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Review Article

Cancer: A Comprehensive Review of Classification, Etiology and Evolving Therapeutic Strategies with Special Emphasis on Chemotherapy

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Cancer is a complex and heterogeneous group of diseases marked by the uncontrolled growth and dissemination of abnormal cells, contributing significantly to global morbidity and mortality. Its origins are multifactorial, involving both environmental influences such as tobacco exposure, radiation, and infectious agents and intrinsic factors like genetic alterations, hormonal disruptions, and immune system irregularities. Based on the tissue of origin, cancers are generally categorized into carcinomas, sarcomas, leukaemia, and lymphomas, each with unique biological characteristics and prognostic outcomes. Treatment approaches vary depending on the cancer type, stage, and molecular profile. Traditional methods include surgical intervention, radiation therapy, and chemotherapy. However, recent innovations have expanded the therapeutic landscape to include targeted treatments, immunotherapies, CAR-T cell therapy, and nanotechnology-based solutions. Chemotherapy remains a foundational systemic treatment, utilizing cytotoxic drugs to disrupt cell division and promote programmed cell death. Despite its effectiveness, chemotherapy poses challenges such as adverse side effects, resistance mechanisms, and lack of specificity. This review aims to provide a detailed exploration of cancer classification and therapeutic modalities, with a particular focus on the evolving role of chemotherapy in oncology. A deeper understanding of these elements is essential for advancing early diagnosis, tailoring personalized treatments, and improving patient outcomes.

Keywords: Cancer, Chemotherapy, Immunotherapy, Targeted therapy.

INTRODUCTION

Cancer encompasses a broad spectrum of diseases defined by the unregulated proliferation and dissemination of abnormal cells. Under physiological conditions, cellular growth, differentiation, and apoptosis are tightly regulated through complex molecular signalling networks. Disruption of these pathways through genetic mutations, epigenetic dysregulation, or environmental insults can trigger malignant transformation. According to WHO and GLOBOCAN 2020 estimates, cancer accounts for approximately one in every six deaths worldwide. The global burden is anticipated to escalate further due to aging populations, rising obesity rates, tobacco use, and environmental carcinogen exposure. In 2020, the five most incident cancers were breast (2.26 million), lung (2.21 million), colorectal (1.93 million), prostate (1.41 million), and non-melanoma skin cancers (1.20 million) [1,2]. Detailed global incidence and mortality data are presented [Table 1]. This review provides a structured synthesis of cancer etiology, histological classification, epidemiological trends, and multimodal treatment strategies, with special emphasis on the pharmacological basis and clinical role of chemotherapy in contemporary oncology.

Epidemiology and Global Burden

Cancer is the second leading cause of death globally [3]. In 2020, an estimated 19.3 million new cases were reported, with nearly 10 million cancer-related deaths. Projections indicate that by 2040, annual incidence may exceed 28 million cases if current trends persist. The distribution of cancer types varies considerably by geographic region, income level, and access to healthcare.

Table 1. Global Cancer Incidence and Mortality Statistics (GLOBOCAN 2020)

Cancer Type	New Cases (millions)	Deaths	% of Cancer Deaths	Mortality Rank
Breast	2.26	685,000	6.9%	2nd
Lung	2.21	1,800,000	18.0%	1st
Colorectal	1.93	916,000	9.4%	2nd
Prostate	1.41	375,000	3.8%	5th
Non-melanoma Skin	1.20	63,700	0.6%	—
Stomach	1.09	769,000	7.7%	4th
Liver	0.91	830,000	8.3%	3rd
Cervical	0.60	342,000	3.4%	—

Etiology and Risk Factors

Carcinogenesis is a multistep process involving the progressive accumulation of genetic and epigenetic alterations that transform a normal cell into a malignant phenotype. The IARC classifies etiological agents into physical, chemical, and biological categories [4]. [Table 2] summarizes the principal carcinogenic agents and their associated malignancies.

