Rapid Identification of Type A Aortic Dissection as a Cause of Acute Ischemic Stroke

Tomoyuki Ohara, MD,* Masatoshi Koga, MD,† Naoki Tokuda, MD,* Eijirou Tanaka, MD,* Hiroyuki Yokoyama, MD,‡ Kenji Minatoya, MD,§ Kazuyuki Nagatsuka, MD,‖ Kazunori Toyoda, MD,* and Kazuo Minematsu, MD*

Background and Purpose: Patients with acute aortic dissection (AAD) sometimes present predominantly with neurological symptoms from cerebral ischemia. Such stroke patients must not receive thrombolysis therapy, which can be fatal. However, patients remain at risk if there is a failure to notice concurrent AAD. We aimed to clarify the characteristics of AAD patients with stroke to identify markers for early AAD detection before thrombolysis. *Methods:* Using the single-center database of Stanford type A-AAD patients between 2007 and 2013, we selected those presenting with acute focal neurological deficits, presumably due to cerebral ischemia. Results of physical, radiological, and blood examinations were assessed in AAD patients with stroke. *Results:* Of 226 AAD patients, 23 (10%) had stroke secondary to AAD. Of the 23 patients, 21 (91%) were primarily examined by stroke physicians and 2 (9%) by cardiologists. Thirteen patients (57%) were potential candidates for intravenous thrombolysis. Only 11 patients (48%) complained of chest/back pain. Positive findings indicating AAD included occlusion or intimal flap of the common carotid artery on carotid ultrasound in 18 (90%) of 20 patients, elevated serum D-dimer values (≥6.9 μg/mL) in 18 (78%) of 23, left hemiparesis as a neurological symptom in 17 (74%) of 23, systolic blood pressure differential above 20 mmHg between the arms in 15 (71%) of 21 patients, and mediastinal widening on chest radiograph in 10 (67%) of 15 patients. All 14 patients who underwent complete evaluation showed 2 or more positive diagnostic findings. *Conclusions:* The combination of physical, radiological, and laboratory findings may be a useful rapid-screening method for AAD as a cause of acute ischemic stroke. **Key Words:** Aortic dissection—thrombolysis—stroke—carotid ultrasound.
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Introduction

Stroke is one of the most serious complications of Stanford type A acute aortic dissection (AAD). A large international AAD registry demonstrated that stroke occurs in 6% of patients with type A AAD, and these patients have poor outcomes compared to those without stroke.[1] AAD patients with stroke sometimes present predominantly with neurological symptoms and not with the typical chest or back pain. This can lead to a delayed diagnosis of AAD.[2,3] Importantly, it has been reported that some patients with stroke secondary to AAD received thrombolysis treatment without prior diagnosis, thus resulting in fatalities.[3-5] Within 1.5 years of the approval of intravenous thrombolysis in Japan, there were 10 reports of

From the *Department of Cerebrovascular Medicine; †Division of Stroke Care Unit; ‡Department of Medical Risk Management; §Department of Cardiovascular Surgery; and ‖Department of Neurology, National Cerebral and Cardiovascular Center.

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Address correspondence to Tomoyuki Ohara, MD, Department of Neurology, Kyoto Prefectural University of Medicine, 465 Kajiicho, Kamigyo-ku, Kyoto 602-0841, Japan. E-mail: ohar21@gmail.com.

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fatalities in stroke patients who received thrombolysis with unrecognized concurrent AAD.[5]

The American Heart Association and other professional societies have proposed a highly sensitive clinical tool that includes pain assessment to identify patients with a high probability of AAD.[6,7] However, this tool seems to be less sensitive in AAD patients with stroke because they more frequently present without acute chest or back pain than AAD patients without stroke.[3,8,9] Therefore, it is critical to establish a rapid-screening method for AAD in possible candidates for thrombolysis.

The purpose of our study was to clarify the clinical and radiological features of stroke patients secondary to AAD to identify useful findings for early AAD detection. This will aid decisions on whether thrombolysis treatment is appropriate for patients with potentially unrecognized AAD.

