

Vascular Endothelial Dysfunction and Diabetic Nephropathy: Never Solves a Problem without Creating Dozens More

Atul Arya, Lakhwinder Singh, Surbhi Rana, Sumeet Gupta and Sidharth Mehan
Punjab Technical University, India

Vascular Endothelial dysfunction (VED) has been encountered to play a central role in the pathogenesis of diabetic nephropathy (DN). Hyperglycemia, altered glucose, fat and protein metabolism lead to vascular endothelial dysfunction. VED results in reduced generation of nitric oxide (NO) demanded for free flow of blood in vessels and increased generation of free radicals mainly reactive oxygen species (ROS). Reduced nitric oxide exacerbates oxidative stress, further promoting endothelial dysfunction. VED has been associated in pathogenesis of hypertension, diabetes mellitus and nephropathy. Long term complication of diabetes mellitus includes retinopathy, neuropathy, cardiomyopathy, nephropathy and dozens more. Based on the clinical evidences showing a relation between endothelial nitric oxide synthase (eNOS) dysfunction and diabetes to promote advanced diabetic nephropathy. VED has been shown to be involved in diabetic nephropathy by inducing glomerular hypertrophy, accumulation of extracellular matrix protein, increased basement membrane thickness, mesangial cell expansion, podocyte loss progressively leading to glomerulosclerosis, tubulointerstitial fibrosis and proteinuria, which ultimately decline glomerular filtration rate (GFR). Thus it is suggested that diabetes-induced VED could be one of the culprits involved in the pathogenesis of diabetic nephropathy.

Biography

Atul Arya is pursuing PhD in Pharmaceuticals Sciences from Punjab Technical University, Punjab, India. He had published more than 12 papers in reputed journals.

atularya07@gmail.com

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