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ORIGINAL RESEARCH

NEUROINTERVENTION

Incidence, Risk Factors and Clinical Implications of Subarachnoid Hyperdensities on Flat-Panel Detector Computed Tomography following Mechanical Thrombectomy in Anterior Circulation Acute Ischemic Stroke Patients

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ABSTRACT

BACKGROUND AND PURPOSE: Flat-panel detector computed tomography (FDCT) immediately after mechanical thrombectomy (MT) can detect complications including early hemorrhagic transformation and subarachnoid hyperdensities (SH). The clinical significance of SH in patients undergoing MT remains unclear.

MATERIALS AND METHODS: We studied 223 patients who underwent MT for anterior circulation stroke, had FDCT performed immediately after the procedure, and had follow-up imaging within 24 hours. SH severity was categorized into 5 grades (SH 0: absent to SH IV: extensive). Baseline and procedural characteristics, as well as outcome measures, were analyzed using group comparisons and multivariable logistic regression analyses.

RESULTS: Overall, 100/223 (45%) of patients showed SH on immediate post-interventional FDCT. The factors associated with an increased SH risk were: medium vessel occlusion or distal vessel occlusion as compared to a large vessel occlusion, a more distal device position, a higher number of device passes, a larger volume of contrast applied, and worse final reperfusion eTICI. Occurrence of SH grade II–IV was independently associated with worse functional outcomes (aOR for mRS 3–6: 2.2, 95% CI 1.1–4.3), whereas patients with SH grade I had similar outcomes to patients without SH.

CONCLUSIONS: Our study identified risk factors for SH, most of which reflect increasingly challenging procedures or more peripheral recanalization attempts. The presence of SH grades II–IV was associated with poorer outcomes, suggesting the need for personalized strategies to reduce its incidence and severity or potentially improve recovery after SH.

ABBREVIATIONS: DVO = distal vessel occlusion; FDCT = flat-panel detector computed tomography; LVO = large vessel occlusion; MVO = medium vessel occlusion; MT = mechanical thrombectomy; SH = subarachnoid hyperdensities

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Disclosure of potential conflicts of interest should be included here.

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SUMMARY SECTION

PREVIOUS LITERATURE: Flat-panel detector computed tomography (FDCT) has gained popularity for its rapid, high-resolution imaging capabilities at disposal in the angiosuite. FDCT can reveal imaging findings occult on conventional DSA, the significance of which remains a matter of interest, especially in the setting of endovascular stroke treatment. SH is one such findings and may be due to true hemorrhage or extravascular contrast medium. Existing literature has addressed newly detected hyperdensities in various brain compartments on CT after endovascular treatment, focusing on their prognostic value and outcome or aiming at differentiating blood from contrast. However, studies focusing on newly detected SH on immediate post-procedure FDCT are still scarce.

KEY FINDINGS: Increased risk of SH on immediate FDCT is seen with MVOs/DVOs, multiple thrombectomy attempts, more distal device position, larger contrast volume used, lower reperfusion scores, and intravenous thrombolysis. Patients with SH grades II-IV had worse outcomes, while patients with SH grade I had similar outcomes compared to patients without SH.

KNOWLEDGE ADVANCEMENT: Our study identified risk factors for SH on FDCT after MT in patients with acute anterior circulation ischemic stroke, primarily related to the complexity of the intervention. SH grades II-IV were associated with worse outcomes, highlighting the potential need for more intensive post-interventional monitoring and individualized treatment strategies.

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INTRODUCTION

Mechanical thrombectomy (MT) has become the standard of care for patients with acute ischemic stroke due to large vessel occlusion.[1,2] Early follow-up imaging studies have identified several complications associated with MT, including arterial vasospasm, dissection, perforation and re-occlusion.[3] Other post-interventional imaging findings include (progressive) infarct demarcation, distal embolization in new territories, hemorrhagic transformation, and subarachnoid hyperdensities (SH).[3] In recent years, the use of flat-panel detector technology has attracted widespread interest within the neuroradiology community. Flat-panel detector computed tomography (FDCT) allows the rapid acquisition of high-resolution images, almost matching the quality of conventional multidetector CT, and is increasingly used during various neurointerventional procedures.[4-8] FDCT acquired during or immediately following MT may reveal imaging findings that are occult on conventional DSA. However, FDCT is at disposal directly in the angiosuite, eliminating the need for patient transfer to a CT suite. If incomplete reperfusion or peri-interventional complications are detected directly in the angiosuite, this may allow to perform additional interventions such as secondary mechanical thrombectomy, intra-arterial lytics, or timely rescue maneuvers, and may potentially also be important for immediate post-interventional patient care (e.g. blood-pressure management or antithrombotic regimen). SH is one such finding and may be related to hemorrhage or contrast staining.[9] However, the prognostic value of SH in patients undergoing MT remains unclear.

