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A Prospective Clinical Evaluation of Olanzapine-Based Antiemetic Regimen in Chemotherapy-Induced Nausea and Vomiting

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Abstract:

Aim: A Prospective Clinical Evaluation of Olanzapine-Based Antiemetic Regimen in Chemotherapy-Induced Nausea and Vomiting

Background: Chemotherapy-induced nausea and vomiting (CINV) is a debilitating side effect of cancer treatment that significantly impairs patient quality of life. While conventional regimens (5-HT₃ antagonists and corticosteroids) are available, many patients still experience inadequate control, particularly in the delayed phase. Olanzapine, an atypical antipsychotic, has emerged as a promising agent due to its broad receptor-blocking activity.[1][2][9][3]

Objective: To prospectively evaluate the clinical efficacy and safety of olanzapine in preventing and managing CINV in patients receiving highly emetogenic chemotherapy (HEC) and moderately emetogenic chemotherapy (MEC).[3]

Methodology: A prospective observational study was conducted over six months in the Medical Oncology department of a tertiary care hospital. The study included 50 adult cancer patients receiving HEC or MEC. Olanzapine (5mg or 10mg) was administered as part of the antiemetic regimen. Outcomes were recorded using the MASCC antiemetics tool and Visual Analogue Scale (VAS).[6]

Results:

- **Demographics:** 70% of participants were female, and 30% were male, with the majority aged between 40–65 years.
- **Common Diagnoses:** Breast cancer was the most frequent diagnosis, followed by lung and ovarian cancers.
- **Efficacy:** A complete response (no vomiting and no rescue medication) was achieved in 68% of patients. Acute and delayed phase control rates were 74% and 61%, respectively.
- **Safety:** The regimen was generally well-tolerated. The most common adverse effect was mild sedation (16%), followed by dizziness.[3][2][11]

Conclusion: Olanzapine-based therapy provides superior control for both acute and delayed CINV, particularly in HEC settings. Its clinical value is underscored by its cost-effectiveness and ability to reduce CINV-related morbidity.[2][3][10]

Keywords: Chemotherapy-induced nausea and vomiting (CINV); Olanzapine; Antiemetic therapy; HEC; MEC; Complete response.

1. INTRODUCTION

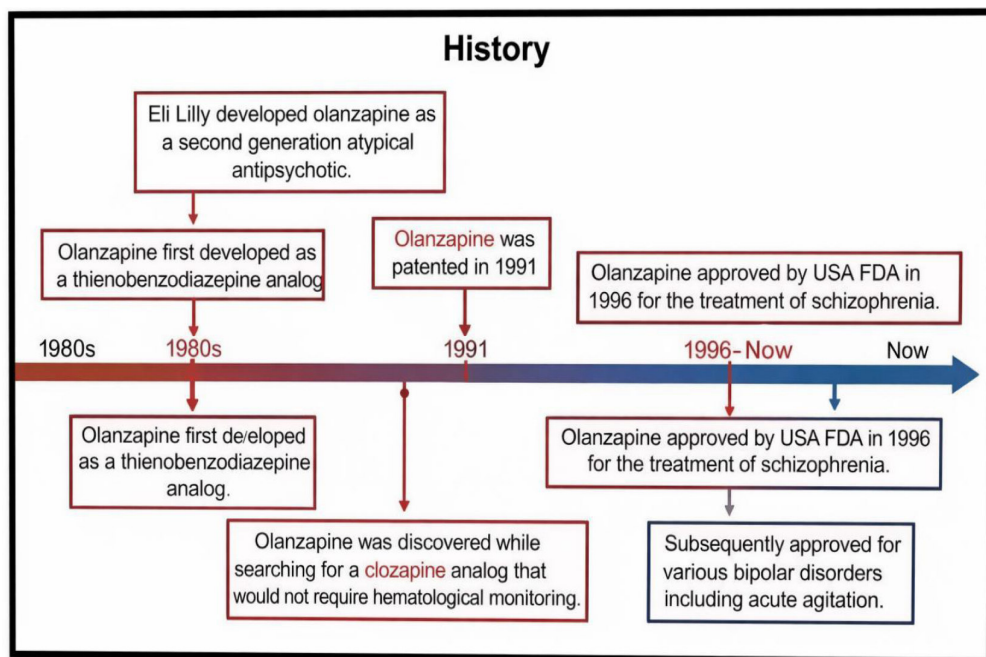
Chemotherapy-induced nausea and vomiting (CINV) is a common and distressing side effect of cancer treatment, affecting nearly 30–50% of patients despite standard antiemetic therapies. It can negatively impact treatment adherence, nutritional status, and overall quality of life. [1][5][9]

CINV involves both peripheral and central mechanisms. The acute phase is mainly mediated by serotonin acting on 5-HT₃ receptors, while the delayed phase is associated with substance P acting on Neurokinin-1 (NK1) receptors.[1][6]

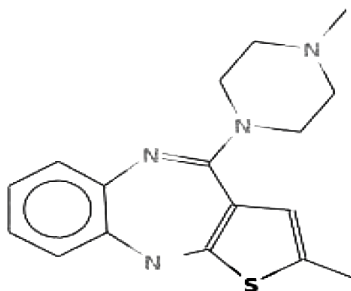
Although conventional triple therapy improves outcomes, it is often limited by cost and incomplete effectiveness. Olanzapine, an atypical antipsychotic, offers a broader approach by blocking multiple receptors involved in the emetic pathway, making it effective in controlling both acute and delayed symptoms.[4][5][8][10][11][2][3]

Recent studies suggest that olanzapine is useful not only as a rescue therapy but also as a preventive agent, particularly for delayed nausea. Its cost-effectiveness further supports its use in clinical practice. [2][3][10]

This study evaluates the efficacy and safety of olanzapine in managing CINV in patients receiving highly and moderately emetogenic chemotherapy. [2][3]



