**Presence of Anterior Temporal Artery Associates with Good Outcome in Acute Atherosclerotic
M1-Middle Cerebral Artery Occlusion**

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Abstract

Introduction We aim to investigate the correlation between presence of Anterior temporal artery (ATA), the first major branch of middle cerebral artery (MCA), on conventional angiography and clinical outcome in patients with acute atherosclerotic M1-MCA occlusion.

Methods Consecutive patients with acute atherosclerotic M1-MCA occlusion from Nanjing Stroke Registry Program (NSRP) between January 2007 and December 2012 were included in this study. All patients underwent MRI followed by conventional angiography. From their data, we analyzed baseline characteristics, infarction patterns, DWI-ASPECTS and collateral circulation. The correlation of ATA presence and good clinical outcome, modified Rankin Scale (mRS) score ≤ 2 , at 3 months was also calculated.

Results In 98 patients meeting entry criteria, the presence of ATA was found in 44 patients. Patients with ATA present were found to have less hypertension ($p=0.042$), lower baseline National Institutes of Health Stroke Scale (NIHSS) ($p=0.043$), more small infarcts in perforating artery territory ($p=0.013$) and a higher number of DWI-ASPECTS ≥ 7 ($p=0.034$). Binary logistic regression analysis showed an adjusted odds ratio of 4.45 for a good outcome in patients with ATA presence (95%CI: 1.52 to 13.03, $p=0.007$).

Conclusions The presence of ATA can be used as a predictor of good outcome in patients with acute atherosclerotic M1-MCA occlusion.

Keywords

stroke; middle cerebral artery occlusion; collateral status; lesion pattern

Introduction

There is an increasing amount of evidence supporting the notion that early identification of patients at risk for bad outcome is of great importance for acute atherosclerotic M1-middle cerebral artery (MCA) occlusion therapeutic decisions. [1] Several factors have been identified as important predictors of these patient outcomes, including stroke severity, patients' age, and infarct size. [2] However, these factors

suffer from a lack of definitive predictive power.[3] Thus, there may be other valuable factors involved in the prediction of good outcome for these patients.

For acute atherosclerotic M1-MCA occlusion patients, collateral circulation may play an important role in influencing recovery through sustaining downstream perfusion distal to the occlusion or through enhancing embolic washout in distal arteries. [4] In addition, collateral circulation can also influence the resultant infarction size and infarction patterns. [5] Currently leptomeningeal anastomoses is considered as an important collateral flow path in M1-MCA occlusion patients. However, collateral flow through other paths, such as small branches of MCA or moyamoya vessels could also be an important factor influencing the progression of patients. [6] The anterior temporal artery (ATA) is the first major branch of MCA. The presence of ATA is often encountered in patients with atherosclerotic MCA occlusion in which it may provide important collateral flow for these patients.

In this study, we hypothesized that presence of ATA associates with good outcome, defined as modified Rankin Scale (mRS) ≤ 2 at 3 months post atherosclerotic M1-MCA occlusion.

Patients and Methods

Study Design and Patient Selection

All patients in our study gave informed consent for participation. This study was approved by the Institutional Review Board of Jinling Hospital (Nanjing, China) and has therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Data for this study was retrieved from the Nanjing Stroke Registry Program (NSRP). [7] From this registry, we included consecutive patients with acute atherosclerotic MCA occlusion between January 2007 and December 2012. All patients were presented with a focal neurological deficit with a duration of >6 hours and <1 week. Patients suspected of M1-MCA occlusion by TCD, MRA or CTA underwent conventional angiography to definite M1-MCA occlusion diagnosis, evaluate the collateral circulation and guild therapy of these patients. MRI scans were performed to determine the lesion distribution and infarct patterns and conventional angiography data was collected to determine the presence of anterior temporal artery and evaluate the collateral circulation.

Patients were included in our study if they had (1) a large-artery atherosclerosis stroke diagnosis in accord with TOAST criterion, (2) acute ischemic lesions distributed within MCA territory or border-zone areas with DWI confirmation in 1 week of symptom onset, (3) conventional angiography demonstrating ipsilateral MCA-M1 occlusion in 2 weeks of symptom onset, (4) present with a focal neurological deficit with a duration of >6 hours and 1 weeks. We excluded patients who had (1) an age of <18 years old, (2) infarcts in multiple vascular territories beyond the unilateral MCA territory, (3) non-atherosclerotic MCA occlusion (eg, Moyamoya disease, radiation-induced arteriopathy, dissection, cardioembolic or arteritis), (4) concomitant tandem ICA and MCA diseases (eg, ipsilateral ICA stenosis $\geq 50\%$), (5) no MRI scan or conventional angiography, (6) lost follow-up.

