Original research

Predictors of tissue infarction from distal emboli after mechanical thrombectomy

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ABSTRACT

Background Distal embolization after endovascular thrombectomy (EVT) is common. We aimed to determine factors associated with tissue infarction in the territories of distal emboli.

Methods This is a retrospective cohort study of consecutive patients with anterior circulation large vessel occlusions who underwent EVT from 2015 to 2021. Patients with Thrombolysis In Cerebral Infarction (TICI) 2b reperfusion and follow-up imaging were identified. Baseline characteristics, procedural details, and imaging findings were reviewed. Primary outcome was categorized according to the occurrence of infarction at the territory of distal embolus on follow-up diffusion-weighted imaging MRI.

Results Of 156 subjects, 97 (62%) had at least one infarction in the territories at risk. Hypertension was significantly more prevalent in the infarct group (83% vs 53%, P=0.001). General anesthesia was more commonly used in the infarct group (60% vs 43%, P=0.037). The median number of distal emboli and diameter of the occluded vessel were similar. After adjusting for confounders, hypertension (aOR 4.73, 95% CI 1.81 to 13.25, P=0.002), higher blood glucose (aOR 1.01, 95% CI 1.00 to 1.03, P=0.023), and general anesthesia (aOR 2.75, 95% CI 1.15 to 6.84, P=0.025) were independently associated with infarction. The presence of angiographic leptomeningeal collaterals predicted tissue survival (aOR 0.13, 95% CI 0.05 to 0.33, P<0.001). 90-day modified Rankin scale (mRS) scores were worse for the infarction patients (mRS 0–2: infarct, 39% vs 55%, P=0.046).

Conclusions Nearly 40% of patients with TICI 2b had no tissue infarction in the territory of a distal embolus. The association of infarction with hypertension and general anesthesia suggests late or post-procedural blood pressure management could be a modifiable factor. Patients with poor leptomeningeal collaterals or hyperglycemia may benefit from further attempts at revascularization.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Distal embolization during endovascular thrombectomy is common with variable tissue outcomes. Prior studies have focused on factors associated with distal emboli after EVT, not factors associated with tissue outcome. There is uncertainty regarding patient and procedural characteristics associated with tissue outcomes after distal embolization.

WHAT THIS STUDY ADDS

⇒ This retrospective review suggests that the presence of leptomeningeal collaterals might favor tissue survival after distal embolization. On the other hand, patients with a history of hypertension, higher admission blood glucose levels, and those who undergo EVT under general anesthesia may benefit from additional attempts to achieve revascularization after distal embolization.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Our study suggests that certain patient and procedural characteristics can be relevant to consider when deciding to perform additional attempts to achieve complete revascularization of distal emboli. Further research focused on the factors that determine tissue survival after distal embolization is suggested.

INTRODUCTION

Endovascular thrombectomy (EVT) has been established as the first-line treatment for patients presenting with large vessel occlusion (LVO) acute ischemic stroke due to its extremely high efficacy.[1] However, the embolization of thrombus fragments during EVT is common (about two in every five cases) and produces lower rates of complete reperfusion, larger final infarct volumes, and worse functional outcomes.[2 3] In general, distal emboli can arise in two ways: due to embolization from the initial target occlusion to a downstream vessel, or due to embolization to a new vascular territory that was not initially affected by the target occlusion.[3] Notably, the outcome of the tissue in the vascular territory affected by the distal emboli is variable, with infarction occurring in about half of the patients.[4] Currently, there are limited data to guide the decision to pursue further EVT or intra-arterial lytic therapy in cases where distal emboli are encountered. Furthermore, the retrieval of distal emboli has been shown to be less effective and carries additional risk for ischemic and hemorrhagic complications.[5 6] Compared with proximal larger vessels, the distal vasculature has smaller diameters and greater tortuosity. Most of the devices used for EVT have been developed for the proximal vessels.

Prior studies have examined the factors associated with the occurrence of distal emboli after EVT, and there is limited knowledge regarding the patient

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and procedural characteristics that may be associated with brain infarction (or survival) in the territories at risk.[2 3] Hence, we aimed to study the characteristics and determine the factors that are associated with infarction secondary to distal emboli after EVT.

