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Temporal muscle thickness and clinical outcomes after thrombectomy for internal carotid artery occlusion

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Abstract

Objectives: Although endovascular thrombectomy (EVT) can help achieve enhanced functional recovery following anterior circulation large-vessel occlusion (ACLVO) stroke, some patients exhibit poor functional outcomes. We assessed the influence of temporal muscle thickness (TMT) on the functional status after 3 months in individuals with ACLVO stroke treated with EVT. *Methods:* This retrospective study analysed adult patients with ACLVO stroke who had undergone EVT between August 2017 and July 2023. We collected clinical, radiological, and laboratory data. The primary functional outcome was the modified Rankin scale (mRS) score 3 months after EVT, and the patients were categorised accordingly into unfavorable (mRS 3–6) and favorable (mRS 0–2) outcome groups. *Results:* Overall, 96 patients (mean age: 69.5 years; 58% men; admission NIHSS: 18.3±5.5) were enrolled. At 3-month follow-up, 74 (77.1%) patients experienced unfavorable functional outcomes. The mean TMT was significantly lower in the unfavorable outcome group compared to the favorable outcome group (5.32 ± 1.90 mm vs. 6.66 ± 1.51 mm, P = .003). In multivariate analysis, thinner TMT and higher NIHSS at 24-36 h post-EVT were independently associated with unfavorable 3-month functional outcomes. Subgroup analyses revealed that TMT was significantly associated with unfavorable outcomes in elderly patients (age ≥70 years, P = .044), those with Internal Carotid Artery (ICA) occlusion (P = .005), particularly in ICA occlusion patients achieving successful recanalization (P = .034).
Conclusions: Thinner TMT was independently associated with unfavorable 3-month functional outcomes in patients with acute ICA occlusion who had undergone EVT, suggesting its potential value as a prognostic marker.

Keywords: Stroke, ischemic; temporal muscle, cerebral arteries, thrombectomy, Modified Rankin Scale

INTRODUCTION

Acute ischemic stroke resulting from anterior circulation large-vessel occlusion (ACLVO) is a primary cause of disability and mortality.[1] Endovascular thrombectomy (EVT) has emerged as a vital therapy for patients with ACLVO, particularly when intravenous thrombolysis fails to adequately restore blood perfusion.[2] Several clinical trials and observational studies have reported that the 3-month modified Rankin scale (mRS) score is a crucial outcome measure in post-EVT follow-up.[3,4] A recent meta-analysis (involving 1,313 patients) reported that the percentage of patients attaining functional independence (mRS 0–2) at the 90-day post-EVT follow-up ranged from 19% to 71%.[5] Owing to the wide variability of functional outcome, identification of factors that influence post-EVT functional outcomes is essential.

Numerous factors have been proposed to predict EVT outcomes in patients with ACLVO, including age, sex, premorbid mRS, National Institute of

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Health Stroke Scale (NIHSS) score, occlusion site, Alberta Stroke Programme Early CT Score (ASPECTS), collateral circulation, diabetes mellitus, hypertension, atrial fibrillation, serum glucose, serum sodium, serum creatinine, EVT time window, modified thrombolysis in cerebral infarction (mTICI) score, and intracerebral haemorrhage (ICH).[6,7] Moreover, various scoring systems, such as the MT-DRAGON (age, initial National Institutes of Health Stroke Scale score, glucose level, pre-stroke mRS score, diffusion weighted imaging-Alberta Stroke Programme Early CT score ≤5) score, GADIS (Gender, Age, Diabetes mellitus history, Infarct volume, and current Smoker) score, Zhang's nomogram, and Totaled Health Risks in Vascular Events (THRIVE) score, have been devised to estimate the 3-month EVT outcome for patients with ACLVO stroke.[8-10] These scoring systems help clinicians identify candidates who may benefit most from EVT, thereby optimizing patient selection and therapeutic outcomes.

Recently, temporal muscle thickness (TMT), which directly correlates with nutritional status and muscle mass, has emerged as a new surrogate marker for predicting functional outcomes, dysphagia, and sarcopenia risk in patients with stroke.[11] Furthermore, TMT has been demonstrated as a prognostic parameter in patients with brain metastasis, primary glioblastoma, and primary central nervous system lymphoma.[12-14] While TMT has been established as a surrogate marker for predicting functional outcomes in general stroke patients, its predictive value may vary across different patient subgroups and stroke characteristics. Therefore, this study aimed to investigate the relationship between TMT and 3-month functional outcomes in ACLVO patients receiving EVT, with particular attention to variations across age groups, sex, and occlusion sites.

