



# Effect of transverse sinus variation on the clinical outcomes of atherosclerotic anterior circulation infarction

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**Background:** Hypoplasia of the transverse sinus (TS) is a common anatomical variation. The aim of this study was to investigate the effects of TS variation (i.e., TS hypoplasia) and no variation (i.e., TS symmetry) and their subgroups on the clinical outcomes of patients with atherosclerotic anterior circulation cerebral infarction (CI).

**Methods:** A total of 75 patients were included in the study and were divided into the no TS variation group and the TS variation group. The TS variation group was further divided into the following subgroups: the TS variation with ipsilateral CI group and the TS variation with contralateral CI group. We retrospectively analyzed the correlations of the endpoint events of patients with large atherosclerotic anterior circulation infarction and TS no variant, and subgroups of TS variants.

**Results:** We found that the diameter of the ipsilateral IJV in patients with TS variants were significantly smaller than those without TS variants, which was statistically significant ( $P < 0.05$ ). The differences in primary endpoint events, secondary endpoint events, and responsible vessel stenosis were not statistically significant when comparing the TS variant and no TS variant groups, and the TS variant subgroup ( $P > 0.05$ ). We found statistically significant differences in the National Institute of Health stroke scale (NIHSS) and Modified Rankin Scale (mRS) scores after 90 days of CI between the total anterior circulation infarct (TACI) TS variant group, the ipsilateral CI TS variant group, and the partial anterior circulation infarct (PACI) TS hypoplasia group and the ipsilateral CI TS variant group ( $P < 0.05$ ). There was a statistically significant difference ( $P < 0.05$ ) between the TS variant group with TACI, the TS variant group with ipsilateral CI, and the TS no variant group and the TS variant with contralateral CI group when comparing patients' mRS scores after 90 days of CI.

**Conclusions:** The diameter of the internal jugular vein (IJV) ipsilateral to the TS variant was significantly smaller than that of the TS no variant. Patients with TACI in the TS variant group and one of its subgroups (the TS variant with ipsilateral CI group) had more severe clinical symptoms and a worse prognosis than patients in the same group with PACI.

**Keywords:** Cerebral infarction (CI); anterior circulation; transverse sinus (TS); variation; outcome prognosis

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## Introduction

Cerebral infarction (CI), also known as ischemic stroke, is a sudden focal neurological dysfunction caused by localized brain tissue necrosis due to impaired blood supply to the brain. CI accounts for about 80% of all strokes, and the incidence of CI is 150–200 in 100,000 people per year, with a recurrence rate of about 10% within 1 year (1,2). CI is currently one of the leading causes of disability and death in China (1,2). According to the clinical staging of CI of the Oxfordshire Community Stroke Project (OCSP), there are total anterior circulation infarcts (TACIs) and partial anterior circulation infarcts (PACIs) (3). According to the trial of ORG 10172 in Acute Stroke Treatment (TOAST) criteria most TACIs and PACIs are large artery atherosclerosis (LAA). LAA forms a large thrombus area and is an important cause of intracranial vascular occlusion. Poor prognosis for massive CI caused by intracranial vascular occlusion (4). Altered vascular structure and hemodynamics have been found to be extremely important factors in the pathogenesis of large artery atherosclerosis.

Cerebral circulation includes the arterial and venous systems. Cerebral arteries can be divided into cortical branches and central branches according to the characteristics of their course and distribution. Cerebral veins include superficial cerebral veins, deep cerebral veins, and venous sinuses (the walls of venous sinuses are composed of dense collagen fibers, which are tough and inelastic, and do not have valves, and venous sinuses include straight, transverse and sigmoid sinuses). Previous studies have focused on intracranial arterial vascular changes, but in fact, veins and venous sinuses predominate the cerebral blood flow and intracranial pressure changes. Currently, due to advances in imaging, the detection of intracranial venous variants is becoming more common, but most patients have no clinical symptoms (5). Transverse sinus (TS) variation refers to bilateral asymmetry of the TS, where one TS duct is smaller than the other or where one TS is absent. TS hypoplasia is the most common type of venous sinus variant in normal subjects and has been considered a physiological variant. However, A previous study (6) have shown that TS hypoplasia impairs cerebral blood flow regulation and has an important role in overall cerebral venous and venous hemodynamics. The TS travels along the attachment margin of the cerebellar curtain, collects blood from the basal surface of the lateral temporal surface, temporal and occipital lobes, and later reaches the posterior lateral part of the temporal bone rock, flows into the sigmoid sinus, and finally converges

