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실험적 초승달토리 콩팥염의 발현에 재조합 Uteroglobulin의 Transglutaminase 억제를 통한 예방 효과

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The Protective Role of Uteroglobin through the Modulation of Tissue Transglutaminase in the Experimental Crescentic Glomerulonephritis

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Backgroud and methods : Tissue transglutaminase (tTG) may induce pro- inflammatory cytokines and produce irreversible end- products, thus promoting renal scarring. It has recently been confirmed that the crescent formation in murine experimental crescentic glomerulonephritis (ecGN) has been inhibited by the administration of recombinant uteroglobin (rUG). However, the ability of UG on tTG modulation has not been thoroughly assessed. In this study, we investigated the feasible protective role of UG in murine ecGN through the modulation of tTG and TGF- β expressions. ecGN was induced by the administration of anti- GBM Ab into C57BL/6 mice.
Results : Both proteinuria and BUN levels were distinctively lower in rUG treated mice compared to those of disease control mice. Glomerular injuries such as mesangial proliferation, matrix production and crescent formation were lessened with the rUG treatment; these findings were parallel with the attenuated expression of tTG and TGF- β . tTG was expressed along with glomerular capillary walls and TGF- β expression appeared mainly on mesangial areas by the induction of ecGN. rUG treatment markedly attenuated the expressions of these proteins in glomeruli without spatial changes. With the addition of LPS to mesangial cells, the expressions of tTG and TGF- β were up- regulated, whilst the addition of cysteamine, tTG inhibitor, attenuated the expression of tTG and TGF- β as well as the cellular proliferation which was further induced by LPS.
Conclusion : we demonstrate for the first time that rUG is able to attenuate the renal injury through the modulation of expressions of tTG and TGF- β in ecGN and further suggest a wide range of feasible molecular targets to reduce the severity of human glomerulonephritis.

Key Words : Uteroglobin, Transglutaminase, Glomerulonephritis (GN)