METHODS

Study setting and participants

This retrospective study enrolled patients with ACLVO who underwent EVT at our institution between August 2017 and July 2023. During the study reference period, the diagnosis of acute ACLVO in all patients was confirmed via computed tomography angiography (CTA). The indications for EVT at our institution were as follows: 1) age: ≥20 years; 2) onset-to-groin puncture time: <8 hours;[15] 3) premorbid mRS: 0–2; 4) NIHSS at admission: 8–30; and 5) occlusion site: internal carotid artery (ICA), M1/M2 segment of the middle cerebral artery (MCA), or A1/A2 segment of the anterior cerebral artery. Premorbid mRS was defined as the status before stroke onset. The indication of EVT was based on the guidelines of the Taiwan Stroke Society and American Stroke Association for EVT in patients with acute ischemic stroke as well as reimbursement regulations of Taiwan's national health insurance.[16] In addition, EVT was contraindicated for patients with any form of intracranial hemorrhage. Three months after EVT, the patients were classified into unfavorable (mRS 3–6) and favorable (mRS 0–2) outcome groups. This study was approved by the Kaohsiung Veterans General Hospital Human Research Committee and was performed in accordance with the Declaration of Helsinki (1975) principles. Informed consent was waived off considering the retrospective nature of the study and the absence of any additional risk to the participants. The flow chart is shown in Figure 1.

Data collection

The following data were collected: demographic information, cardiovascular risk factors (hypertension, diabetes mellitus, atrial fibrillation, hyperlipidemia, and ischemic heart disease), premorbid medication use (antithrombotic therapy, lipid–lowering agents, antiplatelets, and anticoagulants), clinical assessment (blood pressure, NIHSS, pre–EVT intravenous thrombolysis [IVT], and stroke etiology), imaging data (ASPECTS, TMT, and occlusion site), procedure-related characteristics (onset-to-groin puncture time, recanalization degree, and post-EVT ICH), and laboratory data at the time of admission (white blood cell count; estimated glomerular filtration rate; and haemoglobin, hematocrit, platelet, glucose, sodium, potassium, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride levels).

Stroke severity at the time of admission was evaluated using NIHSS, and the stroke etiology was classified according to the criteria of the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification.[17,18] Successful vessel recanalization was defined as an mTICI 2b or 3, and a repeat brain computed tomography (CT) scan was performed at 24 h after EVT.[19] The 3-month clinical outcome after stroke was evaluated using mRS, and favorable functional outcome was defined as mRS 0–2.

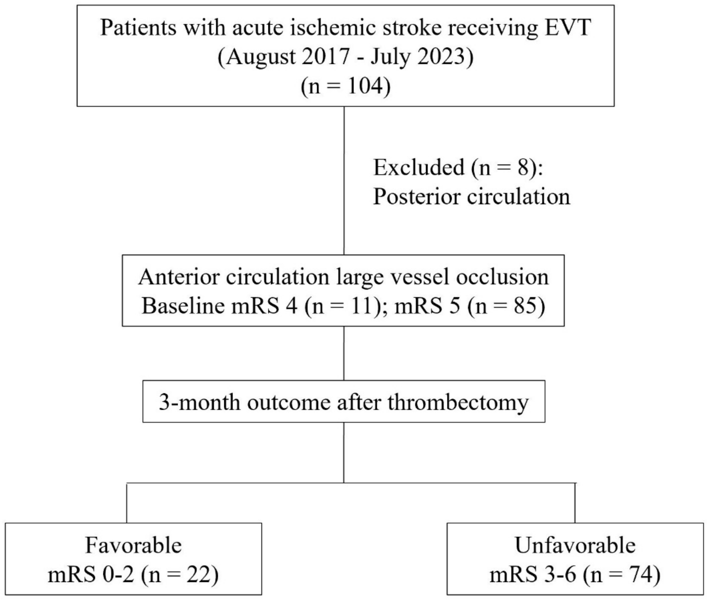

Figure 1. Flowchart summarizing study identification and selection.
Abbreviations: EVT = Endovascular thrombectomy; mRS = Modified Rankin Scale

