Journal of the Neurological Sciences 388 (2018) 23–27

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Cerebrovascular imaging of cerebral ischemia in acute type A aortic dissection

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ARTICLE INFO

Keywords:
Aortic dissection
Stroke
Imaging
MRI
Thrombolysis
Cerebral infarction

ABSTRACT

Background and purpose: Stanford type A aortic dissection (AAD) sometimes causes acute ischemic stroke (AIS) or transient ischemic attack (TIA). There is little understanding of cerebrovascular imaging of AIS or TIA in patients with AAD.

Methods: Consecutive AIS/TIA patients with AAD who were admitted within 4.5 h of onset were reviewed. We compared findings of MRI/MRA between these and consecutive AIS/TIA patients without AAD within 4.5 h of onset.

Results: Seventeen AAD and 249 non-AAD patients were identified. Compared to non-AAD patients, AAD patients had infarcts more frequently in the right anterior cerebral artery (ACA) territory (18% vs. 2%, $P = 0.007$) and the right middle cerebral artery (MCA) territory (71% vs. 29%, $P < 0.001$). There was no difference between the groups regarding whether it was perforator or cortical infarct, single or multiple infarcts, unilateral or bilateral infarcts, or ischemic change extension. On the MRA imaging, the AAD patients more frequently had poor visualization of the right internal carotid artery (ICA) (47% vs. 6%, $P < 0.001$). After adjustment for sex, age and confounding factors, the right ACA territory infarct [odds ratio (OR), 12.2; 95% confidence interval (CI), 1.4–119.4], the MCA territory infarct (OR, 4.9; 95% CI, 1.0–25.0) and poor visualization of the right ICA (OR, 18.1; 95% CI, 4.0–101.9) were independently associated with AAD.

Conclusion: In emergency AIS/TIA patients, right anterior circulation infarct and poor visualization of the right ICA on cerebrovascular imaging are potential imaging markers of AAD.

1. Introduction

Stanford type A aortic dissection (AAD) is a life-threatening disease [1–3]. Patients with AAD often experience neurological symptoms [2–4], and sometimes suffer from stroke. The International Registry of Acute Aortic Dissection (IRAD), a multicenter study, showed that ischemic stroke occurred in 6% of patients in the AAD cohort [3]. We recently reported that AAD were detected in 1.7% of patients who needed acute stroke care management [5]. Neurological symptoms in patients with acute ischemic stroke (AIS) or transient ischemic attack (TIA) pose a challenge as they may obscure typical AAD symptoms such as chest or back pain [2]. Patients with neurological symptoms have a markedly poorer prognosis [2,4,6], possibly missing optimal therapy due to delays in identifying AAD. In addition, AAD in AIS/TIA patients is a contraindication for thrombolytic therapies [7–9], such as intravenous recombinant tissue plasminogen activator (IV rt-PA).

Useful markers or findings are needed to detect AAD in the Emergency Department. In recent studies, plasma D-dimer level, chest

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https://doi.org/10.1016/j.jns.2018.02.044
Received 26 November 2017; Received in revised form 6 February 2018; Accepted 26 February 2018
Available online 27 February 2018
0022-510X/

X-ray and blood pressure abnormalities have been shown useful to detect AAD [10–12]. It has also been reported that carotid ultrasound can detect stroke with AAD based on intimal flap of the common carotid artery (CCA) [13–15]. AAD patients with AIS/TIA are likely to have a right hemispheric event [16]. However, detailed information of common cerebrovascular imaging in patients with AIS/TIA has not been published. Therefore, we investigate the typical cerebrovascular imaging features in AIS/TIA patients with AAD.

2. Material and methods

2.1. Study population

The study compared two groups, AAD and non-AAD patients. The former included consecutive AIS/TIA patients with AAD who were admitted to our institute with neurological signs/symptoms and evaluated with head MRI within 4.5 h of onset from January 2007 to December 2015. The latter included consecutive AIS/TIA patients without AAD who were admitted within the same time window of onset from January 2013 to December 2013. In our emergency department, acute stroke patients were basically assessed by head magnetic resonance imaging (MRI) before IV rt-PA therapy. All patients had bilateral blood pressure measurement of arms and chest X-ray. The diagnosis of AAD was finally confirmed by enhanced-contrast CT after cerebrovascular imaging. Patients were retrospectively identified from the National Cerebral and Cardiovascular Center (NCVC) stroke registry (ClinicalTrials.gov identifier: NCT02251665) and hospital databases. We excluded AAD patients with cardiac pulmonary arrest or near such a state on arrival to the Emergency Department. We also excluded patients who arrived > 4.5 h after onset and those whose condition was too severe to undergo head MRI.

