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New Cerebral Microbleeds After Mechanical Thrombectomy for Large-Vessel Occlusion Strokes

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Abstract: The interval appearance of cerebral microbleeds (CMBs) after endovascular treatment has never been described. We investigated the frequency and predictors of new CMBs that developed shortly after mechanical thrombectomy for acute ischemic stroke, and its impact on clinical outcome.

We retrospectively analyzed patients with large-vessel occlusion strokes treated with Merci Retriever, Penumbra System, or stent-retriever devices. Serial T2∗-weighted gradient-recall echo (GRE) magnetic resonance imaging (MRI) before and 48 A total of 187 consecutive patients with serial GRE were enrolled in this study. CMBs were evident in 36 (19.3%) patients before mechanical thrombectomy. New CMBs occurred in 41 (21.9%) patients after mechanical thrombectomy. Of the 68 new CMBs, 45 appeared in the lobar location, 18 in the deep location and 5 in the infratentorial location. The presence of baseline CMBs was associated with new CMBs after mechanical thrombectomy (OR 5.38; 95% CI 2.13–13.59; $P < 0.001$), no matter whether the patients were treated primarily with mechanical thrombectomy or with intravenous thrombolysis followed by mechanical thrombectomy. Patients with new CMBs did not have increased rates of hemorrhagic transformation, in-hospital mortality, and modified Rankin Scale score 4 to 6 at discharge.

New CMBs are common after mechanical thrombectomy in one-fifth of patients with acute ischemic stroke. Baseline CMBs before mechanical thrombectomy predicts the development of new CMBs. New CMBs after mechanical thrombectomy do not influence clinical outcome.

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Abbreviations: CMBs = cerebral microbleeds, FLAIR = fluid-attenuated inversion recovery, GRE = gradient-recall echo, HT = hemorrhagic transformation, IV = intravenous, mRS = modified Rankin Scale, NIHSS = National Institutes of Health Stroke Scale, PH = parenchymal hematoma, SAH = subarachnoid hemorrhage, TICI = Thrombolysis in Cerebral Infarction, tPA = tissue plasminogen activator, WMH = white matter hyperintensity.

INTRODUCTION

Endovascular therapy with mechanical thrombectomy, particularly using stent retrievers, can provide clinical benefit in patients with large-vessel acute ischemic strokes.[1] White matter hyperintensities (WMHs) are 1 of imaging markers of cerebral small vessel disease and are associated with increased risks of hemorrhagic complications and mortality after reperfusion therapy.[2,3] Cerebral microbleeds (CMBs), another imaging marker of cerebral small vessel disease, are prevalent in patients with stroke and in asymptomatic elderly individuals.[4,5] CMBs may predict increased risks of intracerebral hemorrhage related to antithrombotic therapy.[6] The association of CMBs with hemorrhagic complications in acute stroke patients after intravenous (IV) tissue plasminogen activator (tPA) thrombolysis remains controversial.[7,8] It is not clear whether CMBs are associated with hemorrhagic complications and mortality after mechanical thrombectomy.[9]

New CMBs seen on follow-up MRI can develop slowly in general population of older people, hypertensive patients with intracerebral hemorrhage, patients with cerebral amyloid angiopathy.[10–12] Acute ischemic stroke may trigger the development

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The University of California has patent rights in endovascular retrieval devices for stroke. GRD: consultant—Stryker Neurovascular/Concentric Medical and Covidien. RJ: consultant—Covidien and Medina Medical. ST: consultant—Covidien and Stryker. JLS: consultant—Covidien, CoAxia, Stryker, BrainsGate, Genervon, Grifols, and Lundbeck. SS: consultant—Lundbeck, Paion, Centocor, Inc., Eli Lilly and Co., Concentric Medical, AstraZeneca Pharmaceuticals, Forest Laboratories, Novo Nordisk, Daiichi Asubio Pharmaceuticals, Inc., Omnicare, CoAxia, Nuvelo, Inc., Vernalis, PLC, Photothera, Kendle International, Inc., ev3 Neurovascular, Genervon Biopharmaceuticals, LLC, Cerevast Therapeutics, Covidien, and Stryker. YL: consultant—Blockade Medical. DSL: consultant—Stryker, Covidien and Zoll.
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of new CMBs.[13] In addition, new CMBs can develop rapidly after IV tPA or antiplatelet therapy in acute ischemic stroke.[14–16] New CMBs have also been reported to occur shortly after carotid artery stenting or cardiac valve surgery.[17,18]

Minor trauma from vascular tree traction during mechanical thrombectomy and sudden reperfusion can theoretically lead to new CMBs after endovascular therapy. Little is known of the development of new CMBs develop rapidly after mechanical thrombectomy for acute ischemic stroke, or its impact on clinical outcome. The purpose of the present study was to investigate the frequency and predictors of new CMBs that developed immediately after mechanical thrombectomy for acute stroke, and the association of the presence, burden, and distribution of new CMBs with clinical outcome.