METHODS

Study design and inclusion criteria

We performed a retrospective cohort study of a prospectively maintained database of consecutive patients with acute ischemic stroke due to LVO who underwent EVT at our institution from 2015 to 2021. The following inclusion criteria were used: (1) patients with internal carotid artery or middle cerebral artery occlusions, (2) Thrombolysis In Cerebral Infarction (TICI) score of 2b,[7] and (3) follow-up MRI within 24±12 hours. At our center, the protocol and machinery used for image acquisition have been previously described.[8] Briefly, all the patients were evaluated with a baseline non-contrast CT±CT angiography and CT perfusion. The follow-up MRI was performed 24±12 hours after EVT unless there was a contraindication to MRI. The perfusion imaging was evaluated with a fully automated Rapid software (iSchemaView, Menlo Park, CA) that is used as part of the institutional clinical stroke protocol. Institutional review board approval was obtained (IRB # 201910789), and the need for informed consent was waived due to the retrospective nature of the study. This study is reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[9]

General EVT procedure details

Both conscious sedation and general anesthesia were used. The attending interventional physician made this decision based on factors such as the medical condition of the patient, the patient's ability to remain still, or the presence of a tandem lesion. Some patients were randomized into different anesthesia regimens as part of clinical trials. All EVT cases were performed with the assistance of an attending anesthesiologist and anesthesia team.

All cases were done on biplane neuroangiograhic units (Axiom Artis or Icono, Siemens Healthineers) using transfemoral access, with ultrasound guidance and a micropuncture set. An 8 French sheath was placed, followed by a large bore guide catheter into the common or internal carotid artery. The EVT technique for LVO was performed using a stent retriever, aspiration, or a combination of both. Anteroposterior and lateral digital subtraction angiography (DSA) runs were obtained at the conclusion of the procedure. The patients were then accompanied to the intensive care unit for further management according to current clinical guidelines.[10] The decision to extubate intubated patients was made at the conclusion of the procedure in consultation with the anesthesia faculty.

Clinical data elements

Patient demographics, pre-existing conditions, stroke risk factors, procedural characteristics, and functional outcomes as well as details of the procedure (including the type of anesthesia and devices used) were obtained by review of a prospective institutional LVO database. Functional outcome was evaluated using the modified Rankin scale (mRS) by board-certified vascular neurologists at discharge and 90 days after EVT. A favorable functional outcome was defined by an mRS between 0–2.

Angiographic image assessment

DSA images from the procedure were reviewed for all patients meeting the established inclusion criteria. First, the TICI score was independently adjudicated by two independent investigators blinded to the clinical data. All the patients who had a post-EVT TICI score of 2c (miscategorized as TICI 2b) were excluded. Next, the distal emboli were identified. A distal embolus was defined as an occlusion downstream in the territory of the initial target occlusion. For this study, embolization to a new territory not initially affected by the target occlusion was not included as a distal embolus. The location and diameter of the vessel where the distal occlusion was identified were recorded and measured, respectively. Leptomeningeal collaterals supplying the territory of the target occlusion were graded as absent if no angiographically relevant collaterals were visualized, and present if leptomeningeal anastomoses were filling the territory of the target occlusion by more than half.[11] Finally, any patient with flow-limiting vasospasm after EVT (that might have contributed to subsequent tissue infarction) was excluded.

Determination of infarct at the territory at risk and symptomatic intracranial hemorrhage

MRI diffusion-weighted trace images (DWI) obtained 24±12 hours after EVT were reviewed for infarct presence or absence. The territories at risk were determined through visual inspection by an experienced attending neurointerventionalist (CPD) comparing the lateral image of the final DSA run and the DWI images. If infarction in any of the territories at risk was identified on DWI, the baseline non-contrast CT was reviewed in conjunction with the MRI. The DWI lesions that corresponded to pre-existing infarctions (evidenced in the baseline CT) were excluded. The cases that presented at least one infarct within any of the territories at risk were included in the infarct group. If no infarct was identified, the patient was included in the non-infarct group.