There is a growing body of literature dealing with newly detected hyperdensities in different brain compartments seen on CT after endovascular treatment.[9-28] These mainly focus on their prognostic value and outcome; and/or aim to differentiate blood from contrast staining.[9-28] However, studies concentrating on newly detected SH on FDCT performed immediately after MT for acute stroke are still scarce.[29] Hence, our goal was to investigate the incidence of SH on immediate post-procedural FDCT, identify risk factors for SH, and evaluate the impact of SH on short-term clinical outcome.

MATERIALS AND METHODS

Patient population

All consecutive patients with acute ischemic stroke, who were treated with MT between July 2020 and December 2022 at our tertiary-level center, were retrospectively identified from the stroke database. For inclusion in the present analysis, patients had to meet the following criteria: 1.) anterior circulation acute ischemic stroke, 2.) FDCT performed immediately following the intervention and 3.) follow-up imaging (CT or MRI) within 24 hours. Initially, the decision to obtain post-interventional FDCT was at the discretion of the neurointerventionalist. FDCT tended to be performed more frequently in cases involving more complicated procedures characterized by multiple maneuvers, distal thrombectomies, tandem occlusions, the need for antiplatelet therapy during emergency stenting, intracranial stenosis, peri-interventional dissection in the cervical vessel, the potential administration of adjunctive intra-arterial lytics, and the exclusion of bleeding or other potential complications. However, after the initial phase, all institutional neurointerventionalists systematically obtained FDCT after every acute stroke procedure.[30] This study adhered to the principles outlined in the Declaration of Helsinki and was approved by the local ethics committee (reference ID 231/14, 2019-00547, 2023-00892).

Clinical Data

The clinical data were retrospectively extracted from the prospective stoke database and the electronic medical charts. These included age, sex, , stroke risk factors (hypertension, diabetes mellitus, hyperlipidemia, coronary heart disease, current smoking, atrial fibrillation), medications at baseline (antihypertensives, anticoagulants, antiplatelets, or lipid-lowering medications), baseline NIHSS score, NIHSS at 24h, pre-stroke mRS, mRS at 90 days and administration of intravenous thrombolytics. Distal intracranial ICA and M1 were defined as large vessel occlusion (LVO), M2, A1, and A2 as medium vessel occlusion (MVO), and M3 and A3 as distal vessel occlusion (DVO).[31] MVO and DVO were treated within ongoing trials or after an interdisciplinary discussion based on the severity of focal neurological deficit, vessel occlusion, intravenous thrombolysis eligibility, and technical considerations such as vessel tortuosity and diameter.

All procedures were performed under general anesthesia using a stent-retriever, a direct aspiration catheter, or a combination of the two. Procedural characteristics of the MT, such as the volume of contrast used during the procedure (mL), number of maneuvers/passes, and most distal catheter position (M1, M2, M3, M4, A1, A2, or A3) were registered. Active extravasation seen on DSA was also documented. Reperfusion outcomes were assessed using the eTICI scale, which ranges from 0, indicating no reperfusion, to 3, indicating complete reperfusion (100%) of the target downstream territory. Intermediate grades, 2a, 2b50, 2b67 and 2c, reflect reperfusion levels within the target downstream territory of 1–49%, 50–66%, 67–89%, and 90–99%, respectively.[32]

Imaging analysis

Baseline imaging (CT or MRI), post-procedural FDCT after MT, and early follow-up imaging (CT or MRI) within 24 hours after the procedure were analyzed by a board-certified radiologist (BLS).

Baseline and early follow-up CT included non-contrast CT, CT angiography of the intra- and extracranial arteries and mostly CT perfusion. Baseline and early follow-up MRI studies were performed at a magnetic field strength of 1.5 T or 3T. Stroke protocols mostly included axial DWI and matching ADC maps, axial FLAIR, 3D TOF-MRA of the intracranial arteries, axial SWI, 3D contrast-enhanced T1WI, 3D contrast-enhanced MRA of intra- and extracranial arteries, and MR perfusion.