2.2. Data collection and definition

The collected data included clinical history and presentation, laboratory data, imaging data, treatment, and prognosis. AAD was confirmed by either identifying ascending aorta dissection on chest to abdominal CT (plain or enhanced) or surgical findings. AIS was defined as the persistence of neurological symptoms over 24 h whilst compatible with neuroimaging findings. According to the TOAST (Trial of Org 10,172 in Acute Stroke Treatment) classification [17], stroke etiology was determined in patients without AAD. TIA was defined as transient focal neurological symptoms considered to be of ischemic vascular etiology and with clinical symptoms lasting < 24 h. Mediastinal widening on chest X-ray was defined as a maximal mediastinal width > 86.5 mm, as per a previous study [12]. Systolic blood pressure differential between arms was defined as over 20 mmHg, according to the 2010 guidelines of the American Heart Association and other clinical societies [7]. 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.

2.3. Imaging techniques and classification

All patients underwent 1.5- or 3-tesla MRI in our institute or referral hospitals. We confirmed the findings in conjunction with hospital records. Three-dimensional (3D), time-of-flight magnetic resonance angiography (MRA) of the intra-cranial arteries was performed. Acute ischemic lesions were determined by diffusion weighted imaging (DWI). Infarct location was defined according to the vascular territory: middle cerebral artery (MCA); anterior cerebral artery (ACA); posterior circulation including posterior cerebral artery, basilar artery, and vertebral artery. Based on the imaging, the ischemic change extension was measured using the Alberta Stroke Program Early CT score (DWI-ASPECTS) system (Online supplement) [18,19], noting whether it was either a perforator or cortical lesion, single or multiple infarction(s) and unilateral or bilateral infarct(s). We also measured infarct size (maximum infarct diameter). Visualization of the internal carotid artery (ICA) and vertebral artery to basilar artery (VA-BA) on MRA was also evaluated. Poor visualization of the artery was defined as gross artery signal attenuation when compared to other arteries such as the contralateral artery. We subclassified the ICA poor visualization into two types as decreased or absent signal intensity.

2.4. Statistics

Data are shown as numbers and percentages, mean ± standard deviation (SD), or medians with first and third quartiles. We examined the differences between the groups using Fisher's exact-test for categorical data and Welch's t-test for continuous variables. Logistic regression was used for multivariable analyses of risk factors pertaining to the AAD. Sex, age, and variables with a P value < 0.1 were included simultaneously as confounding factors. Sensitivity, specificity, and area under the curve (AUC) of the receiver operating characteristic (ROC) were evaluated for the independent imaging markers to determine the presence of AAD. All P values were 2-sided, with values < 0.05 considered statistically significant. Statistical analyses were performed using JMP 11.0 (SAS Institute, Cary, NC).

3. Results

3.1. General characteristics and outcomes at discharge

We collected 357 consecutive AAD patients from the NCVC stroke registry and hospital database. Thirty-three were AIS/TIA patients, regardless of admission time window of onset. Of these 33 patients, 28 were admitted to our hospital within 4.5 h of onset and of these 17 underwent head MRI/MRA. The remaining 11 patients, examined only by CT mainly due to unstable condition, were excluded. In this AAD group, there were 15 women (88%), and the mean age was 77 ± 12 years. We also collected 258 AIS/TIA patients without AAD within the same time window of onset from the NCVC stroke registry. Of these, 249 patients underwent head MRI/MRA. The remaining 9 patients, examined only by CT, were excluded. There were 99 women (40%) and the mean age was 73 ± 13 years. We found significant differences in sex, initial systolic and diastolic blood pressure levels, systolic blood pressure differential, the side of hemiparesis, the presence of consciousness disturbance, mediastinal widening and atrial fibrillation between the 17 patients in the AAD group and 249 patients in the non-AAD group (Table 1). Only 5 AAD patients (29%) had chest or back pain. According to the TOAST classification [17], cardioembolism was found in 91 patients (47%), small-vessel occlusion in 20 (10%), large-artery atherosclerosis in 28 (14%), other determined etiology in 24 (13%), and undetermined etiology in 32 (16%) of the 195 patients without AAD diagnosed as AIS. The remaining 54 patients without AAD were diagnosed as TIA. The AAD group had fewer patients with favorable functional outcome defined as modified Rankin Scale of 0 to 2 [12% (2/17) vs. 60% (150/249), $P = 0.004$], and more deaths [29% (5/17) vs. 6% (14/249), $P < 0.001$] at discharge when compared to the non-AAD group.

3.2. Cerebrovascular imaging

Seventeen patients in the AAD group and 249 in the non-AAD group were examined by head MRI and MRA. The main findings are listed in Table 2. Compared to the non-AAD group, patients in the AAD group had infarcts more frequently in the right ACA territory [18% (3/17) vs. 2% (4/249), $P = 0.007$] and the right MCA territory [71% (12/17) vs. 29% (71/249), $P = 0.002$]. There was no significant difference of infarct location in the left MCA territory (18% vs. 38%), the left ACA territory (0% vs. 3%), or the posterior circulation (7% vs. 16%). There was no difference of the infarct distribution regarding whether