Symptomatic intracranial hemorrhage was defined by the evidence of intracranial hemorrhage in the post-EVT MRI study associated with a change of ≥4 points (compared with baseline) in the National Institutes of Health Stroke Scale (NIHSS).

Statistical analysis

Continuous variables were reported as medians with IQR, and categorical variables as frequencies and percentages. Baseline characteristics are summarized for both groups (infarct and non-infarct). The categorical variables of each group were compared using the χ^2 test. Continuous variables were compared using the Wilcoxon rank-sum test.

To identify the predictors of tissue infarction in the territories at risk, we conducted an Akaike information criterion-based stepwise regression procedure on variables selected by preliminary univariable statistical significance ($P<0.05$) and clinical significance based on previous literature reports.[2] A multivariable logistic regression was performed on the model selected by the stepwise regression algorithm. The odds ratio (OR) and 95% confidence interval (95% CI) estimates were obtained from the mixed-effects logistic regression fit applied. All analyses were performed using R software (version 4.1.0) for Windows. $P<0.05$ was considered significant.

RESULTS

Between 2015–2021, 800 patients underwent EVT for LVO. A total of 190 patients had an internal carotid artery or middle cerebral artery initial occlusion, final TICI 2b reperfusion, and a 24±12 hour

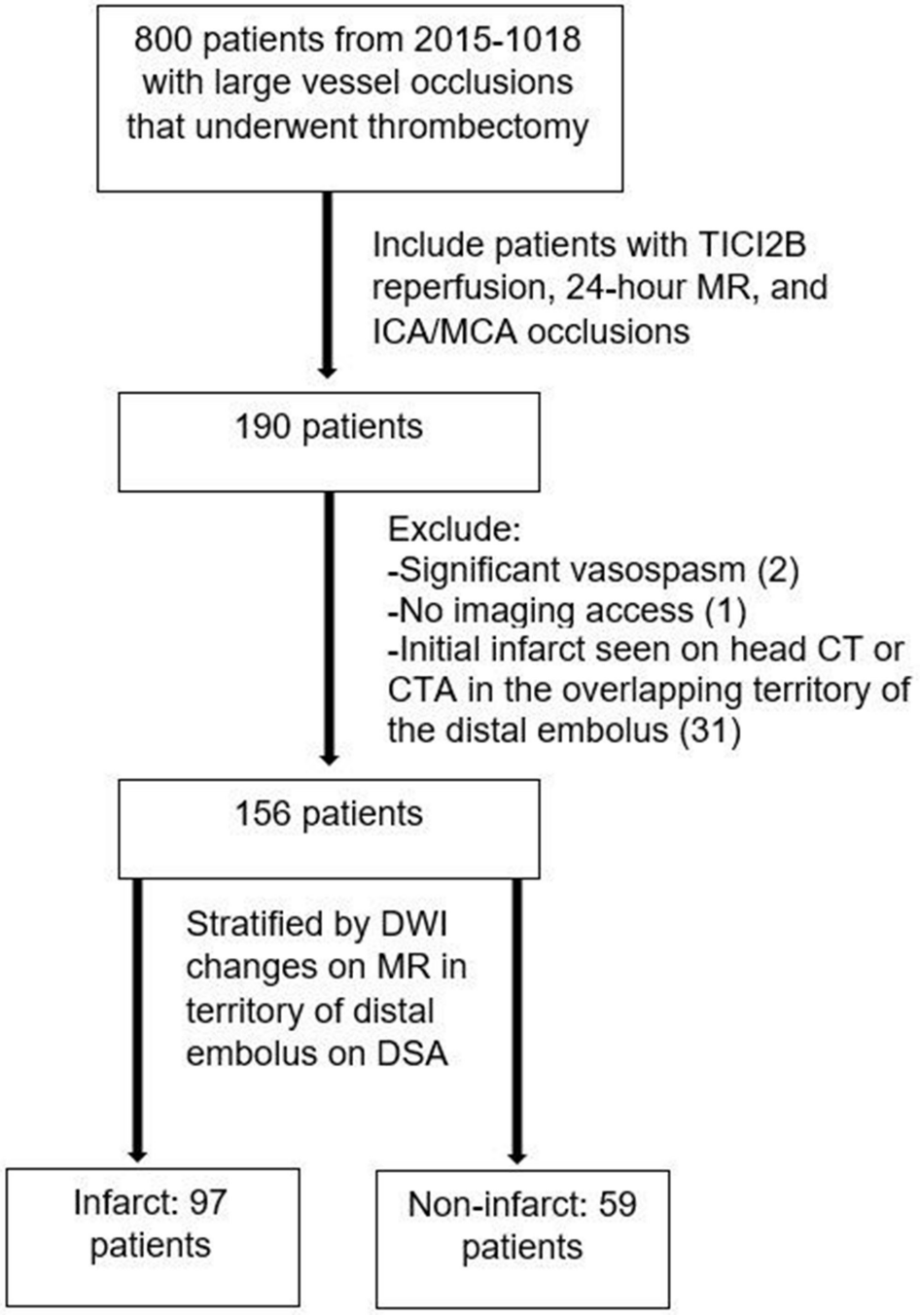

Figure 1 Flow chart of the study cohort selection. CTA, CT angiography; DSA, digital subtraction angiography; DWI, diffusion-weighted imaging; ICA, internal cerebral artery; MCA, middle cerebral artery; TICI, Thrombolysis In Cerebral Infarction.