into the internal jugular vein (IJV) (7). A national study (8) showed that the size of the TS volume is mainly influenced by the blood flow in the superior sagittal sinus, and that most dural arteriovenous fistulas involve the TS, which can cause narrowing of the sinus cavity, resulting in a range of symptoms. We theorize that in the event of intracranial arterial infarction the following two events might occur: (I) congenital TS hypoplasia affects arterial blood return, and its effect over the years contributes to the acceleration of the atherosclerotic process; (II) TS hypoplasia affects arterial return, leading to increased edema in the acute phase of large anterior circulation infarction, which directly affects patient prognosis. In this study, we retrospectively analyzed TS variants in patients with large atherosclerotic anterior circulation infarction to assess their clinical value in terms of risk factors and clinical regression in ischemic stroke. The aim is to provide new ideas for the acute treatment and secondary prevention of patients with CI.

We present the following article in accordance with the STROBE reporting checklist (available at <https://atm.amegroups.com/article/view/10.21037/atm-22-197/rc>).

## Methods

### *Subjects collected*

A total of 75 patients who visited the Department of Neurology of Hongqi Hospital Affiliated to Mudanjiang Medical University and the second hospital affiliated with Mudanjiang Medical University from February 2019 to December 2020, and had an onset time of >48 h were included in this study. All of these patients underwent cranial magnetic resonance diffusion weighted imaging (DWI) or magnetic resonance imaging (MRI) within 48 h of onset, were confirmed to have large atherosclerotic anterior circulation CI, and underwent magnetic resonance venography (MRV) of the head within 48 hours. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by ethics committee of Hongqi Hospital Affiliated to Mudanjiang Medical University (No. 202114), and informed consent was taken from all the patients.

A total of 75 patients were included in the study and were divided into the no TS variation group and the TS variation group. We found in our clinic that there seems to be a specific association between TS variant and complete anterior circulation CI and partial anterior circulation

CI, and we divided them into two subgroups (the TS variation with ipsilateral CI group and the TS variation with contralateral CI group) in order to further verify the potential association.

To be eligible for inclusion in the study, patients had to meet the following inclusion criteria: (I) have been admitted within 48 h of onset and attended outpatient and emergency clinics in accordance with the 2018 Chinese Guidelines for the Diagnosis and Treatment of Acute Ischemic Stroke (9); (II) have a diagnosis of a clear new responsible lesion located in the anterior cerebral circulation as confirmed by a DWI or MRI examination by the same physician; (III) have been classified as having TACI or PACI according to OCSP clinical staging, and have been staged according to the TOAST etiology (patients with the atherosclerotic type of large arteries were included); (IV) have undergone MRV and MRA, and have been diagnosed by the same physician as clearly conforming to the TS variant classification; and (V) have agreed to participate, or have family members that agreed to their participating, in this study, and have signed the informed consent form.

Patients were excluded from the study if they met any of the following exclusion criteria: (I) had a serious medical system disease, such as: severe liver and renal impairment combined with intracranial infection; (II) had a brain tumor; (III) had posterior circulation infarction; (IV) had a cardiogenic embolism; (V) had DWI-negative ischemic lesions; and/or (VI) refused to participate or did not consent to participate in the experiment. A total of 75 patients, with no missing data, were included in this study.

