1. Antiplatelts

Drug name	MOA	Indications	Adverse effects	Route of adminstration
Aspirin	Blocks COX→ inhibits conversion of AA into TXA2.	- Prophylactic in transient cerebral ischemia. - to reduce the recurrence of MI. -in angina.	Hemorrhagic stroke and GIT bleeding	Orally (daily dose of 100 mg).
Cangrelor Ticagrelor NOT prodrugs	ADP receptors blockers		Ticagrelor: bleeding and shortness of breath (dyspnoea)	Ticagrelor(Orally) Cangrelor (IV)
Clopidogrel Ticlopidine Prasugrel Prodrugs	ADP receptors blockers	- prevent vascular events in patients with transient ischemic attacks (TIA). - unstable angina, - prevent thrombotic stroke. - prevent thrombosis in patients undergoing placement of a coronary stent.	- Prasugrel: bleeding Hypertension hypotension atrial fibrillation and bradycardia. - Ticlopidine : Hemorrhage , Leucopenia and Thrombotic thrombocytopenic purpura (TTP). - Clopidogrel (fewer than with ticlopidine): Neutropenia and TTP.	Orally - Ticlopidine: 250 mg BID orally. -Clopidogrel: oral loading dose 300 mg, maintenance dose 75 mg once daily.
Abciximab Eptifibatide Tirofiban	AlII inhibit bridging of platelet by fibrinogen [Glycoprotein IIb/IIIa inhibitors]	- percutaneous coronary intervention (PCTA) & inACSs.	----------	Parenterally
Dipyridamole cilostazol		- with aspirin for prophylaxis in angina. - with warfarin to inhibit embolization from prosthetic heart valves.		----------

MoA
- Inhibits phosphodiesterase → ↑ cAMP → potentiates effects of prostacyclin → platelet inhibition.
- Dipyridamole is also a coronary vasodilator

2. Anticoagulants

Anticoagulants

A) Heparin
B) Low-Molecular-Weight Heparins:
Enoxaparin, dalteparin, tenzaparin
C) Heparinoids:
Danaparoid.
D) Direct & specific thrombin inhibitors:
Hirudin (leech protein), lepiridun, bivalirudin, argatroban, melagatran.
E) Oral direct & specific thrombin inhibitors:
Ximelagatran and Dabigatran
F) Pentasacharide specific Xa inhibitors:
Fondaparinux, Rivaroxaban
F) Warfarin. (vitamin k inhibitor)

old , the most drug lets the patients enter the hospitals in term of bleeding till now

Drug name	MOA	Adverse effects	Antidote	Contraindications
Unfractionated Heparin injectable/infusion	- Activates plasma protease inhibitor antithrombin III (ATIII). - when heparin binds to antithrombin III, it causes the activity of it *100000 folds - The complex (heparin + anti..III) inactivates factors: XIIa, XIa, IXa, Xa,Xa and IIa	1. The major adverse effect is bleeding. 2. Allergy. 3. Increased loss of hair (reversible alopecia). 4. Osteoporosis. 5. Hyperkalemia. 6. Heparin-induced thrombocytopenia (HIT).	protamine	
Low-Molecular Weight Heparins (LMWHs) (Enoxaparin, dalteparin, tenzaparin & ardeparin)	- Bind to & catalyze ATIII. This complex preferentially inactivates factor Xa & minimally affects thrombin so it has lower efficacy than (UFH).	1. Reactions at the injection site: irritation, pain, hematoma, bruising & redness. 2. bleeding. 3. HIT.	protamine	In patient with compromised renal function (because LMWH are eliminated renally)
Warfarin & Coumarin	- It inhibits Vitamin K epoxide reductase and blocks carboxylation of factors VII, IX, & X, & II as well as the proteins C and S.	1. Bleeding– the most dangerous. 2. teratogenic 3. Venous thrombosis (due to ↓activity of protein C) and S 4. Purple toe syndrome (cholesterol microembolization → arterial obstruction) clots might close the small arteries.	Vit K	- Absolute: Pregnancy (teratogenic)

➡️Continuation of anticoagulants

Dabigatran Orally (Onset: 1 hour)	Direct thrombin inhibitor which inhibits: - Both free and fibrin-bound thrombin. - Cleavage of fibrinogen to fibrin. - thrombin-induced platelet aggregation.	1. Bleeding 2. Dyspepsia 3. gastrointestinal upset	Idarucizumab	1. Active pathological bleeding. 2.patients with mechanical prosthetic heart valves
rivaroxaban, apixaban) Orally	Factor Xa inhibitors		Andexanet	1. patients with Prosthetic valve 2. patients with spinal anesthesia or puncture.