MRI. After excluding 34 patients (for vasospasm after EVT, unavailable baseline imaging, or initial infarct in the area of distal embolus), a total of 156 patients were included in the study. A flow chart of the patient inclusion is presented in figure 1.

Clinical and radiographic characteristics

As shown in table 1 and figure 2, in 97 (62%) patients at least one infarct in a territory at risk (with identified distal emboli) was evident on DWI MRI, and in 59 (38%) patients there was no infarct. The median age (infarct, 72 years vs 69 years, P=0.822), proportion of females (51% vs 54%, P=0.652), median baseline NIHSS (infarct, 16 vs 16, P=0.987), and the presenting blood glucose levels (infarct, 129 mg/dL vs 120 mg/dL, P=0.084) were similar. All pre-existing conditions, smoking and pre-morbid medication were similar except for a history of hypertension, which was significantly higher in the infarct group (83% vs 53%, P=0.001).

Median baseline Alberta Stroke Program Early CT Score (ASPECTS) (infarct, 8 vs 8, P=0.474), and median volumes of CT perfusion time-to-maximum >6 s (110 mL vs 105 mL, P=0.357) and >10 s (43 mL vs 40 mL, P=0.388) were not different. The use of intravenous tissue plasminogen activator was similar between groups (infarct, 53% vs 58%, P=0.653). Median time (in minutes) from the last known well to groin puncture (infarct, 302 vs non-infarct, 362, P=0.510) and reperfusion (376 vs 397, P=0.723) were not different. The use of general anesthesia was

Table 1 Comparison of baseline characteristics stratified by the presence of infarct (n=156)

