CONFERENCE ABSTRACT
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MONOAMINE OXIDASES CONTRIBUTE TO ENDOTHELIAL DYSFUNCTION OF THE VASCULAR ACCESS IN HEMODIALYSIS PATIENTS

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OBJECTIVES

Arteriovenous fistulas (AVF) are the 'lifeline' for patients with terminal end-stage renal disease (ESRD) on chronic hemodialysis. AVF maturation failure is a poorly understood process, and is partially caused by endothelial dysfunction due to oxidative stress. Monoamine Oxidases (MAOs) A and B were recently identified as novel sources of vascular oxidative stress.

AIM

To assess the contribution of MAOs to endothelial dysfunction in patients with ESDR with indication of hemodialysis.

MATERIALS AND METHODS

Fragments taken from brachial artery collaterals were harvested from ESRD patients during the surgical procedure that was applied in order to create the AVF in the cubital fossa. The effect of the irreversible MAO-A inhibitor clorgyline (10 μmol/L) and irreversible MAO-B inhibitor selegyline (10 μmol/L) on endothelial-dependent relaxation (EDR) in response to cumulative doses of acetylcholine was studied in isolated phenylephrine-preconstricted rings in the presence of diclofenac (10μmol/L). H2O2 production was analyzed using ferrous oxidation xylenol orange assay. MAO expression was assessed by quantitative PCR.

RESULTS

We showed that both MAO isoforms are expressed in the brachial artery collaterals. Incubation with MAO inhibitors significantly improved EDR and attenuated H2O2 generation in the vascular segments.

CONCLUSIONS

MAO-related oxidative stress might contribute to the primary dysfunction/lack of maturation of the AVF and MAO inhibitors could improve vascular access maturation and longevity in dialysis patients.