

## 당뇨환경이 허혈재관류 손상 이후 진행되는 만성신질환 진행에 미치는 영향?

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### Adverse Effect of Diabetes on the Progression of Chronic Kidney Disease after Ischemia Reperfusion Injury

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**Background:** Diabetic patients have a high risk for chronic kidney disease (CKD) and renal ischemia reperfusion injury (IRI) is known as a cause of CKD. Recently studies showed that acute kidney injury (AKI) is severe in diabetic animal models. However, it is not known whether diabetes accelerates CKD progression after AKI. Therefore, we investigated the influence of diabetes on CKD progression after AKI.

**Methods:** Diabetes was induced by streptozotocin (STZ) and unilateral renal ischemia-reperfusion injury (IRI) model was established in diabetic and non-diabetic C57BL/6 mice at 2 weeks or 8 weeks after STZ treatment. Histological changes in the kidney were evaluated at 3 weeks after IRI in both conditions. The expression levels of genes related to fibrosis and inflammation were determined by qRT-PCR.

**Results:** Tubulointerstitial injury scores were significantly higher in the diabetic IRI group compared with the non-diabetic IRI group. The result of interstitial fibrosis was also consistent with that of tubulointerstitial injury where a significantly higher degree of interstitial fibrosis in the diabetic IRI group was observed. The mRNA expression levels of fibrosis-related genes, TGF- $\beta$ 1, CTGF, collagen Ia-1 and collagen IVa-I, were significantly higher in the non-diabetic IRI group and were raised in diabetic IRI groups. The mRNA expression level of TNF- $\alpha$  was consistent with that of fibrosis in early diabetic status. However, TNF- $\alpha$  and IL-1 $\beta$  expressions had no influence on chronic diabetic status.

**Conclusion:** Our data suggest that diabetes induces renal fibrosis after IRI but is not involved in inflammation.

**Key Words:** 당뇨, 허혈재관류 손상, 신장섬유화

Diabetes, Ischemia reperfusion injury, Renal fibrosis