The Sine Spin FDCT (ARTIS icono biplane) represents the latest generation of cone beam CT. It employs a dual oblique path for image acquisition aimed at minimizing artifacts and optimizing soft tissue brain imaging.[33] FDCT was acquired according to the "7sDCT Sine Spin protocol", details of which have been described previously.[33]

The baseline imaging (CT or MRI) studies were reviewed for the intracranial occlusion site (distal intracranial ICA, M1, M2, M3, A1,

A2, or A3). FDCT studies were evaluated for the presence of SH (yes/no). SH were graded (I–IV) as follows: grade I – hyperdensities in 1 or 2 adjacent sulci; grade II – hyperdensities in 3 or more adjacent sulci, but confined to a single lobe; grade III – diffuse sulcal hyperdensities affecting ≥2 lobes; and grade IV – diffuse sulcal hyperdensities affecting ≥2 lobes and intraventricular extension or extension into basal cisterns (Figure 1). Grading was independently performed by 2 board-certified radiologists (TD and BLS) and disagreements were settled by consensus discussion. If SH was evident on FDCT, the course was reviewed on follow-up imaging (i.e., complete resolution, similar expansion, marked reduction, or clear progression).

ASPECTS was assessed at baseline imaging. If patients underwent CT for baseline imaging, ASPECTS was calculated automatically using CINA-ASPECTS Version 1.4.2.0 (Acivenna.AI, La Ciotat, France), a cloud-based CE-marked deep learning algorithm integrated into an AI orchestration suite (Calantic, v 1.2.0, Bayer, Berlin, Germany). When automated scoring failed, or when patients underwent MRI, ASPECTS was scored by a board-certified radiologist. ASPECTS is a 10-point score used to evaluate early ischemic changes in the hypoperfused area of the middle cerebral artery. A score of 10 indicates the absence of such changes, and one point is subtracted for each standardized brain region involved. For the MRI-based ASPECTS assessment, diffusion-weighted imaging was used and a region had to have diffusion abnormality in 20% or more of its volume to be considered positive for early ischemic changes.[34]

Readers were blinded to outcome, but not to patients' medical history.

Statistical analysis

Descriptive statistics are presented as frequencies and percentages for categorical variables and median with interquartile range (IQR) for continuous variables. Continuous variables were evaluated using Student's t-test or the Wilcoxon-Mann-Whitney U-test, whereas categorical variables were evaluated using Fisher's exact test or the chi-squared test. For more than 2 groups, comparisons for continuous variables were made using either ANOVA or the Kruskal-Wallis test. Inter-rater agreement for different SH categories (0 to IV) is reported as Cohen's Kappa. Multivariable logistic regression was performed to elucidate the influence of selected factors (age, sex, NIHSS on admission, intravenous thrombolysis, number of maneuvers, pre-stroke medication [anticoagulant and antiplatelet], site of occlusion and most distal site of recanalization) on which group of SH was seen (SH 0 vs SH type I–IV).

The association of different SH categories (0 to IV) on several outcomes such as the mRS score at 3 months, dichotomized mRS (0–2 vs 3–6), NIHSS at 24 hours, early neurological deterioration (defined as a change in NIHSS of ≥4 between admission and 24-hour follow-up) and mortality at 3 months (equivalent to mRS 6) was modeled. Ordinal logistic regression was used for the mRS shift analysis. NIHSS at 24 hours was assessed using quantile regression at the 50% quantile (corresponding to the median), whereas binary outcomes were assessed using logistic regression.

Analyses, using inverse probability weighting, of the association of different SH categories on clinical outcomes were adjusted for the following covariates: age, sex, admission NIHSS, occlusion site, baseline ASPECTS, intravenous thrombolysis, pre-stroke mRS, parenchymal hyperdensities seen on FDCT, and final eTICI. Those factors were based on the most robust predictors of functional outcome after stroke.[35] Due to the limited sample size of SH IV (11 cases), the model correction was found to be unstable. Therefore, only a reduced set of variables (age, sex, admission NIHSS, baseline ASPECTS, intravenous thrombolysis, and parenchymal hyperdensities on FDCT) could be included in the analysis for the comparison between SH 0 and SH IV. Results are presented as (adjusted) ORs with 95% CIs for the mRS analyses and logistic regression, and as regression coefficients with 95% CIs for the NIHSS variables. Adjusted regressions were performed with both complete cases and imputation of missing data. Patients who had died at 3 months were assigned a mRS score of 6 and a NIHSS score of 42. The reported P-values were not adjusted for multiple comparisons, which should be taken into account when interpreting the results.