Methods

A total of 226 consecutive Stanford type A-AAD patients within 14 days after onset were admitted to our hospital between 2007 and 2013. The diagnosis of type A AAD was confirmed by contrast-enhanced computed tomography (CT) and/or operative findings. We retrospectively reviewed the medical records to determine the clinical, radiological, and laboratory features in AAD patients. After excluding 45 patients presenting with a shock state or cardiopulmonary arrest because we could not confirm the concurrence of stroke due to global brain ischemia, we selected AAD patients with stroke. Stroke was defined as a focal neurological deficit, presumably due to cerebral ischemia, regardless of the presence of acute ischemic lesions on brain CT or magnetic resonance imaging. We also excluded patients presenting with syncope only. Involvement of dissection in 1 or more major branches of the aortic arch (arch involvement) was evaluated by contrast-enhanced CT or operative findings. Possible candidates for thrombolysis were defined as AAD patients with stroke who presented to our hospital within 4.5 hours after stroke onset and who would receive thrombolysis based on clinical and radiological findings if AAD was misdiagnosed. The present retrospective study was approved by the National Cerebral and Cardiovascular Center's ethics committees and was conducted in accordance with the institutional guidelines.

We focused on the positivity of the following 5 diagnostic findings rapidly available at presentation in AAD patients with stroke: (1) left hemiparesis as a neurological symptom, (2) systolic blood pressure (SBP) differential above 20 mmHg between the arms, (3) mediastinal widening on chest radiograph, (4) intimal flap and/or no flow signal of the common carotid artery (CCA) on carotid duplex ultrasound, and (5) abnormal elevation of D-dimer values. All chest radiographs were taken in a supine anteroposterior manner. Mediastinal widening on chest radiograph was defined as a maximal mediastinal width more than 8.65 cm, which was the optimal cutoff value for the diagnosis of AAD in anteroposterior projection according to a previous study.[10] The cutoff value of SBP differentials, 20 mmHg, was determined according to the 2010 guidelines of the American Heart Association and other clinical societies.[6] Carotid ultrasound was performed in the emergency room in a short time, focusing on the exclusion of AAD. The presence of intimal flap or double lumen sign in the CCA, a direct finding of arterial dissection, and no flow signal in the CCA, suggesting the occlusion of the proximal portion of the CCA or brachiocephalic artery, were evaluated as indicators of AAD. D-Dimer values were measured by a latex agglutination immunoturbidimetric assay (Sekisui Medical Corporation, Ltd., Tokyo, Japan). The upper limit of the normal value was 1.0 μg/mL. Abnormal elevation of D-dimer values was defined as values of more than 6.9 μg/mL, which was the cutoff value for the diagnosis of AAD in patients with acute stroke in a previous study.[11]

Results

Of the 226 AAD patients, we identified 23 AAD patients with stroke. The clinical and radiological features of AAD with stroke are shown in Table 1. Less than half (48%) of the patients complained of chest or back pain. Arch involvement was observed in all patients. The brachiocephalic artery (100%) and the right CCA (83%) were predominantly involved. CT and/or magnetic resonance imaging at presentation revealed acute ischemic lesions in 13 patients (right internal carotid artery territory in 9 patients, left internal carotid artery territory in 1 patient, and 2 or more vascular territories in 3 patients). In the remaining 10 patients, acute ischemic lesions were not identified because of transient ischemic attack in 3 patients, early presentation of patients after stroke onset in 4 patients, or brain imaging not being performed because of emergency surgery in 3 patients. Most AAD patients with stroke (91%) were primarily examined by stroke physicians and only 9% by cardiologists, whereas 98% of AAD patients without stroke were primarily examined by cardiologists. Thirteen patients (57%) were possible candidates for thrombolysis. One of these patients actually received thrombolysis, which eventually resulted in death (Fig 1).

A summary of diagnostic findings for AAD with stroke is shown in Table 2. Carotid ultrasound evaluation was performed in 20 of 23 patients, with 18 (90%) of the 20 patients revealing abnormal findings indicative of AAD. Intimal flap in CCA was observed in 15 patients, and occlusion of the CCA proximal portion was observed in 5 patients. On the other hand, bilateral CCAs were intact on carotid ultrasound examination in 2 patients. All patients had elevated D-dimer values at presentation (median = 29.7 μg/mL, range: 4.2-406.2 μg/mL). Abnormal

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