### **Imaging evaluation**

The imaging system scans were performed using a Philips 3.0T TX magnetic resonance machine from the Netherlands, and T1WI, T2WI, Flair, DWI, and MRV were routinely performed. A TOSHIBA XG790 color ultrasonography machine was used to perform bilateral carotid artery and IJV examinations. The imaging findings were evaluated by an experienced imaging and ultrasonography physician and a neurologist trained to perform a blinded assessment.

### **Diagnostic criteria**

The diagnostic criteria for TS variation were the presence of TS asymmetry suggested by MRV or digital subtraction angiography (DSA) of the head. Asymmetric grading

criteria for TS hypoplasia: The middle segment of the TS was chosen to measure the TS canal diameter (cm) because it is easy to identify and measure. Using MRV, TS was classified as 1 of the following 4 grades according to whether the bilateral TS was symmetrical or not (10): Grade 0: TS bilateral asymmetry  $\leq 10\%$ ; Grade 1: TS asymmetry  $>10\%$  and  $\leq 50\%$ ; Grade 2: TS asymmetry  $>50\%$ ; and Grade 3: TS slender or absent. TS variants were defined as TS developmental asymmetry  $>50\%$  or TS undeveloped, including grades 2 and 3. MRV grading examples are shown in *Figure 1*.

### **Statistical analysis**

SPSS 25.0 statistical software was used to process the data. The measurement data results are expressed as mean  $\pm$  standard deviation ( $\bar{x} \pm s$ ). Comparisons of means between two groups were performed first by a normality test and chi-square test for all statistical indicators. The *t*-test was used to satisfy normal distribution, and the chi-square test and the Wilcoxon rank-sum test were used to compare 2 independent samples if the data does not satisfy the normal distribution curve. The statistical data are expressed as the composition ratio (%). A  $\chi^2$  test was used to compare positive rates. Differences were considered statistically significant if the P value was  $<0.05$ .

