Brachial–Ankle Pulse Wave Velocity as a Predictor of Silent Cerebral Embolism after Carotid Artery Stenting

Tomoaki Murakami, MD,* Hajime Nakamura, MD, PhD,* Takeo Nishida, MD, PhD,* Tomohiko Ozaki, MD,* Katsunori Asai, MD,* Tomoki Kidani, MD,* Yoshinori Kadono, MD,* Manabu Sakaguchi, MD, PhD,† Toshiki Yoshimine, MD, PhD,‡ and Haruhiko Kishima, MD, PhD*

Background: In neuroendovascular therapy, the effect of arterial stiffness on postprocedural cerebral thromboembolism is unknown. In this observational study, we examined the relationship between cerebral thromboembolism after carotid artery stenting and arterial stiffness. *Methods:* From April 2015 to February 2017, we enrolled consecutive patients undergoing scheduled carotid artery stenting in our institution. In all patients, preprocedural brachial–ankle pulse wave velocity was used to assess arterial stiffness, whereas the number of new cerebral ischemic lesions on diffusion-weighted magnetic resonance imaging was assessed after treatment. We also analyzed patient data and details of procedures in patients with carotid artery stenting. *Results:* Twenty-one patients completed the study. The mean brachial–ankle pulse wave velocity was 1879 cm/s. There was no association of cerebral thromboembolisms with age, unstable plaque, protection device, or type of stent. However, the brachial–ankle pulse wave velocity was an independent predictor of cerebral thromboembolisms ($P = .0017$). *Conclusions:* Brachial–ankle pulse wave velocity is predictive of silent cerebral embolisms on diffusion-weighted magnetic resonance imaging after carotid artery stenting.
Key Words: Neuroendovascular therapy—arterial stiffness—silent cerebral embolism—asymptomatic ischemic lesions—carotid artery stenosis—carotid artery stenting—baPWV—predictor.

Introduction

Arterial stiffness was previously reported to be an independent indicator of neurovascular and cardiovascular diseases and events.[1-4] Several methods have been recently developed for the assessment of arterial stiffness, including brachial–ankle pulse wave velocity (baPWV), which is widely used in clinical practice. PWV is calculated as the velocity of the pulse wave between 2 sites of the artery, and provides a measure of arterial stiffness.[5,6] Arterial stiffness assessed using baPWV is particularly useful in large populations, as it is convenient and noninvasive.[7-9] Neuroendovascular therapy is now widely accepted as an alternative to surgery, as technological improvements in neuroendovascular therapy and devices have markedly improved its safety. However, cerebral

From the *Department of Neurosurgery, Osaka University Graduate School of Medicine, Suita, Osaka 565-0871, Japan; †Department of Neurology, Osaka University Graduate School of Medicine, Suita, Osaka 565-0871, Japan; and ‡Global Center for Medical Engineering and Informatics (MEI Center), Osaka University, Suita, Osaka 565-0871, Japan.

Received March 9, 2017; revision received May 8, 2017; accepted May 17, 2017.

Address correspondence to Tomoaki Murakami, MD, Department of Neurosurgery, Osaka University Graduate School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan. E-mail: mmtmtomoaki@gmail.com.

1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2017.05.021

thromboembolism remains a major complication of neuroendovascular therapy.

Progressive arterial stiffness was reported to be an independent predictor for risk of symptomatic stroke.[6] However, the relationship between arterial stiffness and cerebral thromboembolism following neuroendovascular therapy remains unclear. Thus, the aim of the present study was to examine the relationship between cerebral thromboembolism events after neuroendovascular therapy and arterial stiffness using baPWV. In particular, as patients with carotid artery stenosis were reported to exhibit advanced arterial stiffness,[10,11] we examined this relationship for patients with carotid artery stenting (CAS).

Materials and Methods

Ethics approval for the study was obtained by the Osaka University Hospital Institutional Review Board (Approval Number: 15237).

