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Clinical study

Carotid atherosclerosis and arterial peripheral pulse wave velocity in cerebral thrombosis

Tamaki Tomonori [a,*], Sawada Keiko [a], Hayashi Shinkichi [a], Node Yoji [a], Teramoto Akira [b]

[a] Department of Neurosurgery, Nippon Medical School Tamanagayama Hospital, 1-7-1 Nagayama Tama-shi, Tokyo-to, Japan 206-8512
[b] Department of Neurosurgery, Nippon Medical School, Bunkyo-ku, Tokyo, Japan

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Abstract

Stiffening and thickening of the arterial walls are two important components of atherosclerosis. A better understanding of the relationship between stiffening and thickening of the large arteries might lead to optimal strategies for the prevention of cerebrovascular disease. However, there have been few investigations of the correlation between carotid artery wall thickening and arterial stiffness. In this study, we investigated the relationship between arterial stiffness (assessed by the ankle-brachial pulse wave velocity) and carotid plaque (detected by high-resolution real-time B-mode ultrasonography) in patients with cerebral thrombosis. The subjects were 109 patients with cerebral thrombosis aged 40–80 years.

Fifty-six subjects had carotid plaque (27 had low-grade plaques (plaque score < 7.0) and 29 had high-grade plaques (plaque score $\geqslant 7.0$)) and 53 subjects did not have plaques. High pulse wave velocity was found to be significantly associated with existence of carotid plaque ($p < 0.001$), but was not associated with the severity of the plaque ($p = 0.14$) in multivariate logistic regression models. This study shows that the pulse wave velocity is associated with the existence of carotid atherosclerosis, but not with the severity of carotid atherosclerosis, in patients with cerebral thrombosis.

Keywords: Atherosclerosis; Carotid arteries; Ultrasonography; Pulse wave velocity

1. Introduction

Investigation of the relationships between structure and function in the large arteries may lead to a better understanding of the pathophysiology of vascular disease, to improved evaluation of stroke and cardiovascular risks, and thus to the development of more adequate disease prevention strategies. The mechanisms linking arterial stiffness and atherosclerosis are unknown at present.[1–4]

Atherosclerotic changes of the arterial wall include smooth muscle cell proliferation, deposition of lipids and the accumulation of collagen, elastin, and/or proteoglycans. However, it is not known whether (or how) the different stages of plaque development are related to arterial stiffness. Carotid atherosclerosis can be assessed non-invasively using high-resolution B-mode ultrasonography, which gives information about plaque severity, while the arterial pulse wave velocity (PWV) can used for evaluation of arterial stiffness. Recently, a simple and noninvasive method for automatic measurement of the brachial-ankle PWV has been described.[5] The aims of this study were to clarify: (1) the relationship between the presence/absence of carotid plaque and the PWV; and (2) the relationship between the severity of carotid plaque and PWV.

2. Materials and methods

One hundred and nine patients with cerebral thrombosis were recruited from the Department of Neurosurgery at Nippon Medical School, Tamanagayama Hospital, Japan. Cerebral thrombosis was defined as the rapid onset of focal loss of brain function with symptoms lasting for more than 24 hours and with no cause apparent, other than vascular

* Corresponding author. Tel.: +042 371 2111; fax: +81 42 372 7382.
E-mail address: tamakito@nms.ac.jp (T. Tomonori).

