The Association between Hyperintense Vessel Sign and Final Ischemic Lesion Differ in Its Location

Tomoyuki Kono, MD, PhD,* Hiromitsu Naka, MD, PhD,† Eiichi Nomura, MD, PhD,† Naohisa Hosomi, MD, PhD,* Shiro Aoki, MD, PhD,* Eiji Imamura, MD,† Yoshimasa Sueda, MD, PhD,* Tomohisa Nezu, MD,* Tomoya Mukai, MD,* Tomohiko Ohshita, MD, PhD,† Toshiho Ohtsuki, MD, DMSc,* Shinichi Wakabayashi, MD, PhD,‡ and Masayasu Matsumoto, MD, PhD*

Background: The hyperintense vessel sign (HVS) on fluid-attenuated inversion recovery images can frequently be detected in patients with acute cerebral infarction attributable to large artery stenosis or occlusion. The prognostic values and clinical characteristics of HVS remain to be elucidated. The aim of this study was to evaluate the association of HVS with ischemic lesions and severity of neurologic deficit. *Methods:* A total of 96 consecutive acute ischemic stroke patients (54 women, median age 76.5 [range 39-97] years), who had symptomatic severe stenosis or occlusion in the proximal middle cerebral artery that was detected with magnetic resonance angiography within 24 hours of onset, were enrolled. The extent of HVS was graded by a systematic quantitative scoring system (the HVS distribution score) based on Alberta Stroke Program Early Computed Tomographic Score. *Results:* An HVS was detected in 89 patients (93%) at admission, and the patients who displayed wider HVS distribution scores exhibited more severe neurologic deficits at admission ($P < .05$). The follow-up magnetic resonance imaging, which was obtained in 79 patients (82%), was performed an average of 13 days. The association between HVS distribution score and final ischemic lesions was strongly observed ($n = 67$, $P < .05$) but not in the patients with intravenous thrombolysis ($n = 12$, $P = .06$). *Conclusions:* Although the distribution of HVS reflected final ischemic lesion, this association might not apply to the patients with the thrombolysis treatment. The interpretation of HVS distribution score with acute ischemic stroke patients should be discussed dependent on thrombolysis. **Key Words:** Hyperintense vessel sign—acute ischemic stroke—National Institutes of Health Stroke Scale—Alberta Stroke Programme Early CT Score.

Introduction

Hyperintense vessel sign (HVS) on fluid-attenuated inversion recovery (FLAIR) images obtained using magnetic resonance imaging (MRI) can frequently be detected in the major cerebral artery, especially the middle cerebral artery (MCA), of acute ischemic stroke patients.[1-6] HVS is described as focal, tubular, or serpentine hyperintensities that are commonly observed transiently

From the *Department of Clinical Neuroscience and Therapeutics, Hiroshima University Institute of Biomedical and Health Sciences, Hiroshima; †Department of Neurology, Suiseikai Kajikawa Hospital, Hiroshima; and ‡Department of Neurosurgery, Suiseikai Kajikawa Hospital, Hiroshima, Japan.

Received October 9, 2013; revision received November 11, 2013; accepted November 11, 2013.

Conflict of interest: All authors declared no conflict of interest.

Address correspondence to Naohisa Hosomi, MD, PhD, Department of Clinical Neuroscience and Therapeutics, Hiroshima University Institute of Biomedical and Health Sciences, 1-2-3 Kasumi, Minami-ku, Hiroshima 734-8551, Japan. E-mail: nhosomi@hiroshima-u.ac.jp.

1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2013.11.009

in the subarachnoid space. Although HVS has been reported to reflect sluggish or disordered blood flow through the vessels, the exact mechanism responsible for the appearance of HVS and the clinical implications of HVS detection have not been clearly defined. HVS is reported as an indicator of slow blood flow and early ischemia as a result of large-vessel occlusion or stenosis and inadequate collateral circulation.[2] Sanossian et al[7] conducted conventional angiography on acute ischemic stroke patients with HVS, and they noted that HVS was associated with a high grade of collateral circulation. Although the presence of HVS can indicate the presence of collateral flow, collateral flow may not completely account for the presence of HVS because HVS is not observed in all the cases with collateral flow.