Variable	Infarct (n=97)	Non-infarct (n=59)	P value
Age, median (IQR)	72 (61–80)	69 (60–80)	0.822
Female, n (%)	49 (51)	32 (54)	0.652
White, n (%)	89 (92)	58 (98)	0.387
Baseline NIHSS, median (IQR)	16 (10–21)	16 (11–20)	0.987
Glucose, mg/dL, median (IQR)	129 (109–166)	120 (108–142)	0.084
Medical history, n (%)			
Hypertension	80 (83)	31 (53)	**0.001****
Atrial fibrillation	29 (30)	19 (32)	0.762
Diabetes	27 (28)	14 (24)	0.572
Coronary artery disease	20 (21)	7 (12)	0.161
Prior stroke or transient ischemic attack	13 (13)	7 (12)	0.781
Smoking	37 (38)	19 (32)	0.453
Premorbid medications, n (%)			
Antiplatelet drugs	36 (37)	21 (36)	0.848
Anticoagulants	14 (14)	10 (17)	0.673
Anti-diabetic drugs	18 (19)	8 (14)	0.417
Antihypertensives	65 (67)	35 (60)	0.332
Statins	33 (34)	25 (42)	0.295
Pre-EVT imaging			
Baseline ASPECTS	8 (6–9)	8 (7–9)	0.474
CTP time-to-maximum >6 s, mL, median (IQR)	110 (78–147) †	105 (65–135)‡	0.357
CTP time-to-maximum >10 s, mL, median (IQR)	43 (18–87) †	40 (11–79)‡	0.388
Treatment characteristics			
Intravenous tPA, n (%)	51 (53)	34 (58)	0.653
Time from LKW to groin puncture, median (IQR)	302 (242–555)	362 (251–668)	0.510
Time from LKW to reperfusion, median (IQR)	376 (300–604)	397 (303–706)	0.723
General anesthesia, n (%)	58 (60)	25 (43)	**0.037***
Initial occlusion on DSA, n (%)			0.530
Internal carotid artery	14 (14)	7 (12)	
Middle cerebral artery M1 segment	66 (68)	42(71)	
Middle cerebral artery M2 segment	14 (14)	10 (17)	
Middle cerebral artery M3 segment	3 (3)	0 (0)	
Evidence of leptomeningeal collaterals, n (%)	56 (43)	67 (72)	**0.001***
Use of balloon guide catheter, n (%)	3 (3)	1 (2)	0.989
Stent retriever characteristics, median (IQR)			
Diameter, mm	6 (4–6)	4 (4–6)	**0.048***
Length, mm	37 (25–40)	30 (20–40)	**0.046***
Intra-arterial tPA	0 (0)	1 (2)	0.378
Emboli after TICI 2b revascularization			
Number of distal emboli, median (IQR)	1 (1–2)	1 (1–2)	0.667
≥1 distal embolus, n (%)	19 (32)	32 (33)	0.919
Occluded vessel diameter, mm, median (IQR)	1.08 (0.85–1.3)	1.04 (0.8–1.3)	0.662
Follow-up characteristics			
Intracranial hemorrhage, n (%)	10 (10)	6 (10)	0.978
Death, n (%)	22 (23)	7 (12)	0.084
Discharge mRS, n (%)			**0.002***
0–2	13 (13)	20 (34)	
3–6	84 (87)	39 (66)	

Continued

Table 1 Continued

Variable	Infarct (n=97)	Non-infarct (n=59)	P value
90-day mRS, n (%)			**0.046***
0–2	34 (39)§	32 (55)¶	
3–6	53 (61)§	26 (45)¶	

*Statistical significance P<0.05.
†n=61.
‡n=45.
§n=87.
¶n=58.
ASPECTS, Alberta Stroke Program Early CT Score; CTP, CT perfusion; DSA, digital subtraction angiography; EVT, endovascular thrombectomy; LKW, last known well; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; TICI, Thrombolysis In Cerebral Infarction; tPA, tissue plasminogen activator.

more prevalent in the infarct group (60% vs 43%, P=0.037). The location of occlusion was similar between groups, and the most common was the M1 segment of the middle cerebral artery (infarct, 68% vs 71%, P=0.530). The median number of passes was two and did not differ between groups (P=0.742). EVT in the infarct group was performed with a stent retriever with a larger diameter (6 mm vs 4 mm, P=0.048) and longer length (37 mm vs 30 mm, P=0.046). There was no difference in the use of balloon guide catheters (infarct, 3% vs 2%, P=0.989) or the use of intraprocedural intra-arterial (P=0.423) or intravenous (P=0.115) thrombolytic and/or antiplatelet medications between groups. Details about the intraprocedural medications can be found in online supplemental table 1.

The median number of distal emboli (P=0.667) and the presence of more than one distal embolus (P=0.919) were not different. The median diameter of the vessels occluded by the distal emboli was not different between groups (infarct, 1.08 mm vs non-infarct, 1.04 mm, P=0.662). Importantly, the evidence of leptomeningeal collaterals on DSA was significantly higher in the non-infarct group (infarct, 43% vs 72%, P=0.001). Additional details on the occluded branch and the corresponding parenchymal location can be found in online supplemental table 2.