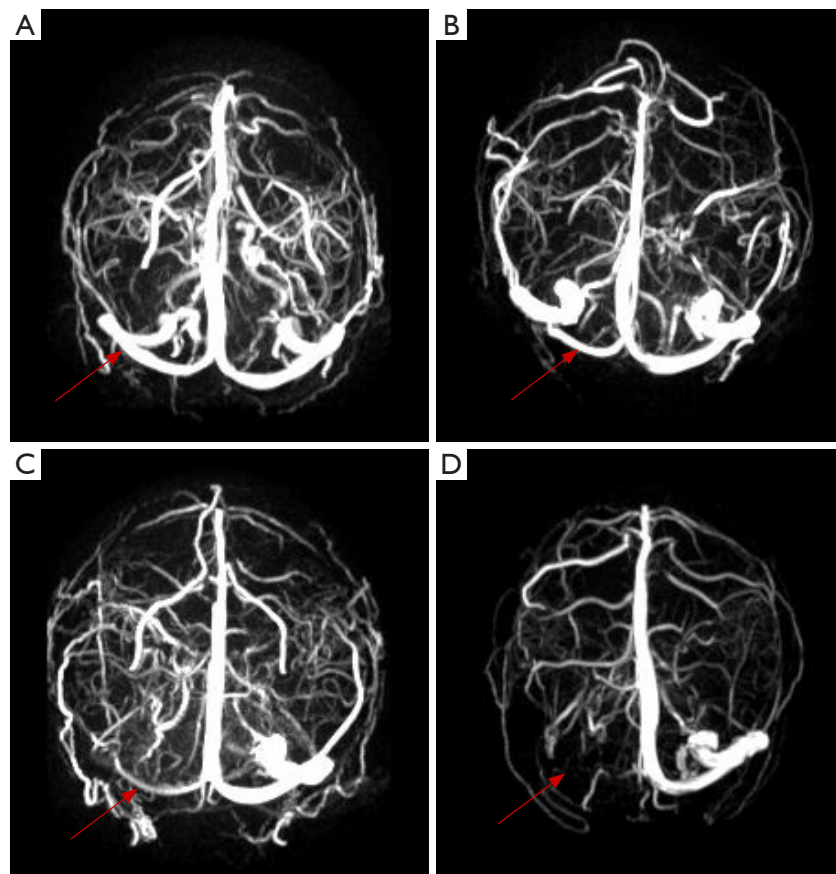
## **Results**

### **Comparison of the demographic and risk factors for LAA-type anterior circulation CI**

A total of 75 patients with LAA anterior circulation infarction were ultimately enrolled in the study. Patients' demographic and risk-factor information are shown in *Table 1* (there was no missing data). The primary endpoint events and secondary endpoint events were not statistically significant ( $P > 0.05$ ) in patients with LAA anterior circulation infarction between the TS hypoplasia and the TS symmetry group and the TS hypoplasia subgroups (see *Table 2*). Some of the cases where Primary endpoint events as well as Secondary endpoint events occurred in the experiment can be seen in *Figure 2*.

### **Significance of IJV diameter asymmetry**

The IJV canal diameter was significantly smaller than that of the TS no variant on the ipsilateral side of the TS



**Figure 1** MRV grading of TS variants. TS was classified as 1 of the following 4 grades using MRV imaging according to whether the TS development was symmetrical or not: I) Grade 0: TS bilateral asymmetry  $\leq 10\%$  (A, arrow); II) Grade 1: TS asymmetry  $>10\%$  and  $\leq 50\%$  (B, arrow); III) Grade 2: TS asymmetry  $>50\%$  (C, arrow); and IV) Grade 3: TS slenderness or absence (D, arrow). MRV, magnetic resonance venography; TS, transverse sinus.

hypoplasia, and the difference was statistically significant when compared to the side without variant TS ( $\chi^2=33.446$ ,  $P<0.05$ ; see *Table 3*).

#### ***Clinical significance of assessing responsible lesions for LAA-type anterior circulation infarcts***

As *Table 4* shows, in patients with LAA-type anterior circulation infarction, intracranial large artery stenosis was not statistically significant between the TS variant and no variant groups ( $P>0.05$ ).

#### ***Clinical regression analysis between patients with LAA anterior circulation infarction and TS variant staging***

In relation to the different variant types of LAA-type complete/partial anterior circulation infarcts in the TS, the NIHSS scores and the 90-day post-infarction mRS scores were significantly different between the TS variant with TACI group and the TS variant with PACI group ( $P<0.05$ ). In the TACI patients, the 90-day post-infarction mRS scores of the TS variant group were significantly different to those of the TS no variant group ( $P<0.05$ ; see *Table 5*). The

**Table 1** General information of patients with LAA-type anterior circulation infarction

Variables	LAA-type anterior circulation pedicle group
Age (years)	61.13±10.05
Sex, n (%)	
Male	47 (62.7)
Female	28 (37.3)
Hypertension, n (%)	45 (60.0)
Diabetes mellitus, n (%)	29 (38.7)
Coronary heart disease, n (%)	14 (18.7)
Stroke history, n (%)	35 (46.7)
Smoking history, n (%)	25 (33.3)
History of alcohol consumption, n (%)	22 (29.3)
HDL (mmol/L)	2.88±0.89
LDL (mmol/L)	2.76±0.78
TC (mmol/L)	5.21±1.50
GLU (mmol/L)	8.76±4.32
UA (μmol/L)	349.22±103.03
FIB (g/L)	2.90±1.46
D-Di (μg/mL)	2.83±1.39
Stroke severity	
ASPECT >7, n (%)	30 (40.0)
ESSEN (score)	2.23±1.40
NIHSS (5–15/16–20), n (%)	30 (40.0)/10 (13.3)
mRS score at admission (score)	3.01±1.30

The data are shown as mean ± standard deviation or n (%). LAA, large artery atherosclerosis; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TC, serumtotalcholesterol; GLU, glutamic acid; UA, uric acid; FIB, fibrinogen; D-Di, D-dipolymer; ASPECT, Alberta Stroke Program Early CT Score; ESSEN, Essen Stroke Risk Score; NIHSS, National Institute of Health stroke scale.

