



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 13 Issue: X Month of publication: October 2025

DOI: https://doi.org/10.22214/ijraset.2025.74840

www.ijraset.com

Call: © 08813907089 E-mail ID: ijraset@gmail.com

ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538

Volume 13 Issue X Oct 2025- Available at www.ijraset.com

Introduction to Adverse Effects of Chemotherapy Agent

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Abstract: Chemotherapy remains a cornerstone in modern cancer management, yet its administration is frequently accompanied by diverse side effects that can undermine both patient wellbeing and adherence to prescribed regimens. This review synthesizes evidence on the primary and most impactful adverse events linkedto chemotherapy agents, exploring the biological mechanisms behind them and outlining contemporary managementapproaches.

Athoroughanalysis of published clinical studies under scores the importance of tailoring supportive care strategies to each individual in order to limit toxicity and improve overall outcomes.

I. INTRODUCTION

Cancer represents a group of disorders defined by unregulated cellular proliferation within bodily tissues or organs. Treatment strategies depend on variables including cancer site, stage, and cellular characteristics. Notably, cancer holdsthedistinction ofbeingtheworld's second most common cause of death.

Alongsidesurgeryandradiotherapy,chemotherapyisapivotaltherapeuticavenue. Advances in pharmaceutical research are continually generating novel anticancer drugs, yet these agents impact both psychological and physical health, manifesting as pain, insomnia gastrointestinal distress, emotional disturbances, and more.

Increasingawarenessofcancerasachronichealthconditionhasledtorecognitionofself-managementand individualized care as essential elements in long-term treatment planning.

A. Types of cancers:

Overonehundreddistinctcancersaffecthumans, arising from various tissue origins. They are commonly categorized as follows:

- 1) Carcinomas:Originatefromepithelialtissues,accountingformostbreast,lung,prostate,andcoloncancers. Sarcomas: Develop from connective tissues such as bone, muscle, or fat.
- 2) Leukemias: Arise in hematopoietic (blood-forming) tissues, leading to abnormal proliferation of blood cells.
- 3) LymphomasandMyelomas:Originatefromcellsoftheimmunesystem, affectinglymphnodesandplasmacells.
- 4) CentralNervousSystemTumours:Includemalignanciesofthebrainandspinalcordsuchasgliomasand medulloblastomas.
- 5) GermCellTumors:Emergefromreproductivecells,oftenaffectingtheovariesortestes.
- 6) Blastoma: Derivedfromimmaturecellsorembryonictissue, more common in pediatric populations (e.g., neuro blastoma, retino blastoma). Frequently occurring cancerty pesbyorganor systemin clude breast, lung, colorectal, bladder, and skin cancers, as well as various leukemias and lymphomas.

B. Classification of Chemotherapy Agent

Chemotherapydrugsarecentraltosystemiccancertherapyand are classified by their molecular structure and activity in targeting malignant cells. Major classes include

- 1) Alkylating Agents: These compounds damage DNAby introducing alkyl groups, causing breaks in DNA strands and ultimately inhibiting cell replication. Common agents include cyclophosphamide and if osfamide.
- 2) Mechanism:AlkylatingagentscreatehighlyreactivecarboniumionsthatbindcovalentlytoDNA,prompting cross-linking, base mispairing, and strand breaks, which together halt DNAsynthesis and cell division.
- 3) Antimetabolites: These drugsmimic natural molecules necessary for DNA and RNA synthesis, thereby interrupting genetic material formation in rapidly proliferating cells. Examples include methotrexate and fluor our acil.
- 4) Anti-TumorAntibiotics:Distinctfromantibacterial agents, this group interferes with DNA replication by directly binding to DNA; doxorubic in and bleomyc in are key representatives.
- 5) TopoisomeraseInhibitors:ThesedisruptenzymesrequiredforDNAstrandunwinding,preventingcelldivision. Drugs like etoposide and irinotecan are in this category.



ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538 Volume 13 Issue X Oct 2025- Available at www.ijraset.com

- 6) MitoticInhibitors(PlantDerivatives):Derivedfromnaturalsources, these agents (e.g., vincristine, paclitaxel) interfere with microtubule function, blocking cell division.
- 7) Corticosteroids:Sometimesincludedinchemotherapyregimenstominimizeinflammationandimmune responses, corticosteroids also help manage treatment side effects.