3. Thrombolytics (Fibrinolytics)

Both protective hemostatic thrombi & target pathogenic thromboemboli are broken down

Drug name	MOA	Indecations
Streptokinase Formed by streptococci	- Combines with plasminogen. The complex cleaves another plasminogen molecule to plasmin.	1. IV for: ▪ Multiple pulmonary emboli ▪ Central deep venous thrombosis (eg, superior vena caval syndrome, ascending thrombophlebitis of iliofemoral vein). ▪ Acute myocardial infarction. ▪ Acute ischemic stroke: tPA should be used within 3 hours after the onset of symptoms. 2. Intra-arterially for: Peripheral vascular disease
Anistreplase	- An acetylated streptokinase-plasminogen complex that cleaves plasminogen to plasmin.	
Urokinase Synthesized by kidney	- Directly cleaves plasminogen to plasmin.	
t-PA (tissue plasminogen activator): Alteplase (recombinant t-PA) Tenecteplase (genetically modified recombinant t-PA → long t1/2) Reteplase (genetically modified recombinant).	- Endogenous direct activator of plasminogen. It preferentially activates plasminogen that is bound to fibrin rather than circulating plasminogen. This specificity reduces systemic bleeding risk, making tPA more accurate and preferable	

Side Effects: Bleeding, Reperfusion Arrhythmia, Hypotension and Hypersensitivity.

4. ALL Drugs

Drug name	MOA	ADR
vincristine	Arrest cell mitosis (M phase)	1. Neuropathy 2. Constipation 3. Nerve Irritation 4. numbness or tingling in the hands and feet.
Glucocorticoid: Prednisone (cortisone)	Higher dose will lead to the killing of T cells.	buffalo hump, DM , peptic ulcers , moon face and hypertension
L- asparaginase	Cause the depletion of asparagine in the blood of the patient, ALL cells cannot synthesize asparagine, which is why we can treat ALL. This depletion leads to the inhibition of protein synthesis in cancerous cells.	All of them are important 1. hyperglycemia secondary to hypoinsulinemia 2. Hypoalbuminemia resulting in peripheral edema or ascites. 3. Decreased production of vitamin K-dependent clotting factors and endogenous anticoagulants such as proteins C and S and antithrombin II. 4. Mild nausea/vomiting 5. Tumor Lysis Syndrome (TLS) this will cause:Hyperkalemia, hyperphosphatemia,hyperuricemia, hypocalcemia, decreased urine output and severe renal insufficiency.
Methotrexate	Inhibition of dihydrofolate reductase.	
Doxorubicin	"Topoisomerase poison": it traps topoisomerase enzymes at the moment they cut the DNA strand, preventing the rejoining of DNA. As a consequence, the cell will die.	cardiotoxicity
6.M.P. (6-mercaptopurine)	A purine analog acts as an antimetabolite.This fake purine enters the DNA chain of both cancer and normal cells, leading to stop replication. (S phase)	
Cytarabine	pyrimidine analog	Dizziness

Chemotherapy for acute leukemias

- Phases of ALL treatment LONG STORY OF 3 YEARS TREATMENT
  - induction
  - intensification
  - CNS prophylaxis
  - maintenance

  } post-remission therapy

1.Induction [four to six weeks]: Vincristine, Glucocorticoid (prednisone, prednisolone or dexamethasone) and L-asparaginase.

2. Consolidation:
1. Methotrexate with mercaptopurine
2. High-dose asparaginase over an extended period
3. Reinduction treatment (a repetition of the initial induction therapy in the first few months of remission).