SUBJECTS AND METHODS

Study Subjects

All consecutive acute ischemic stroke patients with large-vessel occlusion treated by mechanical thrombectomy at a single academic institution between August 2002 and October 2012 were identified from a prospectively maintained database. Patients treated with thrombectomy were either ineligible for IV tPA or refractory to thrombolysis after receiving IV tPA within 4.5 h of stroke onset.[3,19] The mechanical clot retrieval devices included Merci Retriever (Stryker neurovascular, Mountain View, CA), Penumbra System (Penumbra, Inc., Alameda, CA), stent retriever with Solitaire FR device (Covidien/eV3, Dublin, Ireland), or Trevo Retriever (Stryker neurovascular).

Magnetic resonance imaging (MRI) before thrombectomy was routinely performed in all patients without selection biases unless contraindicated. Patients also typically underwent follow-up magnetic resonance (MR) within 48 h after thrombectomy. The multimodal MRI protocol included diffusion-weighted imaging, perfusion-weighted imaging, T2∗ gradient-recall echo (GRE), and fluid-attenuated inversion recovery (FLAIR) sequences. We included patients who had serial T2∗ GRE before and 48 h after thrombectomy. The local institutional review board of University of California at Los Angeles approved the study. Informed consent was obtained from the patient or their representative.

MRI

MRI examinations were performed with a 1.5T Avanto or 3.0T Tim Trio system (Siemens, Erlangen, Germany). On the 1.5T scanner, T2∗ GRE sequences were 5 mm slice thickness with no gap (repetition time, 800 ms; echo time, 15 ms; field of view, 240 mm; 30° flip angle; matrix size, 256 × 144). On the 3.0T scanner, T2∗ GRE sequences were 5 mm slice thickness with slice spacing 6 mm (repetition time, 690 ms; echo time, 20 ms; field of view 240 mm; 30° flip angle; matrix size 320 × 210).[9]

CMBs were defined as punctate, homogeneous, round, hypointense lesions with diameters of up to 10 mm on T2∗ GRE. Mimic lesions were not considered to be CMBs, which included symmetrical hypointensities in the globi pallidi (likely calcification or iron deposition), flow voids from cortical vessels, partial volume artifact from bone, and cavernous malformations.[4,5] New CMBs were defined as CMBs that newly appeared on the follow-up GRE after thrombectomy. Newly appearing CMBs in the acutely infarcted areas were not designated as new CMBs.[13] The presence, burden, and distribution of baseline CMBs and new CMBs were reviewed on the pretreatment and posttreatment T2∗GRE, and recorded using the Microbleed Anatomic Rating Scale and consensus recommendations for neuroimaging standards.[20] Both baseline CMBs and new CMBs were categorized in lobar (frontal, parietal, temporal, occipital, and insular), deep (basal ganglia, thalamus, internal or external capsule, corpus callosum, deep, and periventricular white matter), and infratentorial regions (brainstem and cerebellum). The number of baseline CMBs and new CMBs was categorized as follows: no CMBs, 1 CMBs, 2 to 4 CMBs, and ≥5 CMBs. Two investigators who were blinded to the follow-up images and clinical data independently reviewed MR images. The intra-rater reliability for the presence of CMBs was acceptable (κ values = 0.85).

We used Fazekas scale to score the degree of WMH on baseline axial FLAIR sequences.[3,21] Presence of overall WMH was defined as either deep WMH (Fazekas score ≥2) or periventricular WMH (Fazekas score 3).

Clinical Assessment

We acquired data, including demographic characteristics, vascular risk factors, premorbid medications (antiplatelets, anticoagulants, antihypertensive drugs, and statins), laboratory findings, admission National Institutes of Health Stroke Scale (NIHSS) score, site of arterial occlusion, and time intervals.

