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일측성 허혈/재관류 및 콩팥절제에 의해 유발된 콩팥의 보상성 비대에 있어 활성산소의 역할

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Reactive Oxygen Species is Important Contributor in the Changes of Contralateral Kidney Following Unilateral Renal Ischemia, but not Following Unilateral Nephrectomy

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Unilateral renal injury and unilateral nephrectomy induces the hypertrophy of contralateral kidney. However, the molecular mechanism underlying these alterations remains unclear. We evaluated that reactive oxygen species (ROS) are associated with the hypertrophy of the contralateral kidneys (CKs) caused by unilateral ischemia (UI), which produces pathogenic condition, and unilateral nephrectomy (Ux). Unilateral kidney of mouse was subjected to either 30 min of ischemia or nephrectomy and were administered Mn (III) Tetrakis (1-methyl-4-pyridyl) porphyrin (MnTMPyP, a superoxide dismutase (SOD) mimetic. Ux and UI resulted in kidney hypertrophy, and increased medial area of renal cortical artery in their CKs. UI also increased superoxide production in CK, but Ux did not. UI significantly decreased the levels of manganase SOD (MnSOD) in the CKs, but did not induce significant changes of copper-zinc SOD (CuZnSOD) and catalase expression. In contrast, Ux did not result in significant changes of those enzymes. MnTMPyP administration alleviated the increases of kidney hypertrophy and the medial area caused by UI with the reduction of superoxide production. However, these changes of the CKs induced by Ux did not relieve by MnTMPyP administration. Ux and UI significantly increased angiotensin II type 1 receptor (AT1) in the CKs, but the increased levels of AT1 in Ux was higher than in UI. Although increased AT1 expression of both CKs of UI and Ux prevented by MnTMPyP administration, those of Ux did not complete. It suggests that the kidney hypertrophy and the increase of medial area in the CKs of Ux are not blocked by MnTMPyP treatment is due to the increased AT1 expression compare with those of sham. In conclusion, ROS contributes to the hypertrophy of CKs in UI, but not in Ux, demonstrating that the changes of CKs induced by these two models are governed by different mechanisms.

Key Words : 허혈/재관류 손상, 콩팥절제, 활성산소
Ischemia/reperfusion, Nephrectomy, ROS