2. CHEMISTRY OF OLANZAPINE:



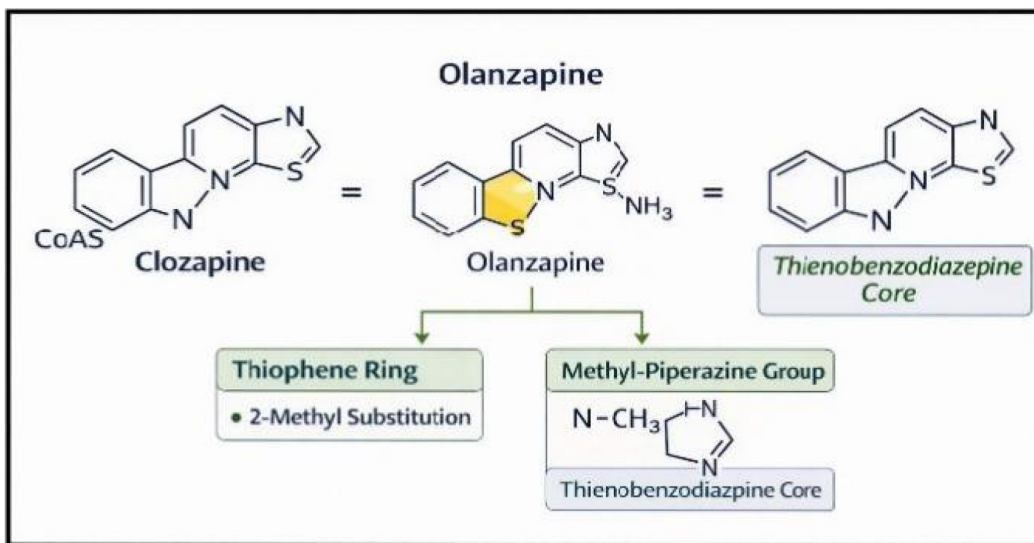
2.1 STRUCTURE OF OLANZAPINE:

Olanzapine (Zyprexa, C₁₇ H₂₀ N₄ S), with a molecular weight of 312.43 g/mol, exists in four crystalline polymorphs and several hydrated forms. Structurally, it is a thienobenzodiazepine derived by replacing a

benzene ring in the dibenzodiazepine scaffold with a theophany ring. As a fully synthetic compound, it is manufactured by condensing a malononitrile derivative with a thienodiazepine precursor. [15][16][11][13]

The methylpiperazinyl-thienobenzodiazepine pharmacophore allows high-affinity binding to D2 and 5-HT_{2A} receptors, a profile distinct from older phenothiazines. This specific arrangement maintains potent antipsychotic effects while reducing the risk of agranulocytosis. Primary metabolism occurs via glucuronidation and oxidation through the CYP1A2 and CYP2D6 pathways. [11][12][15]

2.2 STRUCTURAL ACTIVITY RELATIONSHIP



Activity of Olanzapine is sensitive to the tricyclic thienobenzodiazepine core, the specific substitution on the thiophene ring, and the presence of the methyl-piperazine moiety. The core can be modified to other heterocycles (like Clozapine), but the specific receptor affinity is highly dependent on the shape and electronic properties of these rings. [11][13]

COMMON FOR ALL THIENOBENZODIAZEPINES

Tricyclic Core: Essential for D₂ and 5-HT_{2A} receptor antagonism.

Piperazine Ring: The 4-methyl-1-piperazinyl group is critical for receptor binding.

Diamine Linkage: The nitrogen orientation in the diazepine ring maintains the "atypical" profile.

Lipophilicity: High lipophilicity is required to cross the blood-brain barrier (BBB).

-NH- Group: Serves as a key hydrogen bond donor for binding stability. [11][20]

RING 1 (Thiophene Ring)

Thiophene Replacement: Changing to a benzene ring (Clozapine) alters the safety profile.

2-Methyl Substitution: Increases affinity for D₂ and 5-HT₂ receptors.

Sulphur Atom: Essential for the electronic density required for binding. [11]

RING 2 (Piperazine Substitution)

N-Methyl Group: N - CH₃ is optimal; longer chains decrease receptor affinity.

Saturated Ring: The 6-membered saturated ring is necessary for correct nitrogen spatial. [11]

3. PHARMACOLOGY OF OLANZAPINE:

Olanzapine, also known as a second-generation atypical antipsychotic, functions as a potent antiemetic agent. This category of drugs serves a crucial function in managing and avoiding chemotherapy-induced nausea and vomiting (CINV) in patients. They work by inhibiting multiple neurotransmitter receptors, primarily targeting dopamine D₂, serotonin 5-HT₃, and muscarinic M₁ receptors. [11][2][3]

3.1 PHARMACOKINETICS

ABSORPTION

Olanzapine follows first-order absorption kinetics, primarily occurring via passive diffusion in the gastrointestinal tract. As a highly lipophilic molecule with a molecular weight < 500 Da (consistent with Lipinski's Rule of 5), it exhibits superior intestinal absorption compared to other second-generation antipsychotics. While available in oral (conventional and disintegrating) and intramuscular formulations, oral administration yields an absolute bioavailability of 60–65%. Peak plasma concentrations (C_{max}) are typically reached within 4–6 hours. As a dibasic weak base (pK_a ≈ 4.65 and 7.37), its absorption rate is heavily influenced by its physicochemical properties, including polar surface area and lipophilicity. [20][15][16]

DISTRIBUTION -The drug is extensively distributed into tissues, evidenced by a large volume of distribution (16.4 ± 5.1 L/kg). It is highly protein-bound, primarily to albumin and α_1 -acid glycoprotein. Olanzapine crosses the blood-brain barrier, displaying high affinity for dopamine (D₁, 2, 4), serotonin (5-HT_{2A/C}, 3), histamine (H₁), and muscarinic receptors, with lower affinity for α_2 -adrenergic and GABA sites. [15][16][11]

METABOLISM -Metabolism occurs via first-order kinetics in the liver through both Phase I (oxidation) and Phase II (conjugation) pathways. The primary metabolic route is glucuronidation via UGT1A4 and UGT2B10, forming the major inactive metabolite, 10-N-glucuronide. Cytochrome P450 enzymes, specifically CYP1A2 and FMO3, also contribute to the formation of 4'-N-desmethyl olanzapine and olanzapine N-oxide. Genetic polymorphisms in CYP1A2 and UGT variants significantly contribute to inter-individual variability. Notably, CYP1A2 inducers like smoking or carbamazepine can reduce serum concentrations, necessitating dosage adjustments. [12][15]

EXCRETION-Olanzapine has a mean elimination half-life of approximately 30 hours (ranging from 21–54 hours). Excretion is primarily renal (~60%), with the remainder (~30%) eliminated in faeces, mostly as inactive metabolites. The mean apparent plasma clearance is 26 L/h, though this is significantly higher in males and smokers compared to females and non-smokers. [15][16]

