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Vascular Function of Bioabsorbable Stented Site after Complete Absorption of the Stent

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Background: Drug eluting bioabsorbable stents (DEBSs) represent a new device based therapy for CAD. It has been reported that stented segment becomes reendothelialized after 1 mo and the segments proximal and distal to the stented site are functional at 2 years. In order to examine whether after complete degradation, the DEBS site function was similar to the unstented segments, we performed ex vivo vasomotor function studies using pig coronary arteries.

Methods: Eighteen months after implantation, 10 DEBS sites were assessed for vasomotor function using an organ chamber apparatus. They were stimulated with potassium chloride (KCl), prostaglandin_{2ε} (PGF) and 3 concentrations of endothelin-1 (ET). Endothelium-dependant relaxation (EDR) to substance P (SbP; 0.01 - 100 pM) and endothelium-independent relaxation (EIDR) to sodium nitroprusside (SNP; 0.001 - 10 μM) were assessed following constriction with PGF. Remaining stent segments were fixed for histologic examination.

Results: DEBS sites showed rapid response to low and high concentrations of KCl, PGF and ET. EIDR showed concentration-dependent relaxation to SNP (13.3±4.3%, 21.3±5.6%, 52.7±7.1%, 85.5±5.4% and 100±0%). However, there was no EDR to SbP concentration-dependent stimulation. HE and VM staining showed evidence of SMCs migration across polymer struts and formation of a new abluminal layer was observed. There was complete polymer strut degradation, infiltration of inflammatory cells and minor fibrosis around some DEBS sites. Myocardial degeneration was found in the septum adjacent to the stented sites. Vessel wall within the stented segment was thicker and the lumen was narrower than in the proximal and distal segments.

Conclusion: We have demonstrated for the first time the ex vivo contraction and relaxation responses at DEBS sites to vasoactive agents after complete degradation of the stent. SMCs recovered contractile and relaxing capabilities in this segment but endothelial function was still impaired.