Does excessive salt intake promote progression of atherosclerosis?

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Summary

We have already demonstrated that in aorta from hypertensive Dahl salt-sensitive rats, excessive cholesterol intake results in suppression of NO release which might lead to the initiation of atherosclerosis, but such suppression does not occur in spontaneously hypertensive rats (SHR), an animal model of essential hypertension. These findings suggest that excessive salt intake per se can promote the development of atherosclerosis. In this study, we used SHR to investigate whether excessive salt intake would alter the function of the vascular endothelium and smooth muscle, and also whether excessive salt and excessive cholesterol intake would cause initiation of atherosclerosis. The results obtained were as follows:

1) The blood pressure was markedly increased by a high salt diet (8% NaCl) given for 4 weeks.
2) In the aorta, excessive salt intake impaired not only endothelium-dependent relaxations but also endothelium-independent relaxations. The impairment is thought to be due to the reduced guanylate cyclase activity in smooth muscle cells but not the decreased release of nitric oxide (NO) from endothelial cells.
3) In the renal artery, excessive salt intake impaired endothelium-dependent relaxations but not endothelium-independent relaxations. This functional change is probably due to the increased release of endothelium-derived contracting factors (EDCF) and the decreased release of endothelium-derived hyperpolarizing factors (EDHF), but not a decreased release of NO.
4) These changes in vascular reactivity induced by excessive salt intake were not alleviated by treatment with an anti-hypertensive drug, nifedipine.
5) Intake of excessive cholesterol in addition to excessive salt did not lead to further aggravation of the abnormality of vascular reactivity or the increased blood pressure induced by excessive salt intake, nor was lipid accumulation detected in the arteries.

It has been known that NO is an endogenous anti-atherosclerotic factor. We concluded that in SHR, unlike in Dahl rats, excessive salt intake is not related to the initiation of atherosclerosis via endothelial dysfunction, because the release of NO cannot be suppressed by high blood pressure, high salt or high cholesterol intake.