3.2 PHARMACODYNAMICS

Olanzapine is an atypical antipsychotic characterized by its "multi-acting receptor-targeted" (MARTA) profile. It exerts its therapeutic effects and side-effect profile through potent antagonism across various neurotransmitter systems, including dopaminergic, serotonergic, histaminergic, muscarinic, and adrenergic receptors. [11][12]

DOPAMINERGIC SYSTEM

Olanzapine functions as a selective antagonist at dopamine D₂ receptors, primarily in the mesolimbic pathway. By blocking postsynaptic D₂ receptors, it effectively reduces positive symptoms of psychosis such as hallucinations and delusions. In vivo studies report a striatal D₂ occupancy of 49–63%. A key feature of olanzapine is its relatively loose binding and rapid dissociation from D₂ receptors, which, combined with the inhibition of dopaminergic neurons in the A10 region, contributes to a lower incidence of extrapyramidal side effects (EPS) compared to typical antipsychotics.[11]

SEROTONERGIC SYSTEM

The drug exhibits high affinity for serotonin 5-HT_{2A/C}, 5-HT₃ and 5-HT₆ receptors. Antagonism at the 5-HT_{2A} receptor in the prefrontal cortex is believed to enhance the release of dopamine and glutamate, thereby improving the negative and cognitive symptoms of schizophrenia. Furthermore, its potent antagonist action at 5-HT₃ and 5-HT₆ receptors helps modulate excitatory downstream signaling and may provide additional mood-stabilizing effects. [11]

HISTAMINERGIC SYSTEM

Olanzapine acts as a potent H₁ receptor antagonist. This mechanism disrupts homeostatic signaling in the hypothalamus, particularly affecting the H₁ and GHSR1A (ghrelin) receptor interface. This disruption increases AMP-activated protein kinase signaling, leading to increased appetite, reduced energy expenditure, and significant weight gain. Chronic H₁ blockade is a primary driver of the metabolic syndrome (insulin resistance and dyslipidemia) associated with olanzapine therapy. [11]

MUSCARINIC SYSTEM

Olanzapine demonstrates competitive antagonism across all five muscarinic subtypes (M1- M5), with the highest affinity for M1 (K_i 1.9 nM) and M3. While M1 antagonism helps mitigate EPS, it is also responsible for anticholinergic side effects such as dry mouth, constipation, and potential cognitive impairment or delirium. M3 receptor involvement is further implicated in glucose dysregulation. [11]

Adrenergic System

As a α 1-adrenergic receptor antagonist, olanzapine blocks the effects of norepinephrine on vascular smooth muscle. This inhibition of the IP3/DAG second-messenger pathway often results in cardiovascular side effects, including orthostatic hypotension, dizziness, and reflex tachycardia. [11]

3.3 INDICATION

Primarily indications

To treat schizophrenia and bipolar disorder in combination with other antipsychotic or antidepressants to treat depression associated bipolar disorder I. It is consider as a drug of choice by FDA due to its reduced extrapyramidal side effects or when the symptoms are inadequately controlled. The extrapyramidal side effects of olanzapine is reduced due its high affinity towards 5HT-2A and D2 receptors in the limbic system which help reduce the motor side effects in the nigrostriatal pathway.[11]

Off labelled indications

Chemotherapy induced nausea and vomiting (CINV)

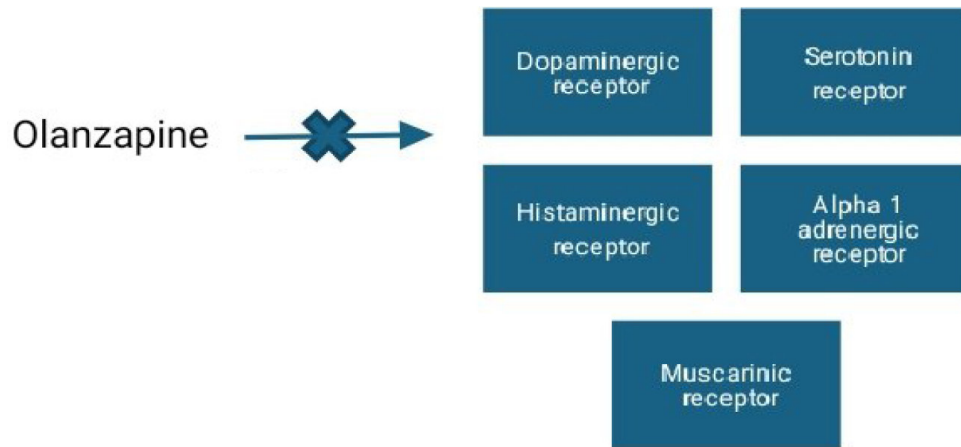
Appetite stimulant [2] [3]