3. Maintenance [2-3 years] usually consists of
1. weekly methotrexate and 2. daily mercaptopurine.

4.CNS prophylaxis: Intrathecal (methotrexate, cytarabine, steroids)
and for adult high-dose systemic chemotherapy (methotrexate, cytarabine, L-asparaginase)

5.AML+CMLDrugs

Drug name	MOA	ADR
Cytosine arabinoside (AML)	An antimetabolite which will enter the nucleotide and act as a false nucleotide and stop DNA polymerase from replicating, it is a cell cycle specific that's why it will stop the cell in the S phase.	Dizziness
Daunorubicin AML	A drug like doxorubicin but the difference is the presence of an OH group within the structure, these two drugs are considered topoisomerase poisons as they capture topoisomerase while it cuts DNA; when topoisomerase cuts the DNA in the process of unwinding DNA coiling.	cardiotoxicity
Thioguanine AML	An antimetabolite that looks like guanine, it incorporates within the DNA and stops DNA polymerase.	
Imatinib (CML)	An inhibitor of the tyrosine kinase domain of the Bcr-Abl oncoprotein and prevents the phosphorylation of the kinase substrate by ATP.	After treatment some mutations would happen like T315I.
Nilotinib or Dasatinib (CML patient has any type of mutations rather than the bad mutation (T315I)).		All of them are important Nilotinib > cardiovascular events Dasatinib > pleural effusion peripheral edema, increase NK cells, skin rash and diarrhea more common with Dasanitib except for progressive peripheral arterial occlusive disease which is more common with Nilotinib (it also causes clotting).
Ponatinib (CML patient has (T315I) mutation)	Can enter the pocket whatever the mutation or the situation is, as it is way more efficient and potent comparing to the other drugs.	Liver problem Heart problems Blockage in arteries and veins Blood clots

1. Fatigue (tiredness) during and after treatment.
2. Soreness at the injection site (if you are having injections under the skin).
3. Women may stop having periods (amenorrhoea), but this may only be temporary.

☆ AML: INDUCTION THERAPY The idea of the (3+7) method refers to using daunorubicin (not a cell cycle specific) for 3 days, followed by 7 days of using cytosin arabinosides (antimetabolite).

☆ AML Consolidation (Following induction into Complete Remission)
Here, we have two choices:
• 3-4 cycles of high dose cytosine arabinoside (HiDAC) administered approximately every 5-6 weeks.
• Bone marrow (peripheral blood stem cell) transplant

6.Treatment of Herpesviruses

Drug name	MOA	Indecations	ADR
Acyclovir (Guanine analogue) Valacyclovir (Acyclovir + ester group)	Acyclovir triphosphate (AcycloGTP) inhibits viral DNA polymerase by: 1. Inhibits viral DNA polymerases electively 2. Incorporated into DNA and terminates synthesis.	1. Treat H. simplex and varicella-zoster Virus. 2. Prophylactically in patients treated with immunosuppressant drugs or radiotherapy who are in danger of infection by reactivation of latent virus. 3. Prophylactically in patient with frequent recurrences of genital herpes.	1. Orally > diarrhea, nausea vomiting and headache. 2. IV > Renal insufficiency and neurologic toxicity.
Ganciclovir	Mechanism like Acyclovir.	1. Active against all Herpes viruses including CMV. 2. Drug of choice for CMV infections: retinitis, pneumonia, colitis.	1. Both Ganciclovir and Acyclovir are teratogenic. 2. Bone marrow suppression (leukopenia Thrombocytopenia. 3. CNS effects (headache, psychosis, coma, convulsions).
Foscarnet (inorganic pyrophosphate analog)	Direct inhibition of DNA polymerase and Reverse Transcriptase very selective for this enzyme Reverse transcriptase is associated with AIDS/HIV	1. CMV retinitis and other CMV infections instead of ganciclovir 2. H. simplex resistant to Acyclovir. 3. HIV	Nephrotoxicity

7. Treatment of respiratory virus infection Influenza A & B

Drug name	MOA	ADR
Neuraminidases inhibitors (Oseltamivir and Zanamivir)	catalyze cleavage of terminal sialic acid residues attached to glycoproteins and glycolipids, a process necessary for release of virus from host cell surfaces thus prevent release of virions from infected cell.	1. Oseltamivir (orally) >Nausea and vomiting. 2. Zanamivir (inhalation)>Exacerbation of reactive airway disease.
Cap-dependent endonuclease inhibitor (Baloxavir marboxil)	Inhibit influenza virus' cap dependent endonuclease activity (cap snatching).	