Table 2. Major Etiological Agents, Exposed Populations, and Associated Cancers

Category	Agent	Exposed Population	Associated Cancer(s)	IARC Class
Physical	Ionizing radiation	Uranium miners, radiologists, Chernobyl survivors	Leukemia, thyroid, lung	IARC Group 1
Physical	Ultraviolet (UV-B/C)	Outdoor workers, sunbed users	Melanoma, BCC, SCC of skin	IARC Group 1
Chemical	Tobacco smoke	Smokers, passive smokers	Lung, oral, bladder, esoph.	IARC Group 1
Chemical	Asbestos	Construction, shipyard workers	Mesothelioma, lung cancer	IARC Group 1
Chemical	Aflatoxins	Contaminated grain/peanut consumers	Hepatocellular carcinoma	IARC Group 1
Chemical	Arsenic (drinking water)	High-arsenic regions (S. Asia, S. America)	Skin, lung, bladder	IARC Group 1
Chemical	Alcohol	Heavy alcohol consumers	Liver, oropharynx, breast	IARC Group 1
Biological	HPV (types 16, 18)	Sexually active individuals	Cervix, oropharynx, vulva	IARC Group 1
Biological	HBV / HCV	Infected individuals	Hepatocellular carcinoma	IARC Group 1
Biological	EBV	Immunocompromised individuals	Burkitt lymphoma, NPC	IARC Group 1
Genetic	BRCA1/2 mutation	Hereditary breast/ovarian cancer families	Breast, ovarian, pancreatic	Germline
Genetic	APC mutation	FAP syndrome carriers	Colorectal cancer	Germline

Histological Classification of Cancer

From a histopathological perspective, cancers are classified according to tissue of origin into six major categories [5,6,7,8]. This classification is of fundamental

prognostic and therapeutic relevance. Table 3 provides a structured overview of cancer categories, cell of origin, primary sites, key subtypes, and estimated proportional incidence.

Table 3. Histological Classification of Cancer by Cell of Origin.

Category	Cell of Origin	Primary Sites	Key Subtypes	% of Cancers
Carcinoma	Epithelial cells	Breast, lung, colon, prostate, cervix	Adenocarcinoma, SCC, transitional cell	80–90%
Sarcoma	Mesenchymal / connective tissue	Bone, muscle, fat, cartilage, tendons	Osteosarcoma, liposarcoma, Ewing sarcoma	1%
Myeloma	Plasma cells (bone marrow)	Bone marrow (multifocal)	Multiple myeloma, plasmacytoma	1.8%
Leukemia	Haematopoietic stem cells	Bone marrow / blood	ALL, AML, CLL, CML	3.5%
Lymphoma	Lymphocytes	Lymph nodes, spleen, extranodal	Hodgkin HL, Non-Hodgkin NHL, cutaneous	5%
Mixed Type	Multiple cell lineages	Uterus, testis, lung, ovary	Carcinosarcoma, teratocarcinoma, adenosquamous	Rare

Carcinoma

Carcinomas are malignant neoplasms arising from epithelial cells and constitute approximately 80–90% of all human cancers. The two principal subtypes are adenocarcinoma (glandular origin; breast, lung, colon, prostate) [9,10,11,12] and squamous cell carcinoma (squamous epithelium; skin, cervix, oropharynx, esophagus) [13,14].

1.2. Sarcoma

Sarcomas originate in mesenchymal connective tissues (bone, cartilage, fat, muscle). They are rare (~1% of adult malignancies) but disproportionately affect children and young adults [15,16]. Key subtypes include osteosarcoma, chondrosarcoma, Ewing sarcoma, and soft tissue sarcoma.

Myeloma

Multiple myeloma is characterised by clonal plasma cell proliferation in the bone marrow, overproduction of monoclonal immunoglobulin (M protein), osteolytic bone lesions, hypercalcemia, renal insufficiency, and immunodeficiency [17].

Leukaemia

Leukemias originate from haematopoietic precursor cells and are classified by cell lineage (lymphoid vs. myeloid) and clinical course (acute vs. chronic). Principal subtypes include ALL, AML, CLL, and CML. CML is pathognomonically associated with the BCR-ABL1 fusion arising from the Philadelphia chromosome translocation [18,19,20].

