Surgical Menopause, Salt Sensitivity, and NO Bioavailability in Women

To the Editor:

We read with great interest the article by Schulman et al1 dealing with the association between the loss of ovarian hormones and the occurrence of salt sensitivity in healthy premenopausal women. The results of their study demonstrated that 38.7% of salt-resistant women developed salt sensitivity after surgical menopause. The authors indicated that the prevalence of salt sensitivity doubled as early as 4 months after surgical menopause. The authors proposed that the loss of ovarian function may unmask a population of women prone to salt sensitivity who would be at higher risk for the subsequent development of hypertension and cardiovascular disease.

Numerous studies have shown that one of the mechanisms underlying the cardiovascular protective effect of estrogen may be the enhancement of NO production. There is evidence demonstrating that vascular endothelial function is markedly influenced by estrogen and is improved by hormone replacement therapy in postmenopausal women. Recently, Scuteri et al3 showed that inhibition of NO bioavailability by asymmetrical dimethylarginine (ADMA) an endogenous NO synthase inhibitor) and a subsequent reduction in endothelial dysfunction might contribute to the increase in blood pressure during salt intake in normotensive postmenopausal women not receiving estrogen. It was also demonstrated that estrogen replacement therapy significantly reduced plasma concentration of ADMA in postmenopausal women. In a study that we presented earlier, it was shown that estrogen-induced improvement of membrane fluidity (the reciprocal value of microviscosity) of erythrocytes was counteracted by ADMA in postmenopausal women. In addition, we showed that amelioration in membrane fluidity of erythrocytes was associated with higher NO metabolite and lower ADMA levels in plasma. The findings might suggest that NO and ADMA might actively participate in the regulation of rheological behavior of cell membranes and microcirculation. In this context, it can be speculated that, in women with higher ADMA levels, the circulatory disorders are more pronounced. The precise role of ADMA in the development of hypertension and cardiovascular disease in women is still unclear. It is possible that estrogen deficiency after menopause might accelerate abnormalities in endothelial function by increasing ADMA levels. Therefore, we would like to know whether the plasma NO metabolite and ADMA concentrations might be associated with the changes in salt sensitivity after surgical menopause in the study by Schulman et al. It would be important to assess more precisely the relationship among estrogen status, salt sensitivity, and NO bioavailability in women after menopause.

Disclosures

None.

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