

Platelets: The Balance between Aggregation and Bleeding

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Thromb Haemost 2019;119:1553.

Platelets are the soldiers of the blood. After they have been released from the megakaryocytes in the bone marrow, they are very well equipped for their tasks: initiation of blood coagulation when there is an interruption of the vessel wall together with the coagulation factors in the blood. However, they cannot differentiate between an interruption of vessel integrity (bleeding) or denudation of the endothelial layer of the vessel wall (e.g., plaque rupture). Much effort has been directed into understanding mechanisms and targets for antiplatelet drugs, and prognostic implications.^{1–3}

The good news is that nowadays we already have established medications that interfere with platelet adhesion and subsequent aggregation, and that we can measure platelet function *ex vivo*.⁴ The closed balance between aggregation on one hand and bleeding on the other is the focus of the theme issue *Platelets* that has been released today.

The first two papers^{5,6} describe platelet function testing in hemostasis in arterial and venous thrombosis: the first on total antithrombotic effects of combination antithrombotic effects in various cardiovascular conditions, and the second on the role of platelet micro-ribonucleic acids in platelet function in stable and unstable coronary artery disease.

Following these papers, clinical issues in antithrombotic therapy are highlighted. First, the role of antiplatelet therapy in primary prevention of cardiovascular disease in a healthy but high-risk growing population: the diabetics.⁷ Combining antiplatelet and anticoagulant therapy is the next topic, when dual or single antiplatelet therapy is combined in high-risk patients with stable coronary artery disease.⁸

Implantation of devices is now current practice in cardiology. Needless to say that the foreign body implants cause activation of the coagulation system including blood platelets. The current evidence is given in a paper on device implantation.⁹

Finally, the use of multiple antiplatelet agents may cause more bleeding than a single drug. The P2Y₁₂ receptor antagonists have found their way in several clinical thrombotic conditions, but they are associated with bleeding when used together with aspirin. Currently, there are reversal strategies

available to antagonize the action of P2Y₁₂ receptor antagonists. This will be discussed in the last chapter of this issue.¹⁰

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Conflict of Interest

F.W.A.V. reports personal fees from AstraZeneca, during the conduct of the study.

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received

August 14, 2019

accepted

August 14, 2019

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Stuttgart · New York

DOI <https://doi.org/>

10.1055/s-0039-1696984.

ISSN 0340-6245.