

## The Role of IL18 on the Progress of Crescentic Glomerulonephritis

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Chronic kidney disease progression is the result of close interaction among renal cells, inflammatory cells and chemokines/cytokines released. Interleukin 18 (IL18), one of the most important innate cytokines produced from macrophages in the early stages of the inflammatory immune response, induces monocyte chemoattractant protein 1 (MCP-1) and interferon  $\gamma$  (IFN- $\gamma$ ), and other Th1 cytokine. For testing the hypothesis that IL18 plays pathogenic role in early stage of crescentic glomerulonephritis, anti-GBM nephritis was induced in C57BL/6 mouse and serial data collected. Kidneys were harvested at days 1, 3, 7, 11 and 16 after inducing glomerulonephritis by intravenous injection of anti-GBM antibody IgG (0.3 mg/g mouse). Proteinuria measured from 24-hour urine before sacrifice showed abrupt increase from day 1, and interstitial fibrosis and glomerular mesangial sclerosis were evident from day 7, consistent with cortical fibronectin level. Various cytokines were measured by ELISA of chopped cortical tissue, and MCP-1 and IL-6 were elevated from day 1 and showed increasing tendency with the disease course, but significant increase of IL-10, IL18, IL-12p70 levels were not evident. TNF- $\alpha$  showed slight elevation at late stage, IFN- $\gamma$  showed none. In this model, MCP-1 and IL-6 showed persistent elevation suggesting pathologic role, but the role of IL18 was not verified.