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Perspective

Advances in Antiplatelet Therapy: Current Concepts and Emerging Targets

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DESCRIPTION

Platelets are small blood cells that play a vital role in stopping bleeding and forming clots at sites of vascular injury. However, platelets can also contribute to the development and progression of atherosclerosis, a chronic inflammatory disease of the arteries that can lead to heart attacks and strokes. Therefore, antiplatelet therapy, which inhibits platelet activation and aggregation, is a key strategy to prevent thrombotic complications in patients with cardiovascular disease.

Current concepts in antiplatelet therapy

The most commonly used antiplatelet drugs are aspirin and P2Y12 receptor antagonists, such as clopidogrel, prasugrel, ticagrelor and cangrelor. Aspirin irreversibly inhibits cyclooxygenase-1 (COX-1), an enzyme that produces Thromboxane A2 (TXA2), a potent platelet activator and vasoconstrictor. P2Y12 receptor antagonists block the binding of Adenosine Diphosphate (ADP) to its receptor on platelets, thereby preventing the activation of downstream signalling pathways that mediate platelet aggregation and secretion.

Aspirin and P2Y12 receptor antagonists are often used together as Dual Antiplatelet Therapy (DAPT) to achieve greater inhibition of platelet function and better clinical outcomes than either drug alone. DAPT is recommended for patients with Acute Coronary Syndrome (ACS), who have an increased risk of recurrent thrombotic events due to plaque rupture or erosion. DAPT is also indicated for patients undergoing Percutaneous Coronary Intervention (PCI) with stent implantation, to prevent stent thrombosis and restenosis.

However, antiplatelet therapy is not without drawbacks. The main limitation of antiplatelet therapy is the increased risk of bleeding, which can be life-threatening and offset the benefits of preventing thrombosis. The risk of bleeding depends on several factors, such as the type, dose and duration of antiplatelet drugs, the presence of comorbidities and concomitant medications, and the patient's genetic profile and adherence to treatment. Therefore, a personalized approach to antiplatelet therapy is needed to balance the risk of bleeding and thrombosis for each individual patient.

Another challenge of antiplatelet therapy is the variability of platelet response to different drugs, which can result in suboptimal inhibition of platelet function or resistance to treatment. Several mechanisms have been proposed to explain this variability, such as pharmacokinetic factors, pharmacodynamics factors, genetic factors, environmental factors and clinical factors. Therefore, methods to monitor platelet function and adjust antiplatelet therapy accordingly are needed to optimize treatment efficacy and safety.

Novel targets for antiplatelet therapy

Despite the advances in antiplatelet therapy, there is still a residual risk of thrombotic events in patients with cardiovascular disease, especially in those with high-risk features or complex lesions. Moreover, there is an unmet need for more effective and safer antiplatelet agents that can overcome the limitations of current drugs. Therefore, novel targets for antiplatelet therapy have been identified and explored in preclinical and clinical studies.

One potential target is the Protease-Activated Receptor 1 (PAR1), which mediates platelet activation by thrombin, the most potent platelet agonist. Thrombin cleaves PAR1 on platelets, exposing a tethered ligand that binds to the receptor and triggers intracellular signalling pathways. Vorapaxar is a selective PAR1 antagonist that has been shown to reduce the incidence of cardiovascular events in patients with a history of myocardial infarction or peripheral artery disease, when added to standard antiplatelet therapy. However, vorapaxar also increases the risk of intracranial hemorrhage, especially in patients with a history of stroke or transient ischemic attack. Therefore, vorapaxar is contraindicated in these patients and should be used with caution in others.

Other novel targets for antiplatelet therapy include kinases and enzymes that regulate intracellular signaling pathways involved in platelet activation, such as Phosphoinositide 3-Kinase (PI3K),

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Protein Kinase C (PKC), Spleen Tyrosine Kinase (Syk) and Cyclooxygenase-2 (COX-2). These targets might offer more selective and reversible inhibition of platelet function, with less impact on haemostasis and bleeding. Moreover, these targets might have additional benefits on vascular inflammation and microcirculation, which are important determinants of atherosclerosis and myocardial ischaemia.

Antiplatelet therapy is a key strategy to prevent thrombotic complications in patients with cardiovascular disease. However, current antiplatelet drugs have limitations, such as increased bleeding risk, variable platelet response and residual thrombotic risk. Novel targets for antiplatelet therapy have been identified and new drugs have been developed to overcome these limitations. These novel targets might provide more effective and safer antiplatelet therapy, as well as modulate thrombo-inflammatory and microcirculatory pathways that are involved in atherosclerosis and myocardial ischaemia. Further research is needed to validate these novel targets and drugs in clinical trials and to establish their optimal use in personalized antiplatelet therapy.