The Circle of Willis and White Matter Lesions in Patients with Carotid Atherosclerosis

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Background: The correlation between cerebral atherosclerosis and white matter lesions (WMLs) in the elderly was controversial in the published articles, where the stenosis was often evaluated by ultrasonography, computed tomography angiography, or magnetic resonance angiography and collaterals were seldom considered. We hypothesized that collaterals influence WMLs. Our study was to explore the relationship between the circle of Willis and WMLs in a retrospective, hospital-based cohort of patients with carotid atherosclerosis. *Methods:* Two hundred eighty-six patients with carotid atherosclerosis were enrolled from the Nanjing Stroke Registry. They underwent magnetic resonance imaging evaluating WMLs and digital subtraction angiography evaluating both carotid atherosclerosis and collateral capacity of the circle of Willis. We tested the association between severe carotid atherosclerosis, the circle of Willis, and WMLs by logistic regression analysis. *Results:* Severity of carotid atherosclerosis was not significantly associated with either periventricular or deep WMLs (P = .656 and .566, respectively). Number of carotid arteries with severe stenosis was not associated with the severity of either periventricular or deep WMLs (P = .721 and .263, respectively). Patency of the communicating arteries (CoA) was not associated with periventricular or deep WMLs (P = .561 and .703, respectively). Advanced age and hypertension were associated with periventricular WMLs (P = .001 and .008, respectively). Advanced age, hypertension, and prior stroke were associated with deep WMLs (P = .049, .048, and .001, respectively). *Conclusions:* The circle of Willis and severe carotid atherosclerosis may not be related to WMLs. Further larger studies are warranted to confirm or refute our findings. **Key Words:** White matter lesions—atherosclerosis—ischemic stroke—collateral.
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White matter lesions (WMLs) in the elderly, also known as leukoaraiosis, appear as irregular hypodensities on computed tomography (CT) and hyperintensities on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences on magnetic resonance imaging (MRI).[1] They are common in aged people and patients with hypertension, depression, and dementia.[2-7] They also predict future stroke and stroke outcome.[8-14] Recent studies have demonstrated that WMLs increase intracerebral hemorrhage in patients treated with tissue plasminogen activator and have a worse functional outcome.[13,15] But their pathogenesis is not fully understood.

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The authors declare that they have no conflict of interest.

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WMLs are often regarded as one type of cerebral small-vessel diseases that include lacunar infarcts, WMLs, microbleed, and so on.[16-18] But some studies have indicated an association between carotid atherosclerosis and WMLs.[5,19,20] Recent study even indicated that carotid artery revascularization could reduce the severity of leukoaraiosis.[21] Chronic cerebral hypoperfusion due to arterial stenosis may be the underlying mechanism of WMLs. It also could induce the collateralization. Cerebral collaterals, mainly including primary and secondary collaterals, are a conserved network of blood vessels to maintain consistent cerebral perfusion.[22] They have been shown to correlate with the risk of stroke and transient ischemic attack.[23,24] However, as the primary collateral, the circle of Willis was seldom considered in exploring the relationship between carotid stenosis and WMLs in previous clinical studies. One of the above studies by Chuang indicated that a complete circle of Willis may protect WMLs, but its sample was small and evaluation of circle of Willis by MR angiography (MRA) was not accurate.[25] So it is necessary to further verify the relationship between collaterals and WMLs in a larger cohort.

Our study was aimed to investigate the relationship of the circle of Willis with WMLs in patients with carotid atherosclerosis in a large, retrospective, hospital-based cohort, in which cerebral atherosclerosis and collateral flow were evaluated by a gold standard, digital subtraction angiography (DSA).

Patients and Methods

Patients

Patients were extracted from the Nanjing Stroke Registry Program between June 1, 2009, and May 31, 2011. The following were the inclusion criteria for the study: (1) aged 65 years or older; (2) detailed clinical evaluation including clinical history, laboratory tests, and electrocardiography; and (3) accomplished MRI and DSA. DSA was performed only if the degree of carotid stenosis evaluated by CT angiography (CTA), MRA, or carotid Doppler was not less than 50%. Exclusion criteria for the study were as follows: (1) dementia; (2) parkinsonism; (3) intracranial hemorrhage; (4) intracranial space-occupying lesion; (5) non-age-related WMLs (eg, multiple sclerosis); and (6) MRI contraindications or known claustrophobia. In addition, our study was to investigate the influence of the carotid atherosclerosis and the circle of Willis on WMLs, so severe atherosclerotic anterior and middle cerebral arteries (no less than 70% stenosis) were excluded. Eventually 286 patients, including 17 transient ischemic attack and 146 acute ischemic stroke, met the above criteria and were included in the study. Informed consents to participate in the study were obtained from all patients. Our study was approved by the Ethics Committee of Jinling Hospital.

Risk Factor Evaluation

Comprehensive clinical examinations including medical history and neurologic examinations were obtained from all patients. The clinical data included age, sex, history of hypertension (defined by the use of an antihypertensive agent before admission or a systolic pressure greater than 140 mm Hg or diastolic pressure less than 90 mm Hg on repeated examinations), diabetes mellitus (defined as a fasting blood glucose level greater than 126 mg/dL or a history of being treated for diabetes mellitus), and hyperlipidemia (defined as a total cholesterol level higher than 200 mg/dL or a low-density lipoprotein cholesterol higher than 130 mg/dL at the time of presentation or a history of treatment). In addition, cigarette smoking (current smoker and those quitting less than 1 year), a previous history of ischemic stroke, and heart disease (defined as a known history or clinical demonstration of any heart disease, including myocardial infarction, angina pectoris, congestive heart failure, or arrhythmia) were noted.

Evaluation of WMLs, the Carotid Atherosclerosis, and the Circle of Willis

All patients enrolled underwent conventional MRI on a 1.5-T system (Signa 1.5-T TwinSpeed, General Electric Medical Systems, General Electric Corporation, Connecticut, USA) on admission. The conventional MRI consisted of transverse T1-weighted imaging (FLASH sequence; repetition time [TR], 280 ms; echo time [TE], 2.5 ms), T2-weighted imaging (turbo spin echo [TSE]; TR, 4000 ms; TE, 100 ms), diffusion-weighted imaging (echo planar imaging [EPI]; TR, 3000 ms; TE, 90 ms), and fluid-attenuated inversion recovery (FLAIR; TR, 8500 ms; TE, 90 ms; and inversion time [TI], 2200 ms) sequences. WMLs were defined as hyperintensities on T2-weighted and fluid-attenuated inversion recovery images. We rated the WMLs according to Fazakas scale. Periventricular hyperintensity (PVH) was rated as 0 = absence, 1 = "caps" or pencil-thin lining, 2 = smooth "halo," and 3 = irregular PVH extending into the deep white matter. Separate deep white matter hyperintensity (DWMH) was rated as 0 = absence, 1 = punctate foci, 2 = beginning confluence of foci, and 3 = large confluent areas.[26-28] When evaluating the WMLs, new (high signal on diffusion-weighted image) and old (definitely low signal on T1-weighted image) infarcts were excluded. If one or both sides of the brain were focally abnormal, estimates were based on the uninvolved side in accord with the principle of symmetry assumed. We dichotomized the WMLs into non-WMLs (0-1 point) and WMLs (2-3 points).

According to the North American Symptomatic Carotid Endarterectomy Trial Collaborators, the degree of carotid stenosis was graded as occlusion, high-grade (70%-99%), moderate-grade (50%-69%), and mild-grade (<50%) stenosis. No less than 70% artery stenosis was

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