TMT measurement

The ipsilateral TMT was measured using a CT scan. All CT and CTA scans were obtained with the following parameters: section thickness and intervals of 0.625 mm each; scan field of view, 320 × 320 mm; and matrix, 512 × 512. The slice thickness was reconstructed to 5 mm, with the window width set at 300 and the window level set at 20. TMT was independently measured by two experienced neurosurgeons using the RadiAnt DICOM Viewer (https://www.radiantviewer.com). The mean of these two measurements was used for analysis. The TMT measurements were taken perpendicular to the temporal muscle's long axis, 5 mm above the orbital roof, employing the sphenoid ridge and Sylvian fissure as anatomical reference points, as described previously.[20,21]

Statistical analyses

Categorical variables were expressed as frequency (percentage), with group differences evaluated using chi-squared test. Continuous variables were expressed as mean ± standard deviation or median (range) and analysed using a two-way analysis. Logistic regression was performed to identify factors associated with the 3-month mRS status, and the results were presented as adjusted odds ratio (aOR) and 95% confidence interval (CI). Variables showing *P* values <.10 in univariate analysis were included in the subsequent multivariate stepwise regression analysis.[22] Subgroup analyses were performed to identify subgroups that demonstrated a more significant association between TMT and functional outcomes. Statistical analysis was performed using SPSS software (version 20.0, IBM Corporation, Armonk, NY, USA), and two-tailed *P* values of <.05 were considered statistically significant.

RESULTS

Clinical characteristics of the study population

A total of 96 patients with acute ACLVO treated with EVT were included; the baseline characteristics of the included patients are summarized in Table 1. The median baseline NIHSS was 18.3 ± 5.5 and the median ASPECTS was 7 (interquartile range 6–8). The vascular occlusion sites were MCA in 55 (57%) and ICA in 41 (43%) patients. A total of 53 patients received IVT with recombinant tissue plasminogen activator before EVT, and the mean onset-to-groin puncture time was 278.3 ± 90.1 min. At the 3-month follow-up, 77% of the patients (74/96) showed unfavorable outcomes (mRS 3–6). The mean TMT was significantly lower in the unfavorable outcome group compared to the favorable outcome group (5.32 ± 1.90 mm vs. 6.66 ± 1.51 mm, *P* = .003). Age, hypertension, diabetes mellitus, TMT, ICH after EVT, 24–36-h

Table 1: Comparison of baseline data according to 3–month functional outcome of patients with large vessel occlusion in anterior circulation stroke underwent endovascular thrombectomy