We collected baseline characteristics for these participants, including sex, age, body mass index (BMI) as well as vascular risk factors and pre-event medication (eg, antiplatelet, antihypertensive and statin). Admission characteristics, including National Institutes of Health Stroke Scale (NIHSS) score, blood glucose, C-reactive protein (CRP), and systolic blood pressure (SBP), and therapeutic methods after admission were also collected.

At the 3-month follow-up, patients were interviewed in the outpatient clinic or by telephone to evaluate clinical outcome. Good outcome at 3 months was defined as modified Rankin Scale score ≤ 2 .

MRI Infarction Patterns and DWI ASPECTS

All patients were scanned within one week of symptom onset with 1.5 Tesla or 3 Tesla MRI scanners and a head coil. Acute infarcts on DWI were defined as hyperintensities on the DWI and hypointensities on the apparent diffusion coefficient map.

DWI lesion patterns and DWI ASPECTS were analyzed by two investigators (Yongkun Li and Wen Sun) who were blinded to clinical and angiography data. The topography of ischemic lesions was determined using published templates. [8] Lesion distribution was analyzed according to their infarct location in which MCA vascular structure were divided into the following territories: perforator, pial, and borderzone. Correspondingly, infarcts were named to match their occurring territory: perforator artery infarct (PAI) refers to striatocapsular infarcts or perforating vessel infarcts of the MCA; pial infarct (PI) refers to infarcts occurring in vascular territories supplied by the main leptomeningeal branches of the MCA;

borderzone infarct (BZI) refers to infarcts occurring in the anterior or posterior cortical BZ or internal BZ of the MCA. In extension, large territorial infarct (LTI) refers to a large ischemic lesion involving the cerebral cortex and subcortical structures in one or more M2 artery territories.

Infarct patterns were divided into two main categories: single territorial lesion and multiple territorial lesions. Therefore, lesions could be categorized to one of the following patterns: single territorial lesions- (1)PAI; (2)PI; (3)BZI; (4)LTI; multiple territorial lesions- (5)PAI+PI; (6)PAI+PI+BZ; (7)PAI+BZ; (8) PI+BZ.

The role of DWI-ASPECTS substituting for lesion volume in acute stroke patients has been well established.[9] To analyze the difference of lesion size on DWI between the two groups, we assessed ASPECTS on DWI according to the method used by Barber et al. [10] ASPECTS includes ten original ASPECTS regions, assigning a score of 1 for a normal and a score of 0 for a region showing ischemic change.

Angiography and Collateral Circulation

To evaluate the collateral circulation and guide therapy of these patients, patients suspected of M1-MCA occlusion by TCD, MRA or CTA underwent conventional angiography within two weeks of symptom onset. We used the Seldinger method via the transfemoral approach and catheterization of the bilateral carotid and vertebral arteries with a 5F selective catheter. MCA occlusion was defined as Thrombolysis in Myocardial Infarction (TIMI) =0. The presence of ATA and the location of MCA occlusion were also evaluated. Informed consent obtained from all patients before angiography.

Collateral flow was assessed with the American Society of Interventional and Therapeutic Neuroradiology (ASITN)/Society of Interventional Radiology (SIR) Collateral Flow Grading System. Two vascular neurologists (Gelin Xu and Renliang Zhang) who were blinded to the clinical and MRI variables independently analyzed the angiographic data. In the event of discrepancies, the final result was adopted by consensus. ASTIN/SIR score with 3-4 was considered as good collateral circulation.

Statistical Analysis

We compared differences between patients with or without ATA using t test or Mann–Whitney test for

continuous variables and Chi-square test or Fisher exact tests for categorical variables as appropriate.

Correlation between DWI-ASPECTS, Distal M1 occlusion, ASTIN/SIR and Presence of ATA was analyzed by calculating Spearman correlation coefficient. To identify independent predictors of favorable outcome, we performed binary logistic regression analysis in which all covariates with a P value <0.1 in a univariate analysis were entered into this logistic regression model. A value of $P<0.05$ was used to indicate statistical significance.