Lymphoma

Lymphomas arise from lymphocytes and are divided into Hodgkin Lymphoma (HL) defined by pathognomonic Reed-Sternberg cells and Non-Hodgkin Lymphoma (NHL), encompassing over 60 biologically distinct subtypes [21,22].

Mixed-Type Tumors

Mixed-type cancers contain neoplastic elements from more than one tissue type or germ cell layer. Notable examples include carcinosarcoma (uterus, ovaries), adenosquamous carcinoma (lung, pancreas, cervix), and teratocarcinoma (testis).

Therapeutic Strategies in Cancer Management

The oncological management of cancer has evolved from single-modality approaches to integrated, multidisciplinary treatment paradigms. [Table 4] provides a comparative overview of the principal treatment modalities, their scope, advantages, limitations, and representative clinical examples.

Table 4. Comparative Overview of Cancer Treatment Modalities

Modality	Scope	Key Advantages	Limitations	Clinical Examples
Surgery	Local / locoregional	Curative; immediate removal	Invasive; limited to localised disease	Appendicectomy (colon), mastectomy (breast)
Radiation Therapy	Local / locoregional	Curative or palliative; organ-sparing	Radiation toxicity; treatment-resistant hypoxic cells	IMRT (prostate), SBRT (lung)
Chemotherapy	Systemic	Broad spectrum; adjuvant/palliative	Non-selective; systemic toxicity; resistance	FOLFOX (CRC), R-CHOP (lymphoma)
Targeted Therapy	Systemic (molecular)	High selectivity; improved OS/PFS	Resistance mutations; high cost	Imatinib (CML), osimertinib (NSCLC)
Immunotherapy	Systemic (immune-based)	Durable responses; memory immunity	Immune-related adverse events (irAEs)	Pembrolizumab (melanoma, NSCLC)
Hormonal Therapy	Systemic (endocrine)	Well tolerated; oral formulations	Limited to hormone-receptor+ tumours	Tamoxifen (breast), enzalutamide (prostate)
Gene Therapy	Systemic / local	Curative potential; precision	Delivery challenges; high cost; early-stage	CAR-T (ALL, DLBCL)
HSCT	Systemic	GvL effect; curative in haematology	High treatment-related mortality; graft failure	Allogeneic HSCT (AML, ALL)

Chemotherapy

Chemotherapy employs cytotoxic agents to interrupt cellular proliferation, predominantly by targeting rapidly dividing cells [23]. Despite its broad clinical utility, chemotherapy is constrained by dose-limiting adverse effects, non-selectivity, and acquired drug resistance. [Table 5] details the principal chemotherapeutic drug classes, their mechanisms of action, representative agents, clinical indications, and cell-cycle specificity [24,25].

Table 5. Classification of Chemotherapeutic Agents by Mechanism of Action

Drug Class	Mechanism of Action	Key Agents	Clinical Indications	Cell Cycle
Alkylating Agents	Covalent DNA cross-linking → replication arrest	Cyclophosphamide, cisplatin, temozolomide, oxaliplatin	Lymphoma, breast, ovarian, glioblastoma	Non-cell cycle specific
Antimetabolites	Competitive inhibition of DNA/RNA synthesis cofactors	Methotrexate, 5-FU, gemcitabine, capecitabine	Colorectal, breast, pancreatic, leukemia	S-phase specific
Topoisomerase I Inhibitors	Prevent re-ligation of single-strand DNA breaks	Irinotecan, topotecan	Colorectal, ovarian, cervical, SCLC	S-phase preferential
Topoisomerase II Inhibitors	Prevent re-ligation of double-strand DNA breaks	Doxorubicin, etoposide, epirubicin	Leukemia, lymphoma, breast, sarcoma	Late S/G2-phase
Taxanes	Microtubule hyperstabilisation → mitotic arrest	Paclitaxel, docetaxel, cabazitaxel	Breast, ovarian, lung, prostate	M-phase specific
Vinca Alkaloids	Tubulin depolymerisation inhibition	Vincristine, vinblastine, vinorelbine	ALL, lymphoma, lung, breast	M-phase specific

Miscellaneous	Multiple / unique mechanisms	Bleomycin, asparaginase, arsenic trioxide	Testicular, ALL, APL	Variable

Targeted Therapy

Targeted therapies exploit specific molecular aberrations in cancer cells, achieving superior selectivity over conventional chemotherapy. [Table 6] summarizes the principal targeted drug classes, molecular targets, representative agents, indications, and predictive biomarkers [26,27,28,29,30,31,32,33,34,35,36].