Final revascularization status was recorded using Thrombolysis in Cerebral Infarction (TICI) score on the angiograms after endovascular treatment. Successful revascularization was defined as TICI 2b-3.[22] CT or GRE MR images at 24 (18–36) h postthrombectomy were reviewed to assess hemorrhagic transformation (HT) and subarachnoid hemorrhage (SAH). HT was classified into hemorrhagic infarct and parenchymal hematoma (PH) using the European Cooperative Acute Stroke Study definition. PH type 2 was defined as dense hematoma >30% of the infarcted area with substantial space-occupying effect.[23] Neurologic status was quantified by the modified Rankin Scale (mRS) score at discharge that ranges from 0 (no symptoms) to 5 (severe disability and bedridden) and 6 (death). Moderate clinical outcome at discharge was defined as mRS ≤3.

Statistical Analysis

We compared baseline and angiographic characteristics of patient with and without new CMBs after thrombectomy. Univariate analysis was performed using the 2-sample *t* test or Mann–Whitney *U* test for continuous variables, and the Fisher's exact and chi-squared tests for categorical variables. After adjustment for age, sex, systolic blood pressure, premorbid antithrombotic drugs use, lytic use, baseline WMH, and baseline CMBs before thrombectomy, which were thought to be potential factors associated with new CMBs, independent factors associated with new CMBs after thrombectomy were assessed in a multiple regression analysis. We further analyzed the association of the presence, burden, and distribution of new CMBs with hemorrhage, in-hospital mortality, and outcome at discharge. The calculation of odds ratio and 95% confidence intervals was assessed in all tests. Statistical analysis was performed using SPSS software, version 20.

RESULTS

During the study period, 263 consecutive patients were treated with mechanical thrombectomy. Of these, 76 patients were excluded for the following reasons: 52 patients did not undergo MR scans before thrombectomy or had

TABLE 1. Patient Baseline Characteristics

Characteristics	New CMBs (+) (n = 41)	New CMBs (−) (n = 146)	*P* Value
Age, y	67 (21)	66 (17)	0.89
Female sex	29 (70.7%)	84 (57.5%)	0.15
Baseline NIHSS, score	17 (7)	18 (7)	0.93
Cardioembolic stroke source	30 (73.2%)	88 (60.3%)	0.15
Systolic blood pressure, mm Hg	149 (30)	155 (30)	0.24
Diastolic blood pressure, mm Hg	78 (16)	83 (18)	0.08
Blood glucose, mg/dL	116 (80–275)	125 (70–417)	0.14
Medical history			
Hypertension	25 (61.0%)	100 (68.5%)	0.45
Diabetes mellitus	12 (29.3%)	29 (19.9%)	0.21
Dyslipidemia	15 (36.6%)	45 (30.8%)	0.57
Atrial fibrillation	19 (46.3%)	57 (39.0%)	0.47
Coronary artery disease	10 (24.4%)	27 (18.5%)	0.39
Previous stroke	8 (19.5%)	24 (16.4%)	0.64
Premorbid medications			
Aspirin	11 (26.8%)	40 (27.8%)	>0.99
Clopidogrel	2 (4.9%)	9 (6.2%)	>0.99
Warfarin	6 (14.6%)	24 (16.7%)	>0.99
Statins	15 (36.6%)	39 (27.1%)	0.25
Antihypertensive drugs	21 (51.2%)	74 (51.4%)	>0.99
Most proximal occlusion site			
Internal carotid artery	9 (22.0%)	47 (32.2%)	0.25
Middle cerebral artery	30 (73.2%)	87 (59.6%)	0.14
Vertebrobasilar artery	2 (4.9%)	12 (8.2%)	0.74
Intravenous tPA failure	14 (34.1%)	53 (36.3%)	0.86
Intra-arterial lytic use	1 (2.4%)	12 (8.2%)	0.30
Mechanical thrombectomy			
Merci retriever	34 (82.9%)	110 (75.3%)	0.40
Penumbra aspiration	4 (9.8%)	22 (15.1%)	0.46
Stent-retriever	3 (7.3%)	14 (9.6%)	>0.99
Intracranial angioplasty or stenting	4 (9.8%)	12 (8.2%)	0.76
Carotid artery stenting	1 (2.4%)	13 (8.9%)	0.31
Attempts to remove clot	2 (1–6)	3 (1–7)	0.54