NIHSS scores and 90-day post-infarction mRS scores were significantly different between the TS variant with ipsilateral TACI group and with the TS variant with ipsilateral PACI group ( $P<0.05$ ), and the 90-day post-infarction mRS scores were also significantly different between the TS variant with ipsilateral TACI group and the TS variant with contralateral TACI group ( $P<0.05$ ; see *Table 6*).

## Discussion

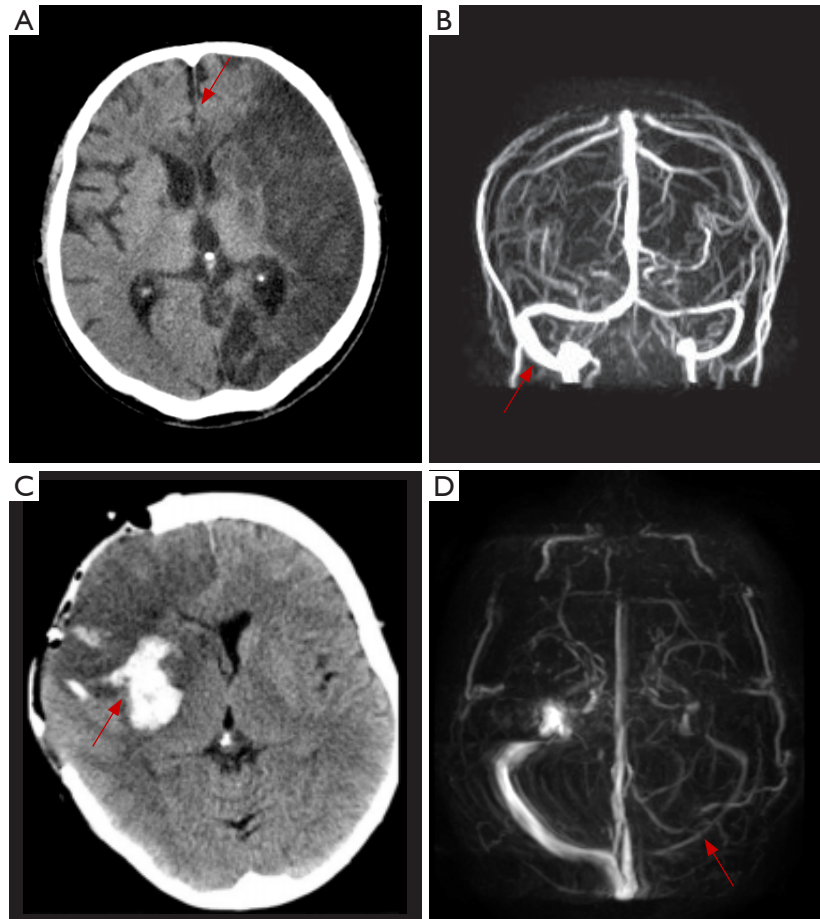
LAA-type anterior circulation infarction is affected by a combination of genetic and environmental factors, and there are many studies on its pathogenesis and pathological process, but research on the contributors of LAA-type anterior circulation infarction is not conclusive. In the present study, consistent with the results of previous studies (11,12), the possible risk factors for the formation of LAA-type anterior circulation infarction were being male (62.7%), hypertension (60%), a history of stroke (46.7%), a NIHSS score of 5–15 (40%), an ASPECT score >7 (40%), diabetes mellitus (38.7%), a history of smoking (33.3%), a mRS score with a mean value in the moderate to high range, a relatively high percentage of these above risk factors, and mean values of serumtotalcholesterol (TC), glutamic acid (GLU), Uric acid(UA), and D-dipolymer (D-Di) higher than normal. In relation to the effects of TC, GLU, UA, D-Di, hypertension, diabetes, and smoking on blood vessels, research has shown that (13) TC abnormalities lead to vascular endothelial cell damage, which in turn induces vascular inflammatory responses, promotes foam cell formation and thus TC is involved in atherosclerosis by promoting the formation of foam cells. Previous studies have shown (14) that UA in serum can contribute to lipoprotein oxidation and lipid peroxidation. UA thickens the vessel wall, elevates blood pressure, and thus leads to vascular remodeling (15). D-Di promotes the proliferation and contraction of smooth muscle and endothelial cells, platelet aggregation, and hemoglobin adhesion, and increases peripheral vascular resistance and blood viscosity (16). A study (17) found that hyperglycemia accelerates the thickening of cerebral atherosclerosis (AS) and the microvascular basement membrane, glycogen deposition, fatty, and hyaline changes, and accelerates atherosclerosis. A study (11) found that harmful substances, such as nicotine and carbon monoxide in tobacco, entering the organism can lead to altered blood rheology, increased shear, damage to vascular endothelial cells and the inhibition of their diastolic function. Houtkamp (18) showed that men have a higher probability of occlusion of large vessels of the anterior circulation (e.g., the internal carotid artery, and middle cerebral artery), which may be associated with the presence of adverse lifestyle habits, such as smoking or excessive alcohol consumption. A history of stroke, NIHSS score, mRS score, and ASPECT showed a possible association with severity in patients with anterior circulation infarction