3.4 MECHANISM OF ACTION IN CINV

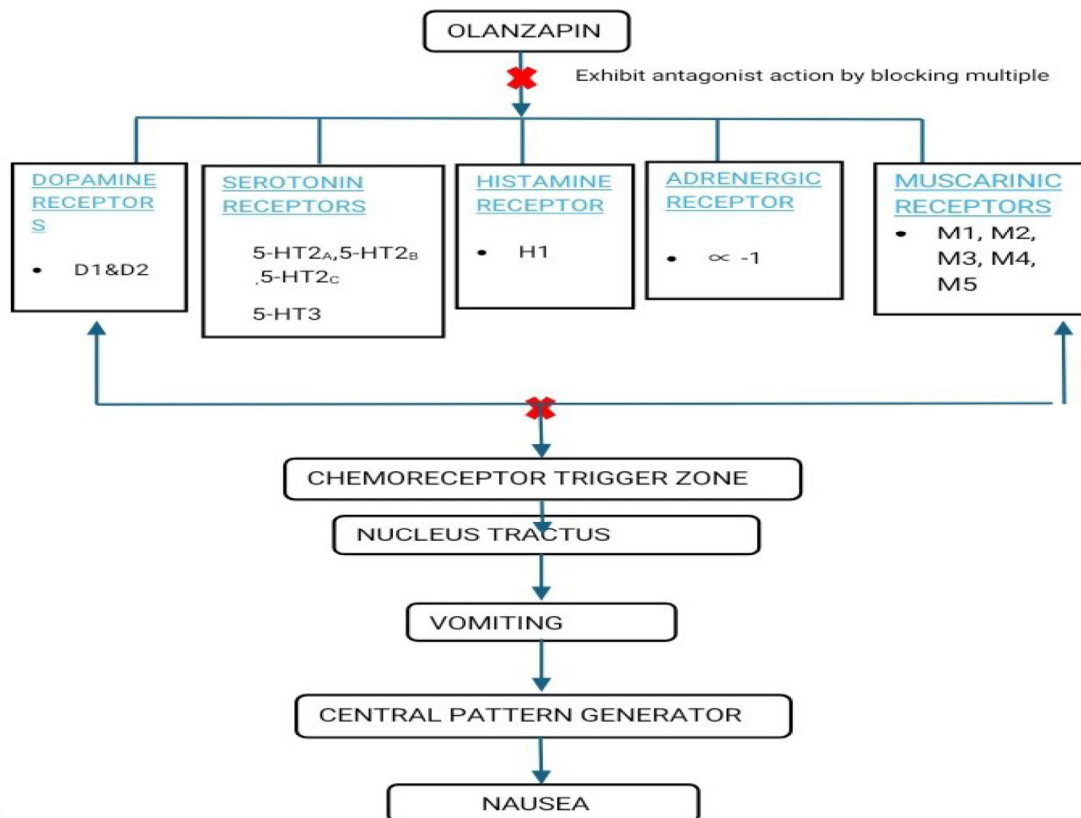
Pathophysiology of Chemotherapy-Induced Nausea and Vomiting (CINV) The physiological basis of nausea involves the conscious awareness of heightened activity within the neural centres controlling the emetic reflex. The vomiting center (VC), located in the medulla oblongata, functions as a central pattern generator (CPG), coordinating autonomic and somatic motor responses to noxious stimuli. The VC integrates emetic signals from four primary sources: the vestibular system, the cerebral cortex, the gastrointestinal tract (GIT), and the chemoreceptor trigger zone (CTZ). [23][24][25]The CTZ, situated in the area postrema on the floor of the fourth ventricle, lies outside the blood-brain barrier. This anatomical positioning allows it to detect emetogenic toxins or chemotherapy-induced mediators within the blood and cerebrospinal fluid. Furthermore, chemotherapy-induced gut dysbiosis—characterized by a reduction in beneficial microbes such as *Faecalibacterium prausnitzii* and *Bifidobacterium*—exacerbates gut inflammation. This disruption of the gut-brain axis amplifies emetic signaling, particularly through the release of serotonin (5-HT). [26][22]

Mechanism of Olanzapine

Olanzapine effectively mitigates CINV through its broad-spectrum antagonism of multiple neurotransmitter receptors involved in the emetic pathway. By targeting dopaminergic (D2), serotonergic (5-HT₃), muscarinic (M1), and histaminergic (H1) receptors within the CTZ and the VC, olanzapine interrupts the transmission of afferent emetic signals. Its efficacy in treating delayed nausea is particularly noted, likely due to its ability to modulate the persistent inflammatory and neurotransmitter imbalances triggered by chemotherapy. [2][3][11]



FLOW CHART OF OLANZAPINE MECHANISM OF ACTION IN CONTROLLING NAUSEA AND VOMITING



3.5 OLANZAPINE INTERACTIONS

Olanzapine shows variable therapeutic response due to factors such as drug–drug and drug–food interactions, pharmacogenomics, special populations, and comorbidities. [15][16]

DRUG–DRUG INTERACTIONS

Olanzapine interacts with several drug classes:

- a) **QTc-prolonging drugs:** Examples: Fluoroquinolones (ciprofloxacin, levofloxacin), macrolides (azithromycin, erythromycin), antifungals (fluconazole, ketoconazole). Mechanism: Additive QT prolongation increases risk of ventricular arrhythmias and torsades de pointes.
- b) **Anticholinergic drugs:** Examples: Amitriptyline, diphenhydramine, atropine. Mechanism: Synergistic muscarinic blockade causing constipation, urinary retention, and cognitive dysfunction.
- c) **CNS depressants:** Examples: Opioids, benzodiazepines, sedating antihistamines. Mechanism: Additive CNS depression leading to sedation and respiratory depression.
- d) **Serotonergic agents:** Examples: SSRIs, SNRIs. Mechanism: Increased risk of serotonin syndrome and neuroleptic malignant syndrome.
- e) **Antihypertensives:** Examples: ACE inhibitors, ARBs, α 1-blockers. Mechanism: Enhanced hypotensive effect causing orthostatic hypotension and falls.
- f) **Dopamine antagonists:** Example: Metoclopramide. Mechanism: Increased extrapyramidal symptoms due to additive D2 blockade. [15][16][11]

DRUG-FOOD INTERACTIONS

Broccoli: Induces CYP1A2, reducing olanzapine levels and efficacy. Alcohol: Increases CNS depression causing sedation and impaired coordination. Grapefruit juice: May increase olanzapine levels, leading to enhanced side effects. [12][15][11]

PHARMACOGENOMIC CONSIDERATIONS

Genetic variations in CYP1A2, UGT1A4/2B10, and ABCB1 influence olanzapine metabolism and plasma levels, affecting efficacy and risk of adverse effects such as metabolic syndrome.[12][15]

SPECIAL POPULATIONS

- a) **Women:** Higher plasma levels due to slower metabolism; may increase prolactin levels. Use in pregnancy is associated with risks like gestational diabetes and low birth weight.
- b) **Pediatrics:** Approved for ages 13–17; requires careful dosing due to higher risk of weight gain and sedation. [15][16][11]

COMORBIDITIES

Hepatic impairment: Requires dose adjustment as olanzapine is metabolized in the liver (CYP1A2). Hypertension & cardiac disease: Increased risk of hypotension and arrhythmias when combined with related medications. [12][15][11]

4. CHEMOTHERAPY-INDUCED NAUSEA AND VOMITING (CINV)

4.1 OVERVIEW

CINV remains a significant side effect affecting 50-60% of patients receiving high emetogenic therapy. It often leads to decreased quality of life, nutritional deficiencies, and treatment non-adherence. Management is guided by three major organizations: MASCC, ASCO, and NCCN [4-6]. [1][9]

4.2 Symptom Definitions

Nausea: An unpleasant throat/epigastric sensation.

Vomiting: Forced expulsion of gastric contents.

Retching: Laboured rhythmic respiratory movements without emesis. [23][24]

4.3 Classification of CINV

Acute: Occurs within 24 hours (peak: 5-6 hours).

Delayed: Occurs >24 hours post-treatment (common with cisplatin; lasts 5-7 days).

