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# 내피전구세포 투여가 만성 신부전 진행에 미치는 영향

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# The Protective Role of Endothelial Progenitor Cells in the Progression of Chronic Renal Failure

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Recently, endothelial progenitor cells (EPCs) have been focused for their capacity to repair damaged endothelial system. Chronic renal failure (CRF) is characterized by glomerulosclerosis, tubulointerstitial fibrosis and vasculosclerosis, which are associated with damaged endothelial cells and impaired angiogenesis. Although many studies have been made to elucidate the role of EPCs on re-endothelialization and angiogenesis, the role of EPCs on the progression of CRF has not been answered thoroughly. EPCs are precursor cells for revascularization which exist in bone marrow, peripheral blood and umbilical cord blood. In this study we evaluated the beneficial role of EPCs using the murine CRF model. CRF was induced in C57BL/6 (B6) and NOD-SCID mice utilizing 5/6 nephrectomy. EPCs were isolated from bone marrow of mice and human umbilical cord blood and were cultured by selective media. Progenitor cells were defined as demonstrating the expression of endothelial cell markers such as CD34, CD31 Sca-1, eNOS and KDR-2. The serial changes of renal function and histological features were monitored in every 4 weeks till 20 weeks after disease induction with/without EPCs injection. In CRF mice, the level of serum BUN increased steadily over time and creatinine was higher as compared with age matched control mice. CRF mice that were adoptively transferred with bone marrow derived BM-EPCs and human cord blood derived endothelial progenitor cells (hUCB-EPCs) revealed less deterioration of renal function as well as less severe histologic changes compared to those of disease control mice. Urine protein excretion in B6 mice markedly increased after 5/6 nephrectomy till 20 weeks of disease, but BM-EPC injection decreased the amount of proteinuria down to normal control. After the adoptive transfer of BM-EPCs, CD31 and vWF positive cells were well preserved in glomeruli. The expressions of proinflammatory cytokines and adhesion molecules were also down-regulated with the injection of BM-EPCs. In conclusion, we have shown that BM-EPCs and hUCB-EPCs infusion after the induction of CRF was able to protect the progression of kidney disease through the preservation of healthy endothelial structures in kidney and less inflammation at the site of damage.

**Key Words :** 만성 신부전, 내피전구세표, 신기능 보호
CRF, EPC, Renoprotection