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일측요관폐쇄 백서모델에서 Erythropoietin의 효과

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Erythropoietin Attenuates Renal Injury in an Experimental Unilateral Ureteral Obstructed Rat Model

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Introduction : Erythropoietin (EPO) has recently been shown to exert important cytoprotective and antiapoptotic effects in ischemic brain injury, infarted heart, CyclosporinA (CsA)-induced nephrotoxicity and IR renal injury. We examined that EPO attenuates also renal injury in the Unilateral Ureteral Obstructed (UUO) rat model by anti-apoptotic and anti- inflammatory actions.

Methods : We divided Sprague-Dawley (SD) rats into four groups as sham group, UUO for 3 day group, EPO-treated sham group and EPO-treated UUO for 3 day group. EPO treatment dose was 3,000 U/Kg/day and it was administrated daily from 2 days before operation to post-operation 3day via intraperitoneal route. We compared the datas from competitive RT-PCR for TGF-β, TNF-α, MCP-1, osteopontin (OPN), Fas and Bcl-2, Western-blotting for caspase 3, light microscopic finding with H&E staining, and immunohistochemistry of TGF-β, ED-1 and caspase 3 of each groups.

Results : The mRNA levels of TGF-β, TNF-α, MCP-1, osteopontin and Fas of the EPO-treated UUO group showed a significant decrease than the UUO group ($p<0.05$).The mRNA level of Bcl-2 in the EPO-treated UUO group showed a significant increase than the UUO group ($p<0.05$). The activity of active caspase 3 in the EPO-UUO groupshowed a significant decrease than the UUO group ($p<0.01$). Light microscopically, interstitially infiltrated inflammatory cells and myofibroblasts were significant decreased in the EPO-treated UUO group than the UUO group ($p<0.05$). In immunohistochemistry, the EPO-treated UUO group showed significantlydecreased reactions for TGF-β, ED-1, and caspase-3 than the UUO group ($p<0.05$).

Conclusion : EPO exerts renal protective effects in anexperimentally UUO rat model by anti-apoptotic action via suppression of Fas gene expression, activation of Bcl-2 gene expression and inhibition of caspase-3 activity and by anti-inflammatory action via suppression of TGF-β, TNF-α, MCP-1 and OPN gene expression and inhibition of cellular TGF-β and ED-1 production, not by hematopoietic benefits in vivo.