail patients. Benzodiazepines increase the likelihood of sedation, confusion, and falls, and should be used sparingly. Dose adjustment, clinical monitoring, and individualized risk–benefit evaluation are essential when prescribing these agents in older adults.

Radiotherapy to the upper abdomen is considered to pose a moderate emetogenic risk, and prophylaxis with an anti-5-HT3 agent (II, A) and optional dexamethasone (II, C) is recommended [35]. In the case of concomitant chemoradiotherapy, antiemetic prophylaxis should be tailored to the chemotherapy regimen's emetogenic risk [35].



Klempner SJ, Pazo-Cid RA, Lonardi S, Swanson L, Arango MJ, Enzinger P, et al. Consensus guidance for prevention and management of nausea and vomiting in patients treated with zolbetuximab + chemotherapy: a RAND/UCLA modified Delphi panel study. ESMO Gastrointestinal Oncology. 2025;7:100131. https://doi.org/10.1016/j.esmogo.2024.100131

Fig. 2 Expert consensus algorithm for the prevention and management of zolbetuximab-induced nausea and vomiting [43]. *5-HT3* 5-Hydroxytryptamine Type 3 Receptor, *NCCN* National Comprehensive Cancer Network, *NK1* Neurokinin-1 Receptor, *PI* prescribing information

With regards to vomiting related to the anatomical and/or functional features of GC, management strategies include endoscopic stenting, surgical bypass or gastric decompression in cases of obstruction, as well as the use of prokinetics such as metoclopramide in patients with gastroparesis [39, 40].

Integrative and non-pharmacological therapies lack methodological quality in most publications, which makes it difficult to establish a strong recommendation for their role in antiemetic prophylaxis [41]. Within this section we can mention acupuncture and electro-acupuncture (complementary techniques for the treatment of nausea and vomiting, particularly in acute emesis; II, B); acupressure (studies suggest a reduction in nausea); progressive muscle relaxation and relaxation techniques (guided imagery; II, B); and nutrition. Nutritional counseling on healthy eating and an individualized dietary plan provided by a dietitian or other healthcare professional is recommended (II, B) [24]. Some recommendations, based on observational studies with small sample sizes and heterogeneous designs with respect to the types of cancer included and dietary interventions evaluated, are as follows: prioritize a soft and fractionated diet, with smaller and more frequent meals; eat slowly, chew well and rest post-meal; follow a Mediterranean diet, consume ginger-infused beverages (they have antioxidant and anti-inflammatory properties that help reduce chemotherapy-induced oxidative stress); include cold and dry foods (e.g., toast, crackers, and bread); avoid the consumption of fatty, highly seasoned or irritating foods; maintain adequate hydration; and consume adequate protein (may stimulate gastrin secretion, promote normal gastric rhythms and reduce nausea), fats and carbohydrates.

Recommendations

- Initiate prophylactic antiemetic treatment in patients at high and moderate risk of emesis.
- Consider combining different antiemetics to achieve optimal emesis control.
- Monitor treatment response and adjust the type and dosage of antiemetics based on emesis severity and patient response.

Conclusions

Effective emesis management in GC requires individualized risk assessment, integrating tumor-related complications and treatment-associated emetogenicity to optimize patient care. In clinical practice, combined antiemetic regimens that include anti-5-HT3 agents, dexamethasone and anti-NK1 agents, with the possible addition of olanzapine, have proven effective in the prevention of both acute and delayed episodes of emesis.

Additionally, non-pharmacological interventions, such as nutritional counseling, can complement pharmacological strategies and enhance treatment tolerance. It is essential to tailor antiemetic prophylaxis to the patient's individual characteristics (age, sex, and medical history) and the treatment's emetogenic risk, following evidence-based therapeutic algorithms to optimize adherence and continuity of oncological management.

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Declarations

Conflict of interest AFM: receives honoraria for speaker's bureau participation and serving on advisory boards form Amgen, Servier, Merck, Roche, Astellas, Daichi, Lilly, Astra Zeneca, Merck Sarp &Dome (MSD) and Bristol-Myers Squibb (BMS). Payments of travels and congress registration for Astra Zeneca, Daichi and Roche. IMD: receives honoraria for speaker's bureau participation, and serving on advisory boards from AMGEN, AstraZeneca, Bristol-Meyers Squibb (BMS), Merck Sharp & Dohme (MSD), Lilly and Sanofi. Payment of travels and congress registration fees from AMGEN, Merck Sharp & Dohme (MSD), Merck KGaA, Bristol-Meyers Squibb (BMS), AstraZeneca, IPSEN and ADVANZ PHARMA SPAIN, S.L.U. NML: receives honoraria for speaker's bureau participation and serving on advisory boards from AAA (a Novartis company), AMGEN, AstraZeneca, Bristol-Meyers Squibb (BMS), Merck Sharp & Dohme (MSD), Servier, Takeda. Payment of travels and congress registration fees from AAA (a Novartis company), ADVANZ PHARMA SPAIN, S.L.U. AMGEN, Merck Sharp & Dohme (MSD), Roche and Servier. VA: receives honoraria for speaker's bureau participation and serving on advisory boards form Lilly. Beigene, Organon, Astellas, Merck Sarp &Dome (MSD) and Bristol-Myers Squibb (BMS). Payments of travels and congress registration for Merck and MSD. BGP: receives honoraria for speakers'bureau participation and serving on advisory boards from Amgen, AAA (a Novartis company), Servier, Roche Farma, Lilly. Novartis, Eisai, Merck Sharp & Dohme (MSD) and Bristol-Myers Squibb (BMS). Payment of travels and congress registration fees from Servier, Novartis, Merck, Roche Farma, Ipsen, Lilly, Merck Sharp & Dohme (MSD), Amgen and Bristol-Myers Squibb (BMS). PJF: receives honoraria for speakers'bureau participation, and serving on advisory boards from Astellas, AstraZeneca, Bristol-Myers Squibb (BMS), Esteve, Merck Sharp & Dohme (MSD), Novartis, Nutricia, Pfizer, Rovi, Servier, Takeda, and Viatri and research grants from Astellas, AstraZeneca, BMS, and MSD.

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