**Table 2** Comparison of endpoint events between TS variant group (TS hypoplasia group) and TS no variant group (TS symmetry group)

Group project	TS no variant group (n=16)	TS variant group		
		Total (n=59)	TS hypoplasia ipsilateral to the infarct (n=41)	TS contralateral hypoplasia to the infarct (n=18)
Primary endpoint events, n (%)				
Malignant middle cerebral artery infarction				
Centerline shift of more than 10 mm	0	2 (3.3)	2 (4.8)	0
Cerebellar curtain herniation symptoms	0	0	0	0
Secondary endpoint events, n (%)				
Combined new cerebrovascular events	0	1 (1.7)	0	1 (5.6)
Combined CI hemorrhage conversion	1 (6.3)	0	0	0

TS, transverse sinus; CI, cerebral infarction.



**Figure 2** Primary endpoint events, secondary endpoint events, and TS variants in patients with anterior circulation infarction. CT scan of the head of a 75-year-old male showing a malignant middle cerebral artery infarction with a midline shift of more than 10 mm (A, arrow), and a MRV scan showing ipsilateral TS developmental variation (B, arrow). The CT scan of the head of a 62-year-old male showing a combined hemorrhagic transformation after an anterior circulation infarction (C, arrow), and a MRV scan showing a developmental variation of the contralateral TS (D, arrow). TS, transverse sinus; MRV, magnetic resonance venography.

**Table 3** Relationship between TS hypoplasia and the lateral canal diameter of the IJV

IJV (n=52)	TS hypoplasia (n=52)	
	Left	Right
Left	22	2
Right	8	20
$\chi^2$ values	33.446	
P values	<0.01	

The IJV is divided into the left and right sides by the reduced diameter of the IJV. TS, transverse sinus; IJV, internal jugular vein.

of the LAA type. Consistent with the findings of Zhang and others (12,18), the NIHSS score is an independent predictor of adverse clinical outcomes in patients with LAA anterior circulation infarction, and most patients with LAA anterior circulation infarction have large vessel stenosis, and due to stenosis and hypoperfusion, platelet clots, and fibrin accumulate in the arterial wall, leading to thrombosis. In addition high stroke-related scores are more likely to lead to large vessel occlusion, which in turn aggravates clinical symptoms.

We treated patients with atherosclerotic anterior

**Table 4** Comparison of responsible stenosis between TS variant group and TS no variant group

Group project	TS no variant group (n=16)	Total (n=59)	TS variant group	
			TS hypoplasia ipsilateral to the infarct (n=41)	TS contralateral hypoplasia to the infarct (n=18)
Middle cerebral artery stenosis	7	24	17	7
Anterior cerebral artery stenosis	4	15	8	6
Internal carotid artery stenosis	3	9	8	1
No intracranial large artery stenosis	2	11	8	4

TS, transverse sinus.

