Understanding adverse blood vessel remodeling following stenting

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Atherosclerosis is a leading cause of heart attacks and stroke. The narrowing of blood vessels that is caused by atherosclerosis can be treated with angioplasty or stenting to improve blood flow. However, the stenting process induces deleterious remodeling of the blood vessel that can increase thrombosis risk, limiting the use of this strategy.

In an article published in the *Journal of Clinical Investigation*, a research team led by Ziad Ali of the Columbia University Medical Center now provides new insights into the pathological remodeling that occurs following blood vessel stenting.

In patients, they show that a gene network regulated by the antioxidant enzyme glutathione peroxidase-1 (GPX1) was downregulated in atherosclerotic plaques and a gene variant in the receptor tyrosine kinase ROS1 was associated with adverse effects of stenting. In a mouse model, loss of the antioxidant GPX1 promoted both oxidative and reductive stress, which in turn led to elevated ROS1 activity.

Their study suggests ROS1 merits further exploration as a therapeutic target to improve the treatment of flow-limiting atherosclerosis.

More information: Oxido-reductive regulation of vascular remodeling by receptor tyrosine kinase ROS1, *J Clin Invest.* 2014;124(12):5159–5174. DOI: 10.1172/JCI77484