Each classutilizes a unique mechanism to selectively target neoplastic cells but can also impact normal rapidly dividing cells, accounting for the broad spectrum of side effects encountered during chemotherapy.

II. CHEMOTHERAPY AGENT ADVERSE EFFECTS WITH ITS MECHANISMOFACTION

A. Alkylatingagent: Adverse Effects and Mechanistic Basis

Class Effects- Bone marrow suppression (most common and dose-limiting) Gastrointestinal distress (nausea, vomiting, mucositis) Alopecia Secondary malignancies (notably AML, due to mutagenic effects) Organ-specific toxicities depending on the drug: Cyclophosphamide \rightarrow bladder, Busulfan \rightarrow lungs, skin, Nitrosoureas \rightarrow CNS Dacarbazine \rightarrow liver and marrow

Quick Mnemonic for Distinct Toxicities - Cyclophosphamide→Cystitis(fromacrolein) Busulfan → Breathless (lung fibrosis) Nitrosoureas→ Neural toxicity

Procarbazine→Peculiarreactions(disulfiram-like)

Drug	MajorAdverseEffects&Rationale	Mechanismof Toxicity		
Carmustine/Lomustine (Nitrosoureas)	Myelosuppression-Hemorrhagic cystitis(acroleinbyproduct)-SIADH (rare)-Secondarymalignancies (e.g., leukemia,bladdercancer)	FormDNA crosslinks, especiallyin rapidlydividing cells;toxic metabolites		
		(acrolein) cause additionaltissue irritation.		
Busulfan	, ,	Causes alkylation and cross-linking of DNA, impairing replication in hematopoieticand epithelial tissues.		
Carmustine/Lomustine (Nitrosoureas)		Cross the blood– brain barrier and formDNA– protein crosslinksinneural andtumortissues.		
Dacarbazine/ Procarbazine	Myelosuppression-Nausea and vomiting- Leukemogenicpotential- Disulfiram-like reaction and MAO inhibition (esp. procarbazine)	Methylatingagents that damage DNA through free radicals and alkylation.		
Melphalan/Chlorambucil	Myelosuppression-Secondary leukemia (long-term use)-GI irritation	Classic alkylators binding DNA guanineresidues→ impaired replication.		



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B. Antimetabolites-AdverseEffects

Drug	MajorAdverseEffects&Rationale	MechanismofToxicity				
Methotrexate(MTX)(Folate	Bonemarrowdepression(↓WBCs, RBCs,	Blocks dihydrofolate				
antagonist)	platelets)-Mouth ulcers (mucositis)-	reductase, suppressing DNA				
	Liverinjury(†LFTs, long-term fibrosis risk)-	synthesis in				
	Rarely, pulmonary fibrosis rapidlydividingtissu					
	bone marrow, mucosa					
		and hepatic cells.				
5-Fluorouracil(5-FU) (Pyrimidine	Myelosuppression-Hand–foot	Converted to FdUMP,				
analog)	syndrome(pain,redness,peeling on	inhibiting thymidylate				
	palms/soles)-Diarrhea, mucositis-	synthase→faultyDNA				
	Photosensitivity	formation \rightarrow destruction of				
		tissues				
		withhighcell turnover.				
Cytarabine (Ara-C)	Severe pancytopenia-GI irritation (nausea,	IncorporatedintoDNA; blocks				
(Pyrimidineanalog)	vomiting, diarrhea)-Neurotoxicity(ataxia,	DNApolymeraseanddisrupts				
	slurred speech, esp. high doses/elderly)-	synthesis in proliferating cells.				
	Conjunctivitis	_				

Gemcitabine(Pyrimidine analog)	Bonemarrowsuppression-Flu- like syndrome-	Acts a	is a	chain
	Rare interstitial lung injury	terminatord	ſΑ	
		replication,	damaging	
		proliferating	g mar	row and
		pulmonary	cells.	
6-Mercaptopurine(6-MP) (Purine	Myelosuppression-Hepatic damage	Converted	by	HGPRT;
analog)		interferes w	ith purin	e synthesis
		\rightarrow	toxic	to
		hematopoie	ticandliv	er
		cells.		

