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Folic Acid 유발 급성 신손상에서 Tubular CD36 overexpression의 영향

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Effects on Tubular CD36 overexpression in Folic Acid induced Acute Kidney Injury

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36 The incidence of AKI has increased, most likely due to a trend toward older, more severely and chronically ill patients admitted to the hospital. AKI may progress to end-stage renal disease, and even subclinical episodes of AKI, which are common, may also progress to end-stage renal disease. Multiple risk factors such as preexisting kidney disease, hyperglycemia, obesity, dyslipidemia, hypertension, old age, and inflammation may be associated with increased incidence to end-stage renal disease after acute kidney injury. Recently, several studies suggest that acute kidney injury may be important cause of chronic kidney disease. However, it is not known well how acute kidney injury progress to chronic kidney disease excepting preexisting kidney disease. So we investigated the effects of CD36 overexpression in acute kidney injury, which is modulated in clinical conditions such as hyperglycemia, hyperlipidemia, and chronic inflammation. We used human CD36-overexpressed mice in proximal tubules to elucidate the effects of CD36 in acute kidney injury by folic acid administration. Kidneys were removed 48 hours after folic acid administration. Human CD36-overexpressed mice showed more severe pathological findings, and more increased and prolonged mRNA expression in inflammatory, fibrosis, and apoptotic markers. These results suggest that CD36-overexpressed clinical conditions such as diabetes and hyperlipidemia are vulnerable to acute kidney injury and may be potential risk factors of progression to chronic kidney disease after acute injury.

Key Words: Folic acid, CD36, AKI