LOCAL INTRACORONARY ROSIGLITAZONE TREATMENT INDUCES A VASOPROTECTIVE GENE EXPRESSION PROFILE AND COMPLETE REENDOTHELIALIZATION IN A PORCINE IN STENT-RESTENOSIS MODEL

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Background: Implantation of Drug-eluting stents (DES) is associated with delayed reendothelialization and endothelial dysfunction and thus may contribute to in stent-restenosis and late stent thrombosis. Rosiglitazone activates „PPARgamma“, a transcription factor involved in the control of inflammation, apoptosis and cell proliferation. We hypothesized that local rosiglitazone treatment using coated stents (RDES) improves reendothelialization by different gene expression of vasoactive and vasoprotective genes, regulating vascular tone, growth and endothelial function in comparison to Bare Metal Stent (BMS).

Methods: In 5 pigs, RDES and BMS were randomly implanted in coronary arteries with an overexpansion (balloon/stent to artery ratio 1.3-1.5:1) in order to disrupt the endothelial layer and to induce inflammation and neointimal hyperplasia. The expression of vasoactive and vasoprotective genes (eNOS, C-natriuretic Peptide (CNP), adiponectin, endothelin-1) were evaluated after 3 months by quantitative real time RT-PCR. Additionally, endothelial expression of CD31 was analyzed by immunohistochemistry.

Results:

Conclusions: 3 month after implantation of RDES not only reendothelization was complete, but also the endothelium appears functional, supported by a beneficial vascular gene expression profile in comparison to BMS. Intracoronary rosiglitazone treatment therefore may prevent in stent-restenosis and late stent thrombosis.