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flow in the popliteal artery of patients with peripheral vascular disease (PVD). IPC promotes local vasodilatation and arteriogenesis.

Methods: The aim is to investigate whether it has a systemic remote effect on distant arteries and this effect is mediated through circulating nitric oxide. Fifteen patients with PVD, mean age 73.8 with range (61-84), ABPI <0.9, superficial femoral artery stenosis $>50\%$, and 15 healthy volunteers, mean age 57 range (37-75) were randomised for forearm hyperaemia IPC for an hour and sublingual glyceryl trinitrate (S/L GTN). 15 healthy volunteers were controls. Using duplex ultrasound, the BA diameter was measured at 1-minute post-hyperaemia, 30 and 60 minutes during pump use. At 16 minutes post-pump cessation, 2 puffs S/L GTN were administered. Venous blood sample collected at baseline, 5 minutes and 30 minutes of the pump use to be analysed for nitric oxide.

Results: The percentage change of BA diameter among 30 candidates at 1 min post-hyperaemia was 3.3% ($p < 0.05$) (Wilcoxon), and at 30 and 60 minutes of IPC was 1.5% ($p < 0.05$) and 3% ($p < 0.05$) respectively. The response to GTN was 17.1% ($p < 0.05$). The difference between the controls and other groups was statistically significant. The nitric oxide level in patients group did increase significantly at 30 minutes $p = 0.028$ while healthy volunteers the level remained steady.

Conclusion: IPC produces a significant dilatation of the BA and has thus a systemic effect on the arterial system. This is a novel finding. This effect is mediated via nitric oxide released from lower extremity vessels as a result of their exposure to the increased shear stress generated through the IPC.

Radio Protective RP105 Protects against Vein Graft Disease and Lesion Stability Via Dampening of Inflammatory Responses

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Introduction: Vein grafts are often used to bypass atherosclerotic lesions; however, patency rates are troublesome due to the development of vein graft disease. Deficiency of toll like receptor (TLR)4, a key initiator of inflammatory signalling, results in reduced vein graft disease. As TLR4 signalling is regulated by the accessory molecule Radio-Protective 105 (RP105), we aimed to investigate the effects of RP105 on vein graft disease.

Methods: Vein graft surgery was performed on Rp105−/− mice ($n = 13$) and C57BL/6 mice ($n = 11$), as well as on Ldlr−/−/Rp105−/− mice ($n = 11$) and Ldlr−/− mice ($n = 11$) fed a western type diet, 28 days later lesion size and composition was analysed. Furthermore, in vitro experiments on smooth muscle cells and mast cells were performed.

Results: A 90% increase in vein graft lesion size was observed in Rp105−/− mice. Lesion size did not differ between Ldlr−/−/Rp105−/− mice and Ldlr−/− mice, but interestingly, we detected a significant increase in the number of unstable lesions and intraplaque haemorrhage upon RP105 deficiency. In both experimental setups, an increase in lesional macrophages was seen. Peritoneal Rp105−/− macrophages showed an increase in proliferation. Rp105−/− smooth muscle cells and bone marrow derived mast cells secreted increased levels of the monocyte chemoattractant CCL2. In both the Rp105−/− and Ldlr−/−/Rp105−/− vein grafts the amount of lesional CCL2 was significantly increased, as well as the number of activated perivascular mast cells.

Conclusion: Together, these data indicate that RP105 has a protective role in vein graft disease by dampening the inflammatory effect, since RP105 deficiency results in an increased inflammatory response and exacerbated CCL2 production by both mast cells and smooth muscle cells.

Morphological and Stent Design Risks Factors to Prevent Migration Phenomena and Type 1a Endoleak for Thoracic Aneurysm: A Numerical Analysis

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Introduction: The primary mechanically related problems of endovascular aneurysm repair are migration and type Ia endoleaks. They occur when there is no effective seal between the proximal end of the stent-graft and the vessel. In this work, we have developed several deployment simulations of stent parameters using the finite element method (FEM) to investigate the contact stiffness of a nitinol stent in a realistic Thoracic Aortic Aneurysm (TAA).

Methods: The following factors associated with these complications were evaluated: (1) Proximal Attachment Site Length (PASL), (2) stent over-sizing value (O%), (3) different friction conditions of the stent/aorta contact, and (4) proximal neck angulation. Then, the numerical observations are used as a guide to optimize the stent design in such neck morphology to strengthen the contact and prevent migration or endoleak type Ia.

Results: The simulation results show that PASL >18 mm is a crucial factor to prevent migration at a neck angle of 60°, and the smoothest contact condition with low friction coefficient ($\mu = 0.05$). The increase in O% ranging from 10% to 20% improved the fixation strength. However, O% $\geq 25\%$ at 60° caused eccentric deformation and stent collapse. Higher coefficient of friction $\mu > 0.01$ considerably increased the migration risk when PASL $= 18$ mm. No migration was found in an idealized aorta model with a neck angle of 0°, PASL $= 18$ mm and $\mu = 0.05$.

The optimized stent results showed better contact stability to resist the migration. They also showed a good compromise of stent design requirements (flexibility and stiffness). Moreover, the new design can also prevent the risk of folding or collapse of stent struts by mitigating the energy of eccentric deformation caused by high angulation and oversizing.