Results

Patients and Clinical Characteristics

In our study, a total of 103 patients met entry criteria with only 5 patients losing follow-up. Baseline characteristics of patients are shown in **Table 1**. Among them, forty-four (44/98, 44.9%) patients were found to have ATA present while fifty-four (54/98, 55.1%) patients were found to have ATA absent. The anterior temporal artery absent (ATAA) group was found to higher rates of hypertension than the anterior temporal artery present (ATAP) group (72.2% vs 52.3%, $p=0.042$). Other risk factors and baseline characteristics (including SBP, CRP and blood glucose) between the two groups were similar. Additionally, the baseline National Institutes of Health Stroke Scale (NIHSS) on admission is significantly higher in the ATAA group than that of the ATAP group ($p=0.043$). Furthermore, baseline pre-event medication and medication received during hospitalization between these two groups were similar.

Infarct Characteristics and Collateral Circulation

To investigate whether the volume of infarcts varies between these two groups, we analyzed patient DWI-ASPECTS through MRI. Data in **Table 1** shows that the number of patients with $ASPECTS \geq 7$ of the ATAA group is smaller than that of the ATAP group ($p=0.034$). The number of lesions and the distribution of lesions in all patients were also analyzed (**Figure 1**), in which the presence of multiple lesions were more common in the ATAA group than the ATAP group (81.5% vs 63.3% $p=0.047$). Pertaining to vascular territorial distribution of infarcts, PAI were similar between the two groups, however, small perforating artery infarcts ($<2\text{cm}$) occurred more frequently in patients with ATA present than those without (22.7% vs 5.6% $p=0.013$); large perforating artery infarcts ($\geq 2\text{cm}$) were more common

in the ATAA group (44.4% vs 25.0% $p=0.046$) whereas PI and BZI occurrence showed minimal differences between the two groups.

With regard to multiple territorial lesions, the most frequently occurring lesion pattern in the ATAA group is the concomitant PAI and BZ infarct (10/54, 18.5%) while the ATAP group varied in occurrence: 2 (4.5%), 6 (13.6%), 3 (6.8%), 6 (13.6%) patients had PAI+PI, PAI+PI+BZI, PAI+BZI, PI+BZI respectively. There was no difference in collateral flow between the two groups (**Table 1**).

Good Clinical Outcome at 3 Months

At 3 months, 50/98 patients (51.0%) had a good clinical outcome. As shown in Table 2, the good outcome group had a lower baseline NIHSS score, higher prevalence rate of ATA, more patients with ASPECTS ≥ 7 , and more single BZ territory lesions on DWI than the group with bad outcome. Hence, the presence of ATA (OR, 4.45; 95% CI, 1.52 to 13.03; $P=0.007$) is independently associated with good outcome (**Table 2**). As shown in Table 3, presence of ATA correlated well with high DWI-ASPECTS and distal M1 occlusion.

Discussion

Our study found that in acute atherosclerotic M1-MCA occlusion, patients with ATA present have smaller lesion volume, a higher number of small perforating artery infarcts, better baseline neurological function, and less multiple lesion occurrence rates. Hence, the presence of ATA was determined to be independently associated with good clinical outcomes.

Intracranial atherosclerosis disease is the most common cause of ischemic stroke in China.[11] In patients with intracranial atherosclerosis artery occlusion, collateral flow plays an important role in maintaining downstream territory perfusion. For M1-MCA occlusion, primary collateral circulation is established via the leptomeningeal anastomoses (LMAs) from the anterior cerebral artery (ACA) and posterior cerebral artery (PCA). In previous studies, the presence of ATA in M1-MCA occlusion on CTA identifies a group of patients with reduced case fatality.[12] Recent study [13] also assessed leptomeningeal collateral status with whole-brain dynamic time-resolved computed tomography angiography (CTA). Unlike a conventional angiography, this technique visualizes pial arterial filling in all vascular territories, thereby

providing additional hemodynamic information. It might be better at estimating collateral status. Our study included patients between January 2007 and December 2012, we couldn't analysis collateral status with whole-brain dynamic time-resolved computed tomography angiography in the last few years. Then, conventional digital subtraction angiography (DSA) was performed to evaluate collateral circulation and guild therapy of these patients. Although we did ICA injections to analysis ACA-MCA collateras and vertebral injection to measure PCA-MCA collaterals, we couldn't estimate these collateral status simultaneously like whole-brain dynamic time-resolved CTA. So, further studies about collateral circulation could be based on whole-brain dynamic time-resolved CTA. Our results showed that our patients have similar collateral circulation from ACA, PCA, and LMA, which indicates that there may be other mechanisms influencing patient outcome.

