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LETTER TO THE EDITOR

A Deficient VEGF Enhances Endothelial Cell Loss and Impaired Renal Function

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Keywords VEGF, circulating endothelial cell, renal microvascular disease, hemodynamics, vasodilators

Dear Sir,

We as well as others have noted that a significant number of patients have impaired renal function without any previous known risk factors or precedent history of renal disease. All of the 11 patients observed during the past few years showed a significant impairment of renal function with a mean value of glomerular filtration rate of 69 ± 22 (vs. control 116 ± 15 mL/min/1.73 m^2; $p < 0.01$) and a mean value of fractional excretion of magnesium (FE Mg) of 7.6 ±10 (vs. control 1.6 ± 0.2%; $p<0.09$). Such renal functional impairment was associated with intrarenal hemodynamic abnormalities, namely, reductions in renal plasma flow (337 ± 85 vs. control 567 ± 50 mL/min/1.73 m^2; $p < 0.001$) and peritubular capillary flow (268 + 64 vs. control 450 ± 43 mL/min/1.73 m^2; $p < 0.001$). Indeed, the reduction in peritubular capillary flow reflects glomerular endothelial dysfunction, which has recently been demonstrated to be the crucial determinant of tubulointerstitial fibrosis or renal disease progression.[1]

The dysfunctioning glomerular endothelium commonly encountered in chronic kidney disease patients is usually induced by circulating toxins such as oxidative stress or proinflammatory cytokines.[2] It is interesting to observe greater endothelial cell loss, which is reflected by an increased number of circulating endothelial cells (mean 2286 ± 1506 vs. control 60 ± 66 cells/mL). Concomitant to this notion, all eleven patients also expressed a very low value of vascular endothelial growth factor (VEGF) (mean 36 ± 35 vs. control 355 ± 167 pg/mL). Because VEGF is an essential growth factor for endothelial cell growth and proliferation, the deficient VEGF would impede the reparative process of vascular injury and therefore promote the advancement of vascular pathology. A VEGF deficiency in conjunction with enhanced circulating endothelial cell loss would likely explain the undulant clinical manifestation of renal microvascular disease associated with renal functional impairment in this particular group of patients. A restoration or stabilization of renal function can be achieved by correcting the hemodynamic maladjustment with multidrug vasodilators.[3]

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