8. Antiretroviral agents

Drug name	MOA	ADR
Azidothymidine (Zidovudin (AZT))	Potent antagonist of reverse transcriptase and causes chain termination.	1. Toxic to bone marrow, for example, it causes severe anemia and leukopenia. 2. Headache is also common
Didanosine (Dideoxyinosine) Doctor didn't mention it	Act as chain terminators and inhibitors of reverse transcriptase.	(Orally) Their main toxicities are pancreatitis, peripheral neuropathy, GI disturbance, BM depression.
Non-nucleosideNon-competitive RT inhibitors (Nevirapine and Delavirdine)	1. Bind to viral RT, inducing conformational changes that result in enzymes inhibition. 2. Combination therapy with AZT	1. RASH 2. CNS effects (e.g. sedation, insomnia, vivid dreams, dizziness, confusion, feeling of "disengagement").
Protease Inhibitors (Saquinavir, and Ritonavir). All end with navir	- Responsible for cleavage of viral polyprotein into number of essential enzymes (reverse transcription, polymerase). Have significantly altered the course of the HIV disease. - All are reversible inhibitors	(Orally) 1. GI disturbances, hyperglycemia and they interact with Cytochrome p450. 2. Buffalo hump

9. Anti-malrial

Drug name	Type	MOA	Adverse effects	Contraindication
Chloroquine	Blood schizontocidal	- It is accumulated in parasite lysosomes and inhibits digestion of hemoglobin by the parasite and thus helps reduce its supply of amino acids. - It also inhibits haem polymerase - the enzyme that polymerises toxic free haem to the innocuous hemozoin.	1. At high doses, gastrointestinal upset,pruritus, headaches. 2. visual disturbances. 3. Parenteral administration can result in hypotension and cardiac arrhythmia, convulsions.	psoriasis or porphyria
Quinine and Quinidine	- Blood schizonticide - Gametocidal against Pvivax and Povale	- kill malaria parasites during their blood stage. - prevent the spread of malaria by reducing the number of parasites in infected individuals, thereby lowering the risk of transmission to mosquitoes (gametocidal).	1. Cinchonism [tinnitus, headache, nausea, dizziness, flushing, and visual disturbances]. 2. Therapeutic doses may cause hypoglycemia.	----------
Proguanil(Chloroguanide)	Erythrocytic schizonticide	- Inhibits plasmodial DHFRase in preference to the mammalian enzyme. - Current use of proguanil is restricted to prophylaxis of malaria in combination with chloroquine.		----------
Mefloquine	----------	- Inhibition of the haem polymerase. (like chloroquine).	1. nausea, vomiting, dizziness, sleep and behavioral disturbances, epigastric pain, diarrhea,	Epilepsy, psychiatric disorders, arrhythmia, cardiac conduction defects

➡️Continuation of Anti-malrial

			abdominal pain, headache, rash, and dizziness. 2. Neuropsychiatric toxicities	
Primaquine	- Primary and latent hepatic stages of P. vivax and P. Ovale - Gametocidal		1. nausea, epigastric pain, abdominal cramps, and headache, and these symptoms are more common with higher dosages and when the drug is taken on an empty stomach. 2. induce hemolytic anemia in patients with G6PD.	- a history of granulocytopenia or methemoglobinemia, in those receiving potentially myelosuppressive drugs (eg,quinidine), - Avoided in pregnancy & G6PD
Artemisinin derivatives (Artemether Arteether Artesunate) Used only in combination	Blood schizonticide	- Have peroxide configuration responsible for its action. - Short Duration of action.		----------

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