Anticipatory: A learned response triggered by sensory cues (smell, sight) from previous cycles; managed with benzodiazepines.

Chronic: Persistent vomiting lasting beyond 5 days.

Refractory: Emesis occurring despite optimal prophylactic treatment. [1][9][7]

4.4 Risk Factors

Patient-Related: Female sex, younger age, history of motion sickness/pregnancy emesis, low alcohol intake, and poor prior CINV control.

Treatment-Related: Dose, schedule, and specific emetogenicity of the antineoplastic agent. [1][8][4][6]

4.5 Emetogenic Potential (ASCO/NCCN Scale)

High (HEC) : >90% risk.

Moderate (MEC): 30-90% risk.

Low: 10-30% risk.

Minimal: <10% risk. [4][6]

4.6 Standard Treatment Protocols

HEC: Triple therapy (5-HT₃ receptor antagonist + NK₁ receptor antagonist + Dexamethasone) [4-6].

MEC: Double therapy (5-HT₃ antagonist + Dexamethasone) [4-6].

Low/Minimal: Single agent (typically a corticosteroid) or no routine prophylaxis [4-6].

4.7 Pathophysiology

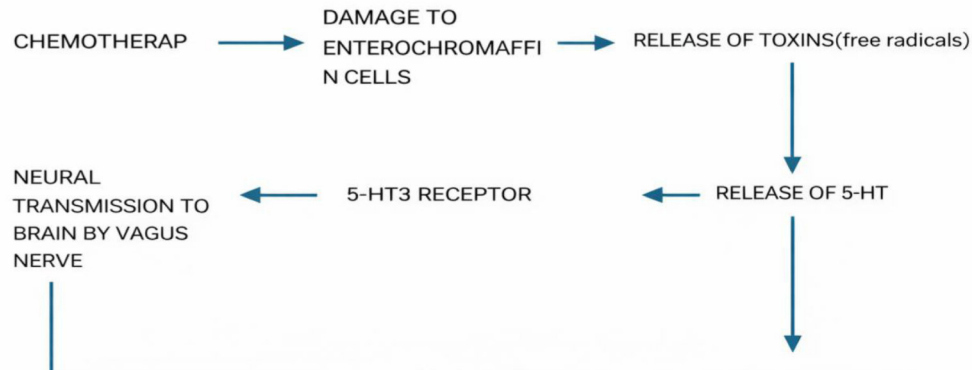
CINV involves two primary neural pathways:

Peripheral Pathway (GI Tract): Chemotherapy damages enterochromaffin cells in the GI mucosa, releasing serotonin and Substance P. These signals travel via the vagal nerve to the CNS.

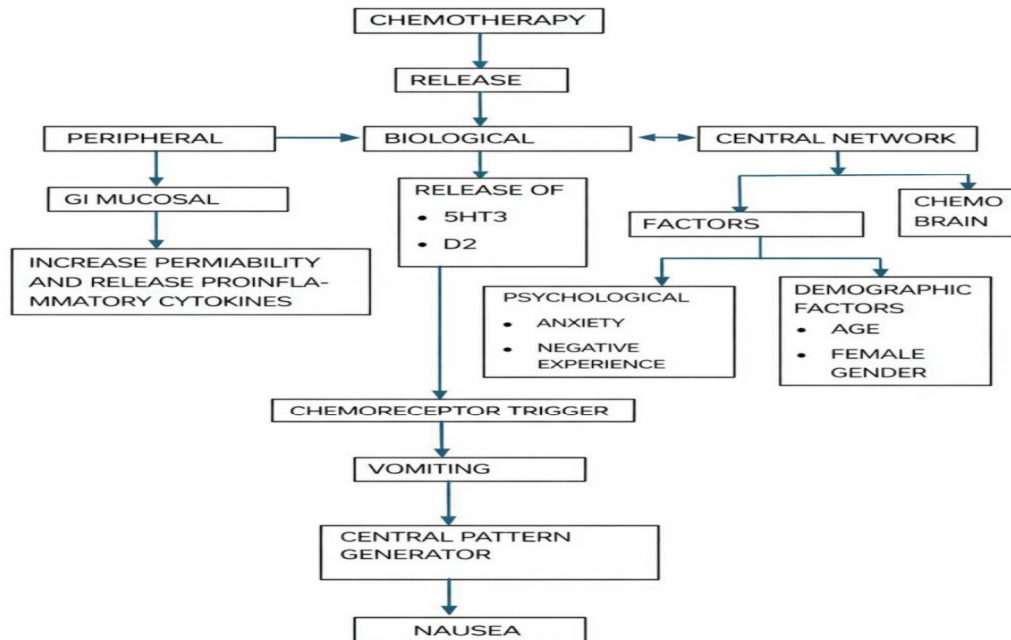
Central Pathway (Brain): Chemoreceptor Trigger Zone (CTZ): Detects blood-borne toxins via 5-HT₃, NK₁, and D₂ receptors. Vomiting Centre (VC): Coordinates the motor

Act of emesis. Limbic System: Mediates the psychological and anticipatory aspects of nausea. [24][25][1][26][23]

MECHANISM FLOWCHART OF CHEMOTHERAPY INDUCED NAUSEA AND VOMITING IN GUT



PHYSIOLOGY INVOLVED IN CHEMOTHERAPY INDUCED NAUSEA AND VOMITING



5. MATERIALS AND METHODS

5.1 STUDY DESIGN

A prospective observational clinical study was conducted to assess the effectiveness and safety of an olanzapine-based antiemetic regimen in preventing chemotherapy-induced nausea and vomiting (CINV).

