Flow (shear stress)-mediated remodeling of resistance arteries in diabetes

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Résumé en anglais
Shear stress due to blood flow is the most important force stimulating vascular endothelium. Acute stimulation of the endothelium by shear stress induces a vasodilatation mainly due to the release of nitric oxide (NO) among other relaxing agents. After a chronic increase in blood flow (shear stress), the endothelium triggers diameter enlargement, medial hypertrophy and improvement of arterial contractility and endothelium-mediated dilation. Shear stress-mediated outward remodeling requires an initial inflammatory response followed by the production of reactive oxygen species (ROS) and peroxinitrite anions, which activate MMPs and extracellular matrix digestion allowing diameter expansion. This outward remodeling occurs in collateral growth following occlusion of a large artery. In diabetes, an excessive ROS production is associated with the formation of advanced glycation end-products (AGEs) and the glycation of enzymes involved in vascular tone. The balance between inflammation, AGEs and ROS level determines the ability of resistance arteries to develop outward remodeling whereas AGEs and ROS contribute to decrease endothelium-mediated dilation in remodeled vessels. This review explores the interaction between ROS, AGEs and the endothelium in shear stress-mediated outward remodeling of resistance arteries in diabetes. Restoring or maintaining this remodeling is essential for an efficient blood flow in distal organs.

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