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Stem Cell Transplantation, immunotherapy and cell therapy

NON-CANONICAL COAGULATION PLATELETS FUNCTION: MONOCYTE-PLATELET INTERACTION A BRIDGE BETWEEN INFLAMMATION AND COAGULATION

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Introduction: Platelet activation leads to the release of several molecules with immunoregulatory functions. While these factors play a limited role in thrombosis, they have a significant impact on modulating host immune responses. Monocytes recognize microbial components and respond by producing different pro- and anti- inflammatory cytokines and chemokines. Importantly, their interaction with platelets can induced an anti-inflammatory phenotype. Studies have shown that activated platelets bind to monocytes predominantly via P-selectin and subsequently trigger downstream signalling pathways that alter monocyte phenotype. In the present study we aimed to assess platelets-monocytes interaction, as well as activity of platelets and monocytes, in patients with different bacterial infection and in healthy controls.

Methods: Ten patients with various bacterial infections and ten healthy control subjects were enrolled. CytoFLEX SRT cytometry was performed on whole blood using the following antibodies CD41-PC7 and CD62P-selectin-PE (CD62P) to identify platelets; CD14-APC to identify monocytes; and the tetra-panel (CD45-FITC/CD4-RD1/CD8-ECD/CD3-PC5) to identify lymphocytes. Monocyte were identified on side scatter properties and positive CD14. Platelet–monocyte aggregates (MPAs) were assessed by double CD62P and CD14 positivity. Platelet activation were evaluated by expression of P-selectin and CD41. Immunofluorescence microscopy was performed on whole blood smear using CD41-FITC, CD62P-Alexa Fluor 647, and CD14-Alexa Fluor 594 antibodies.

Results: Whole blood CytoFLEX SRT proved to be an effective method for distinguishing MPAs of varying sizes from free circulating platelets. The level of MPAs showed a significant positive correlation with surface expression of P-selectin and was significantly lower in patients compared to healthy controls ($p < 0.001$). Activated platelets were higher in patients than in controls ($p < 0.001$).

Conclusions: Preliminary results obtained suggest that increased levels of MPAs could have a potential pro-inflammatory role and could be a valuable biomarker to assess inflammatory status, although the patient cohort is limited. Promoting the formation of MPAs, it may be possible to induce a shift in monocytes toward an anti-inflammatory phenotype, contributing to the long-term resolution of inflammation. Moreover, given the high number of activated platelets in patients, potential pro-thrombotic risks should be further investigated. Finally, further studies of monocyte characterization could help more to identify the monocyte platelets interactions and their function. Understanding the regulatory mechanisms of the platelet–monocyte network could provide the basis for the development of new drugs and patient-specific therapeutic approaches.