Common Chemotherapies

Class	Drugs	Mechanism	Used in	Pharma/ Metabolism/ Excretion	Side effects		Antidote/ Co-treatment	Genomic Bio-marker
					Short-Term	Long-Term		
Anthracyclines	Doxorubicin Daunorubicin Idarubicin Mitoxantrone	Antibiotic from Streptomyces bacteria; Intercalates between DNA/ RNA hybrids in replication.	Leukemia Sarcomas Lymphoma	Liver	Myelosuppression Mucositis Skin reactions (hand-foot syndrome)	Heart failure (dose-dependent)	Dexrazoxane may be used in limited cases for patients at highest risk of developing cardiotoxicity	-
Asparaginase	PEG- Non-PEG (Erwinia)-	Bacterial enzyme, converts asparagine to aspartic acid and ammonia. Inhibits protein synthesis	ALL AML	PEG half life 5-7 days, Non-PEG half life <24 hours	Anaphylaxis Coagulo-pathy/ Thrombosis Hyper-ammonemia Encephalo-pathy Hemorrhagic pancreatitis Transaminitis	-	-	-
Vinca alkaloids	Vincristine Vinblastine Vinorelbine	Inhibits mitotic M phase by preventing microtubule function	ALL Lymphoma Sarcoma, CNS NBL WT	Liver	Neurotoxicity Peripheral neuropathy SIADH Constipation Seizures Hypotension	-	Stool regimen	-

Legend:
Diseases: ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; BTs, brain tumors; NBL, neuroblastoma; WT, Wilms tumor
Side effects: SIADH, syndrome of inappropriate ADH; N/V/D, nausea/vomiting, diarrhea
Genes: DHFR, dihydrofolate reductase; MGMT, O-6-methylguanine-DNA methyltransferase; UGT1A1, UDP glucuronosyltransferase 1; TPMT, thiopurine S-methyltransferase
Other: IT, intrathecal; PEG, polyethylene glycol

Common Targeted Therapies

Drug	Mechanism	Used In	Pharma/ Metabolism/ Excretion	Side Effects		Antidote/ Co-Treatment	Pharmaco-genomic Biomarkers
				Short-Term	Long-Term		
Imatinib	Kinase inhibitor of BCR-ABL fusion, PDGFR and c-Kit proteins	Ph+ ALL GIST CML	Liver	Nausea Diarrhea Myalgias	Cardiac toxicity, delayed linear growth (pre-pubescent)	-	BCR-ABL fusion PDGFR mutation
Dasatinib	Inhibitor of ABL, Src, c-Kit kinases	Ph+ ALL CML	Liver	Myelo-suppression Pleural effusion	Pulmonary hypertension	-	BCR-ABL fusion
Sorafenib	Multi-kinase inhibitor (BRAF, VEGFR, PDGFR, FLT3)	FLT3+ AML RCC Liver tumors	Liver	Hemorrhage Electrolyte wasting (low PO4, Ca, K) Myelo-suppressio Cardiac toxicity	-	-	FLT3 internal tandem duplication in AML

Common Targeted Therapies

Drug	Mechanism	Used In	Pharma/ Metabolism/ Excretion	Side Effects		Antidote/ Co-Treatment	Pharmaco-genomic Biomarkers
				Short-Term	Long-Term		
Crizotinib	Inhibitor of ALK, ROS1, and NTRK1 kinases	Lymphoma NBL Others	Liver	Nausea Vomiting Diarrhea	-	-	Mutation or fusion of ALK, ROS1, NTRK1
Rituximab	Monoclonal antibody against CD20 (B-cell lineage marker)	ALL Lymphoma	-	Infusion reactions Cytokine release syndrome Pulmonary toxicity	Reactivation of viruses	-	-
Dinutuximab (ch14.18)	Monoclonal antibody against GD2 glycolipid	NBL	-	Capillary leak syndrome Hypotension Neuropathic pain Hyper-sensitivity reactions	-	-	-
Chimeric antigen receptor (CAR) T cells	Engineered patient T cells expressing modified CD19 receptors, which kill B-lineage cells	B-ALL	-	Cytokine release syndrome (fevers, myalgias, capillary leak/ hypotension, resp. failure) Encephalo-pathy	B cell aplasia	Tocilizumab (IL6R antagonist) for severe CRS	-

Oncologic Emergencies

Tumor Lysis Syndrome (TLS)

Definition	• An oncologic emergency that is caused by massive tumor cell lysis and the release of large amounts of intracellular contents (**potassium, phosphate, and uric acid**) into the systemic circulation • Most often occurs after the initiation of cytotoxic therapy in patients with high-grade lymphomas (particularly the Burkitt subtype) and ALL • Can also occur spontaneously and with other tumor types that have a high proliferative rate, large tumor burden, or high sensitivity to cytotoxic therapy
Pathogenesis	• Rapid lysis of tumor cells releases large amounts of intracellular contents (potassium, phosphate, and nucleic acids) into circulation leading to hyperkalemia, hyperphosphatemia, secondary hypocalcemia, hyperuricemia. • Purines are metabolized to hypoxanthine and xanthine, and then to uric acid via xanthine oxidase. • Uric acid is poorly soluble in water leading to crystal precipitation and deposition in the renal tubules and AKI. • Allopurinol competitively inhibits xanthine oxidase, blocking the metabolism of hypoxanthine and xanthine to uric acid. Xanthine is less soluble than uric acid so allopurinol can exacerbate AKI. • Cancer cells have ~4X higher Phos than normal cells. Hyperphosphatemia can lead to secondary hypocalcemia and renal calcium phosphate precipitation. Hypocalcemia may also cause cardiac arrhythmias. • Elevated uric acid and phosphate worsen the severity of AKI (increases precipitation of each other)

Oncologic Emergencies continued on next page →