

1. Heparins

MOA: Catalyzes ATIII mediated breakdown of Xa and Thrombin
USE: TX/PPX of DVT and PE
SE: Bleeding (aPPT), HIT (type II), Hypoaldosteronism (Type 4 RTA), osteoporosis
Enoxaparin
LMWH - long half life, renal elimination
Fondaparinux - Inhibits Xa, Less HIT
Antidote - Protamine SO₄ (less effective on LMWH, Fondaparinux), PCC

2. Warfarin

MOA: Inhibit Vit. K epoxidase reductase → inhibit gamma carboxylation of II, VII, IX, X, proteins C and S
SE: Bleeding (PT), initial Hypercoag. – protein C (give w/ heparin), Skin necrosis, Teratogen (crosses placenta)
PK: Increases efficacy: CYP inhibitors—Cimetidine (PK), Albumin binders – EtOH, Lasix (PK), Anti-coags – aspirin, heparin (PD), Long term Antibiotics (PD), Decreases efficacy: Cyp inducers – Barbs, Rifampin (PK) absorb-Cholestyramine (PK), Vit K – Green veggies (PD)
Antidote – Vit K., Fresh frozen plasma (immediate)

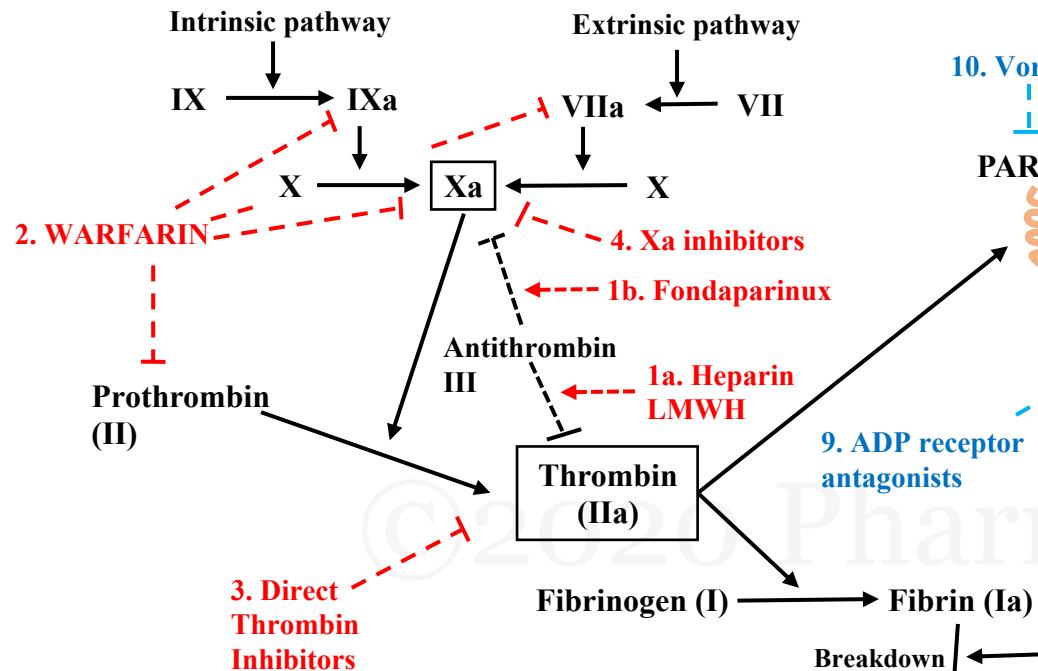
3. Direct Thrombin inhibitors

MOA: Bind and inhibit Thrombin
USE: Used to in pts with HIT
Lepirudin – PAR, CI: Renal disease
Bivalirudin – PAR, CI: Liver & renal
Argatroban – arginine derivative
Dabigatran – Oral, P-GP substrate
Antidote - Idarucizumab

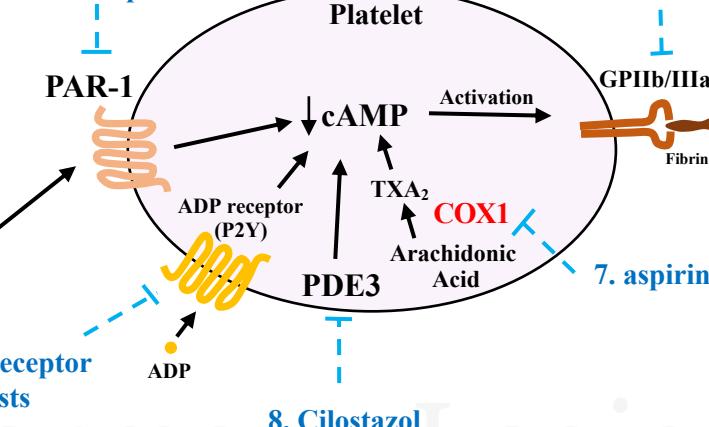
4. Factor Xa inhibitors

MOA: inhibits Xa-mediated thrombin activation
Rivaroxaban – used in pts with A. fib
Apixaban - used in pts with A. fib
Edoxaban
Betrixaban
DDI: P-GP substrate
Antidote – Andexanet, PCC

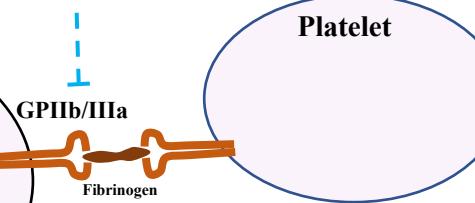
Red Thrombus



10. Vorapaxar



11. Glycoprotein IIb/IIIa inhibitors



White Thrombus

7. Aspirin

MOA: Irreversibly inhibit COX1
SE: GI bleeds, Salicylate toxicity, pseudo-allergy (↑Leukotriene synthesis), Metabolic acidosis (↑Gap), tinnitus

8. Cilostazol

MOA: Inhibits PDE3 → ↑cAMP
SE: Coronary steal syndrome - vasodilation

9. ADP receptor antagonists

MOA: Inhibits ADP-mediated decreased cAMP - irreversible

Ticlopidine – SE: TTP, neutropenia
Clopidogrel – CI: omeprazole

Prasugrel – prodrug – irreversible

Ticagrelor – added to aspirin for CAD

10. Vorapaxar

MOA: Platelet protease activated receptor-1 antagonist

SE: hemorrhage

CI: pts w/ TIA, stroke

Fibrinolytics

5. Plasminogen activators

MOA: facilitate conversion of plasminogen to plasmin (↑PT/↑PTT)

t-PA

Alteplase

Tenectiplase

Use: lyse clots– ischemic stroke, DVT/PE

CI: Head trauma/bleeding, hypertension

6. Plasminogen inhibitors

MOA: prevent conversion of plasminogen to plasmin

Aminocaproic acid

Tranexamic acid

Plasminogen activator inhibitor-1

Use: Patients with hemophilia A

5. Plasminogen Activators

MOA: Inhibit fibrin-mediated platelet aggregation
SE: Thrombocytopenia
Abciximab – monoclonal antibody
Tirofiban - IV
Eptifibitide – IV

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11. GP IIb/IIIa inhibitors