Data are represented as mean (standard deviation), median (interquartile range) or number (percentage).
CMBs = cerebral microbleeds, NIHSS = National Institutes of Health Stroke Scale, tPA = tissue plasminogen activator.

noninterpretable MR images; 19 patients did not undergo MR scans after thrombectomy; 10 patients had no clinical outcome data because of inter-hospital transfer within 24 h after endovascular procedure. A total of 187 patients who had serial T2∗ GRE before thrombectomy and 48 h after thrombectomy were included for the final analysis. One hundred seventy-three patients (92.5%) had anterior circulation strokes. Mean age was 66.5 ± 17.6 years and 113 (60.4%) were women. Mean presentation NIHSS score was 17.5 ± 6.7 points. One hundred forty-four patients (77.0%) were treated primarily with Merci Retriever, 26 patients (13.9%) with Penumbra System, and 17 patients (9.1%) with stent retriever. Seventy-five patients (40.1%) were administered IV or intra-arterial (IA) tPA, including IV tPA in 67 patients (35.8%), IA tPA in 8 patients (4.3%), and IV combined with IA tPA in 5 patients (2.7%).

On initial GRE images before thrombectomy, 72 baseline CMBs were observed in 36 patients (19.3%). One hundred eighty patients (96.3%) had follow-up MR examinations within 24 h after thrombectomy. On postthrombectomy GRE images, new CMBs were identified in 41 patients (21.9%). Baseline CMBs were not observed on follow-up GRE in 2 patients. Baseline characteristics were similar between patients with new CMBs and those without CMBs (Table 1). A total of 68 new foci of microbleed were identified among patients with new CMBs. The median number of new CMBs was 1 (interquartile range 1–6). Twenty-two patients (11.8%) had 1 new CMB, 18 patients (9.6%) had 2 to 4 new CMBs, and only 1 patient (0.5%) had ≥5 new CMBs. Lobar new CMBs were present in 33 patients (17.6%), deep new CMBs in 15 patients (8.0%), infratentorial new CMBs in 4 patients (2.1%), and mixed new CMBs in 10 patients (5.3%). More than half of the new CMB lesions (n = 45) were located in lobar regions, whereas the other lesions were in either deep (n = 18) or infratentorial (n = 5) regions. New CMBs with ipsilateral vessel occlusion treated by thrombectomy were presented in 38 patients (92.7%). The number and locations of new CMBs are shown in Table 2.

Patients with new CMBs more often had the baseline CMBs before thrombectomy than those without new CMBs (41.5% vs 13.0%; $P < 0.001$). There were no significant differences in the baseline WMH and time intervals factors between patients with and without new CMBs (Table 3).

Factors independently associated with new CMBs after thrombectomy are shown in Table 4. In a multiple regression analysis after adjustment for age, systolic blood pressure,

TABLE 2. Distribution and Number of New Cerebral Microbleeds

	Number		
Location	**Ipsilateral Thrombectomy**	**Contralateral Thrombectomy**	**Total**
Lobar regions (n = 33)			
Frontal	22	4	26
Parietal	5	0	6*
Temporal	7	0	7
Occipital	4	1	5
Insula	1	0	1
Deep regions (n = 15)			
Basal ganglia	5	0	5
Thalamus	3	0	3
Internal capsule	2	0	2
External capsule	2	0	2
Corpus callosum	0	1	1
Deep periventricular white matter	2	3	5
Infratentorial regions (n = 4)			
Brainstem	1	0	1
Cerebellum	2	0	4*
Total	56	9	68*

* Three new cerebral microbleeds (CMBs) including 1 parietal and 2 cerebellum CMBs were found in 2 patients with basilar artery occlusions treated by thrombectomy.

premorbid antithrombotic drug use, lytic use, and baseline WMH, the presence of baseline CMBs before thrombectomy was associated with new CMBs after mechanical thrombectomy (OR 5.38; 95% CI 2.13–13.59; $P < 0.001$), no matter whether the patients were treated primarily with thrombectomy (OR 5.12; 95% CI 1.66–15.80; $P = 0.005$) or with IV tPA followed by thrombectomy (OR 7.75; 95% CI 1.33–45.06; $P = 0.02$).

