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Changes of Type B Endothelin Receptor in Rats with Puromycin Aminonucleoside-induced Nephrotic Syndrome

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Both endothelin (ET) and nitric oxide (NO) systems play a role in concert in determining the degree of renal sodium excretion. We investigated whether sodium retention associated with nephrotic syndrome is related with an altered role of these systems. Male Sprague-Dawley rats were treated with puromycin aminonucleoside (PAN, 180 mg/kg, iv), whereas the control group received vehicle only. They were examined at days 7 and 14, i.e., sodium retaining and compensatory stages, respectively, of nephrotic syndrome. The expression of ET and NO system components was determined by real-time PCR and western blot analysis. At day 7 after PAN injection, the urinary excretion of sodium was decreased, along with the development of ascites and positive sodium balance. The mRNA expression of ET-1 was increased in the inner medulla, whereas that of type B ET receptor (ETRB) decreased. The expression of neuronal NO synthase was decreased. At day 14, the urinary excretion of sodium did not differ from the control. The expression of ETRB and neuronal NO synthase resumed their control values. On the other hand, the expression of ETRA, and endothelial and inducible NO synthase proteins did not differ from the control at either stage. These findings suggest that the downregulation of ETRB and neuronal NO synthases may contribute to the development of sodium retention in nephrotic syndrome. A functional recovery of these systems may promote sodium excretion in the later stage.

Key Words : 신증후군, ETB수용체, 산화질소
Nephrotic syndrome, Endothelin-B receptor, Nitric Oxide