Participants and Data Collection

We prospectively enrolled 21 consecutive adult patients scheduled for CAS in the Department of Neurosurgery at Osaka University Hospital between April 2015 and February 2017. We measured preprocedural baPWV in all patients. Patient data including age, sex, body mass index, and personal medical history of hypertension, dyslipidemia, diabetes, current or past smoking habits, preoperative blood pressure, heart rate, and laboratory data were assessed in patients undergoing CAS using medical records. Plaque details were recorded according to plaque imaging with magnetic resonance imaging (MRI) and angiograms. Access route, aortic arch plaque, and details of the carotid stenosis lesion (lesion morphology, North American Symptomatic Carotid Endarterectomy Trial %, and lesion length) were assessed with a preprocedural diagnostic angiogram. Details of the procedure, including anesthesia, sheath size, catheter size, distal embolic protection device, method of proximal protection, stent type, number of new cerebral ischemic lesions on diffusion-weighted (DW)-MRI, and the modified Rankin Scale at discharge, were also collected.

BaPWV Measurement

BaPWV was measured using a volume-plethysmographic apparatus (BP-203RPE III; Colin-Omron, Co, Ltd, Tokyo, Japan) before the CAS procedure. This device simultaneously records PWV, blood pressure, electrocardiogram, and heart sounds. Each patient lay in the supine position and was examined using electrocardiographic electrodes attached to both wrists, a microphone for detecting heart sounds placed on the left edge of the sternum, and cuffs wrapped around both brachia and both ankles. After a few minutes' rest, the patients' pulse volume waveforms at the brachium and ankle were recorded. The time delay between the wave front of the brachial waveform and that of the ankle waveform was measured (ΔTba). The distance between each sampling point and the heart of each patient was calculated automatically using the individual height. BaPWV was calculated as the difference between the heart–ankle distance and the heart–brachium distance, divided by ΔTba.[9] After examination of both the left and the right sides, the higher baPWV was chosen for statistical analyses.[12]

MRI and Magnetic Resonance Angiography

MRI was scheduled at 1 day after CAS. Imaging was performed on either a 1.5-T system (Ingenia; Philips Healthcare, Best, the Netherlands), a 3.0-T Discovery system (Discovery MR750 and MR750w; GE Healthcare, Waukesha, WI), or a 3.0-T Achieva system (Achieva; Philips Healthcare). New ischemic lesions after CAS in the whole brain were evaluated with DW-MRI and apparent coefficient maps by experienced neuroradiologists who were blinded to all clinical details, and were compared with preprocedural MRI. Similarly, preprocedural assessment of carotid plaques in patients undergoing CAS was performed using MRI plaque imaging, which included time-of-flight imaging and T1- or T2-weighted black-blood imaging. If hyperintense lesions on time-of-flight imaging or T1- or T2-weighted black-blood imaging were detected in the CAS lesion, this lesion was considered an unstable plaque.

CAS Procedure

Acetyl salicylic acid (100 mg/d) and clopidogrel (75 mg/d) or cilostazol (200 mg/d) were administered for a minimum of 7 days before CAS. Access route to the carotid stenotic lesion was assessed carefully by angiogram and computed tomography angiogram before the procedure, and the most appropriate access route was selected, including the brachial approach and the femoral approach. We used local anesthesia unless there was a particular reason for sedation, such as the patients could not be kept at rest. Where possible, a transcranial Doppler was attached to each patient's head. An experienced neuroendovascular team performed all CAS procedures. A heparin bolus was administered after sheath insertion to increase the activated clotting time to >300 seconds. A guiding catheter was introduced into the common carotid artery proximal to the carotid stenosis. Two types of distal embolic protection devices were used: the GuardWire (Medtronic, Santa Rosa, CA) or the FilterWire EZ (Boston Scientific, Natick, MA). Predilatation of the stenotic lesion was performed with a suitable balloon catheter. Two different types of stents were placed into the stenotic lesion: the Precise (Johnson & Johnson, Cordis, Minneapolis, MN) or the Wallstent (Boston Scientific). Postdilatation was performed with an appropriate balloon catheter.