Role of Estrogen and Endothelium in Migraine in Young Women

To the Editor:

We read with great interest the recent article by Tietjen and colleagues dealing with migraine and endothelial activation in young women. The results of their study demonstrated a strong relation between migraine and endothelium-related biomarkers, including von Willebrand factor activity, high-sensitivity C-reactive protein, and total nitrite/nitrate concentration, in a young, relatively healthy cohort of women. The authors suggest that, in premenopausal women with migraine, there is evidence of increased endothelial activation, a component of endothelial dysfunction. They further propose that an understanding of the role of endothelium in migraine may provide a rationale for multiple avenues for stroke prevention in migraineurs.

Numerous studies have shown that estrogen may have beneficial effects on cardiovascular functions. One of the mechanisms underlying the protective effect of estrogen may be the enhancement of nitric oxide production. There is evidence showing that vascular endothelial function is markedly influenced by estrogen and improved by hormone replacement therapy in postmenopausal women. In an in vitro study presented earlier, we demonstrated that 17β-estradiol increased membrane fluidity (a reciprocal value of membrane microviscosity) of erythrocytes and improved the rigidity of cell membranes in women through the nitric oxide- and cGMP-dependent mechanism. Because abnormalities in membrane microviscosity could cause a disturbance in rheological behavior and microcirculation, these findings indicate that estrogen deficiency might be involved in the pathophysiology of vascular complications in women. On the other hand, it has been demonstrated that endothelial function varied during the menstrual cycle in premenopausal women, suggesting that sex hormone status would have a crucial role in cardiovascular risk and other disease processes in premenopausal women. Kawano et al also demonstrated a menstrual cyclic variation of myocardial ischemia in premenopausal women. Therefore, we would like to know whether menstrual cycle-related endogenous estrogen status might be associated with the endothelial biomarker levels and the magnitudes of migraine in young women in the study of Tietjen and colleagues. It would be important to assess more precisely the relationships between estrogen status and endothelial function during the menstrual cycle and their contribution to the pathogenesis of migraine in young women.

Disclosures

None.

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