The Impact of Absent A1 Segment on Ischemic Stroke Characteristics and Outcomes

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Background: A1 segment is the proximal portion of anterior cerebral artery. Absence of the A1 segment can compromise anterior cerebral collateral blood flow. Few studies have examined the association of an absent A1 segment and ischemic stroke outcome. We sought to determine the association between A1 absence and affected vessel territory, stroke volume, and outcomes among patients with acute ischemic stroke (AIS). *Methods:* A retrospective review of prospectively identified patients with AIS from July 2008 to March 2013 was performed. Patients without intracranial vascular imaging were excluded. We compared patients with absent A1 to patients with bilateral A1 segments in terms of demographics, stroke severity (as measured by National Institute of Health Stroke Scale [NIHSS]), vascular distribution, and in-hospital mortality using the chi-square test and logistic regression. *Results:* Of the 1146 patients with AIS and intracranial vascular imaging, 5.9% patients (n = 68) had absent A1. Compared with other AIS patients, those with absent A1were older (65 vs. 63 years old, respectively, $P = .016$). There was no difference between groups in terms of the vascular distribution or the side of the stroke. The median volume of the infracted tissue was similar across the groups even when it was stratified according to the Treatment of Acute Stroke Trial classification. Patients with an absent A1 had twice higher odds of in-hospital mortality (odds ratio, 2.4; 95% confidence interval, 1.1-5.2; $P = .028$); however, significance was lost after adjusting to age, NIHSS at baseline, and glucose on admission. Other outcome measures were similar across the groups. *Conclusions:* In our sample, patients with an absent A1 segment did not have a specific vascular distribution, larger infarct volume, or worse outcomes. **Key Words:** Stroke—ischemic—cerebral circulation—mortality—magnetic resonance angiography—computed tomography angiography.

Introduction

Anatomically intact circle of Willis is associated with better outcome in patients with acute ischemic stroke (AIS).[1] Because only 42%-50% of healthy individuals are believed to possess a complete circle of Willis configuration, it is made apparent that variations in circle of Willis anatomy and ultimately collateral capacity are relatively common in the population.[2-4]

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Previous anatomic and radiographic studies have indicated that congenital abnormalities of the A1 segment are commonly observed. The reported prevalence of hypoplasia of the A1 segment in healthy individuals varies between 1% and 13%.[4-7] The complete absence of the A1 segment is much less common than A1 segment hypoplasia, ranging from 1% to 6%.[5,8-10]A prior study has demonstrated that the incidence of A1 segment hypoplasia is significantly higher in patients with AIS than in healthy controls, suggesting that impaired collateral blood flow due to A1 segment hypoplasia may be a contributing factor for AIS.[5] Collateral support in the anterior portion of the circle of Willis is provided through interhemispheric blood flow across the anterior communicating artery providing the opportunity for reversal of flow in the proximal anterior cerebral artery (ACA) in the setting of internal carotid artery (ICA) occlusion, adding to cerebral blood flow provided by the existing extracranial collaterals.[11,12] Previous studies have shown that in the case of absent A1, both ACAs are perfused by 1 ICA with associated lower flow rate through the other ICA on the side of absent A1.[13] Several computational models of cerebral flow dynamics have shown that A1 absence had the most considerable negative impact on blood flow of any common circle of Willis anomaly.[13-15]

In the light of the previous research, we hypothesized that absent A1 segment is associated with higher rates of ACA territory infarcts, larger stroke volumes, and worse outcomes.

Methods

Patients

We conducted a retrospective analysis on prospectively identified patients who presented consecutively to our medical center with AIS between July 2008 and March 2013. Patients who did not receive cerebrovascular imaging during their clinical course (magnetic resonance angiography [MRA], time-of-method [TOF], or computed tomographic angiography [CTA]) were excluded from analysis. Clinical variables were abstracted from our prospective institutional review board–approved stroke registry.[16]

Definitions

Using MRA and CTA, we classified patients into 2 groups: patients with an absent A1 and patients who had intact bilateral A1 segments. An intact A1 segment was defined as an A1 segment that extended from the ICA to the ipsilateral ACA (A2 segment). Complete absence of the A1 segment was indicated in patients with lack of communication between the ICA and the ipsilateral ACA. Patients with hypoplastic A1 segment with atretic communication between ICA and ACA were classified as intact A1. The categorization of stroke etiology was determined using Treatment of Acute Stroke Trial [TOAST] classification.[17] Patients who were discharged home and to inpatient rehabilitation were considered to have a favorable discharge disposition. Neuroworsening was defined as an increase in National Institute of Health Stroke Scale [NIHSS] by 2 points within 24 hours.[18]

Imaging

CTA and MRA were interpreted by a trained research fellow (A.S.). A1 classification was confirmed by a board certified vascular neurologist (S.M.S.). We used the image processing program Centricity PACS (GE Healthcare Milwaukee, WI) to calculate the stroke volume for patients with absent A1 segment and a random sample of patients with intact A1 segment. The infarct area was manually tracked using the region of interest tool on each diffusion-weighted imaging slice, then multiplied by the slice thickness.

Statistical Analysis

We compared admission demographics, initial stroke severity as measured by the baseline NIHSS, vascular distribution of infarction, TOAST classification, and outcomes in patients with absent A1 and patients with intact A1. Categorical data were compared using the Pearson chi-square or Fisher exact test, where appropriate. Continuous data (presented as medians with ranges) were compared using Wilcoxon rank sum test. All tests were performed at the $\alpha = .05$ level and were 2-sided. As this was an exploratory analysis, no adjustments were made for multiple comparisons.[19] The retrospective chart review was approved by the Institutional Review Board at the Tulane University (IRB protocol number 297713-1).

Results

Of 1243 AIS patients who presented to our hospital during the study period, 1146 patients met the inclusion criteria. Of these, 44.2% (507 of 1146) were women and 66.3% (760 of 1146) were black. The median age was 63 years. In our sample, we detected absent A1 in 5.9% (68 of 1146). Of these, 44 were on the right side and 24 on the left side.

MRA was performed in 1064 patients; of these, 427 patients also had CTA conducted during their hospital stay. MRA was able to correctly identify 25 of 25 absent A1 segments that were detected using CTA and correctly ruled out absent A1 in 399 of the 402 ruled out by CTA. Using CTA as the gold standard, the sensitivity of MRA for detecting absent A1 in our sample was 100% (95% confidence interval [CI], 83.4%-100%) while specificity was 99% (95% CI, 97.6%-99.8%). We found no association