The successful revascularization rates with TICI score 2b or 3 were similar between patients with new CMBs and those without new CMBs (41.5% vs 39.0%). Patients with and

TABLE 3. Univariate Comparison of Radiological Characteristics and Angiographic Outcome Between Patients With and Without New CMBs

Characteristics	**New CMBs (+) (n = 41)**	**New CMBs (−) (n = 146)**	***P* Value**
Baseline CMBs, %	17 (41.5%)	19 (13.0%)	<0.001
Baseline CMBs distribution			
Strictly lobar, %	6 (14.6%)	5 (3.4%)	0.02
Strictly deep, %	7 (17.1%)	5 (3.4%)	0.005
Strictly infratentorial, %	0 (0.0%)	2 (1.4%)	>0.99
Mixed, %	4 (9.8%)	7 (4.8%)	0.26
Baseline CMBs number, %			
1	11 (26.8%)	11 (7.5%)	0.002
2–4	5 (12.2%)	8 (5.5%)	0.16
≥5	1 (2.4%)	0 (0.0%)	0.22
Baseline WMH, %	21 (51.2%)	54 (37.2%)	0.15
Baseline WMH distribution			
Deep WMH	14 (34.1%)	37 (25.5%)	0.32
Periventricular WMH	21 (51.2%)	47 (32.4%)	0.04
Baseline CMBs coexisting with overall WMH, %	13 (31.7%)	8 (5.5%)	<0.001
Baseline CMBs coexisting with deep WMH, %	7 (17.1%)	6 (4.1%)	0.009
Time intervals, h			
Onset to prethrombectomy MR	4.2 (2.3)	4.6 (2.2)	0.38
Onset to groin puncture	6.4 (4.0)	6.0 (2.2)	0.57
Procedure duration	1.6 (0.5)	1.7 (0.7)	0.50
Procedure end to postthrombectomy MR	7.0 (5.4)	6.1 (4.1)	0.22
Onset to reperfusion	7.1 (2.3)	7.1 (2.3)	>0.99

CMBs = cerebral microbleeds, WMH = white matter hyperintensity.

TABLE 4. Multivariate Analysis of Predictors of New Cerebral Microbleeds After Thrombectomy

	Overall Thrombectomy (n = 187)		Thrombectomy and IV tPA (n = 67)		Thrombectomy Without IV tPA (n = 120)	
Variables	**Odds Ratio (95% CI)**	***P* Value**	**Odds Ratio (95% CI)**	***P* Value**	**Odds Ratio (95% CI)**	***P* Value**
Baseline CMBs	5.38 (2.13–13.59)	<0.001	7.75 (1.33–45.06)	0.02	5.12 (1.66–15.80)	0.005
Baseline WMH	2.14 (0.84–5.46)	0.11	1.44 (0.29–7.25)	0.66	2.63 (0.80–8.65)	0.11
Age	0.98 (0.95–1.01)	0.18	0.98 (0.94–1.04)	0.55	0.98 (0.95–1.01)	0.20
Sex	1.77 (0.77–4.03)	0.18	1.36 (0.36–5.09)	0.65	2.20 (0.74–6.55)	0.16
Systolic blood pressure	0.99 (0.98–1.00)	0.16	1.00 (0.97–1.02)	0.72	0.99 (0.97–1.01)	0.15
Premorbid antithrombotic drugs use	0.92 (0.41–2.05)	0.84	0.93 (0.23–3.67)	0.91	0.98 (0.36–2.70)	0.97
Lytic use	0.89 (0.40–1.97)	0.77	–	–	–	–

CI = confidence interval, CMBs = cerebral microbleeds, tPA = tissue plasminogen activator, WMH = white matter hyperintensity.

without new CMBs had similar rates of either any HT (34.1% [14 of 41] vs 47.3% [69 of 146]) or any PH (14.6% [6 of 41] vs 17.8% [26 of 146]). Subgroup analysis according to the distribution (lobar, deep, infratentorial, or mixed) and number (1, 2–4, or ≥5) of new CMBs showed no difference in the rates of HT or PH between patients with new CMBs and those without new CMBs. The associations of new CMBs with HT and PH are shown in Table 5. Patients with new CMBs had similar rates of either PH-2 (2.4% [1 of 41] vs 4.8% [7 of 146]) or SAH (17.1% [7 of 41] vs 21.2% [31 of 146]) when compared with those without new CMBs. The associations of new CMBs with PH-2 and SAH are shown in Table 6.

