

# Summary of imp. Points in Antiplatelet Drugs lecture



	Mechanism of action	Drug	ROA
(1)	<p>Inhibition of prostaglandin metabolism, TXA2 is inhibited</p> <p>(Arachidonic acid pathway inhibitors)</p>	<p>Aspirin ((Acetylsalicylic Acid)</p> <ul style="list-style-type: none"> <li>- Commonly used</li> <li>- can lead to GIT disorders (PUD)</li> <li>- Aspirin Resistance: !!!imp</li> </ul> <p>A- in 5%-75%</p> <p>B- recurrent thrombosis while on antiplatelet therapy. Due to inhibition of only ONE ( TXA2)of many factors leading to thrombus formation</p>	Oral
(2)	<p>Inhibition of ADP-induced platelet aggregation</p> <p>(Antagonist of ADP receptors)</p> <p>(Slow onset of action (3 - 5 days).</p>	<p>1-Clopidogrel      2- Ticlopidine</p> <p>1 is :</p> <p>A- better than 2 (less ADRs, potent &amp; longer duration of action )</p> <p>B- Alternative prophylactic therapy to aspirin</p> <p>C- works by reducing the “stickiness”</p> <p>D-Combination with aspirin may be recommended</p>	Oral
(3)	<p>GP IIb / IIIa receptor antagonists (Inhibitors)</p> <p>(inhibits all the pathways of platelet activation (Final common pathway).)</p>	<p>Abciximab (imp. One , monoclonal antibody , used in Acute coronary syndrome before surgery)</p> <p>Tirofiban      Eptifibatide</p>	I.V Imp.
(4)	<p>Phosphodiesterase 3 (PDE) inhibitors / adenosine uptake inhibitors ( increasing of cAMP lead to inhibition of Plt aggregation)</p>	<p>Dipyridamol (When give alone it has little or no beneficial effect , combined with aspirin + warfarin)</p> <p>Cilostazol</p>	Oral