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횡문근융해증에 의한 급성신손상 쥐에서 MAPKs와 Bcl-2 경로를 통한 N-acetylcysteine의 효과

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N-acetylcysteine Attenuates Rhabdomyolysis-induced Acute kidney Injury via MAPKs and the Bcl-2-related Protein Pathway in Rats

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Rhabdomyolysis-induced acute kidney injury (AKI) accounts for about 10% to 40% of all cases of AKI. It is known that N-acetylcysteine (NAC) is effective in various experimental renal injury models; however, little information is available for the rat model of glycerol-induced rhabdomyolysis. In this study, we hypothesize that NAC plays a renoprotective role via the anti-apoptotic pathway by a direct and/or indirect mechanism. Male Sprague-Dawley rats were divided into four groups: (1) saline control group, (2) NAC (150 mg/kg, i.v.) treated group, (3) only glycerol (50%, 8 ml/kg, i.m.) treated group and (4) NAC plus glycerol treated group. Rats were sacrificed at 24 hours after glycerol injection and the blood and renal tissues were harvested. Glycerol administration caused severe renal dysfunction, which included marked renal oxidative stress, significantly increased blood urea nitrogen (BUN) and serum creatinine levels. Histopathological findings such as cast formation and tubular necrosis after glycerol treatment confirmed renal impairment. Additionally, we noted a marked activation of ERK and JNK, but not p-38, in the glycerol-treated group. We also observed high expression of Bax and Bad but only weak expression of Bcl-2 and Bcl-xL in the glycerol-treated group. However, NAC pretreatment significantly improved renal function and decreased the activation of ERK, JNK, Bax, and Bad whereas it increased Bcl-2 and Bcl-xL. These results demonstrate that NAC protects against renal dysfunction, morphological damage and biochemical changes via the anti-apoptotic pathway in glycerol-induced rhabdomyolysis model in rats.

Key Words : 횡문근융해증, 급성신손상, N-아세틸시스테인
Rhabdomyolysis, Acute kidney injury, N-acetylcysteine