All statistical analyses were performed with R 4.3.1[36] and/or Stata 17.0

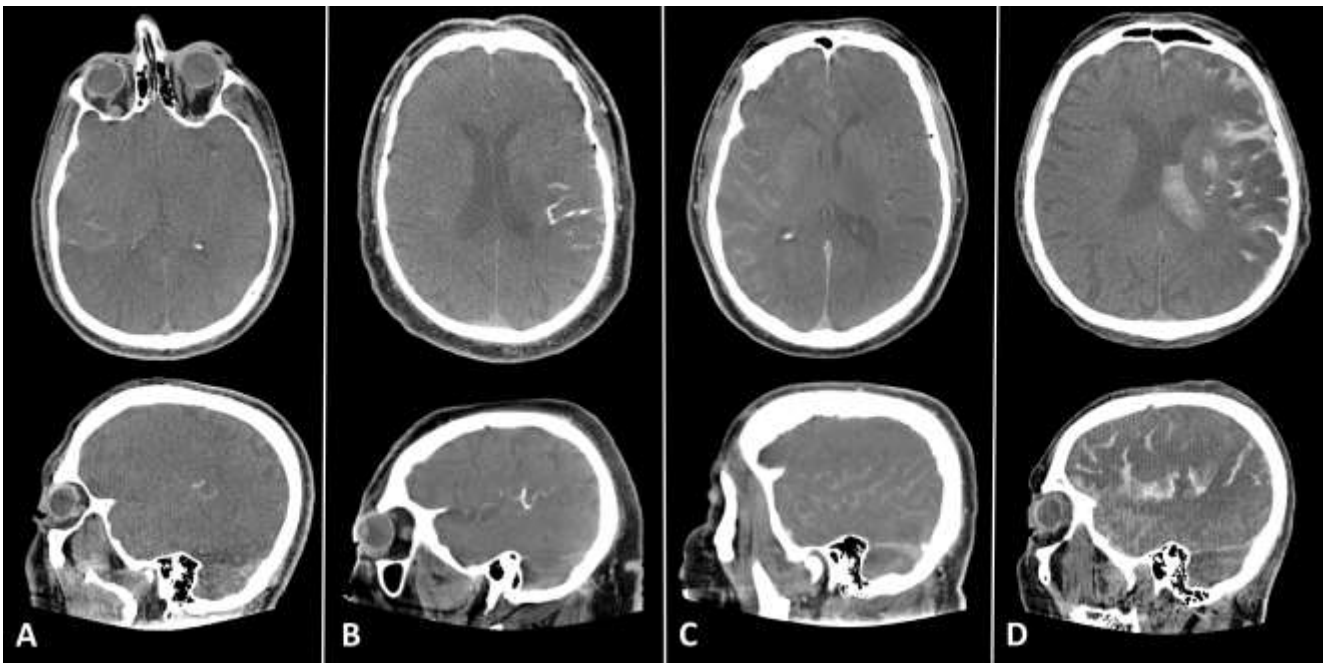


FIG 1. Examples of subarachnoid hyperdensities (SH) grade I to IV. (A) SH grade I with hyperdensities in 2 neighboring sulci. (B) SH

grade II with hyperdensities in more than 2 neighboring sulci, but confinement to one lobar area. (C) SH grade III with diffuse sulcal hyperdensities affecting >2 lobes. (D) SH grade IV with diffuse hyperdensities affecting >2 lobes and intraventricular extension.