Variable	Total n=96 (%)	3–month mRS 0–2 n=22 (%)	3–month mRS 3–6 n=74 (%)	p–value
Demographic characteristics				
Age, y (mean ± SD)	69.5±12.5	64.2±11.1	71.0±12.5	.025
Male	58 (60)	14 (64)	44 (60)	.725
Height, cm	162.7±9.5	164.6±8.8	162.2±9.7	.294
Weight, kg	66.1±13.8	70.1±13.1	64.9±13.9	.118
Body mass index, kg/m2	24.8±4.0	25.7±3.2	24.6±4.2	.252
Vascular risk factors				
Hypertension	31 (32)	3 (14)	28 (38)	.039
Diabetes mellitus	14 (15)	0 (0)	14 (19)	.035
Atrial fibrillation	53 (55)	13 (59)	40 (54)	.677
Hyperlipidemia	21 (22)	2 (9)	19 (26)	.143
Ischemic heart disease	17 (18)	4 (18)	13 (18)	.947
Medication use history				
Antithrombotic therapy	23 (24)	3 (14)	20 (27)	.261
Lipid lowering agent	16 (17)	2 (9)	14 (19)	.348
Antiplatelet	29 (30)	3 (14)	26 (35)	.066
Anticoagulant	16 (17)	3 (14)	13 (18)	1.000
Clinical assessment				
Diastolic blood pressure, mmHg	66.1±21.1	72.8±24.1	64.1±19.8	.090
Systolic blood pressure, mmHg	125.4±29.3	131.3±30.3	123.6±29.0	.284
Baseline NIHSS, score	18.3±5.5	16.7±5.3	18.8±5.5	.110
ASPECTS (median, IQR), score	7 (6–8)	7 (6–8)	8 (6–8)	.613
TMT, mm	5.63±1.9	6.66±1.51	5.32±1.90	.003
Onset–to–groin puncture time, min	278.3±90.1	266.4±88.4	281.8±90.9	.483
IVT	53 (55)	15 (68)	38 (51)	.163
Stroke etiology				.160
Atherosclerotic	31 (32)	4 (18)	27 (37)	
Cardioembolic	52 (54)	13 (59)	39 (53)	
Others	13 (14)	5 (23)	8 (11)	
Vessel occlusion				
Lesion – ICA	41 (43)	9 (41)	32 (43)	.846
Left side occlusion	58 (60)	15 (68)	43 (58)	.396
Successful recanalization (mTICI 2b–3)	77 (80)	19 (86)	58 (78)	.409
Symptomatic ICH	14 (15)	0 (0)	14 (19)	.035
24 to 36–h NIHSS, score	15.7 ±10.4	7.5±5.6	18.1±10.3	<.001
Laboratory data on admission				
WBC (x1000/Cumm), n=96	8.6±2.9	8.6±2.5	8.6±3.1	.989
Haemoglobin (g/dL)	13.4±2.3	13.8±1.9	13.3±2.4	.421
Hematocrit (%)	40.5±6.2	41.2±5.6	40.4±6.4	.586
Platelet (x1000/Cumm)	216.9±69.7	227.7±75.8	213.7±68.0	.410
Glucose (mg/dL), n=96	146.6±57.6	119.6±23.4	154.6±62.2	.011
Creatinine (mg/dL), n=96	1.3±1.3	1.1±0.4	1.4±1.4	.396
eGFR (ml/min/1.73), n=96	66.5±25.3	67.6±24.4	66.2±25.7	.815
Sodium (mmol/L), n=96	137.5±3.2	138.1±1.8	137.4±3.5	.348
Potassium (mmol/L), n=96	4.1±0.6	3.9±0.4	4.1±0.7	.115
HDL–C (mg/dL), n=83	42.2±12.6	37.4±7.7	43.5±13.4	.068
LDL–C (mg/dL), n=83	96.2±40.7	95.6±24.3	96.3±44.3	.943
TG (mg/dL), n=83	137.4±92.1	150.7±72.6	133.2±97.6	.463

Abbreviation: mRS = Modifid Rankin Scale; NIHSS = National Institutes of Health Stroke Scale; ASPECTS = Alberta Stroke Programme Early CT Score; IQR = interquartile range; TMT = temporal muscle thickness; IVT = intravenous recombinant tissue–type plasminogen activator therapy; MCA = middle cerebral artery; mTICI = modified thrombolysis in cerebral infarction; ICH = intracranial haemorrhage; WBC = white blood cells count; eGFR = estimated glomerular filtration rate; HDL–C = High Density lipoprotein cholesterol; LDL–C = Low Density lipoprotein cholesterol; TG = triglyceride

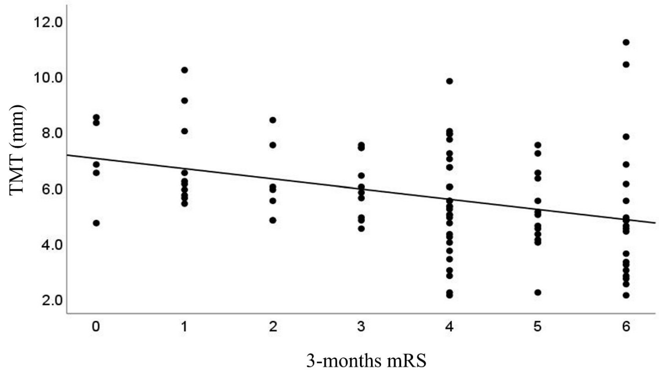

Figure 2. Correlation between temporal muscle thickness and 3-month mRS in patients with ACLVO stroke after EVT.
Abbreviations: TMT = temporal muscle thickness; mRS = modified Rankin Scale

NIHSS after EVT, and serum glucose level at the time of admission were significantly associated with functional outcomes at the 3-month follow-up. These factors were all negatively associated with functional outcomes: older age, hypertension, diabetes, thinner TMT, ICH after EVT, higher 24-36h NIHSS, and elevated serum glucose levels.

