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Correlation between cervical artery kinking and white matter lesions

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ABSTRACT

Objective: To explore the correlation between cervical artery (i.e., extracranial carotid artery and vertebral artery) kinking and white matter lesions (WMLs).

Patients and methods: In total, 147 patients who underwent magnetic resonance imaging (MRI) of the head and computed tomography angiography (CTA) of the head and neck were included in this study. The severity score of WMLs in each patient was evaluated using MRI data. The extent and location of cervical artery kinking were determined and recorded using CTA. The correlation between cervical artery kinking and WMLs was analyzed using logistic regression and Dunnett's method.

Results: The age at which diabetes and carotid artery (common carotid artery and extracranial internal carotid artery) kinking develop and the rate of incidence were significantly higher in patients with WMLs than in those with non-WMLS ($P < 0.05$, $P < 0.01$), but there was no significant difference in the incidence of vertebral artery kinking. Logistic regression analysis indicated that carotid artery kinking (OR = 6.144, 95% CI: 2.702–13.970), age (OR = 1.055, 95% CI: 1.007–1.106), and diabetes (OR = 4.636, 95% CI: 1.421–15.126) were risk factors for WMLs ($P < 0.01$). The Pearson correlation coefficient was 0.753 ($P < 0.01$), thereby suggesting that there is a positive correlation between the severity of WMLs and the number of carotid artery kinks.

Conclusion: Carotid artery kinking may be an independent risk factor for WMLs.

1. Introduction

White matter lesions (WMLs), commonly called cerebral white matter hyperintensity or leukoaraiosis, are manifestations of cerebral small vessel disease [1]. Furthermore, WMLs include periventricular white matter hyperintensity (PVWMH) and deep white matter hyperintensity (DWMH). The incidence of WMLs in the elderly population is approximately 20%, which is significantly higher than the incidence reported in young people [2]. Moreover, it is widely regarded to be associated with stroke, dementia, and a high risk of death [3]. Many studies [4,5] have suggested that the main cause of WMLs is chronic cerebral ischemia that is caused by low blood perfusion and microvascular dysfunction. Anatomically, large vessels are considered to be the "upstream" blood vessels that feed the smaller blood vessels throughout the brain. Some studies [6,7] have shown that certain neck macrovascular diseases, such as carotid atherosclerotic stenosis, may lead to chronic ischemia of the distal small vessels, thereby ultimately producing WMLs. Atypical cervical artery elongation is another type of neck macrovascular disease whose relationship with WMLs is not clear. Weibel [8] divided atypical elongation arteries into three categories: arterial tortuosity, in which arteries assume a "C," "U," or "S" shape; coiling, in which there are one or more arterial loops; and kinking, wherein tortuous arteries are folded into an acute angle (single or double Z-shaped). It is currently believed that kinking and ischemia are related, while tortuosity and coiling do not contribute to this condition. Some scholars [9] believe that internal carotid artery kinking is significantly related to the development of transient ischemic attacks as well as stroke and that after surgical treatment of arterial kinking, patient symptoms significantly improve. Another study [10] has also hypothesized that arterial kinking with hypertension may be associated with intracranial ischemia. However, the relationship between cervical artery kinking and WMLs is unclear. The purpose of this study was to investigate the correlation between WMLs and cervical artery kinking and to explore the pathogenesis of WMLs.

2. Materials and methods

2.1. Objects

We selected the patients from the Neurology Department of the First Affiliated Hospital of Beng Bu Medical College from December 2013 to December 2014. These patients were included if they had undergone

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magnetic resonance imaging (MRI) of the head and computed tomography angiography (CTA) of the head and neck. Patients were excluded if they exhibited or were diagnosed with intracranial and extracranial vascular stenosis or non-vasculogenic white matter lesions, such as hereditary leukodystrophy, autoimmune central nervous system demyelination, toxic demyelinating disease, infectious demyelinating encephalopathy, and other diseases. Furthermore, patients were excluded if they had received treatment that may cause WMLs within a month, such as a radiotherapy, chemotherapy, immunosuppressive agent use, or hormone therapy. This study was conducted with approval from the Ethics Committee of Bengbu Medical College. Written informed consent was obtained from all participants.

2.2. General information collection

The patients were selected on the basis of with the inclusion and exclusion criteria. We collected patient information, including age, sex, stroke, hypertension, diabetes, coronary heart disease, triglycerides, cholesterol, smoking, alcohol consumption, and other medical histories.

2.3. Diagnosis of cervical artery kinking

The cervical artery refers to the extracranial carotid artery, which is composed of the common carotid and extracranial internal carotid arteries, and the vertebral artery. Patients received intravenous injection of the contrast medium iopromide 370 (90–100 ml at an infusion rate of 5 ml/s). Arterial images of the head and neck were obtained using GE 64-slice spiral CT (General Electric Company, Fairfield City), which imaged from the aortic arch plane to the cranial vault. Raw data were sent to the ADW4.4 workstation for surface reconstruction (CPR), volume rendering (VR), and maximum intensity projection (MIP) analysis. According to the Weibel criteria [8], a twist angle of less than 90° was defined as kinking, and instances of arterial tortuosity and coiling were excluded. The position and number of kinks were recorded.

