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IL-17 결핍 마우스에서 실험적 반월성 사구체신염의 약화

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Attenuation of Experimental Crescentic Glomerulonephritis in IL-17 Deficient Mice

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Crescentic glomerulonephritis (GN) is the most severe form of glomerulonephritis, and progresses rapidly to end-stage renal diseases unless treated adequately. T cells play a major role in the pathogenesis of crescent GN, and Th1 mediated adaptive immunity has been known as a major player in this process. Recently, subset of T cells producing IL-17 (Th17) is identified and known as a major T cell subset in the pathogenesis of several autoimmune diseases such as experiment autoimmune encephalitis and collage induced arthritis. We induced experimental crescentic GN by administrating anti-glomerular basement membrane (GBM) antibodies to Balb/c and IL-17 deficient Balb/c mice. Renal dysfunction and severity of proteinuria were significantly reduced in IL-17 deficient mice compared to wild type mice after 3 and 7 days of disease induction. The deposit of immunoglobulin and crescent formation in glomeruli were also reduced in IL-17 deficient mice. These changes were accompanied with the reduction of C3 and TGFβ deposition in glomeruli. In addition, IL-17 deficient mice showed the attenuation of renal tubular damages and sclerosis. With the development of crescentic GN, mRNA expression of IL-1β, TGF-β 1, IL-10, and STAT3 increased in the kidney of wild type mice, but these changes were attenuated in IL-17 deficient mice. These findings suggest that IL-17 play an important role in the development of autoimmune induced crescentic GN, and might be the therapeutic target of crescentic GN.

Key Words : 실험적 반월성 사구체신염, IL-17, T 세포
Experimental crescentic GN, IL-17, T cells