Independent covariates of 3-month unfavorable outcomes after EVT

In univariate logistic analyses, older age (aOR, 1.04; 95% CI, 1.00–1.09; $P = .03$), hypertension (aOR, 3.86; 95% CI, 1.05–14.22, $P = .043$), thinner TMT (aOR, 0.68; 95% CI, 0.52–0.90; $P = .006$), higher 24–36-h NIHSS after EVT (aOR, 1.20; 95% CI, 1.09–1.32; $P < .001$), and higher serum glucose level at the time of admission (aOR, 1.02; 95% CI, 1.00–1.04; $P = .017$) were associated with 3-month unfavorable outcomes. Multivariate logistic regression analyses conducted with stepwise selection of variables revealed that thinner TMT (aOR, 0.63; 95% CI, 0.42–0.96; $P = .03$) and higher 24–36-h NIHSS after EVT (aOR, 1.21; 95% CI, 1.08–1.35; $P = .001$) were independently associated with 3-month unfavorable outcomes (Table 2). As illustrated in Figure 2, the scatter plot demonstrates a negative correlation between TMT and 3-month mRS scores ($R^2 = .118$), indicating that patients with thinner temporal muscle thickness tend to have poorer functional outcomes at 3 months.

Subgroup analyses

In subgroup analyses stratified by age, sex, vessel occlusion site, and recanalization degree, it was found that thinner TMT was significantly associated with unfavorable outcomes in the following subgroups: patients age ≥70 years, females, males, ICA occlusion, successful recanalization (mTICI 2b-3), and poor recanalization (mTICI 0-2a). Further analysis of the ICA occlusion group stratified by recanalization degree revealed that thinner TMT contributed to unfavorable outcomes in patients with successful recanalization after EVT (Table 3). As shown in Figure 3, TMT exhibited significant negative correlations with 3-month mRS scores in both successful recanalization (mTICI 2b-3,

A

B

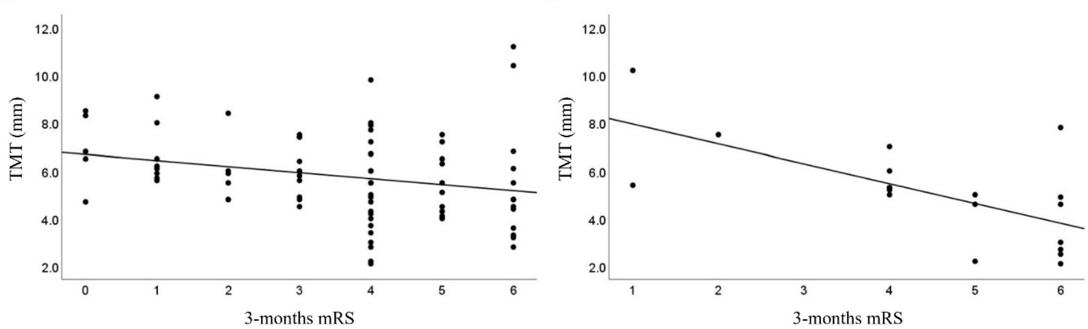

Figure 3. Association between TMT and 3-month mRS in patients with A: successful recanalization (mTICI 2b-3) and B: poor recanalization (mTICI 0-2a).
Abbreviations: TMT = temporal muscle thickness; mRS = modified Rankin Scale

Table 2: Logistic regression analyses for 3–month unfavorable outcome (mRS 3–6) initial indicators in patients with acute ischemic stroke treated with endovascular thrombectomy