C. Anti-tumoragent-Adverseeffects:

Drug	MajorAdverseEffects&Rationale	MechanismofToxicity
VincaAlkaloids(Vincristine, Vinblastine)	Neurotoxicity (peripheral neuropathy, more common with vincristine)-Myelosuppression (vinblastine)-Constipation (autonomicneuropathy)	Disrupt microtubule formation,impairingmitosis.
Taxanes(Paclitaxel, Docetaxel)	Neutropenia-Peripheral neuropathy- Hypersensitivityreactions (particularly paclitaxel)	



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Anthracyclines(Doxorubicin,	Cardiotoxic	ity (do	ose-	dependent	Intercalate	into	Г	NA,
Daunorubicin)	congestive	hea	art	failure)-	generatefreeradi	calscausing	oxid	ative
	Myelosuppı	ression			damage.			
	-Alopecia-N	Aucositis						
Bleomycin	Pulmonary	fibrosis	(main	concern)-	InducesDNAstra	andbreaks	via	free
	Skin	hy	perpig	mentation-	radical formation	n.		
	Minimalmy	elosuppre	ession					

D. TopoisomeraseInhibitors-AdverseEffects

Drug/Class	MajorAdverseEffects& Rationale MechanismofToxicity
Etoposide / Teniposide	Myelosuppression (especially leukopenia)-Block topoisomerase II, preventingre-
(TopoisomeraseII inhibitors)	Alopecia-Mucositis and GI irritation-Secondary ligation of DNA strands → accumulation
	acute leukemia (typically after prolonged use) of double-strand breaks → cell death in
	dividing cells.
Topotecan/ Irinotecan	Severe diarrhea (early form due to InhibittopoisomeraseI,resulting insingle-
(TopoisomeraseI	cholinergiceffects;lateformdueto strandDNAbreaksthat
	mucosaldamage)-Neutropenia impairreplicationand
inhibitors)	(dose-limiting)-Nausea and vomiting-transcription.
	Fatigueandweakness

$E. \quad \textit{MitoticInhibitors} (\textit{Plant-DerivedAgents}) \!\!-\!\! \textit{AdverseEffects}$

Drug	MajorAdverseeffects&rationale	MechanismofToxicity						
VincaAlkaloids(Vincristine,	Vincristine – Peripheral neuropathy	Bind to β-tubulin and prevent						
Vinblastine, Vinorelbine)	(sensory + motor),	microtubule						
	constipation, jawpain, SIADH; minimal	polymerization→haltcell division at						
	marrow toxicity Vinblastine / Vinorelbine	metaphase.						
	– Myelosuppression (dose-							
	limiting),mucositis,mild neurotoxicity.							
Taxanes (Paclitaxel,	Myelosuppression (mainlimiting factor)-	Stabilizemicrotubules, preventing						
Docetaxel, Cabazitaxel)	Peripheral neuropathy-Hypersensitivity	their depolymerization \rightarrow mitotic						
	reactions (from solvent; reduced	arrest in metaphase.						
	withnewerformulations)-Nail and hair	•						
	changes (alopecia, nail discoloration)-Fluid							
	retention,							
	especiallywith docetaxel							
Podophyllotoxins(Etoposide,	Myelosuppression-Mucositis	Inhibit topoisomerase II and						
Teniposide)	-Secondary leukemia (rare,	interfere with microtubule dynamics						
	relatedtoDNAdamage)-Hair loss	(technicallyoverlapswith						
		topoisomeraseinhibitors).						

III. EFFECTS METHODS OF IDENTIFICATION OF ADVERSEEFFECTS

1) ClinicalTrials and Pharmacovigilance: During clinicaltrials, adverse effects are meticulouslyrecorded and classified using scales like the Common Terminology Criteria forAdverse Events (CTCAE) by the National Cancer Institute. Post-marketing surveillance identifies rare and long-term effects not observed intrials.



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- 2) Patient-ReportedOutcomes(PROs):ToolslikethePatient-ReportedOutcomesMeasurementInformation System (PROMIS) enable patients to self-report symptoms, offering real-world insights.
- 3) BiomarkersandImaging:Biochemicalmarkers(e.g.,liverenzymesforhepatotoxicity)andimaging Techniques (e.g., echocardiography for cardiotoxicity) help identify subclinical damage.
- 4) HealthcareProviderMonitoring:Regularassessmentofhematological,hepatic,renal,andcardiac functions provides objective data on drug-indused organ damage.