Predictors of tissue infarction and secondary outcomes

In the multivariable logistic regression analysis (table 2), after adjusting for confounders, the variables that remained associated with tissue infarction were hypertension (adjusted OR (aOR) 4.73, 95% CI 1.81 to 13.25, P=0.002), higher blood glucose (aOR 1.01, 95% CI 1.00 to 1.03, P=0.023), and general anesthesia (aOR 2.75, 95% CI 1.15 to 6.84, P=0.025). Of note, the

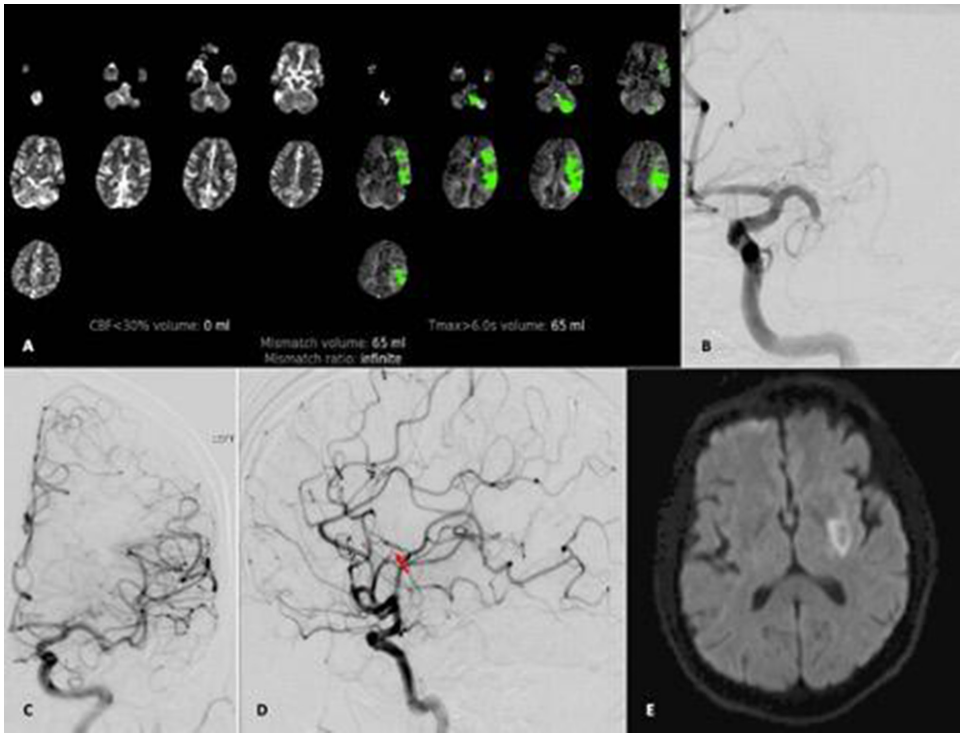

Figure 2 Illustrative case. (A) Baseline CT Rapid software (iSchemaView) output showing the area of penumbra without infarct core on the left hemisphere. (B) Digital subtraction angiography run, AP view of the left internal carotid artery showing a large vessel occlusion at the M1 segment of the middle cerebral artery. (C) Post-endovascular thrombectomy (three passes) run, AP view showing TICI 2b reperfusion. (D) Lateral view showing a persistent occlusion due to distal emboli. (E) 24 hour diffusion-weighted MRI showing acute infarction in the left putamen/globus pallidus. AP, anteroposterior; TICI, Thrombolysis In Cerebral Infarction.

Table 2 Multivariable analysis of the predictors of infarct at the territory at risk

