Thromboses and stenoses of permanent vascular access appear to be a serious hazard for patients with end-stage kidney disease on programmed haemodialysis” Kalinin et al (2019).

Abstract:

Thromboses and stenoses of permanent vascular access appear to be a serious hazard for patients with end-stage kidney disease on programmed haemodialysis. Relapses of these pathological conditions are the cause of repeated hospitalization, secondary surgical interventions and may eventually lead to impossibility of carrying out procedures of haemodialysis. Often, vascular access dysfunction occurs for no apparent reason, thus underlying the importance of studies aimed at revealing additional factors of intravascular thrombogenesis and neointimal formation in a vascular access, including the works dedicated to studying genetic predictors of the development of the above-mentioned complications. The authors examined herein the role of polymorphisms of the genes of endothelin-1 (END-1), nitric oxide synthase-3 (NOS-3), angiotensinogen-2 (AGT-2), angiotensinogen-1 (AGT-1), angiotensinogen 2 receptor type 1 (AGTR1), mitochondrial superoxide dismutase-2 (SOD-2), catalase (CAT) superoxide dismutase-1 (SOD-1) and angiotensin converting enzyme (ACE) in the functional state of permanent vascular access in patients on dialysis. The obtained results demonstrated direct cause-and-effect relationships between polymorphisms lys-198 asn in the END-1 gene, C60T, T58C in the SOD-2 gene and the function of vascular access. The presence of END-1 gene lys-198 asn polymorphism in a homozygous state (allele 1) was associated with a high risk of an unsatisfactory condition of permanent vascular access (p=0.019).

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Genetics and functionality of vascular access devices in dialysis | 2