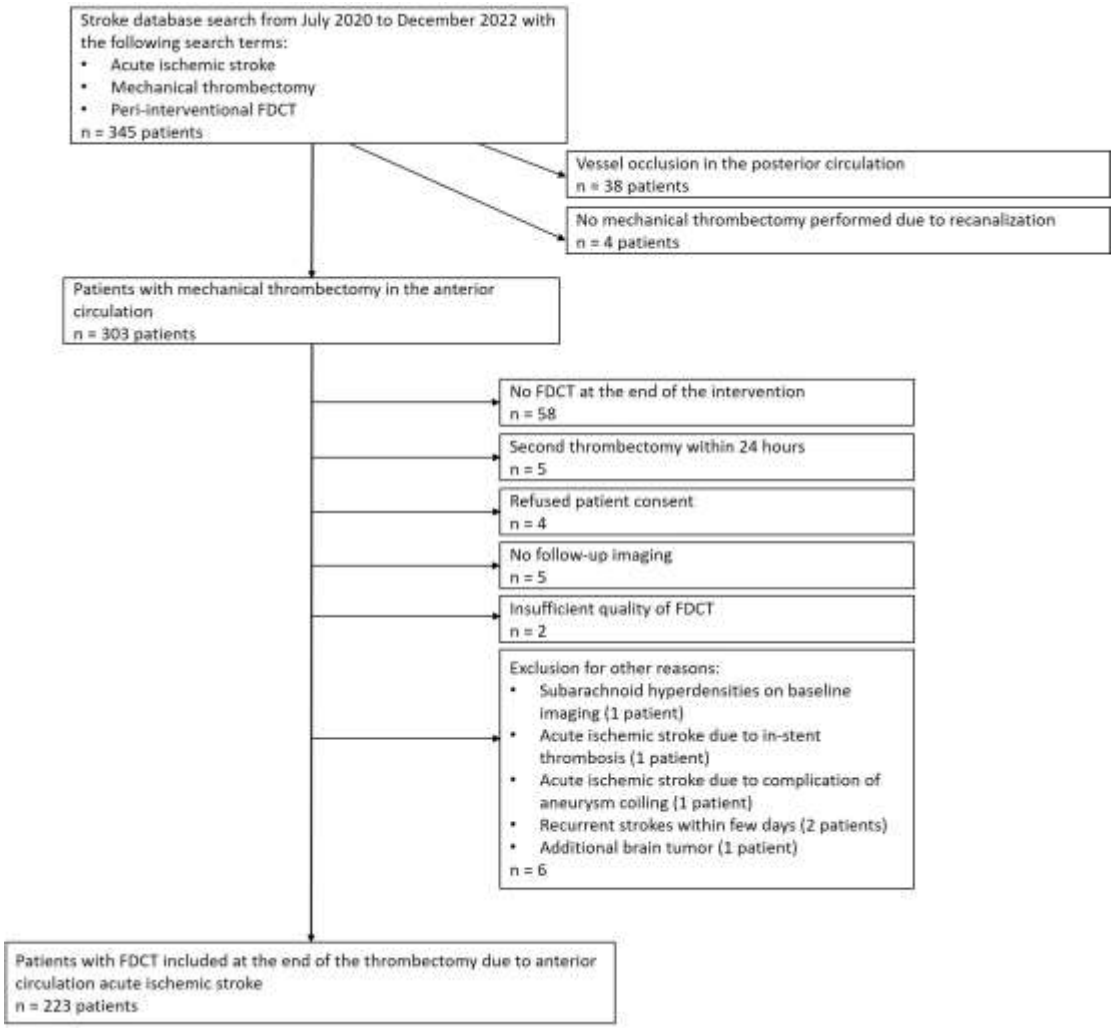


FIG 2. Flowchart depicting patient selection process

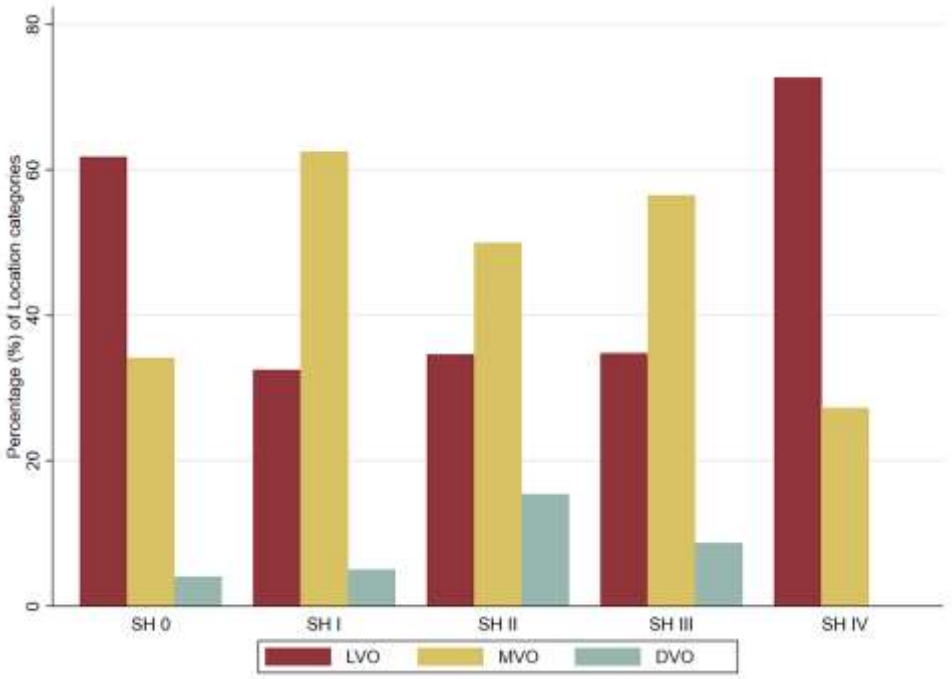
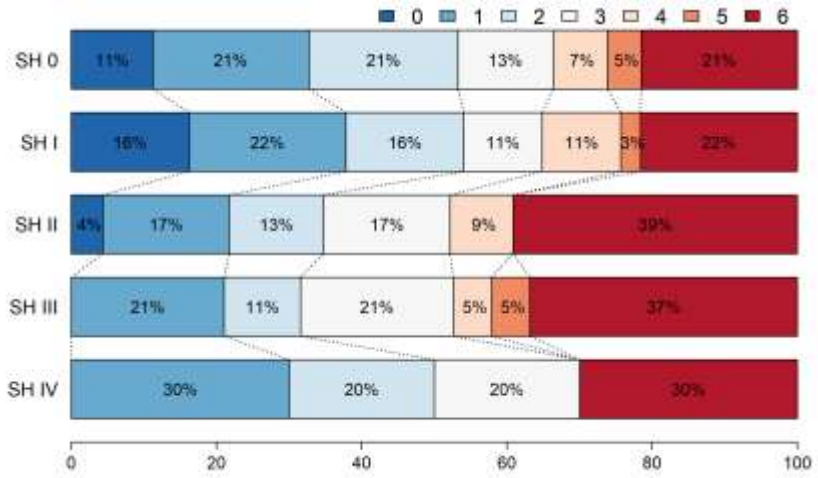