Variable	Univariate		Multivariate	
	OR (95% CI)	p–value	aOR (95% CI)	p–value
Age, y	1.04 (1.00–1.09)	.030		
Male	1.19 (0.45–3.20)	.725		
Body mass index, kg/m2	0.93 (0.83–1.05)	.250		
Hypertension	3.86 (1.05–14.22)	.043		
Diabetes mellitus	–			
Atrial fibrillation	0.81 (0.31–2.14)	.677		
Hyperlipidemia	3.46 (0.74–16.18)	.116		
Ischemic heart disease	0.96 (0.28–3.31)	.947		
Antithrombotic therapy	2.35 (0.63–8.79)	.206		
Lipid lowering agent	2.33 (0.49–11.17)	.289		
Antiplatelet	3.43 (0.93–12.69)	.065		
Anticoagulant	1.35 (0.35–5.24)	.665		
Diastolic blood pressure, mmHg	0.98 (0.96–1.00)	.096		
Systolic blood pressure, mmHg	0.99 (0.97–1.01)	.284		
Baseline NIHSS, score	1.08 (0.98–1.19)	.113		
ASPECTS	0.94 (0.71–1.24)	.649		
TMT, mm	0.68 (0.52–0.90)	.006	0.63 (0.42–0.96)	.030
Onset–to–groin puncture time, min	1.00 (1.00–1.01)	.479		
IVT	0.49 (0.18–1.35)	.168		
Stroke etiology				
Atherosclerotic	Ref			
Cardioembolic	0.44 (0.13–1.51)	.194		
Others	0.24 (0.05–1.10)	.066		
Lesion– ICA				
Left side occlusion	0.65 (0.24–1.78)	.398		
Successful recanalization (mTICI 2b–3)	0.57 (0.15–2.18)	.414		
Symptomatic ICH	–			
24 to 36–h NIHSS, score	1.20 (1.09–1.32)	< .001	1.21 (1.08–1.35)	.001
WBC (x1000/Cumm)	1.00 (0.85–1.18)	.989		
Haemoglobin (g/dL)	0.92 (0.74–1.14)	.417		
Hematocrit (%)	0.98 (0.90–1.06)	.582		
Platelet (x1000/Cumm)	1.00 (0.99–1.00)	.407		
Glucose (mg/dL)	1.02 (1.00–1.04)	.017		
Creatinine (mg/dL)	1.54 (0.55–4.36)	.413		
eGFR (ml/min/1.73)	1.00 (0.98–1.02)	.813		
Sodium (mmol/L)	0.92 (0.78–1.09)	.345		
Potassium (mmol/L)	2.10 (0.83–5.28)	.115		
HDL–C (mg/dL)	1.05 (1.00–1.11)	.073		
LDL–C (mg/dL)	1.00 (0.99–1.01)	.942		
TG (mg/dL)	1.00 (0.99–1.00)	.462		
		Hosmer–Lemeshow test	χ^2 =2.448	.864

Abbreviation: mRS = Modified Rankin Scale; OR = odds ratio; aOR = adjusted odds ratio; NIHSS = National Institutes of Health Stroke Scale; ASPECTS = Alberta Stroke Programme Early CT Score; IQR = interquartile range; TMT = temporal muscle thickness; IVT = intravenous recombinant tissue–type plasminogen activator therapy; MCA = middle cerebral artery; mTICI = modified thrombolysis in cerebral infarction; ICH = intracranial haemorrhage; WBC = white blood cells count; eGFR = estimated glomerular filtration rate; HDL–C = High Density lipoprotein cholesterol; LDL–C = Low Density lipoprotein cholesterol; TG = triglyceride

Table 3: Subgroup analyses of the association between the thickness of temporal muscles and 3–month functional outcome

Variable	Total	mRS 0–2	mRS 3–6	p–value
Age				
< 70 years	63.8±19.2	68.6±16.1	61.4±20.4	.228
≥ 70 years	49.0±15.8	61.2±11.7	47.4±15.7	.044
Sex				
Female	48.7±13.8	58.4±7.3	46.1±13.3	.017
Male	61.2±20.6	71.2±16.6	58.1±20.9	.036
Vascular occlusion site				
ICA	52.8±17.6	67.0±20.2	48.8±14.8	.005
MCA	58.9±19.7	66.2±11.3	56.6±21.3	.124
Recanalization				
Successful (mTICI 2b–3)	57.7±18.4	64.9±13.5	55.3±19.2	.047
Poor (mTICI 0–2a)	50.7±20.9	77.0±24.1	45.8±16.8	.013
Recanalization of ICA occlusion				
Successful (mTICI 2b–3)	56.5±16.0	63.9±17.3	49.2±14.6	.034
Poor (mTICI 0–2a)	62.9±25.0	78.0±33.9	47.9±16.1	.072

Abbreviation: mRS = Modified Rankin Scale; ICA = internal carotid artery; MCA = middle cerebral artery; mTICI = modified thrombolysis in cerebral infarction

P = .047) and poor recanalization (mTICI 0-2a, P = .013) groups, with a more pronounced TMT difference observed in the poor recanalization group. In the subgroup analysis focusing on ICA occlusion patients, this correlation remained significant in the successful recanalization group (P = .034) but not in the poor recanalization group (P = .072).

DISCUSSION

In this retrospective cohort study, we investigated the relationship between TMT and 3-month functional outcomes in patients with acute ACLVO stroke who were treated with EVT. A thinner TMT on the ipsilateral side of cerebral vessel occlusion was found to be independently linked to unfavorable 3-month functional outcomes. Subgroup analyses revealed that this association was significant in patients aged ≥70 years, irrespective of their gender, presence of ICA occlusion, irrespective of recanalization degree, especially ICA occlusion patients exhibiting successful recanalization after EVT.

The association between sarcopenia and poor stroke functional outcomes is well established.[20, 23, 24] Stroke patients with sarcopenia, as measured by TMT in CT scans, often show poor mRS and an increased risk of dysphagia. The assessment of sarcopenia encompasses both muscle mass and muscle strength.[25] TMT serves as a dependable indicator of muscle mass and strength in stroke patients.[26,27] Brain CT is routinely conducted in patients with acute ischemic stroke, and measuring CT-TMT provides an uncomplicated, noninvasive assessment approach for sarcopenia. Regarding the cutoff value of Temporal Muscle Thickness (TMT) and its relationship with sexes, current literature provides limited direct data. Our subgroup analyses aligned with these findings, showing that males generally had thicker TMT compared to females. Katsuki *et al*. identified prognostic TMT thresholds in subarachnoid haemorrhage patients: 4.9 mm for females and 6.7 mm for males, suggesting potential sexual differences.[28] Importantly, we found that TMT was significantly associated with 3-month mRS in both sexes, indicating its prognostic value in stroke patients across sexes. Furthermore, our subgroup analyses demonstrated a negative correlation between TMT and 3-month mRS in patients aged ≥70 years, which may be attributed to the significant reduction in muscle mass observed in this age group.[29] Alawieh *et al*. found that ischemic stroke patients over 80 years old have a higher risk of mortality, unfavorable outcomes, and post-operative bleeding after receiving EVT.[30] Based on these findings, we speculate that muscle mass may be one of the critical factors affecting the prognosis of stroke treatment in elderly patients. Furthermore, TMT measurements exhibited consistent prognostic trends in both male and female patients, regardless of their age. Nozoe *et al.* demonstrated a similar correlation between TMT and sarcopenia risk, which was independently associated with stroke functional outcomes in male and female patients aged >65 years.[23]

Beyond serving as a sarcopenia indicator, our findings suggest that TMT may reflect underlying vascular health and influence stroke outcomes. This hypothesis is supported by recent research demonstrating the connection between muscle mass and vascular function. For instance, Campos *et al*. found that sarcopenia is independently associated with subclinical atherosclerosis and endothelial dysfunction in very elderly individuals, demonstrating that lower muscle mass significantly correlates with higher coronary calcium scores.[31] Additionally, Rodriguez *et al*. and Dvoretskiy *et al*. have established that muscle mass is closely related to vascular health, a critical factor in stroke prognosis.[32,33] We therefore propose that TMT, while specifically measuring temporal muscle mass, could serve as a proxy indicator of overall vascular health status and thereby influence post-stroke outcomes. Our findings support this concept, as the association between TMT and 3-month mRS remained significant after adjusting for confounders. Furthermore, this relationship aligns with our current understanding of how favorable vascular conditions, particularly collateral circulation, can protect penumbral tissue from progressing to infarction.[34]