The rate of procedure-related vessel perforation was not significantly different in patients with new CMBs compared with those without new CMBs (4.9% vs 4.8%). The rates of other complications, including vessel dissection, vasospasm, device fracture, and groin hematoma were not significantly different.

The associations of new CMBs with clinical outcomes are shown in Table 7. The rate of in-hospital mortality was not significantly higher in patients with new CMBs than in those without new CMBs. Subgroup analysis according to the distribution (lobar, deep, infratentorial, or mixed) and number (1, 2–4, or ≥5) of new CMBs did not show any difference in the rates of in-hospital mortality between patients with new CMBs and those without new CMBs. There was no difference in the rates of moderate clinical outcome (mRS score 0–3) at discharge between patients with new CMBs and those without new CMBs.

DISCUSSION

Our large study with series GRE MRI showed that baseline CMBs are present in 19% of patients with acute ischemic stroke before mechanical thrombectomy. New CMBs could develop rapidly after mechanical thrombectomy in approximately 22% of patients. Baseline CMBs before thrombectomy suggested an almost 4-folds of increased risks of new CMBs after the procedure. New CMBs after thrombectomy were not associated with an increased rate of bleeding and poor outcome.

The rapid development of new CMBs after mechanical thrombectomy in our study was consistent with recent studies of IV tPA or antiplatelet therapy for acute ischemic stroke. New CMBs developed in 13% of acute ischemic stroke patients within 1 week of stroke onset.[13] Two studies reported that approximately 5% of patients with acute stroke had new CMBs after IV tPA therapy within 24 h after treatment.[14,15] New CMBs could also occur shortly in 8% of patients after carotid

TABLE 5. Association of New Cerebral Microbleeds With Hemorrhage After Thrombectomy

	Comparison of HT			Comparison of PH		
Characteristics	**No HT (n = 104)**	**HT (n = 83)**	***P* Value**	**No PH (n = 155)**	**PH (n = 32)**	***P* Value**
Any new CMBs, %	27 (26.0)	14 (16.9)	0.16	35 (22.6)	6 (18.8)	0.82
New CMBs distribution						
Lobar, %	20 (19.2)	13 (15.7)	0.57	27 (17.4)	6 (18.8)	0.80
Deep, %	13 (12.5)	2 (2.4)	0.01	14 (9.0)	1 (3.1)	0.47
Infratentorial, %	2 (1.9)	2 (2.4)	>0.99	2 (1.3)	2 (6.2)	0.14
Mixed, %	7 (6.7)	3 (3.6)	0.52	7 (4.5)	3 (9.4)	0.38
New CMBs number, %						
1	14 (13.5)	8 (9.6)	0.50	19 (12.3)	3 (9.4)	0.77
2–4	12 (11.5)	6 (7.2)	0.46	15 (9.7)	3 (9.4)	>0.99
≥5	1 (1.0)	0 (0.0)	>0.99	1 (0.6)	0 (0.0)	>0.99

Data are represented as number (percentage). CMBs = cerebral microbleeds, HT = hemorrhagic transformation, PH = parenchymal hematoma.

TABLE 6. Association of New Cerebral Microbleeds With Parenchymal Hematoma Type 2 and Subarachnoid Hemorrhage After Thrombectomy

	Comparison of PH Type 2			Comparison of SAH		
Characteristics	**No PH-2 (n = 179)**	**PH-2 (n = 8)**	***P* Value**	**No SAH (n = 149)**	**SAH (n = 38)**	***P* Value**
Any new CMBs, %	40 (22.3)	1 (12.5)	>0.99	34 (22.8)	7 (18.4)	0.66
New CMBs distribution						
Lobar, %	32 (17.9)	1 (12.5)	>0.99	26 (17.4)	7 (18.4)	>0.99
Deep, %	15 (8.4)	0 (0.0)	>0.99	15 (10.1)	0 (0.0)	0.04
Infratentorial, %	3 (1.7)	1 (12.5)	0.16	3 (2.0)	1 (2.6)	>0.99
Mixed, %	9 (5.0)	1 (12.5)	0.36	9 (6.0)	1 (2.6)	0.69
New CMBs number, %						
1	22 (12.3)	0 (0.0)	0.60	17 (11.4)	5 (13.2)	0.78
2–4	17 (9.5)	1 (12.5)	0.56	16 (10.7)	2 (5.3)	0.54
≥5	1 (0.6)	0 (0.0)	>0.99	1 (0.7)	0 (0.0)	>0.99