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occlusion. Patients with any of the following conditions were excluded: cerebral hemorrhage, transient ischemic attacks, subarachnoid hemorrhage, embolic stroke secondary to heart disease and brain tumors. Based on the clinical characteristics and brain CT and/or MRI findings, patients with cerebral thrombosis were classified into groups with cortical infarction (CI) or subcortical infarction (SCI). The CI group was composed of patients with clinical evidence of cortical deficits in whom CT and/or MRI showed an infarct involving the cerebral cortex in the carotid artery territory without evidence of cardiogenic embolism. The SCI group was composed of patients in whom CT and/or MRI showed a small infarct in the subcortical carotid artery territory that was responsible for the symptoms. Patients with combined cortical and subcortical involvement on imaging were included in the CI group. The age of the patients ranged from 40 to 80 years. We evaluated risk factors that were considered standard in the Framingham study.[6] Hypertension was defined as a systolic blood pressure > 140 mmHg or a diastolic blood pressure > 90 mmHg, or that the patient was using antihypertensive medication. Diabetes mellitus was as defined by the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus.[7] Hyperlipidemia was defined as a fasting serum total cholesterol level > 230 mg/dL, low density lipoprotein (LDL) cholesterol > 140 mg/dL, triglycerides > 150 mg/dL, high density lipoprotein (HDL) cholesterol < 40 mg/dL, or that the patient was using lipid lowering medication. The clinical characteristics of the patients are summarized in Table 1. High-resolution B-mode ultrasonography was performed with a 7.5 MHz duplex scanner (Hitachi EUB-555, Hitachi, Japan). A carotid intima-media thickness (IMT) of < 1.1 mm was defined as indicating the existence of atheromatous plaque. To assess the severity of atherosclerosis, we used a plaque score that was calculated as the total thickness of all plaques in both carotid arteries (Fig. 1).[8,9] We separated low-grade plaques (PI < 7.0) from high-grade plaques (PI ⩾ 7.0). A new device, the AT-form PWV/ABI (Nippon Colin Co., Ltd., Komaki, Japan), was used to simultaneously measure the bilateral brachial and tibial arterial pressure waveforms using a volume-plethysmographic apparatus (Fig. 2).[10] This device records the phonocardiogram, electrocardiogram, pulse volume, and arterial blood pressure at both the left and right arms and ankles. The brachial-ankle PWV was then calculated from the time for the waveform to travel between the right arm and both ankles. The distance between the right

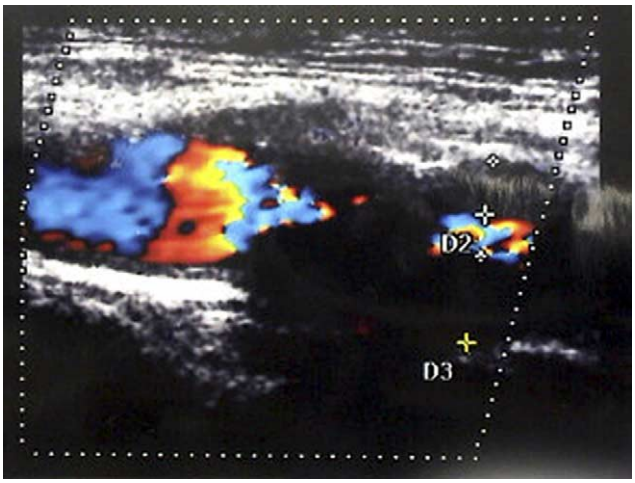

Fig. 1. Ultrasound duplex image shows a carotid plaque and severe stenosis in a 67-year-old man. His plaque score was 9.7.

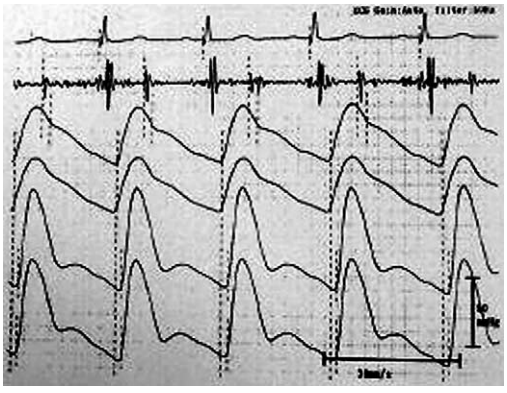

Fig. 2. The pulse wave at bilateral brachial arteries and ankles in a 75-year-old man with a high pulse wave velocity (2954 cm/sec).

Table 1
Clinical characteristics of subjects in three groups

	Carotid plaque			
	None	Low-grade plaque	High-grade plaque	*p*-value
Cerebral thrombosis type (SCI/CI)	26/27	18/9	13/16	0.351
No. of cases	53	27	29	0.120
Age (years)	63 ± 9	66 ± 7	68 ± 7	0.632
Sex (male/female)	39/14	17/10	20/9	0.354
Initial systolic BP	143.5 ± 20.7	140.0 ± 16.6	144.8 ± 20.7	0.157
Hypertension	41	23	25	0.220
Blood cholestrol (mg/dl)	230.0 ± 40.0	231.7 ± 77.9	251.7 ± 56.3	0.164
Hyperlipidemia	39	19	23	0.115
Blood glucose (mg/dl)	117.6 ± 33.7	155.6 ± 60.9	162.3 ± 62.9	0.256
Diabetes mellitus	22	14	15	0.573
BMI	21.1 ± 2.5	20.9 ± 2.3	21.1 ± 2.5	0.438
Smoking history	32	22	25	0.376

Data are mean ± S.D. or case number. BP, blood pressure; BMI, body mass index; CI, cortical infarction; SCI, subcortical infarction.