

Novel hydrophilic stent coating inhibits platelet adhesion in vitro

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Purpose: Platelets react to foreign body surfaces and induce blood coagulation. Therefore, during neurovascular interventions with implantation of stents and flow diverters, the standard of care includes the dual antiplatelet therapy (DAPT) with ASA and clopidogrel, followed by antiplatelet monotherapy with ASA indefinitely. Although the risk for stent thrombosis is reduced, the risk for hemorrhagic complications increases and there are patients, for which DAPT is not suitable at all. A potential solution for this problem is the application of an antithrombogenic coating on the device surface. Such a coating should reduce the thrombogenicity of the implant itself, hence making DAPT obsolete. The purpose of this study was to evaluate the antithrombogenic effect of two hydrophilic stent coatings.

Materials and Methods: Due to their highly complex geometry, analysis of platelet adhesion on stents is challenging. Therefore, two different hydrophilic coatings (HPC-I + HPC-II) were applied on NiTi plates. Platelet adhesion was examined during whole blood contact *in vitro*. Uncoated NiTi plates served as control. In detail, SEM analysis, CD61 immunofluorescence microscopy and phase analysis was performed.

Results: The uncoated (bare) and the HPC-I coated specimens were completely covered with adherent CD61 positive cells, whereas HPC-II coated specimens exhibited a significant antithrombogenic effect. On the HPC-II coated specimens only very few CD61 positive cells were detected (Fig. 1). This effect was true for all blood donors. Quantitative Phase Analyses revealed a highly significant difference in the "platelet coated area" on the bare ($48.61 \pm 7.3\%$), the HPC-I ($40.19 \pm 8.9\%$) compared to the HPC-II coated ($1.12 \pm 0.4\%$) specimens (Mean \pm SE; Fig. 1).

Conclusion: A new hydrophilic polymer coating was tested, which inhibits platelet adhesion *in vivo* significantly. The HPC-II coating – when applied on a flow diverter stent – could reduce the thrombogenicity of the implant itself, hence making DAPT obsolete.

Figure 1: Representative fluorescence micrographs of uncoated (bare) and differently coated (HPC-I and HPC-II) NiTi specimens. The specimens were incubated in whole blood for 10 min under dynamic conditions. Adherent platelets were stained with a CD61 antibody (yellow). Bottom right: Quantitative Phase Analysis of the area coated with CD61 positive cells (Mean \pm SE, asterisks denote significance at $p \leq 0.05$; *** = $p \leq 0.001$; Kruskal-Wallis and DUNN Posttest; $n = 15$).

Figure 1:

