Antiplatelet medications

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Antiplatelet medications (also **antiaggregans**) inhibit **primary homeostasis** and thus prevent the forming of **primary platelet thrombus** (platelet plug). They are mainly used as profylaxis for arterial thrombosis.

Common indications:

- secondary prevention of myocardial infarction
- secondary prevention of ischemic stroke and transitory ischemic attack (TIA)

Mechanism of action

We can classify antiplatelet drugs based on the mechanism of action:

- irreversible inhibitors of COX on platelets and endothelial cells
- inhibitors of platelet ADP receptors
- inhibitors for fibrinogen (gpIIb-IIIa) receptors
- inhibitors of platelet cyclic nucleotide phosphodiesterase

Irreversible inhibitors of COX on platelets and endothelial cells

Acetylsalicic acid (*aspirin*) is most commonly used antiplatelet medication. ASA irreversibly inactivates cyclooxygenase enzyme (COX-1) in platelets and thus stops the synthesis of prothrombic Thromboxane A2. By blocking COX-2 in endothelial cells it blocks the synthesis of vasodilating and *antiplatelet* PGI2 in endothelial cells. The therapy aims to block *only* the aggregating agent TXA2 while maintaining PGI2 secretion. This is achieved by administering low doses of ASA (100-300 mg/day), because of the different affinity of ASA to COX-2 and COX-2. At these doses, irreversible blockade of platelet cyclooxygenase occurs and lasts throughout the life of the platelet, approximately 7-8 days.

Inhibitors of platelet ADP receptors

These medications inhibit adenosine diphosphate induced (ADP-induced) platelet aggregation. They are administered to patients who do not tolerate the ASA for various reasons (eg. peptic ulcer) and to patients in whom we need dual antiplatelet therapy. This group includes: *clopidogrel*Template:HVLP, *ticlopidine* and new substances *ticagrelor*Template:HVLP a *prasugrel*Template:HVLP.

- Ticlopidine is not used anymore because of potential hemotoxicity.
- Ticagrelor and prasugrel in combination with ASA are used for antiplatelet therapy in arterial interventions (especially before and after percutaneous coronary artery interventions).

Inhibitors for fibrinogen (gpIIb-IIIa) receptors

Glycoproteins gpIIb-IIIa are fibrinogen receptors on the platelet surface. By blocking these receptors, we inhibit the final step of platelet activation. This group includes: *abciximab*Template:HVLP, *tirofiban* a *eptifibatide*Template:HVLP. *Abciximab* is a fragment of the chimeric monoclonal antibody and is indicated for patients who do not respond to conventional therapy and to prevent cardiac complications in percutaneous interventions. *Tirofiban* is a non-peptide antagonist that is indicated for unstable angina pectoris. The cyclic heptapeptide *eptifibatide* has similar indications. The importance of gpIIb-IIIa inhibitors decreased after the introduction of *ticagrelor and prasugrel* into clinical practice.

Inhibitors of platelet cyclic nucleotide phosphodiesterase

This group includes *dipyridamole*. It is a coronary vasodilator that does not show antiplatelet effects *in vitro*. It increases cAMP levels in platelets and reduces platelet adhesion to damaged endothelium. In therapy, it is most often used in combination with another antiplatelet agent.

Links

Related articles

- Hemostasis
- Acetylsalicic acid
- Procvičování:Antiagregans

Literature

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