

Monocyte–Platelet Complexes in Myocardial Infarction: Sub-Sets and Platelet-Derived Microvesicles Matter

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Thromb Haemost 2018;118:1854–1855.

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Atherosclerosis is a chronic lipid-driven inflammatory disease of the arterial wall characterized by the involvement of the innate and adaptive immune system.^{1–3} Low-density lipoproteins enhance a series of pro-inflammatory reactions perpetuating the activation, recruitment and transmigration of different innate immune cells (monocytes, mast cells, neutrophils, natural killer cells and dendritic cells). Although the contribution of circulating monocytes is essential, acquired immunity, mainly performed by T cells (Th1- and Th2), is also critically involved in atherosclerosis lesion progression. Monocytes and macrophages are very versatile, and depending on the local micro-environment they can assume different phenotypes and functional characteristics, a termed referred to as ‘polarization’ (a reversible process).^{4,5}

Platelets are released into the circulation as cytoplasmic fragments of bone marrow megakaryocytes and circulate in the blood stream for 7 to 10 days without interacting with other blood elements and/or the vascular wall. Upon endothelial injury or rupture of an atherosclerotic plaque, platelets become activated and anchor on the damage vessel wall.⁶ Rapid platelet recruitment induces thrombosis and target organ ischaemia. In the heart, atherothrombosis induce acute coronary syndromes (ACSs). The initial platelet tethering is mainly mediated by the glycoprotein Ib alpha (GPIb α) receptor (the main binding region of GPIb/IX/V platelet complex) that also contains binding sites for leukocyte integrin macrophage-1 antigen and P-selectin, which favours further platelets and leukocyte recruitment.⁶ Activated monocytes can form complexes with activated platelets by specific interaction of P-selectin on activated platelets with P-selectin glycoprotein ligand-1, which is expressed by monocytes and further stabilized by additional integrin adhesion between these cells. In 1991, the dynamics of leukocyte–platelet adhesion and platelet–platelet interaction in the whole blood were first described and since then

the impact of these interactions in ACS has been analysed in different studies.⁷ More recently, increased platelet–monocyte counts have been associated with different monocyte sub-sets and levels of activation. In this issue, an interesting paper by Elena Vasilieva’s group investigated not only platelet–monocyte and monocyte sub-set interactions in acute myocardial infarction but also how platelet-derived microvesicles participate in these interactions.⁸ Indeed, platelet-derived extracellular vesicles have been demonstrated to contribute to thrombosis and be markers of active disease in different types of patients even when patients were treated as per guidelines.^{9,10}

Conflict of Interest

None.

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received

October 4, 2018

accepted

October 4, 2018

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Stuttgart · New York

DOI <https://doi.org/10.1055/s-0038-1675242>.
ISSN 0340-6245.

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