	Univariable			Multivariable		
Variable, n (%)	OR	95% CI	P value	aOR	95% CI	P value
Age	1.01	0.99 to 1.03	0.543			
Use of antiplatelet drugs	1.07	0.55 to 2.11	0.848			
History of hypertension	4.25	2.07 to 8.99	**<0.001***	4.73	1.81 to 13.25	**0.002***
Blood glucose value	1.01	1.00 to 1.02	**0.026***	1.01	1.00 to 1.03	**0.023***
Use of general anesthesia	2.01	1.04 to 3.93	**0.038***	2.75	1.15 to 6.84	**0.025***
Stent retriever diameter	1.37	0.99 to 1.90	0.060	1.49	0.98 to 2.29	0.062
Number of passes	0.96	0.77 to 1.21	0.714			
Number of emboli	1.28	0.79 to 2.15	0.338			
Occluded vessel diameter, mm	1.31	0.46 to 3.82	0.608	2.99	0.76 to 12.81	0.126
Evidence of leptomeningeal collaterals	0.21	0.09 to 0.43	**<0.001***	0.13	0.05 to 0.33	**<0.001***

*Statistical significance P<0.05.
aOR, adjusted OR.

evidence of leptomeningeal collaterals persisted as a protective factor (aOR 0.13, 95% CI 0.05 to 0.33, P<0.001).

Post-EVT symptomatic intracranial hemorrhage (infarct, 10% vs 10%, P=0.978) and death (infarct, 23% vs 12%, P=0.084) were not different. However, mRS at discharge (mRS 0–2: infarct, 13% vs 34%, P=0.002) and at 90 days (mRS 0–2: infarct, 39% vs 55%, P=0.046) were different.

DISCUSSION

In this study of patients with anterior circulation LVO who had a TICI 2b reperfusion after EVT and distal emboli arising from the initial target occlusion, we evaluated the factors associated with tissue infarction in the territories at risk. Distal emboli resulted in infarction in more than half of these patients (62%), similar to what has been reported in the literature.[2 4] Patients who presented with a higher admission blood glucose, had a history of hypertension, and underwent EVT under general anesthesia were more likely to present at least one infarction secondary to distal emboli. However, the presence of leptomeningeal collaterals (on DSA) in the territory at risk was associated with a lower risk for infarction.

The increased risk of infarction with the use of general anesthesia may be related to lower blood pressure during general anesthesia than during conscious sedation, leading to less leptomeningeal collateral flow and an increased risk of infarction. Diastolic blood pressure may be an important factor for distal emboli washout or lysis, and there is a need for further characterization of the relationship between general anesthesia and brain perfusion.[2] The association between a history of hypertension and infarction may be explained by autoregulation and a right shift of the autoregulatory curve in patients with long-standing hypertension.[12] These patients may be more vulnerable to reduced cerebral blood flow at the lower range of normal blood pressure. Alternatively, these patients may have poor collaterals owing to hypertension-related injury to small arterioles.[13] In our study, we found that 70% of the distal emboli with some degree of leptomeningeal collateral supply to the territory at risk did not result in any stroke in that territory, compared with 45% of those without leptomeningeal collateral supply.

Higher glucose levels were associated with infarction from distal emboli. Hyperglycemia is a well-recognized factor that increases the risk of tissue infarction.[14] Higher glucose levels have previously been associated with a decreased chance of recanalization following pharmacologic thrombolysis and poorer functional outcomes following thrombectomy.[14–17] These patients may be less likely to achieve meaningful reperfusion of the embolus than those with normoglycemia. However, trials of better glucose control have failed to show a functional outcome benefit.[18]

The most relevant aspect to address is to define what should be considered an optimal reperfusion at an individual patient level. While TICI 2b is commonly accepted as optimal, previously published data suggest that the presence of distal emboli after EVT for the target LVO is associated with worse functional outcomes.[3 19] Our data suggest that this is likely driven by the subgroup that goes on to have an infarct, as we found that functional outcomes were worse in the infarct group. Garcia-Tonel *et al* evaluated the clinical and functional outcomes in 459 patients with LVO and found that a lower degree of recanalization (TICI 2b) and a higher number of passes were associated with worse clinical outcomes (mRS >2).[20] Additionally, in a different study the same group found that the need for additional passes to achieve a higher degree of recanalization led to worse outcomes due to potential clot fragmentation and embolization.[21] Furthermore, the growth of the infarcted tissue (and worse clinical outcomes) has been shown to be higher in patients where a TICI 2b reperfusion is achieved when compared with those with TICI 2c.[22] Considering this evidence, we note that the identification and revascularization of distal emboli (when possible) can certainly contribute to improved clinical outcomes.