IV. FORMULATION STRATEGIES TO REDUCE ADVERSE EFFECTS OF CHEMO THERAPY DRUGS

Drug	Formulation method	Formulation type	Typeofcancer	Reducedadverse effects	Biomarker	Outcome
Doxorubicin	Surface PEGylation and lipid vesicle entrapment	PEG- Liposomal (Doxil)	Ovarian, Kaposi's sarcoma, Multiple myeloma	Loweredcardiac damage and bone marrow suppression	Folate receptor expression	Extended circulation andtargeted drug accumulation
Daunorubicin	Lipid bilayer encapsulation	Liposomal (DaunoXome)	Kaposi's sarcoma	Reduced systemic cytotoxicity	Enhanced permeability andretention (EPR)	Improved tolerability and drug retention
Irinotecan	PEG-lipid vesicle composition	PEG- Liposomal (Onivyde)	Pancreatic carcinoma	Less gastrointestinal irritation	TumorEPR mechanism	Prolonged drug availability and improved overall survival
Paclitaxel	Albumin-based nanoparticle conjugation	Albumin- bound particles (Abraxane)	Breast,Lung, Pancreatic	Avoidance of solvent- induced allergies	SPARC- binding protein	Enhanced absorption and reduced hypersensitivity
Cytarabine	Controlled- releaselipid suspension	Liposomal (DepoCyt)	Lymphomatous meningitis	Decreased neurotoxic events	CSF-residing malignant cells	Sustained CSF concentration with f ewer hospital visits
Vincristine		Liposomal (Marqibo)	Acute lymphoblastic leukemia	Diminished neurotoxicity	CD44 ligand- mediated targeting	Increased stabilityand therapeutic activity
Docetaxel	Amphiphilic polymer micellization	PEG-PDLLA micelle system	Lungandbreast carcinomas	Lower hypersensitivity, reducedsystemic damage	Tubulin affinityand HSP90 linkage	Slow release andimproved intracellular accumulation
Cisplatin	PEG-stabilized lipid vesicle	PEG- Liposomal (Lipoplatin)	Lung,Breast, Gastric	Reducedrenal and auditory toxicity	Transferrin receptor targeting	Enhanced tumor selectivity



ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538 Volume 13 Issue X Oct 2025- Available at www.ijraset.com

					andsafety n	nargin
Doxorubicin (Folate- targeted)	Ligand-based nanocarrier	Folate-linked liposomalNP		Decreasednon- specific distribution	Greater selectivity attenuated target reactivity	tumor and off-
Paclitaxel (HA-targeted)	Hyaluronic acid- modified nanoparticle	HA- Liposomal system	Breast, Colorectal	. *	Improved therapeutic and outcome	index patient

Summary of strategies – Liposomal drug carriers modify biodistribution and exploit the enhanced permeability andretention (EPR)effect, therebylowering systemic toxicity; notable examplesincludeDoxiland DaunoXome. Polymeric micelles enhance the stability of poorly soluble drugs such as docetaxel and enable controlled release, contributing to reduced adverse effects. Albumin-bound nanoparticle systems, likeAbraxane, improve drug

Solubilityandreducehypersensitivitylinkedtoorganicsolvents. Activetargetingnanocarriersutilizeligandssuch as folate, transferrin, or hyaluronic acid to enhance tumor-specific accumulation while minimizing off-target interactions. Biomimetic nanostructures, incorporating cancer cell membranes, promote immune evasion and improved biocompatibility. Altogether, these innovative formulations highlight the importance of nanocarrier design, surface engineering, and targeted delivery strategies in lowering chemotherapy-induced toxicity without compromising therapeutic efficiency.

V. MANAGEMENT AND MITIGATION STRATEGIES

- 1) PreventiveMeasures: Antiemetics (e.g., Ondansetron). Pre-treatment hydration for nephrotoxicity.
- SupportiveCare:Growthfactors(e.g.,Filgrastimforneutropenia).
 3.Lifestyle Adjustments: Diet, exercise, and psychological support.

