Insight Platelets promote lymphocyte adhesion to extracellular matrix components under arterial flow conditions......

Platelets guide lymphocytes to vascular injury sites

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In this issue of *Thrombosis and Haemostasis*, Spectre et al. (1) demonstrate that (activated) platelets promote lymphocyte adhesion to extracellular matrix components under arterial flow conditions, thereby further connecting thrombotic processes to inflammation and immunity.

At sites of vascular injury, platelets adhere and aggregate on exposed subendothelial extracellular matrix (ECM) components and thereby form a plug that seals the wound or eventually occludes a diseased vessel, e.g. at sites of atherosclerotic plaque rupture (2). Besides this essential role in haemostasis and thrombosis, platelets are increasingly recognised to also orchestrate inflammatory processes, a combination of events best described by the term “thrombo-inflammation” (3).

Several studies have shown that platelets can directly interact with lymphocytes and that this interaction is important for the modulation of adaptive immunity (3). However, lymphocytes are also abundantly found in atherosclerotic plaques and it is not clear whether they are recruited there by direct interactions with exposed plaque ECM components or through a platelet-dependent mechanism. Spectre and co-workers now show in a series of elegant experiments that platelets are essentially required to support lymphocyte adhesion to different ECM components under arterial flow conditions *ex vivo* (500 s⁻¹). Interestingly, the nature of the adhesive substrate largely determined the ability of the attached platelets to recruit specific lymphocyte subsets. For example, while platelets attached to collagen or fibrinogen, but not von Willebrand factor (vWF) efficiently recruited CD4+ and CD8+ T cells, all three substrates primed platelets to recruit NK cells in this system. The authors further provide evidence that the differential expression of adhesion molecules, most notably PSGL1 (CD162) and Mac-1, on different lymphocyte subpopulations, which interact with P-selectin and GPIb on platelets, respectively, may contribute to this selectivity, but further studies will be required to address this in more detail.

Together, these observations provide a possible mechanism how platelets promote chronic inflammatory diseases such as atherogenesis, and may also serve as a basis for a better understanding of acute “thrombo-inflammatory” disease states, such as ischaemic stroke where platelets and T cells have been identified as essential pathogenic factors but their mechanistic link has not been identified (3).

Conflicts of interest
None declared.

References
