P-161

황화수소가 생쥐의 신장 허혈/재관류 손상의 회복을 촉진한다

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Hydrogen Sulfide Accelerates the Recovery of Ischemia/reperfusion-injured Kidney in Mice

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Kidney ischemia/reperfusion (I/R) induces tubular epithelial cell damage, inflammation, and production of reactive oxidative species, resulting in acute kidney injury (AKI). If recovery of damaged tubular cells is inadequate, kidney progresses fibrosis, leading to ultimately renal functional disorder. Hydrogen sulfide (H2S), a novel endogenous gaseous molecule, regulates a variety of cellular signals involved in injury and repair. This study aimed to identify the role of H2S and its producing enzymes in the recovery of kidney following I/R injury. Mice were subjected to 30 minutes of bilateral renal ischemia. Some mice were daily administered NaHS, an exogenous H2S donor, and propargylglycine (PAG), an inhibitor of the H2S producing enzyme; cystathionine gamma-lyase (CSE), during recovery period. Ischemia decreased H2S level along with reductions of H2S-producing enzymes activity and expression. These decreases did not return to normal level until 8 days after ischemia. BrdU incorporation dramatically increased in the tubular epithelial cells and interstitial cells of the kidney after I/R. NaHS administration accelerated the increase of BrdU-incorporated tubular cells, whereas it inhibited the increase of BrdU-incorporated interstitial cells, indicating NaHS treatment accelerates the restoration of damaged tubules. In consistency with the increase of tubular cells, NaHS administration accelerated reforming tubules with the acceleration of normalization of plasma creatinine (PCr). In contrast, PAG delayed reforming tubules and normalization of PCr. NaHS treatment facilitated the balance of redox status disrupted by I/R. Our findings demonstrate that H2S accelerates the recovery of I/R-induced kidney damage, suggesting that the H2S-producing transsulfuration pathway plays an important role in kidney repair after acute injury.

Key Words: CBS, CSE, 황화수소, 허혈, 활성화 산소종
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