Cerebrovascular Disease in Type 2 Diabetic Patients Without Hypertension

To the Editor:

The close relationship between diabetes mellitus and arteriosclerosis of the cerebral arteries has recently been reported. However, in these studies, the subjects included older patients with hypertension, which itself is a significant risk factor for arteriosclerosis of the cerebral arteries. Thus, previous studies on the effect of diabetes on the development of sclerosis were confounded by hypertension and aging. Therefore, we examined relatively young patients with diabetes mellitus who did not have hypertension in order to clarify the influence of diabetes mellitus itself on the development of sclerosis of the cerebral arteries.

The subjects included 30 patients with type 2 diabetes mellitus (DM). The subjects with DM did not have hypertension (systolic blood pressure <140 mm Hg, diastolic blood pressure <90 mm Hg) and had no history of cerebral infarction, diabetic retinopathy, diabetic neuropathy, or diabetic nephropathy. Among the 30 diabetic patients, 4 were being treated with insulin injections, 10 with oral hypoglycemic agents, and 16 with dietary therapy alone. The control group (C) consisted of 20 healthy male subjects (10.0%) of the control group. The incidence of LA was significantly higher in the diabetic group than in the control group (P<0.05). These findings suggest that diabetes mellitus itself contributes to the development of sclerotic lesions in cerebral arteries.

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Response

We read with great interest the study of Nagata et al concerning the influence of diabetes mellitus on the development of sclerosis of the cerebral arteries. The authors studied a group of patients with uncomplicated diabetes mellitus type 2 and a group of control subjects comparable for age and sex. The 2 groups of subjects were studied by means of MRI to assess the presence of silent lacunar lesions and by means of magnetic resonance angiography and ultrasoundographic scanning of the intracranial and extracranial arteries to assess the presence of atherosclerosis. Moreover, transcranial examination was also performed to measure carotid artery intima-media thickness (IMT) and to evaluate carotid plaques. Authors found that either the incidence of lacunar lesions or the incidence of atherosclerosis was significantly higher in the diabetic group than in the control group. These findings suggest that diabetes mellitus itself (even in relatively young subjects without hypertension and any other diabetic organ damage) contributes to the development of sclerotic lesion in cerebral arteries. Both silent lacunar lesions and increased carotid artery intima-media thickness have been found independent risk factors for stroke in older population. Thus, the study of Nagata et al outlines an important role of diabetes as an independent risk factor for the development of atherosclerosis. In our study concerning carotid artery IMT evaluation in lacunar versus nonlacunar infarcts, we did not find increased carotid artery IMT values in the subtype of first-ever symptomatic lacunar infarcts. However, we think that a comparison with our previous study is difficult. In fact, we evaluated first-ever stroke patients, not homogeneous for vascular stroke risks, in order to investigate whether common carotid IMT measurements may help to identify different subtypes of ischemic stroke patients. Moreover, we did not look at the presence of possible coexistent, previous, silent lacunar lesions in the 2 groups (namely lacunar hypertension than in the control group (P<0.05). These findings suggest that diabetes mellitus itself contributes to the development of sclerotic lesions in cerebral arteries.
versus nonlacunar) of patients. Nagata et al looked at the relationship between diabetes, as a vascular risk factor, and the presence of silent lacunar lesions and cerebral atherosclerosis. However, stroke patients were not included in their study. It has been hypothesized that 2 types of lacunar infarcts with different prognosis could be distinguished, namely those with silent lacunar lesions and those without such lesions. Two types of underlying small-vessel pathology could underlie these lesions, although they might not be mutually exclusive. In addition, de Jong et al recently found that patients with a single symptomatic lacunar stroke had better prognosis over time than those with concomitant silent lacunar lesions. We think that a prospective study on the evaluation of carotid artery IMT in lacunar patients with a single symptomatic lacunar stroke and those with concomitant silent lacunar lesions might add further insights on this issue.

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