Aims & Scope:

Cardiovascular diseases (CVD) currently account for nearly half of noncommunicable diseases (NCDs). Among the cardiovascular risk factors (CVRF) associated with CVD, some cannot be modified such as family history and age, while others, like high cholesterol, high blood pressure and diabetes, can be modified. Endothelial dysfunction stimulates increased platelet adhesion to endothelial cells, the first step in platelet activation. Platelets are nuclear pieces of megakaryocyte cytoplasm approximately 1.5-3 μm in diameter. The normal function of circulating platelets during the haemostatic process is to stop blood loss after a tissue trauma. Platelet activation leads to the release of pro-inflammatory, mitogenic and pro-apoptotic molecules, and cytotoxic agents, in addition to the interaction with leukocytes and endothelial cells. Then secreted products from activated platelets lead to the recruitment of further platelets into the growing aggregate. Thus, interaction of activated platelets with the dysfunctional endothelium is an important contributor to atherothrombosis and the development of CVD. Modulation of dysregulated endothelium and activated platelets is largely done so using prescribed pharmacological agents such as aspirin and glycoprotein inhibitors in the primary and secondary prevention of coronary events in high risk individuals. However, healthy people (middle aged and older), smokers and people under stress can also display impaired platelet and endothelial activities.

Alterations in platelet hemostatic properties, as well as their role in the immune response, may favor the expansion of damage besides well-known activities on the development of atherothrombotic plaques.

There exist different antiplatelet therapies, with significant platelets membrane and intracellular targets as well as final responses. In addition, combination of drugs therapies is also being applied with different success and under continuous discussion. Overall, these transform platelets in good targets for atherothrombotic drugs as well as for prevention or treatment of CVD.

- Analyze the role of platelets as target for the development of antithrombotic drugs
- Identify intracellular and membrane platelets’ targets to modulate platelet Aggregation
- Discuss the effects and development of drugs directed to platelets function

Schedule:

- Manuscript submission deadline: April 2020

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