**Table 5** Comparison of NIHSS scores and 90-day post-infarction mRS scores among different variant types of TS in patients with TACI/PACI anterior circulation infarction

Project grouping	TACI		PACI	
	TS symmetry	TS hypoplasia	TS symmetry	TS hypoplasia
NIHSS scores	13.33±4.62	12.67±4.97*	3.00±1.41	4.05±3.47
mRS scores	3.00±0.00	3.48±0.75*^	1.43±1.62	1.77±1.34

\*, TS variant with TACI group vs. TS variant with PACI group,  $P < 0.05$ ; ^, in TACI patients,  $P < 0.05$  in the TS variant group compared to the TS no variant group. NIHSS, National Institute of Health stroke scale; mRS, Modified Rankin Scale; TS, transverse sinus; TACI, total anterior circulation infarct; PACI, partial anterior circulation infarct.

**Table 6** Comparison of NIHSS scores and 90-day post-infarction mRS scores between different types of TS variants in patients with TACI/PACI anterior circulation infarction.

Project grouping	TACI		PACI	
	TS hypoplasia ipsilateral to the infarct	TS contralateral hypoplasia to the infarct	TS hypoplasia ipsilateral to the infarct	TS contralateral hypoplasia to the infarct
NIHSS scores	12.93±5.58*	12.14±3.81	4.07±3.54	3.27±2.15
mRS scores	3.57±0.76*^	3.29±0.76	1.74±1.29	1.55±1.51

\*, TS variant with ipsilateral TACI group compared to TS variant with ipsilateral PACI group,  $P < 0.05$ ; ^, TS variant with ipsilateral TACI group compared to TS variant with contralateral TACI group,  $P < 0.05$ . NIHSS, National Institute of Health stroke scale; mRS, Modified Rankin Scale; TS, transverse sinus; TACI, total anterior circulation infarct; PACI, partial anterior circulation infarct.

circulation infarction with or without concomitant TS variants with almost identical treatment, i.e., conventional cerebrovascular disease therapeutic measures, such as antiplatelet aggregation, lipid lowering, and improvement of cerebral circulation. In relation to the primary and secondary endpoint events for patients in this study with LAA anterior circulation infarction, there was no statistically significant difference between the TS variant group and the no variant group, and between the TS variant group with ipsilateral CI and the TS variant group with contralateral CI ( $P>0.05$ ). This may be related to the short study period, the small number of patients enrolled, and possible biases in data collection.

Indicators of hemodynamic assessment in the IJV include blood flow and vessel diameter, and some studies have reported that an asymmetrical IJV diameter also leads to hemodynamic abnormalities (19,20). As the values related to blood flow in patients were not obtained during the IJV ultrasound performed in this study due to technical limitations, only the canal diameter was evaluated. In evaluating the IJV canal diameter, we found that the percentage of canal diameter asymmetry was about 48%, and when analyzed with the side of the TS variant, the results suggested that the IJV canal diameter on the same side as the TS variant was significantly smaller, which is also consistent with the findings of previous studies (21). The IJV is the largest deep branch of the jugular vein, and is an important pathway for cerebrospinal fluid and cerebral venous return. Structural changes in the vein can lead to endothelial denudation or extracellular matrix disruption, resulting in blood flow stagnation and vessel wall damage (22). TS hypoplasia leads to a decrease in return blood flow, a significant increase in the velocity of blood flow within the IJV under the effect of gravity, a decrease in the pressure within the IJV, a greater pressure difference between the lumen and outside the lumen, and a relatively greater compression of the vessel by extra-luminal structures that cannot maintain the luminal structure, leading to a reduction in the diameter of the IJV or even collapse. In the next study, more cases should be included and venous hemodynamic indices should be obtained.