There are many limitations of our study. Some of these emboli may have been present before the procedure. However, the etiology of emboli does not impact the choice to attempt revascularization. Another limitation is that the angiography area is two-dimensional while the MRI volume is three-dimensional. To assess whether distal emboli caused infarction, we visually assessed the distal emboli territory on DSA and looked at where we would expect an infarct on MRI. There is inherent subjectivity in this process. However, we purposefully underestimated the number of emboli that do not infarct by considering any infarct in the territory at risk as infarct presence, regardless of whether the infarct was smaller or larger than what one would expect from the perfusion defect on DSA. In addition, some of these infarctions may have already occurred before the occurrence of the distal embolus (pre- or intra-procedural). We did not have good data on intra-procedural or post-procedural blood pressure to be able to compare between general anesthesia and sedation patients.

This analysis was limited to patients with TICI 2b results. We excluded those with TICI 2c, as these distal emboli are unlikely to be treated owing to the very small caliber of the occluded vessels and the near complete reperfusion. We also excluded TICI 2a patients, as most of these are patients with persistent large (proximal M2) branch occlusions. Finally, our ability to define leptomeningeal collateralization may have been liberal and incomplete. We considered any retrograde filling of an occluded branch on DSA as leptomeningeal collateral flow. This is a liberal definition and includes a wide range of collateralization. It was also incomplete, as collateral flow from posterior cerebral arteries was not assessed for the majority of patients unless there was good filling of the posterior cerebral artery via the posterior communicating artery on the carotid artery injection.

CONCLUSIONS

Our study suggests that patients with the presence of leptomeningeal collaterals in the parenchymal territories with a distal embolus after EVT is likely to survive. Further intervention may not be necessary in these cases. However, in patients with a history of hypertension or higher admission glucose levels, the territories with distal emboli after EVT were more likely to infarct, and this may be a factor that weighs toward further attempts for revascularization. Our data reinforce the need for further characterization of the effect of general anesthesia on brain perfusion during EVT. Finally, we found worse functional outcomes in the cases with distal emboli after EVT. These findings may help guide decisions on whether to attempt revascularization of distal emboli after thrombectomy in these patient populations.

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Contributors EF, JV-S, NHF, and CBZ participated in data acquisition, editing figures and tables, and revision and final approval of the manuscript. YL participated in the data analysis of the study. CD and SO-G participated in the concept of the study, acquired the data, critically revised the manuscript, and approved the final work. CD is responsible for the overall content and is the guarantor of the entire work. He accepts full responsibility for the work and conduct of the study, had access to the data, and controlled the decision to publish.

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SUPPLEMENTAL MATERIAL

Table 1. Comparison of the use of intraprocedural medications stratified by the presence of infarct (n = 156).

Medications, n (%)	**Infarct (*n* = 97)**	**Non-infarct (*n* = 59)**	***p*-value**
Intra-arterial			.423
Tirofiban	2 (2)	2 (3)	
Tissue plasminogen activator	0 (0)	1 (2)	
Intravenous			.115
Tirofiban	3 (3)	0 (0)	

Table 2. Comparison of characteristics of the distal emboli between infarct and non-infarct groups (n = 222).

Variable	Infarct (*n* = 129)	Non-infarct (*n* = 93)	*p*-value
Vessel occluded, n (%)			.164
Anterior cerebral artery	3 (2)	4 (4)	
Posterior cerebral artery	3 (2)	1 (1)	
MCA M2 segment	19 (15)	8 (9)	
MCA distal M2 segment	4 (3)	2 (2)	
MCA M3 segment	42 (33)	21 (23)	
MCA distal M3 segment	22 (17)	28 (30)	
MCA M4 segment	36 (28)	29 (31)	
Brain parenchyma location, n (%)			.436
Medial	3 (2)	4 (4)	
Frontal	17 (13)	16 (17)	
Parietal	68 (53)	41 (44)	
Temporal	28 (22)	27 (29)	
Occipital	4 (3)	2 (2)	
Insula	9 (7)	3 (3)	

Abbreviations: MCA, middle cerebral artery.