5.2 SOURCE OF DATA AND MATERIALS

- Patient consent forms
- Data collection forms and case reports
- Chemotherapy treatment charts
- Laboratory reports (CBC, LFT, RFT, serum electrolytes)
- Medication administration records

5.3 INCLUSION CRITERIA

- Patients aged 18–75 years with solid or hematological malignancies
- Receiving moderately or highly emetogenic chemotherapy (MEC/HEC)
- Receiving olanzapine (alone or with 5-HT₃ antagonists and dexamethasone)
- ECOG ≤ 2
- Adequate hepatic and renal function
- BMI ≥ 18 kg/m²
- Acceptable baseline blood glucose levels
- At least one chemotherapy cycle completed

Assessments:

- Nausea: Visual Analogue Scale (VAS)
- Vomiting: CTCAE grading
- Sedation levels and blood glucose monitoring

5.4 EXCLUSION CRITERIA

- Age < 18 years
- Hypersensitivity to olanzapine or antipsychotics
- Severe comorbid conditions (cardiac, hepatic, renal failure)
- Pregnant or lactating women
- History of tardive dyskinesia or neuroleptic malignant syndrome
- Uncontrolled diabetes (HbA1c > 9%)
- Concurrent investigational antiemetic therapy

5.5 METHOD OF DATA AND COLLECTION

Data were collected from:

- Patient interviews and medical records
- Demographics, diagnosis, and TNM staging
- Chemotherapy and antiemetic regimen details
- Comorbidities and concurrent medications
- Clinical observations (nausea, vomiting, sedation)
- CINV phases: acute (0–24 h), delayed (24–120 h), overall

5.6 Data Collection Procedure

- Sociodemographic and clinical data collected during oncology visits
- BMI calculated using height and weight
- Vital signs recorded at baseline
- Nausea and vomiting assessed at each chemotherapy cycle
- Complete response evaluated for acute, delayed, and overall phases
- Sedation assessed using Epworth Sleepiness Scale
- Adverse effects (somnolence, weight gain, hyperglycemia, EPS, QTc changes) were monitored

STUDY SAMPLE SIZE

Cancer patients on chemotherapy: 50 patients

DURATION OF THE STUDY

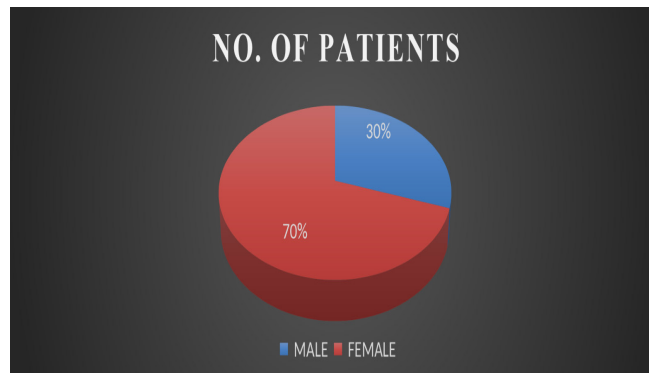
Study was conducted for 6 months

PLACE OF THE STUDY

CARE group of hospitals, hi-tech city, Hyderabad.

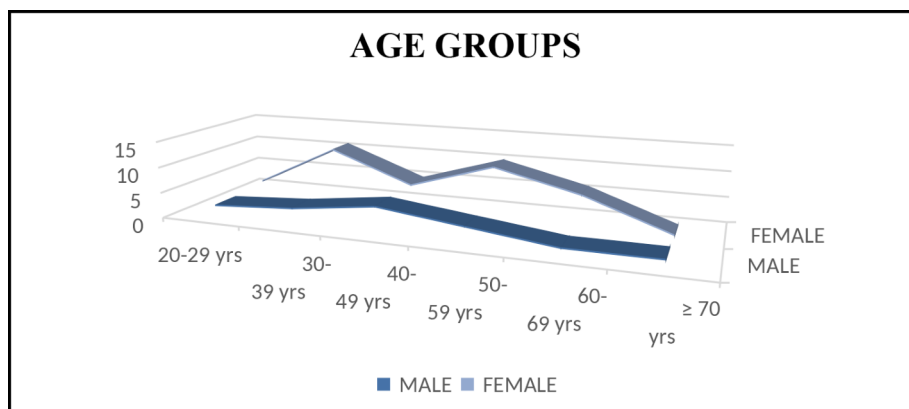
RESULTS

TABLE 1:- Dissemination of Patient Based of Patient Based on Sex



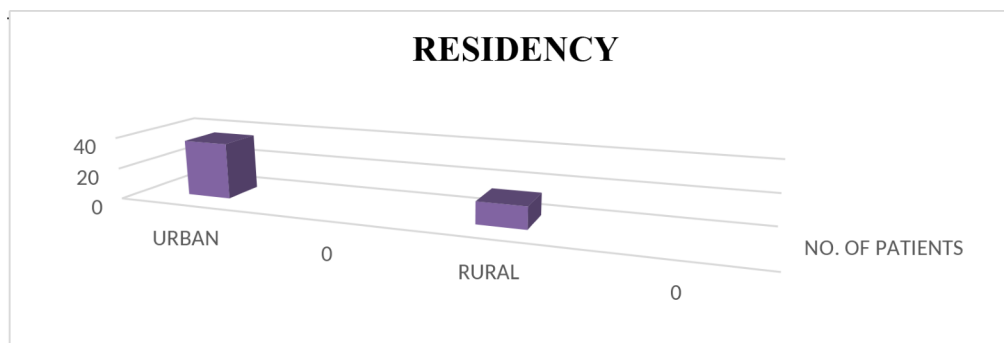
Out of a sample of 50 patients, 15 (30%) were male and 35 (70%) were female with diagnosis of breast cancer. Therefore, this study indicates a higher proportion of female patients.

TABLE 2:- DISSEMINATION OF PATIENTS BASED ON THE AGE



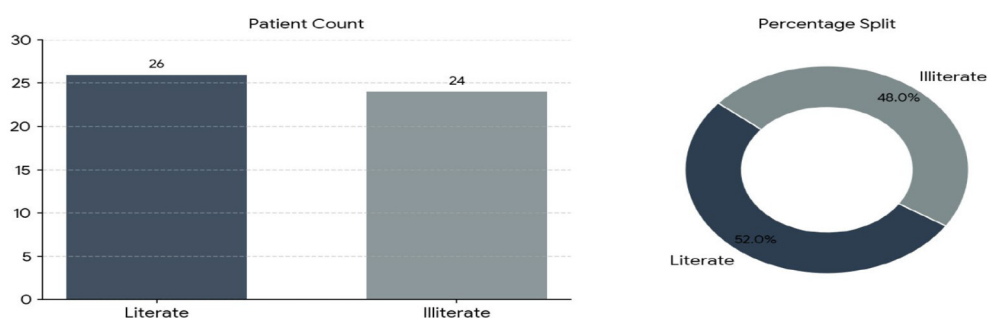
The study's findings indicate that the highest proportion of the sample (33%) falls within the 30-39 age range.