FIG 3. Baseline intracranial occlusion site (large vessel occlusion [LVO], medium vessel occlusion [MVO] and distal vessel occlusion

[DVO]) stratified by subarachnoid hyperdensities (SH).

A



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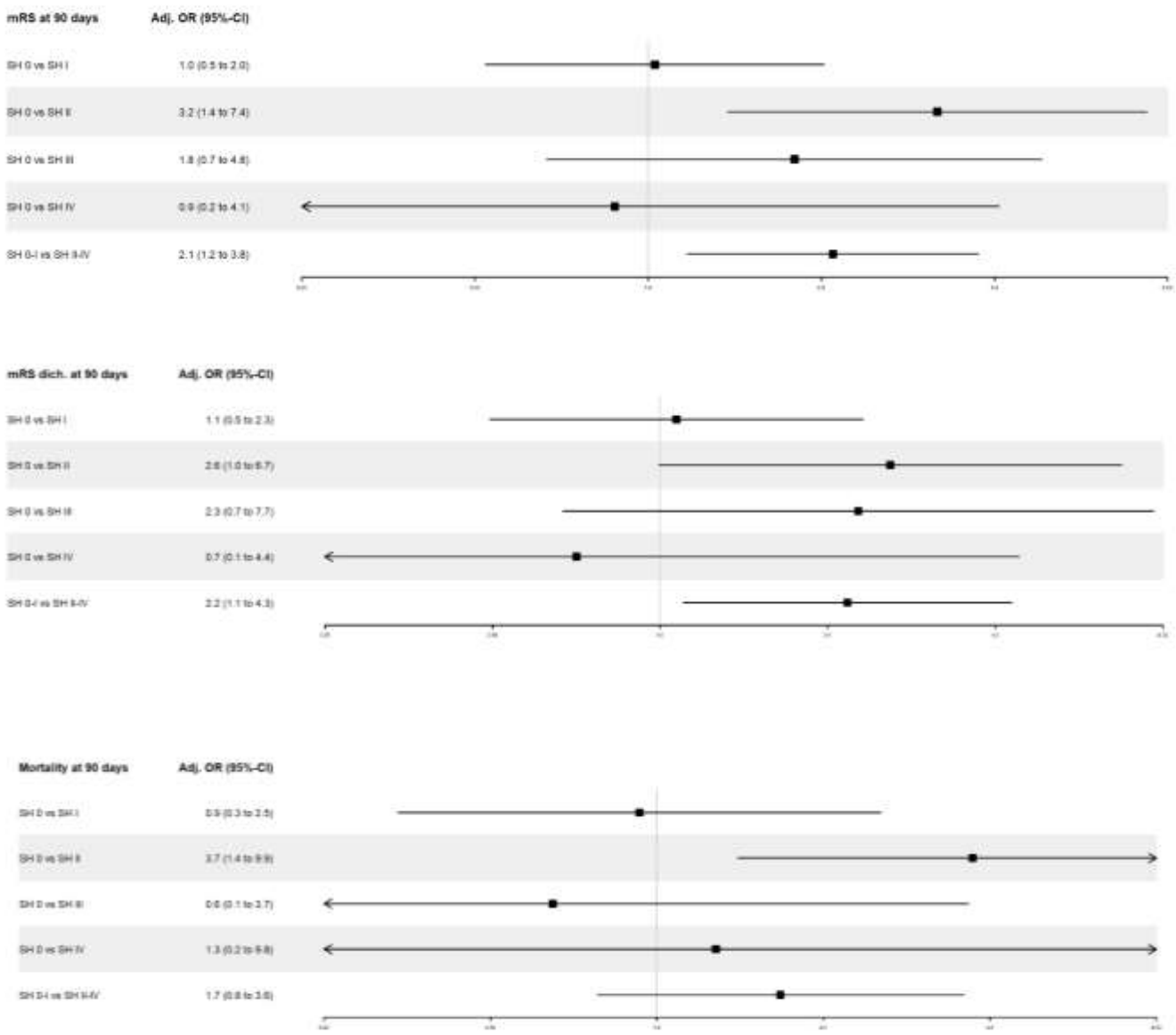