2.4. WMLS diagnosis and severity score evaluated

The T1WI, T2WI and FLAIR sequences were collected using the Siemens MAGNETOM Verio 3.0T MRI (Siemens AG, Munich, Germany) scanner. WMLs were defined as a continuous halo-like high signal around the ventricle in the FLAIR sequence (PVWMH), or point or patchy lesions in the deep white matter area (DWMH). The severity score of PVWMH and DWMH were evaluated in accordance with the revised Fazekas criteria [11]. The total score was obtained by adding the two scores: the (1) PVWMH score: 0 point, no lesions; 1 point, periventricular thin lesions; 2 points, periventricular smooth halo-like lesions; or 3 points, lesions into the deep white matter area; and the (2) DWMH score: 0 points, no lesions; 1 point, point-like lesions; 2 points, partial fusion lesions; or 3 points, severe fusion lesions.

2.5. Statistical analysis

All data were analyzed using SPSS17.0 software. The *t*-test and chi-square test were used to perform single factor quantitative and qualitative analysis. Multivariate logistic regression analysis was then performed on the statistically significant single-factor variables ($P < 0.05$). An analysis of variance and Dunnett's method were used to analyze the correlation between the kinking of arteries and the severity of WML. $P < 0.05$ was assumed to indicate a statistically significant association.

Table 1
Univariate analysis of WML [n; percentage (%)] ($\bar{x} \pm$ SD).

Variable	White matter lesions		χ^2 or t	P
	No (n = 56)	Yes (n = 91)		
Age	54.65 ± 9.65	62.3 ± 9.39	−4.747	0.000
Sex			0.421	0.516
Male	32 (57.1)	47 (51.6)		
Female	24 (42.9)	44 (48.4)		
Stroke			3.534	0.060
No	21 (37.5)	21 (23.1)		
Yes	35 (62.5)	70 (76.9)		
Diabetes			11.846	0.001
No	51 (91.1)	60 (65.9)		
Yes	5 (8.9)	31 (34.1)		
Hypertension			0.139	0.709
No	27 (48.2)	41 (45.1)		
Yes	29 (51.8)	50 (54.9)		
Coronary heart disease			3.250	0.071
No	39 (69.6)	75 (82.4)		
Yes	17 (30.4)	16 (17.6)		
Triglycerides	1.79 ± 0.96	1.72 ± 0.56	0.559	0.577
Cholesterol	4.44 ± 1.18	4.69 ± 1.09	1.308	0.193
Smoking			0.292	0.589
No	37(66.1)	64 (70.3)		
Yes	19(33.9)	27 (29.7)		
Drinking			0.184	0.668
No	43 (76.8)	67 (73.6)		
Yes	13 (23.2)	24 (26.4)		
Carotid artery kinking			22.488	0.000
No	41 (73.2)	30 (33.0)		
Yes	15 (26.8)	61 (67.0)		
Vertebral artery kinking			3.135	0.077
No	39 (69.6)	50 (54.9)		
Yes	17 (30.4)	41 (45.1)		

3. Results

3.1. Comparison of baseline and univariate analysis

We collected a total of 147 patients. The mean age was 59.63 ± 12.63 years, and 79 (53.7%) of the patients were male and 68 (46.3%) of the patients were female. Among the patients, 91 (61.9%) had WMLs and 56 (38.1%) had non-WMLs. There were 76 patients (51.7%) with carotid artery kinking, 58 patients with vertebral artery kinking (39.5%), and 43 patients (39.3%) with both carotid and vertebral artery kinking. The age and the incidence of diabetes and carotid artery (i.e., common carotid artery and extracranial internal carotid artery) kinking were significantly higher in patients with WMLs than in those with non-WMLs ($P < 0.05$, $P < 0.01$); however, there was no significant difference in the incidence of vertebral artery kinking (Table 1; Figs. 1 and 2).

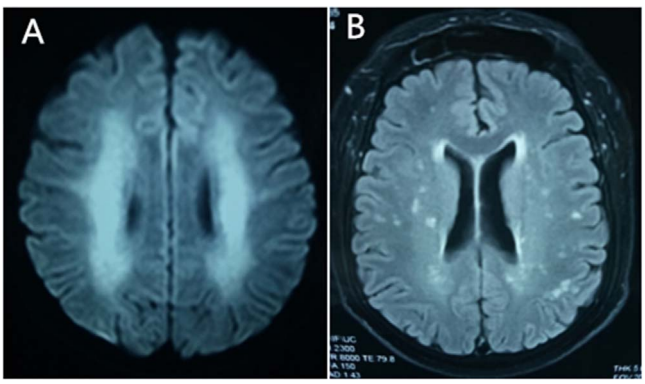

Fig. 1. A: It shows the periventricular white matter hyperintensity (PVWMH); B: It shows the deep white matter hyperintensity (DWMH).