The contribution of vascular endothelium to the progression of chronic kidney disease

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Abstract

Although historically endothelial contribution to chronic kidney disease (CKD) had been neglected, recent investigations provide conclusive evidence of its role in maintaining tissue homeostasis, supporting tissue regeneration and, when dysfunctional, instigating development and progression of tissue fibrosis. These findings are of critical importance for the development of nephrosclerosis and progression of CKD. Three endothelial pathways involved in the progression of CKD include stress-induced premature senescence of endothelial cells, endothelial-mesenchymal transition, and the loss of the endothelial surface layer. These abnormalities orchestrate the development of proteinuria, pro-inflammatory microenvironment, microvascular rarefaction, pro-fibrotic state, and regenerative failure. Therapeutic strategies to overcome endothelial cell dysfunction and its renal consequences will be discussed.

Biography:

Michael S Goligorsky, MD, PhD, holds the Alvin I Goodman Chair in Nephrology and is Professor of Medicine, Pharmacology and Physiology, Academic Chief of Renal Division, and Director of Renal Research Institute at the New York Medical College. After completing residency and fellowship, he joined the faculty of the State University of New York at Stony Brook (1988). He became a Professor of Medicine and Physiology in 1997 and named an Honorary Professor at the University College London (1998). In 2002 he was recruited by the New York Medical College to inaugurate Renal Research Institute. In 1991, he was elected to the American Society of Clinical Investigations; in 2002 elected to the American Association of Physicians. MSG serves as an Associate Editor for Am J Pathology, Am J Physiology: Cell and a Topic Editor for Nephrology, Dialysis, and Transplantation. His research interests include: the mechanisms of endothelial dysfunction as a harbinger of atherosclerotic, diabetic, and hypertensive vascular damage; stress-induced premature senescence (SIPS) of endothelial cells and the role of lysosomal dysfunction in this process; mechanisms of functional incompetence of endothelial progenitor cells (EPC) in chronic kidney disease; mechanisms of Alarm Signaling by an ischemic organ; and proteomic analysis of the urine in kidney disease.