Table 6. Major Targeted Therapy Classes, Molecular Targets, and Clinical Indications

Drug Class	Molecular Target	Representative Agents	Key Indications	Predictive Biomarker / Notes
EGFR inhibitors	EGFR	Erlotinib, gefitinib, osimertinib	NSCLC (EGFR-mutant)	Exon 19 del / L858R mutation required
HER2-directed	HER2/ErbB2	Trastuzumab, pertuzumab, T-DXd	Breast, gastric (HER2+)	HER2 IHC 3+ or FISH amplification
BCR-ABL1 TKIs	BCR-ABL1 fusion	Imatinib, dasatinib, ponatinib	CML, Ph+ ALL	Philadelphia chromosome t(9;22)
VEGF/VEGFR inhibitors	VEGF / VEGFR	Bevacizumab, sunitinib, sorafenib	RCC, HCC, colorectal, GIST	Anti-angiogenic mechanism
CDK 4/6 inhibitors	CDK4/CDK6	Palbociclib, ribociclib, abemaciclib	HR+ HER2− breast cancer	Used with aromatase inhibitor
PARP inhibitors	PARP-1/2/3	Olaparib, niraparib, rucaparib	BRCA-mutant ovarian/breast	Synthetic lethality mechanism
BRAF/MEK inhibitors	BRAF V600 / MEK	Vemurafenib, dabrafenib, trametinib	BRAF V600E melanoma, NSCLC	Combination recommended to prevent resistance
PD-1/PD-L1 ICIs	PD-1 / PD-L1	Pembrolizumab, nivolumab, atezolizumab	Multiple solid tumors	PD-L1 TPS / TMB as predictive biomarkers
ALK inhibitors	ALK fusion	Alectinib, brigatinib, lorlatinib	ALK-rearranged NSCLC	EML4-ALK fusion most common

Immunotherapy

Cancer immunotherapy harnesses the host immune system to eliminate tumor cells. Approved modalities include immune checkpoint inhibitors (anti-CTLA-4, anti-PD-1/PD-L1), CAR-T cell therapies, bispecific T-cell engagers (BiTEs), and cancer vaccines. CAR-T therapy has demonstrated durable complete remissions in relapsed/refractory B-cell ALL and diffuse large B-cell lymphoma [37,38].

Hormonal Therapy

Endocrine therapy is the standard of care for hormone receptor-positive cancers. ER-positive breast cancer is managed with selective estrogen receptor modulators (tamoxifen), selective estrogen receptor degraders (fulvestrant), or aromatase inhibitors (anastrozole, letrozole). Prostate cancer employs LHRH agonists/antagonists (leuprolide, degarelix) or androgen receptor antagonists (enzalutamide, apalutamide) [39,40].

Gene Therapy and HSCT

Gene therapy encompasses oncogene suppression, tumor suppressor gene restoration, suicide gene therapy, and CRISPR-Cas9-mediated genome editing. Hematopoietic stem cell transplantation (HSCT) enables dose-intensified chemotherapy with haematopoietic rescue and confers graft-versus-leukaemia (GvL) immune effects in allogeneic settings [41,42,43,44,45].

CONCLUSION

Cancer represents a diverse group of diseases unified by dysregulated cellular growth and the potential for systemic dissemination. This review has systematically outlined the etiological determinants, histological classification schema, and principal treatment modalities currently employed in oncology. Chemotherapy, while central to cancer treatment, must be contextualized within a broader multimodal framework that increasingly incorporates targeted therapy, immunotherapy, and precision medicine. Continued investment in translational oncology research, biomarker discovery, and international healthcare equity is essential to improving survival outcomes and quality of life for cancer patients globally.

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