ATTENUATED VASOCONSTRICTOR RESPONSES TO CORONARY ARTERIAL ASPIRATE FROM PATIENTS WITH PACLITAXEL ELUTING STENT VERSUS BARE METAL STENT IMPLANTATION FOR SAPHENOUS VEIN AORTO-CORONARY BYPASS STENOSIS

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Background: Implantation of bare metal stents (BMS) induces the release not only of particulate debris, but also of soluble vasoconstrictors which contribute to microvascular impairment. We have now addressed the potential attenuation of such vasoconstriction by use of paclitaxel eluting stents (PES).

Methods: Using a distal protection/aspiration device, coronary arterial blood was retrieved before and during stent (n = 14 BMS, n = 14 PES) implantation in patients with saphenous vein aorto-coronary bypass stenosis and analyzed for plasma serotonin and thromboxane B2 concentrations. Vasoconstriction of rat mesenteric arteries with intact (+E) and denuded (-E) endothelium in response to coronary arterial or aspirate plasma was quantified and normalized to that by potassium chloride (KClmax=100%).

Results: Coronary arterial plasma before stent implantation induced a vasoconstriction of 30-43% of KClmax, which was independent of endothelial integrity. Serotonin-release was 2.2±0.5 μmol/l with BMS and 2.0±0.4 μmol/l with PES, thromboxane B2-release was 26±5 pg/ml with BMS and 22±8 pg/ml with PES. BMS-aspirate plasma induced a vasoconstriction of 68±18% (+E) or 93±14%(-E), respectively. In contrast, PES-aspirate plasma induced only minor vasoconstriction (+E: 8±3, -E: 12±5% of KClmax). Addition of paclitaxel to BMS-aspirate plasma attenuated vasoconstriction. PES-aspirate induced microtubular condensation in immunofluorescence microscopy.

Conclusion: Aspirate from PES implantation attenuates vasoconstriction, possibly secondary to microtubular stabilization.