The prognostic values and clinical characteristics of HVS remain to be elucidated. Several reports have shown that the presence of HVS is associated with a larger infarct volume and a higher National Institutes of Health Stroke Scale (NIHSS) score.[2,8] However, Lee et al[9] reported that the presence of HVS on the distal MCA was associated with a smaller infarct size and lesser initial stroke severity in the patients with proximal MCA occlusion who underwent intravenous thrombolysis.

We have hypothesized that there may be an association of HVS with ischemic lesion and severity of neurologic deficits. Additionally, those associations may be different depending on HVS location and the intravenous thrombolysis treatment.

Methods

Study Design

The hospital review board of Suiseikai Kajikawa Hospital approved the study protocol. We prospectively evaluated consecutive acute ischemic stroke patients who were admitted to Suiseikai Kajikawa Hospital between January 2008 and December 2010. We included patients (n = 188) who had symptomatic severe stenosis or occlusion in the MCA (M1 or M2 portion) that was detected with MRI and magnetic resonance angiography (MRA) within 24 hours from the stroke onset. Symptomatic severe stenosis and occlusion, which corresponded to the patient's neurologic deficits, were evaluated based on MRA. To analyze HVS, we excluded patients with occlusion of the internal carotid artery proximal to the symptomatic intracranial ischemic lesion (n = 63) and patients with low-quality MR images (n = 29). After the exclusions, this study included 96 patients with MCA severe stenosis or occlusion and acute ischemic stroke in the area of interest. Follow-up MRI was performed approximately 14 days after the first MRI to evaluate the sizes of the final ischemic lesions. Ischemic stroke subtypes were classified by 2 stroke neurologists using the Trial of Org 10172 in Acute Stroke Treatment criteria.[10] The clinical severity of the ischemic stroke was evaluated using the NIHSS at admission.

MRI Protocol

All the MRI studies were performed with two 1.5-T MRI scanners (Magnetom Symphony Advanced or Avanto, Siemens Medical Systems, Erlangen, Germany). The baseline and follow-up MRIs were performed using diffusion-weighted imaging (DWI), FLAIR imaging, and intracranial MRA. DWI was performed using the echo-planar spin-echo imaging technique with the following parameters: the repetition time was 3500 ms, the echo time was 98 ms, the b value was 1000 s/mm^2, the slice thickness was 5 mm, the matrix size was 256 × 256, and the field of view was 230 mm. Ultimately, 16-20 contiguous axial sections parallel to a line through the anterior and posterior commissures were obtained. The FLAIR images were acquired with commercially available 2-dimensional sequences and balanced across field strength for the ability to identify the chronic ischemic parenchyma. The FLAIR images were obtained with the following parameters: the repetition time/echo time = 10,000/107 ms, the inversion time = 2500 ms, and 20 continuous slices with .9 × .9 × 7-mm resolution for a total acquisition of 2 minutes and 25 seconds. MRA was obtained using a 3-dimensional time-of-flight gradient-echo technique for intracranial arteries.

Imaging Analysis

The presence of MCA severe stenosis or occlusion was assessed using MRA. Severe stenosis was defined as a signal loss on MRA, and occlusion was defined as the lack of a signal in the distal artery on MRA. The distribution of ischemic lesions at admission was evaluated using the Alberta Stroke Program Early Computed Tomographic Score (ASPECTS) in 10 regions with DWI (DWI-ASPECTS).[11] A template of 2 axial DWI slices with markers for the 10 regions being scored by the DWI-ASPECTS was provided. The distribution of the final infarct lesions in the subacute stage was scored based on FLAIR images according to the ASPECTS (FLAIR-ASPECTS). The FLAIR-ASPECTS was carefully estimated, considering that there was a difference between white matter lesions before stroke onset and final infarct lesions. HVS was defined as a linear or serpentine-appearing hyperintensity in the subarachnoid space on FLAIR images that corresponded to a typical arterial course. The extent of HVS was graded by a systematic quantitative scoring system (the HVS distribution score, Fig 1). The HVS-positive area was divided into the Sylvian fissure (S) and 6 MCA perfusion areas (M1-M6) that coincided with the cortex based on the ASPECTS divisions. When HVS was detected, regions that were identical to the ASPECTS divisions were counted to yield an HVS distribution score. In this study, the maximal HVS distribution score was 7, which indicated that HVS was detected in all MCA areas including the Sylvian fissure (S and M1-M6 areas based on ASPECTS division). An