While direct evidence of temporal muscle's ECA supply contributing to intracranial blood flow via anastomoses is limited, several studies have documented the extensive network of anastomoses between extracranial and intracranial vessels as potential compensatory mechanisms.[35,36] The secondary collateral circulation, primarily leptomeningeal collaterals, can be rapidly recruited during acute ischemia, as demonstrated in rat models where these collaterals reached maximal vasodilatation within 12 seconds of ICA occlusion.[36] The clinical significance of collateral circulation has been well established, with recent studies showing that good collateral status can extend the EVT time window beyond 6 hours.[37] In line with the importance of collateral circulation, we observed that the physiological reserve reflected by TMT becomes particularly crucial when primary circulation is compromised, as evidenced by the more pronounced TMT disparity between outcome groups in patients with poor recanalization. Specifically regarding the anatomical basis, the blood flow in the temporal muscle is supplied by the superficial temporal artery (STA) and maxillary artery (MA), both originating from the external carotid artery.[38,39] There are numerous arterial connections between the STA–MA complex and the ICA. The TMT may suggest the developmental wellness of the STA–MA complex and hence implies the STA–MA complex's physiological reserve of compensatory collateral flow support in the clinical setting of acute ICA occlusion (Figure 4). This observation explains why TMT contributes to the outcome of ICA occlusion rather than MCA occlusion, as the MCA occlusion site is distal to this collateral anastomosis. However, the helpfulness provided by the STA–MA complex appears to be temporary. In our subgroup analysis of the ICA occlusion group, while TMT was significantly associated with outcomes in those who achieved successful recanalization after EVT, this association was

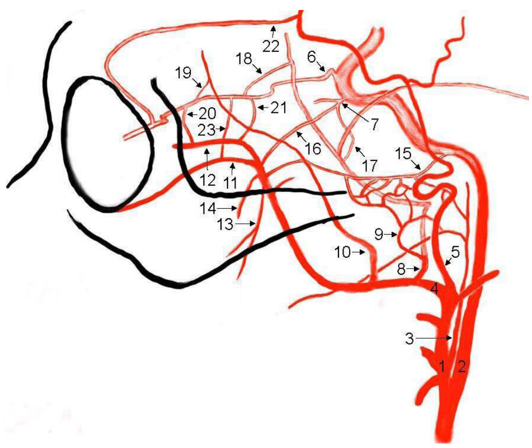

Figure 4. A schematic illustrating potential anastomoses between the superficial temporal artery–maxillary artery (STA–MA) complex and internal carotid artery (ICA).
1: external carotid artery; 2: ICA; 3: ascending pharyngeal artery; 4: MA; 5: STA; 6: ophthalmic artery(OA); 7: inferolateral trunk (ILT); 8: middle meningeal artery (MMA); 9: accessory middle meningeal artery; 10: deep temporal artery; 11: infraorbital artery(IOA); 12: sphenopalatine artery; 13: descending palatine artery; 14: posterior superior alveolar artery; 15: vidian artery; 16: artery of foramen rotundum; 17: anastomosis of ILT and MMA; 18: lacrimal artery; 19: distal lacrimal artery; 20: anterior ethmoidal artery; 21: posterior ethmoidal artery; 22: supraorbital artery; 23: anastomosis of OA and IOA. Artery 15 to artery 23 represent the connection between the STA–MA complex and ICA.

not significant in the group with unsuccessful recanalization (mTICI 0-2a). This finding suggests that the STA–MA complex plays a reinforcing role, but its ability to sustain collateral support is limited when normal blood flow is not restored promptly. As demonstrated by Al-Dasuqi *et al*., while collateral status significantly influences infarct volume after stroke, successful reperfusion ultimately determines functional outcomes.[40]

This study had some limitations. First, this was a retrospective study with a relatively small sample size. However, we conducted a power analysis (using Shieh-O'Brien large sample approximation) based on TMT and poor functional outcomes (power: 0.857). Second, we did not analyze specific occlusion locations within the ICA territory, which might influence collateral patterns and outcomes. Third, our cohort's relatively prolonged onset-to-groin puncture time (278.3 ± 90.1 min) potentially impacted the 3-month functional outcomes, particularly given the time-sensitive nature of collateral circulation effectiveness. Future research should focus on interventions to improve overall nutritional status and muscle mass, which may in turn increase TMT and potentially lead to better outcomes in ACLVO stroke patients undergoing EVT.

In conclusion, thinner TMT indicated an independent association with poor 3-month functional outcomes of EVT in patients with ACLVO stroke. TMT demonstrated stronger predictive value in elderly patients (age ≥70 years) and particularly in those with ICA occlusion who achieved successful recanalization after EVT. These findings suggest TMT might serve as a practical indicator of vascular health and potential stroke outcomes.

DISCLOSURE

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