FIG 4. (A) Distribution of modified Rankin scale scores (mRS) at 90 days for patients without subarachnoid hyperdensities (SH), and patients with SH I–IV on flat-panel detector computed tomography. (B) The association of different grades of subarachnoid

hyperdensities (SH 0 to IV) with mRS at 3 months, mRS dichotomized (0–2 vs 3–6), and mortality (equivalent to mRS 6). Analyses were carried out using multivariable ordinal/logistic regression adjusting for prespecified confounders (see methods).

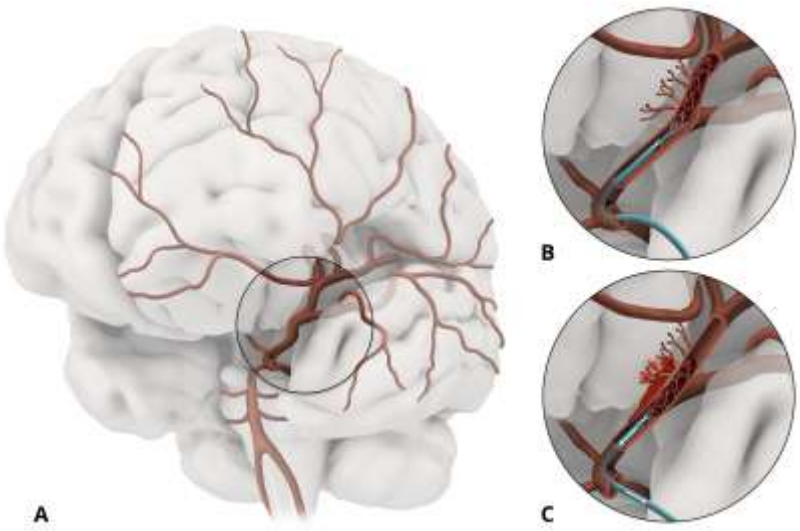

FIG 5. Possible mechanism leading to subarachnoid hyperdensities.
(A) Ventrolateral view of the brain and circle of Willis. (B) Close-up showing a proximal M2 occlusion with an inserted stent-retriever. (C) Close-up during the retrieval of the stent-retriever. During navigation, smaller vessels (M2 and beyond) tend to straighten more than proximal vessels. The perforators are exposed to excessive forces during thrombectomy due to stretching and may be sheared off leading to subtle extravasation, which is occult on standard DSA but may be detected on FDCT as subarachnoid hyperdensities, in this case, a subarachnoid hemorrhage.

RESULTS

Cohort

Between July 2020 and December 2022, 223 patients (113/223 (50.7%) women; median age 75.5 years, IQR 63.3, 83.1) met the inclusion criteria (Figure 2). This represents approximately 1/3 of all patients undergoing anterior circulation MT at our tertiary center during this period. Throughout the study period, individuals with FDCT tended to have a lower pre-stroke mRS, a higher prevalence of MVOs or DVOs, lower ASPECTS on baseline imaging, an increased frequency of symptom onset either unknown or observed on wake-up, and a higher 24-hour NIHSS in contrast to patients for whom no FDCT was acquired (Supplementary Table 1).