TABLE 3 DISSEMINATION OF PATIENTS BASED ON THE RESIDENCY



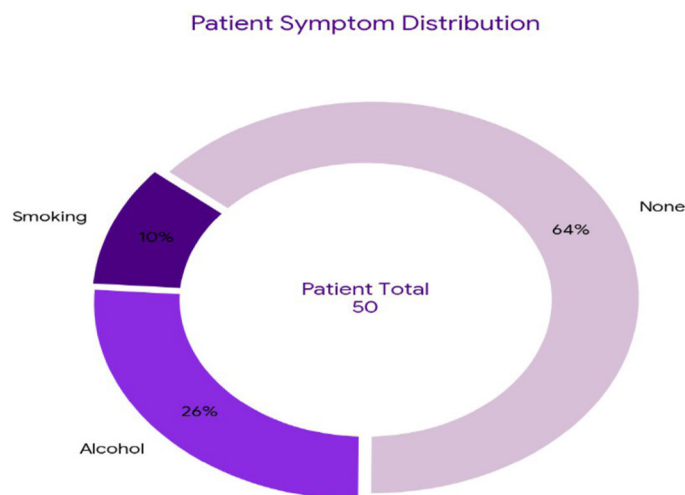
The criteria included patient habitation, comparing urban and rural areas. The data indicates a higher percentage of patients from urban areas.

TABLE 4:- DISSEMINATION OF THE PATIENTS BASED ON THE EDUCATION



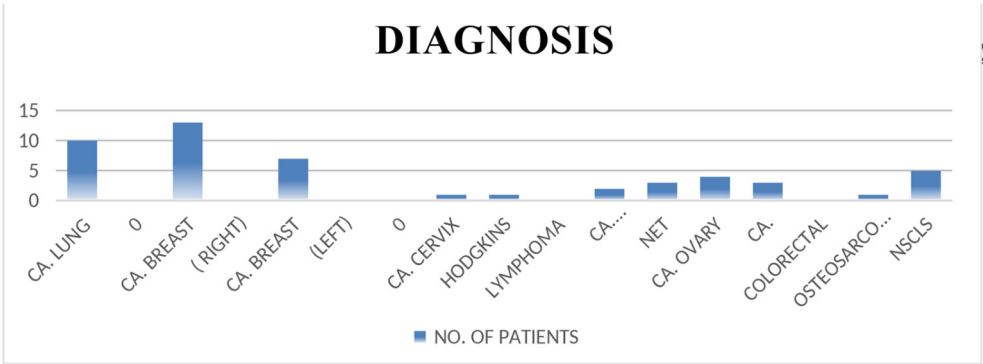
The chart shows a fairly even distribution, with literate patients slightly outnumbering illiterate patients by two individuals.

TABLE 5:- DISSEMINATION OF PATIENTS BASED ON LIFESTYLE FACTORS



The graph present the social history of the patient. A total of 4 patients reported, smoking 5 had a history of alcohol consumption, and 54 patients reported no relevant social history.

TABLE 6:- DISSEMINATION OF PATIENTS BASED ON THE DIAGNOSTIC



The research was conducted based on patient's diagnosis, which included Ca. Lung, Ca. Breast, Ca. Cervix, Hodgkin's Lymphoma, Ca. Endometrium, NET, Ca. Ovary, Ca. Colorectal, Osteosarcoma and NSCLS. The highest percentage of patients -26%- were diagnosed Ca. Breast (Right), while the lowest percentages, 2% were observed in subjects with Ca. Cervix, Hodgkin's Lymphoma and Osteosarcoma.

TABLE 7:- DISSEMINATION OF PATIENTS BASED ON THE RISK FACTORS

Observational studies were conducted on patients with risk factors. Hypertension were observed in 13 patients, Diabetes mellitus in 12 patients, Hypothyroidism in 7 patients and 27 patients is with no comorbidities.

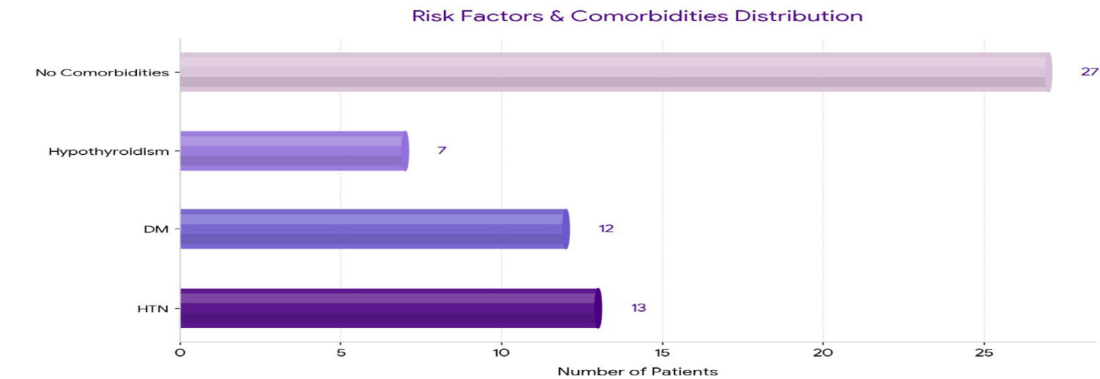


TABLE 8:- DISSEMINATION OF PATIENTS BASED ON TREATMENT REGIMEN

Among the 50 cases collected patients are highly encouraged with HEC regimen when compared to MEC as the rate of complete response shown more with HEC therapy.