Before undergoing conventional DSA, patients in our study also underwent MRI and DWI to evaluate lesion size and infarct pattern. DWI-ASPECTS is a semi-quantitative estimation of lesion size, in which it has been shown to correlate well with actual DWI lesion volume. [9,14] Lesion size on DWI has been demonstrated to be associated with stroke severity and has also been studied as an important predictor of functional outcome. [14,15] We analyzed DWI-ASPECTS to investigate the difference of lesion volume between the two groups. From our results we found that the presence of ATA was associated with higher DWI-ASPECTS. As we know, ATA is the first major branch of the MCA, it provides blood supply to the polar and anterolateral portions of the temporal lobe [16] .Therefore, the presence of ATA in M1-MCA occlusions may negate proximal occlusion of the MCA trunk, which has been identified previously as an angiographic factor predicting malignant MCA infarction.

Lesion pattern is another factor affecting the association between ATA presence and outcome. Previous studies suggested that advanced injury in the basal ganglia resulted in worse dysfunction and disability at discharge among patients with M1-MCA occlusion. [17-19] Our results show that PAI occurrence is smaller in the ATAP group than the ATAA group. This can be accounted by the difference in M1-MCA occlusion position, in which the ATA commonly arises proximal to or opposite to the origin of the lenticulostriate arteries. In acute atherosclerosis M1-MCA occlusion patients, ATA presence suggests that only the smaller perforator artery territory is involved[20] . Furthermore, ATA presence can also sustain downstream perfusion from MCA occlusions, involving smaller downstream MCA territory.

A combination of local branch occlusion and embolism, with or without hemodynamic compromise, is a common pathogenesis of stroke resulting from MCA disease[21]. Our data also provides evidence that multiple DWI lesions, which may be markers of embolism, are more common in the ATAA group. These lesions on DWI may be caused by an embolism originating from the proximal MCA lodging itself in the distal MCA. Additionally, perfusion deficits caused by MCA occlusion may contribute to the development of embolic infarction through impaired clearance of emboli. In ATAP patients, ATA can partially maintain perfusion, leading to enhanced clearance of emboli in the MCA territory.

There are two main strengths in our study: the first being that we have only included patients with acute atherosclerotic MCA infarction, allowing us to maintain the consistency of our patients. The second being that all our patients have underwent both conventional DSA and MRI scan. Conventional DSA is considered to be the “gold standard” for determination of ATA presence as well as collateral flow evaluation while DWI is the most sensitive diagnostic modality for detecting acute infarction and lesion pattern[21]. Therefore, these methods allow us to better explore the potential mechanisms underlying the relationship between ATA presence and good outcome.

While we found that patients with ATA generally had better anterior flow than ATAA group patients, we did not have CT-Perfusion or MRI-Perfusion results to confirm the link between ATA presence and better perfusion. Therefore in the future studies, the association between ATA presence and downstream perfusion areas from MCA occlusion site worthy of further investigation. Additionally, although we collected patients in a prospective stroke registry, this study was a single-center study and was limited by a small sample size.

Ethical Standards and Patient Consent

We declare that this study has been approved by the Institutional Review Board of Jinling Hospital (Nanjing, China) and has therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. We declare that all patients gave informed consent prior to inclusion in this study.

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Conflict of interest

We declare that we have no conflict of interest.

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Figure legends

Fig 1 Lesion patterns and angiography characteristics of patients in different groups

A, A 63-year-old man presented with right hypesthesia 2 days before MRI study. DWI showing acute left BZ infarction.

B, Angiography of Figure 1-A showing left M1-MCA occlusion and presence of ATA.

C, A 47-year-old man had sudden onset of aphasia and right hemiplegia 4 days before MRI scan. DWI showing acute large territory infarction of left MCA.

D, Angiography of Figure 1-C showing left M1-MCA occlusion and ATA is absent.