Intracranial vascular stenosis in this study was not statistically significant between types in the TS no variant group and TS variant group, and between the TS variant with ipsilateral CI group and the TS variant with contralateral CI group ( $P>0.05$ ), which may be related to the small number of patients enrolled in the study and the moderate to high clinical severity. The question of whether

the abnormalities in venous system morphology resemble the consequences of an incomplete Willis loop needs to be studied longitudinally.

In the present study, patients with TACI in the TS variant group and one of its subgroups (i.e., the TS variant with ipsilateral CI group) had more severe clinical symptoms and a worse prognosis than patients in the same group with PACI. In a study by Kaartinen *et al.* (23), 3 of 9 patients with middle cerebral artery infarction who presented with lethal edema, cerebral angiography showed ipsilateral TS atresia and IJV dysplasia, and in addition to infarct volume, poor ipsilateral cranial vein drainage was found to be associated with TS dysplasia or occlusion and with early lethal edema in MCA infarction. And, we also found that poor ipsilateral intracranial venous drainage was consistent with findings leading to a more severe clinical prognosis. Additionally, another study (24) found that among these four clinical types of OCSF, patients with TACI had heavy disease and neurological deficits on admission, a large infarct size, poor prognosis (especially thrombosis of the more proximal intracranial vessels), and a high disability rate. The NIHSS score is an important tool for evaluating the severity of neurological deficits of patients, and the 90-day post-infarction mRS score is a measure of the neurological recovery of patients after CI. A lower score indicates less paralysis and a better prognosis. Munuera *et al.* (25) found that patients with ipsilateral TS occlusion or dysplasia in CI had a poor prognosis, and the incidence of ipsilateral IJV occlusion or dysplasia was comparable to that of patients with a good prognosis. The ratio of infarct contralateral TS occlusion or dysplasia or contralateral IJV occlusion or dysplasia was similarly distributed between patients with poorer prognosis and those with good function. TS variation with ipsilateral CI affects the prognosis of LAA-type anterior circulation infarction. Our study also reaffirms the idea that patients with TS variation with ipsilateral CI present with more severe symptoms in the clinical setting, and also that TS variation affects the prognosis of CI. This may be because the venous collapse and elevated retrograde pressure caused by venous sinus variation can lead to increased permeability, edema, and secondary hemorrhage within the damaged tissue. In ischemia, altered venous drainage can increase resistance and lead to greater edema. Poor venous outflow on the affected side is associated with poor arterial collateral circulation, and it is unclear whether venous sinus and/or IJV atresia or dysplasia actually restrict ipsilateral venous outflow and contribute to the development of early fatal



edema after middle cerebral artery infarction. The majority of patients enrolled in this study had moderate infarction, and the relatively small number of patients with TACI may have affected some of the statistical results.

This study clarified the risk factors for the development of LAA anterior circulation infarction, and analyzed correlations between the factors related to the clinical prognosis of patients with LAA anterior circulation infarction, and compared the TS variant group to the TS no variant group, and TS variant subgroups. Similar to previous studies, our findings suggest that we should pay more attention to the venous system in the future, and it is especially important to expand the sample size, increase the number of hemodynamic measurements, and conduct longer longitudinal studies. In-depth studies on the mechanisms of LAA-type anterior circulation infarction, and effective treatments also need to be conducted. We are of the view that more data, collected over a longer period, and more analysis studies should be conducted in the future.

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### Footnote

*Reporting Checklist:* The authors have completed the STROBE reporting checklist. Available at <https://atm.amegroups.com/article/view/10.21037/atm-22-197/rc>

*Data Sharing Statement:* Available at <https://atm.amegroups.com/article/view/10.21037/atm-22-197/dss>

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <https://atm.amegroups.com/article/view/10.21037/atm-22-197/coif>). The authors have no conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised

in 2013). The study was approved by ethics committee of Hongqi Hospital Affiliated to Mudanjiang Medical University (No. 202114), and informed consent was taken from all the patients.

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