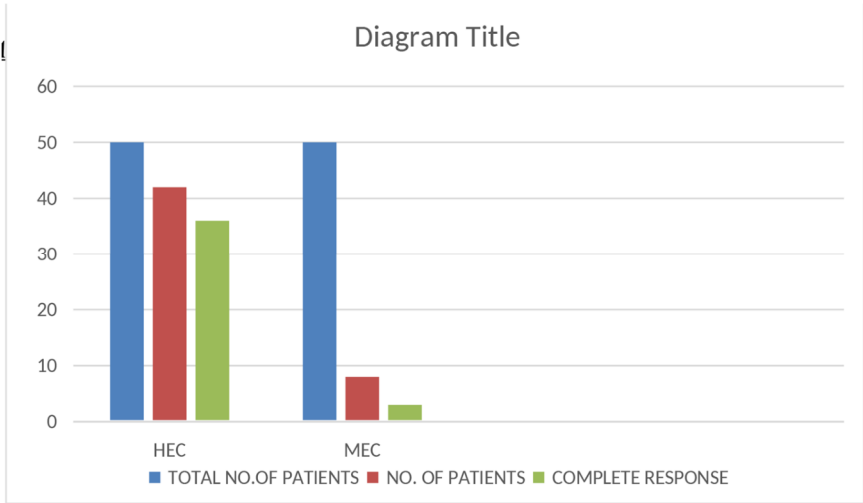
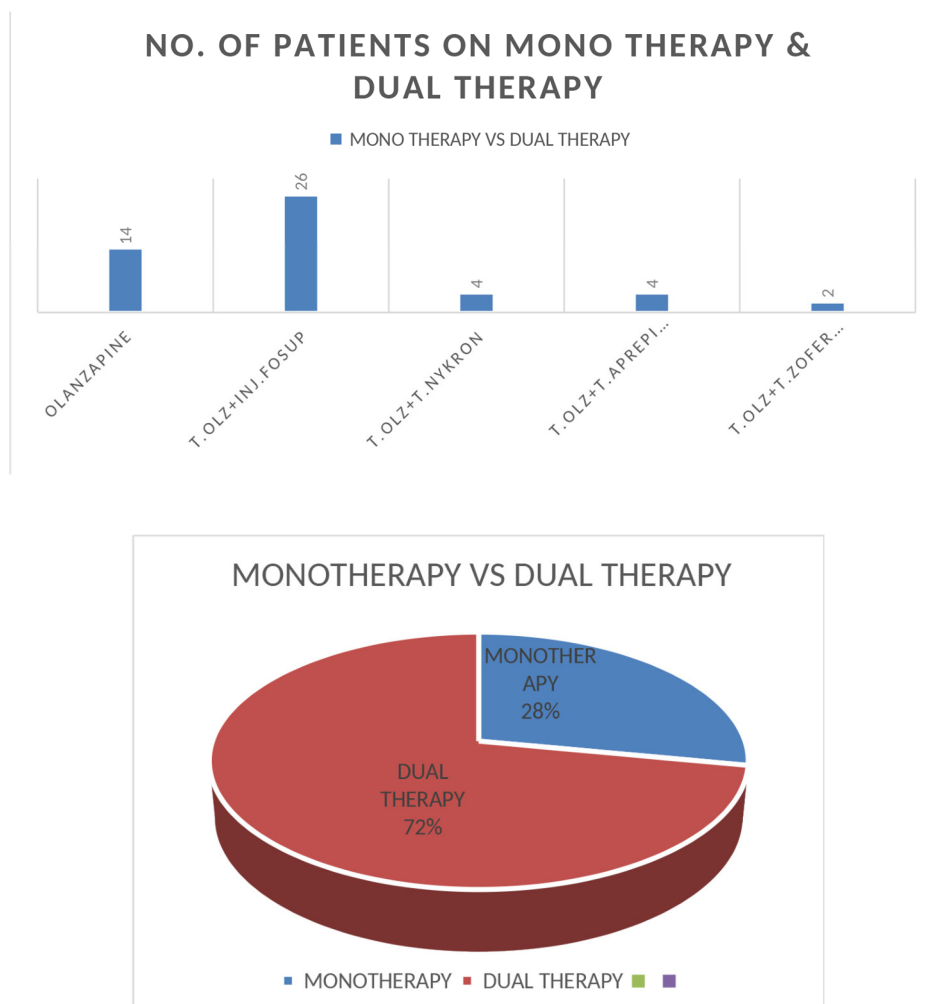


TABLE 9: DISSEMINATION OF PATIENTS BASED MONOTHERAPY VS COMBINATION THERAPY

Among the 50 patients as the results showed that the number of patients on combination therapy were around 36 and showed a good response rate in controlled CINV when compared to the other 14 who were on monotherapy to treat CINV in the early cycles majorly.



DISCUSSION

- Chemotherapy-induced nausea and vomiting (CINV) remains a common and distressing complication that negatively affects patient comfort, treatment adherence, and quality of life.
- The study aimed to evaluate the efficacy and safety of an olanzapine-based antiemetic regimen in chemotherapy patients.
- A total of 50 patients were included, with a higher proportion of females (70%) compared to males (30%), likely due to increased breast and gynecological cancer cases.
- Most patients were in the 30–39 years age group, indicating a predominance of middle-aged individuals undergoing chemotherapy.
- Body weight distribution followed expected patterns, with females mainly between 50–70 kg and males between 70–80 kg.
- A greater number of patients were from urban areas than rural areas, suggesting better access to oncology services in urban regions.
- Educational status was nearly equal among patients, showing that CINV affects individuals irrespective of literacy levels.

- The majority of patients had no history of smoking or alcohol use, though some reported these habits, which may influence health status.
- Breast cancer was the most common malignancy, followed by lung cancer and non-small cell lung cancer, with other cancers occurring less frequently.
- Hypertension and diabetes mellitus were the most common comorbidities, although many patients had no associated conditions.
- Olanzapine demonstrated high effectiveness, with most patients achieving complete response (no vomiting) and very few experiencing severe symptoms.
- Symptom control was better in the acute phase (0–24 hours) than in the delayed phase (24–120 hours), though overall outcomes remained satisfactory.
- Many patients required rescue antiemetics, indicating the need for additional support in some cases, especially with highly emetogenic chemotherapy.
- The regimen was well tolerated, with minimal side effects; somnolence was the most common, followed by mild dizziness and dry mouth.
- Common chemotherapy drugs used included paclitaxel, carboplatin, and cyclophosphamide, which have high emetogenic potential.
- Overall, olanzapine was found to be effective and safe in reducing the severity and frequency of CINV, improving patient outcomes.

CONCLUSION

The present prospective clinical study assessed the efficacy and safety of an olanzapine-based antiemetic regimen in patients experiencing chemotherapy-induced nausea and vomiting.

The findings demonstrate that olanzapine is effective in preventing and controlling CINV, with a large proportion of patients experiencing complete relief from vomiting and only a small percentage reporting moderate or severe symptoms. The drug showed effectiveness in both the acute and delayed phases of CINV.

In addition, the treatment was well tolerated by most patients, with only a small number experiencing mild adverse effects such as somnolence, dizziness, and dry mouth.

Based on these observations, it can be concluded that olanzapine is a safe and effective antiemetic agent that can significantly improve the management of chemotherapy-induced nausea and vomiting in cancer patients.

Further research involving larger study populations and extended monitoring periods is recommended to validate these findings and to explore the long-term benefits of olanzapine in different chemotherapy regimens.

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