Table1 Baseline characteristics of patients with or without anterior temporal artery

Characteristics	ATAA(n=54)	ATAP(n=44)	<i>p</i> Value
Age(mean±SD)	58.70±11.70	59.86±9.16	0.593
Male n(%)	39(72.2)	33(75.0)	0.757
Risk factors			
Hypertension n(%)	39(72.2)	23(52.3)	0.042*
Diabetes n(%)	13(24.1)	12(27.3)	0.718
Hyperlipidemia n(%)	15(27.8)	6(13.6)	0.090
Ever smoker n(%)	24(44.4)	22(50.0)	0.584
Alcohol consumption n(%)	16(29.6)	10(22.7)	0.441
Previous stroke/TIA n(%)	10(18.5)	6(13.6)	0.515
CAD n(%)	7(13.0)	4(9.1)	0.546
Baseline NIHSS score(median, IQR)	8.5(6.0-13.0)	7.0(4.0-10.0)	0.043*
BMI	24.64(22.79-25.75)	24.01(22.58-25.27)	0.287
SBP mmHg(median, IQR)	141(130.0-148.50)	136.5(125.0-153.75)	0.247

Blood glucose, mg/dl(median, IQR)	98.1(87.75-109.8)	95.40(81.00-126.00)	0.614
CRP(median, IQR)	2.95(1.08-5.20)	1.95(1.15-5.60)	0.702
Pre-event medication n(%)			
Antiplatelet therapy at onset	19(35.2)	12(27.3)	0.402
Statin therapy at onset	15(27.8)	8(18.2)	0.265
Antihypertensive therapy at onset	23(42.6)	15(34.1)	0.390
Medication therapy in hospital n(%)			
Double antiplatelet therapy	45(83.3)	32(72.7)	0.203
Statin therapy	53(98.1)	41(93.2)	0.217
Antihypertensive therapy	37(68.5)	22(50.0)	0.062
Distal M1 occlusion	11(20.4)	25(56.8)	<0.001
Good collateral flow on DSA n(%)	7(13.0)	5(11.4)	0.810
ASITN-SIR=0	5(9.3)	2(4.5)	0.454
ASITN-SIR=1	21(38.9)	13(29.5)	0.334
ASITN-SIR=2	21(38.9)	24(54.5)	0.122

ASITN-SIR=3	4(7.4)	2(4.5)	0.688
ASITN-SIR=4	3(5.6)	3(6.8)	1.000
ASPECTS $\geq$ 7 on DWI n(%)	24 (44.4)	29(65.9)	0.034*
Multiple infarcts on DWI n(%)	44(81.5)	28(63.6)	0.047*
Infarct involving perforating artery territory n(%)			
Small(<2cm)	3(5.6)	10(22.7)	0.013*
Large($\geq$ 2cm)	24(44.4)	11(25.0)	0.046*
Infarct involving pial artery territory n(%)	26(48.1)	18(40.9)	0.474
Infarct involving Border-zone territory n(%)	32(59.3)	21(47.7)	0.255
Single territorial lesion n(%)			
1.PAI	7(13.0)	10(22.7)	0.204
2.PI	9(16.7)	4(9.1)	0.271
3.BZI	8(14.8)	6(13.6)	0.868
4.Large territorial	3(5.6)	7(15.9)	0.092
Multiple territorial lesions n(%)			

5.PAI+PI	3(5.6)	2(4.5)	1.000
6.PAI+PI+BZI	7(13.0)	6(13.6)	0.922
7.PAI+BZI	10(18.5)	3(6.8)	0.089
8.PI+BZI	7(13.0)	6(13.6)	0.922

* $P < 0.05$

Abbreviations: TIA=transient ischemic attack, CAD= coronary artery disease, IQR= inter quartile range, BMI=body mass index, SBP= systolic blood pressure, CRP= C-reactive protein, DSA= digital subtraction angiography, DWI= diffusion weighted imaging, PAI= perforator artery infarct, PI= pial infarct, BZI= borderzone infarct

