As a key mediator of vascular and cardiac maladaptive remodeling, mineralocorticoid receptor (MR) plays a pivotal role in vascular fibrosis and intimal hyperplasia (IH) and is potentiated locally in hemodialysis vascular access following diverse injuries, like barotrauma, cannulation and shear stress" Chen et al (2018).

Abstract:

Hemodialysis vascular access dysfunction is a common and intractable problem in clinical practice with no definitive therapy yet available. As a key mediator of vascular and cardiac maladaptive remodeling, mineralocorticoid receptor (MR) plays a pivotal role in vascular fibrosis and intimal hyperplasia (IH) and is potentiated locally in hemodialysis vascular access following diverse injuries, like barotrauma, cannulation and shear stress. MR-related genomic and non-genomic pathways are responsible for triggering vascular smooth muscle cell activation, proliferation, migration and extracellular matrix overproduction. In endothelial cells, MR signaling diminishes nitric oxide production and its bioavailability, but amplifies reactive oxygen species, leading to an inflammatory state. Moreover, MR favors macrophage polarization towards a pro-inflammatory phenotype. In clinical settings like post-angioplasty or stenting restenosis, the beneficial effect of MR antagonists on vascular fibrosis and IH has been validated. In aggregate, therapeutic targeting of MR may provide a new avenue to prevent hemodialysis vascular access dysfunction.

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Reference:
Chen, B., Wang, P., Brem, A., Dworkin, L., Liu, Z. and Gong, R. (2018) Mineralocorticoid receptor: A hidden culprit for hemodialysis vascular access dysfunction. EBioMedicine. December 5th. .

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