VI. FUTURE PROSPECTIVE

- 1) AdvancesinIdentification:NoveldrugdesignslikeDRP-104,whichareactivatedspecificallyintumor tissues but not in normal tissues, aim to reduce side effects by targeting delivery and activation mechanisms, thus minimizing damage to healthy cells .Scoring systems such as the Cardiotoxicity Score (CardTox-Score) have been developed to predict chemotherapy-induced myocardial toxicity using clinical, biochemical, and echocardiographic data, enabling proactive risk assessment before therapy .Longitudinal approaches like the Multiple Overall Toxicity(MOTox)scoreevaluatetoxicitycycle-by-cycleduringchemotherapy, which can inform timely interventions to manage cumulative adverse effects .
- 2) Research and Monitoring: Hospital-based pharmacovigilance and monitoring of adverse drug reactions (ADRs)byclinicalpharmacistsimproveearlydetectionandreporting, which reduces health and economic burdens associated with chemotherapy toxicity. There is ongoing emphasis on understanding and controlling specific side effects such as delayed nausea and vomiting by studying molecular pathways involved, leading to better antiemetic therapies.
- predictive Future Trends :Integration of models using patient-specific data tailor chemotherapy regimensminimizingadverseeffectsisanemergingtrend, asseen with models predicting haematological toxicity in lung cancer. Advancesinunderstandingsystemicbystandereffectscausedbychemotherapy-inducedDNAdamageand inflammation may lead to new preventive measures. Enhanced patient self-reporting and real-world data collection on adverse effects will contribute to refined risk models and personalized management strategies. Overall, the future holds promise in combining precision medicine, predictive analytics, novel drug formulations, and comprehensive monitoring platforms to better identify and mitigate chemotherapy adverse effects, improving patient outcomesandqualityof life.



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VII.APPLICATION

- 1) Clinical Applications: Hospital and Infusion Center SettingsModern chemotherapy formulations like Doxil(PEG-liposomaldoxorubicin)andAbraxane(albumin-boundpaclitaxel)areadministeredinclinical oncology units where patients receive targeted infusions with reduced monitoring requirements due to their improved safety profiles.
- Personalized Medicine Implementation: These formulations enable biomarker-driven treatment selection, whereon cologists use molecular testing to identify patients with folatereceptor over expression,
 SPARC protein levels, or CD44 expression to optimize drugselection and improve the rapeutic outcomes.
- Combination Therapy Protocols: The reformulated drugs facilitate multi-agent chemotherapy regimens with improved tolerability. For example, GEM/DOX liposome combinations have demonstrated superior survivaloutcomescomparedtosingleagenttreatments, extending median survival by 3.4 times compared to controls.
- OutpatientTreatmentExpansion:Enhancedsafetyprofilesallowforambulatorycaredelivery,reducing hospitalization requirements and enabling patients to receive treatment in outpatient settings with fewer side effect-related complications Pediatric and Geriatric Oncology:Reduced toxicity formulations are particularly valuable in vulnerable populations where traditional chemotherapy doses must be limited.
 - Liposomalvincristine(Marqibo)exemplifiesthisapplicationinpediatricacutelymphoblasticleukemia.
- 2) Therapeutic Applications
- Tumor-Specific Targeting: Advanced formulations exploit the enhanced permeability and retention (EPR) effectins olid tumor, allowing preferential drug accumulation in cancer ous tissues while sparing healthy organs.
- ControlledDrugRelease:Sustained-releaseformulationsmaintaintherapeuticdrugconcentrationsover extended periods, reducing dosing frequency and improving patient compliance while minimizing peak-related toxicity.
- Overcoming Drug Resistance: Nanoparticle systems can bypass multidrug resistance mechanisms by altering cellular uptake pathways and drug efflux patterns, potentially restoring sensitivity to previously ineffective agents. Microenvironment-Responsive Delivery: Intelligent delivery systems respond to tumor-specific conditions such as acidic pH or elevated lactate levels (up to 40-fold higher than normal tissue), enablingprecisedrugreleaseatthetargetsite.
 ImmunogenicCellDeathEnhancement: Certain formulations promote immunogenic cell death, recruiting immune cells to tumor sites and potentially enhancing the body's natural anti-cancer response beyond direct cytotoxic effects.

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