Table 2 Factors associated with good clinical outcome

	mRS $\leq$ 2(n=50)	mRS>2(n=48)	<i>p</i> Value	OR	95%CI	<i>p</i> value
Age(mean $\pm$ SD)	59.44 $\pm$ 10.22	59.00 $\pm$ 11.08	0.839			
Male n(%)	38(76.0)	34(70.8)	0.563			
Risk factors						
Hypertension n(%)	30(60.0)	32(66.7)	0.494			
Diabetes n(%)	13(26.0)	12(25.0)	0.910			
Ever smoker n(%)	25(50.0)	21(43.8)	0.535			
Alcohol consumption n(%)	12(24.0)	14(29.2)	0.563			
Hyperlipidemia n(%)	9(18.0)	12(25.0)	0.399			
Previous stroke/TIA n(%)	6(12.0)	10(20.8)	0.237			
CAD n(%)	6(12.0)	5(10.4)	0.804			
Baseline NIHSS score (median, IQR)	5(4.0-8.0)	11.5(7.25-15.0)	<0.001*	0.76	0.65-0.90	0.001
BMI (median, IQR)	24.6(22.9-25.5)	24.2(22.5-25.6)	0.477			
SBP mmHg (median, IQR)	141(125.0-150.0)	140(135.0-157.0)	0.492			

Blood glucose, mg/dl (median, IQR)	95.4(82.35-109.80)	98.1(82.8-125.1)	0.391			
CRP (median, IQR)	1.90(0.98-3.93)	3.00(1.40-7.28)	0.110			
Pre-event medication						
Antiplatelet therapy at onset n(%)	13(26.0)	18(37.5)	0.221			
Statin therapy at onset n(%)	11(22.0)	12(25.0)	0.726			
Antihypertensive therapy at onset n(%)	20(40.0)	18(37.5)	0.800			
Medication therapy in hospital						
Double antiplatelet therapy n(%)	37(74.0)	40(83.3)	0.260			
Statin therapy n(%)	47(94.0)	47(97.9)	0.327			
Antihypertensive therapy n(%)	29(58.0)	30(62.5)	0.649			
Presence of ATA	31(62.0)	13(27.1)	0.001*	4.45	1.52-13.03	0.007
Distal M1 occlusion	22(44.0)	14(29.2)	0.128			
Good retrograde collateral flow on DSA n(%)	5(10.0)	7(14.6)	0.489			
ASITN-SIR=0	5(10.0)	2(4.2)	0.436			
ASITN-SIR=1	17(34.0)	17(35.4)	0.883			

ASITN-SIR=2	23(46.0)	22(45.8)	0.987			
ASITN-SIR=3	1(2.0)	5(10.4)	0.108			
ASITN-SIR=4	4(8.0)	2(4.2)	0.678			
ASPECTS $\geq$ 7 on DWI n(%)	39(78.0)	14(29.2)	<0.001*	2.65	0.87-8.07	0.086
Multiple infarcts on DWI n (%)	34(68.0)	38(79.2)	0.211			
Infarct involving perforating artery territory n(%)						
Small(<2cm)	9(18.0)	4(8.3)	0.158			
Large($\geq$ 2cm)	15(30.0)	20(41.7)	0.228			
Infarct involving pial artery territory n(%)	21(42.0)	23(47.9)	0.556			
Infarct involving Border-zone territory n(%)	23(46.0)	30(62.5)	0.101			
Single territorial lesion						
PAI territorial lesion n (%)	11(22.0)	6(12.5)	0.214			
PI territorial lesion n (%)	9(18.0)	4(8.3)	0.158			
BZ territorial lesion n (%)	10(20.0)	4(8.3)	0.099*	1.25	0.30-5.25	0.761
Large territorial lesion n (%)	3(6.0)	7(14.6)	0.161			

Multiple territorial lesions

PAI+PI territorial lesion n (%)	4(8.0)	1(2.1)	0.362
PAI+PI+BZ territorial lesion n (%)	4(8.0)	9(18.8)	0.117
PAI+BZ territorial lesion n (%)	5(10.0)	8(16.7)	0.331
PI+BZ territorial lesion n (%)	4(8.0)	9(18.8)	0.117

* $P < 0.1$

Abbreviations: TIA=transient ischemic attack, CAD= coronary artery disease, IQR= inter quartile range, BMI=body mass index, SBP= systolic blood pressure, CRP= C-reactive protein, DSA= digital subtraction angiography, DWI= diffusion weighted imaging, PAI= perforator artery infarct, PI= pial infarct, BZ= borderzone infarct

Table 3 Spearman correlation analysis between DWI-ASPECTS, Distal M1 occlusion, ASTIN/SIR and Presence of ATA

	DWI-ASPECTS	Distal M1 occlusion	ASITN/SIR
Presence of ATA			
R	0.223*	0.376*	0.117
p	0.027	0.000	0.250
N	98	98	98

* $P < 0.05$