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Local RAS in the Renal Drainage Lymph Vessels Involves a Therapeutic Target for Salt-Sensitive Hypertension.

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Summary

The renal lymphatic system contributes to the regulation of intra-renal fluid homeostasis as well as pathogenesis of acute kidney disease, kidney fibrosis, polycystic kidney disease, hypertension, and kidney implantation. Roles of local renin-angiotensin-system (RAS) in the renal drainage lymph vessels (RDLV) still is not clear, although the kidney is the most important organs of pathogenesis in salt-sensitive hypertension via RAS imbalance. The purpose of the present study is to investigate that a high salt diet (4 weeks) for rats alter function of RDLV in response to angiotensin I or angiotensin II. Wistar rats (male, 4 weeks) were divided to normal salt diet (NSD) and high salt diet (HSD, 8% NaCl) groups. After 4 weeks treatment with NSD or HSD, we isolated the RDLV and lymph nodes in the iliac and renal regions, and then examined pharmacological (video-microscope system), biochemical (real time PCR), and immunological (FACS) studies. Angiotensin I (1-100 nM) or angiotensin II (1-100 nM) dose-dependently constricted RDVL of NSD and HSD rats. HSD significantly enhanced the angiotensin I-induced constriction of RDLV and slightly upregulated mRNA revels of angiotensin type1a receptors, angiotensinogen, and angiotensinogen converting enzyme (ACE) in the wall of RDLV. The total number of lymphocytes in the renal lymph nodes of HSD rats were slightly less than those of NSD rats. We conclude that HSD may reduce distensibility of RDLV and suppress lymph drainage from the intra-renal space through the angiotensin I-mediated constriction of RDLV, suggesting that HSD influence cell population in the renal drainage lymph nodes of rats.