AFFECT OF ANTISENSE MCP-1 IN MSPGN RATS

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Objective: To further clarify whether the antisense MCP-1 have renal protective role in MsPGN rats.

Method: Rats were induced mesangial proliferative glomerulonephritis by injected Thy-1 antibody through tail vain. The infected mesangial cells were transferred the rat kidney via renal artery injection detected by in suit hybridization. 24h urinary protein, serum albumin, cholesterol, blood urea nitrogen and serum creatinine were checked by automatic biochemistry analyzer method. Histopathological examination for light microscopy counted by a semiquantitative score. To assess the glomerular hypercellularity, the number of nuclei was counted in ten glomeruli and expressed as the number in per glomeruli. The MCP-1, TGF- β_1 protein secretion were measured by immunohistochemical in paraffin sections. Using RT-PCR evaluate the level of mRNA formation of MCP-1, CCR2, TGF- β_1 in kidney.

Result: In MsPGN rats the infected mesangial cells were detected by in suit hybridization, and the positive cell were stain in glomerular. In antisense group, the glomerular lesion, total cell and 24h urinary protein excretion reduce than that of control group. The level of mRNA formation of MCP-1, CCR2, TGF- β_1 decreased. The MCP-1 positive cell in paraffin sections also decreases. But the TGF- β_1 positive cell in paraffin sections still high compare with control.

Conclusion: Mesangial cell can transfer MCP-1cDNA into kidney. Antisense MCP-1 can reduce glomerular proliferation and damage through dresease mRNA formation of MCP-1, CCR2, TGF- β_1 in MsPGN rats. MCP-1 induce extracellular matrix synthesis part by TGF- β_1 .