Data are represented as number (percentage). CMBs = cerebral microbleeds, PH = parenchymal hematoma, SAH = subarachnoid hemorrhage.

artery stenting.[18] The development of CMBs was attributable to blood–brain barrier (BBB) disruption and small arteries or arteriole rupture.[4,5] Sudden reperfusion and hemodynamic changes after mechanical thrombectomy could aggravate the BBB disruption. Some of the new CMBs were located in the insular regions and could be related to traction injury and rupture of small perforating arterioles during mechanical thrombectomy. The latter 2 mechanisms could explain the higher rate of new CMBs in IV tPA plus mechanical thrombectomy patients in this study than IV tPA alone patients in previous studies. The rate of new CMBs shortly after thrombectomy would take more than 5 years for a general population of elderly patients to accumulate.[10]

In our study, only baseline CMBs suggested increased risks of developing new CMBs after either thrombectomy or IV tPA followed by thrombectomy. The association of baseline CMBs with new CMBs was consistent with the results in recent studies of IV tPA.[14,15] Several studies found that other factors such as baseline WMH, vascular risk factors, systolic blood pressure as predictors of new CMBs.[24–30] Our data show periventricular WMH rather than deep WMH was associated with new CMBs after mechanical thrombectomy.

The finding that new CMBs were irrelevant to intracranial hemorrhage after mechanical thrombectomy in our study was supported by a recent IV tPA study. In that series of 121 acute stroke patients, new CMBs was associated with neither any HT nor symptomatic hemorrhage after IV tPA therapy.[15] In another series of 224 acute stroke patients, patients with new CMBs had an increased risk of symptomatic hemorrhage after IV tPA therapy.[14]

Our study had limitations. The data were collected using GRE sequence, which was typically acquired using 5 mm slices. Some of new CMBs could thus be present but missed on the pretreatment study due to partial volume averaging. Susceptibility-weighted imaging sequences on a 3.0T MR scanner would be superior to GRE sequences on sensitivity and reliability of CMBs detection.[31] In addition, most of the patients in this study were treated with old generation of mechanical thrombectomy devices, which had significantly more traction of the vascular tree and longer procedure time than the current stent retriever devices. In our cohort, the rate of new CMBs was not significantly differences in patients treated with stent retriever than in those treated with old generation of thrombectomy devices (17.6% [3 of 17] vs 22.4% [38 of 170];

TABLE 7. Association of New Cerebral Microbleeds With Outcomes After Thrombectomy

	Modified Rankin Scale 0–3			In-Hospital Mortality		
Characteristics	**No (n = 115)**	**Yes (n = 72)**	***P* Value**	**No (n = 160)**	**Yes (n = 27)**	***P* Value**
Any new CMBs, %	25 (21.7)	16 (22.2)	>0.99	40 (25.0)	1 (3.7)	0.01
New CMBs distribution						
Lobar, %	18 (15.7)	15 (20.8)	0.43	32 (20.0)	1 (3.7)	0.05
Deep, %	11 (9.6)	4 (5.6)	0.41	15 (9.4)	0 (0.0)	0.13
Infratentorial, %	4 (3.5)	0 (0.0)	0.30	3 (1.9)	1 (3.7)	0.47
Mixed, %	7 (6.1)	3 (4.2)	0.74	9 (5.6)	1 (3.7)	>0.99
New CMBs number, %						
1	15 (13.0)	7 (9.7)	0.64	22 (13.8)	0 (0.0)	0.05
2–4	9 (7.8)	9 (12.5)	0.32	17 (10.6)	1 (3.7)	0.48
≥5	1 (0.9)	0 (0.0)	>0.99	1 (0.6)	0 (0.0)	>0.99

Data are represented as number (percentage). CMBs = cerebral microbleeds.

$P = 0.77$). It would be interesting to see if gentler manipulation of the vascular tree and shorter procedure time could reduce the number of CMBs after procedure in a larger prospective study.

CONCLUSIONS

Our findings showed that new CMBs were common after mechanical thrombectomy in one-fifth of patients with acute stroke. Baseline CMBs before thrombectomy was associated with increased risks of new CMBs in patients treated with either thrombectomy or IV thrombolysis followed by thrombectomy. Patients with new CMBs after thrombectomy did not increase the risk of hemorrhage and poor outcome.

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