Presence and subtypes of SH

Post-procedural FDCT revealed SH (grade I–IV) in 100 cases (45%). Two FDCTs were of insufficient quality to identify and classify SH due to extensive beam hardening artifacts and aliasing artifacts, respectively (Figure 2). Forty patients had SH grade I, 26 patients grade II, 23 patients grade III, and 11 patients grade IV. The first-line technique at our tertiary center is a combined stent retriever and distal aspiration thrombectomy. Seven patients (7/223, 3%) were treated with aspiration catheter alone. Two patients (2/7, 29%) had SH (both grade I). Inter-rater agreement was very good (Cohen's kappa 0.94, 95% CI 0.86 to 1.00).The evolution of SH on follow-up imaging differed between groups (P = 0.004; Supplementary Table 2); with more severe SH grades (II-IV) less often showing complete resolution within 24 hours.

Factors associated with occurrence of SH

Most baseline factors were similar between patients with and without SH, as shown in Table 1. Baseline characteristics stratified by all SH groups are shown in Supplementary Table 3. There was a difference in the baseline occlusion site between the groups with and without SH, with overall MVO/DVO occurring more frequently (62% vs 38%, P<0.001, Table 1 and Figure 3) in patients with SH I–IV.

Analysis of procedural characteristics found that patients with SH grade I–IV had a higher number of thrombectomy passes (2 [1, 4] vs 1 [1, 2]; P < 0.001), more often had medium (68% vs 59%) or distal vessels (28% vs 17%) (P < 0.001) as the most distal microcatheter site, were less likely to have higher eTICI scores (P = 0.014), and received more contrast during the procedure (133 ml [100, 190] vs 110 ml [90, 160]; P = 0.035) than patients without SH (Table 2). The incidence of active extravasation seen on DSA increased at higher SH grades (SH I, 0/40 [0%]; SH II, 1/26 [4%]; SH III, 4/23 [17%]; SH IV, 8/11 [73%], P < 0.001; Supplementary Table 3). There was also a trend toward a higher rate of intravenous thrombolysis in patients with SH (52.0% vs 39.8%; P = 0.07, Table 1), which reached significance when all SH groups were stratified separately (P = 0.019, Supplementary Table 3).

The findings of the multiple logistic regression analysis indicated elevated odds of presenting with SH grades I–IV in patients with more thrombectomy passes (OR per pass 1.5, 95% CI 1.2–1.9) and after receiving intravenous thrombolysis (OR 2.0, 95% CI 1.1–3.8). When examining the most distal device site, location in medium (OR 3.8, 95% CI 1.2–12.3) or distal vessels (OR 4.5, 95% CI 1.2–16.9)

was associated with higher odds of presenting with SH. A similar tendency was observed for patients with a baseline occlusion site classified as MVO or DVO (OR 1.8, 95% CI 0.9–3.5).

Functional outcome

In the unadjusted analyses, we sought to identify potential associations of SH with several outcomes. When comparing patients without SH separately with every SH group individually, the presence of the worst grade (IV) was associated with higher odds (3.9 95% CI 1.1–13.9) of experiencing early neurological deterioration. In the next step, grouping individuals with similar point estimates and hence comparing SH II–IV to SH 0–I groups was associated with worse functional outcomes (ordinal shift; OR 1.9, 95% CI 1.1–3.4; dichotomized mRS 3–6, OR 2.0, 95% CI 1.0–3.8) and higher mortality (OR 2.1, 95% CI 1.1–4.2), in the SH II–IV group.

In the adjusted analyses, we observed partially a tendency towards increasing point estimates, indicating higher odds of adverse outcomes in the SH II, III, and IV groups. The SH II group showed increased odds of a higher mRS at 90 days (aOR 2.7, 95% CI 1.2–6.0) and an increased risk of 90-day mortality (aOR 3.0, 95% CI 1.2–7.6) compared to the SH 0 group. In a next step, combining groups with similar point estimates and hence comparing SH II–IV to SH 0–I was associated with worse functional outcomes (ordinal shift aOR 2.0, 95% CI 1.2–3.6; dichotomized mRS 3-6, aOR 2.1, 95% CI 1.1–4.0) in the SH II–IV group (Figure 4).

The results of the unadjusted, adjusted and grouped comparisons are shown in Figure 4, Supplementary Table 4 and Supplementary Figure 1, respectively.

DISCUSSION

Our study found that almost half of the patients undergoing MT due to anterior vessel occlusion showed SH on immediate post-interventional FDCT. An increased risk of SH is associated with MVOs/DVOs, multiple thrombectomy attempts, a more distal device position, a larger volume of contrast applied, lower reperfusion scores, and after receiving intravenous thrombolysis. Patients with SH grade II–IV had overall worse outcomes, whereas patients with SH grade I had a comparable outcome to patients without SH on FDCT.

There is a growing body of literature addressing the topic of newly detected hyperdensities after MT. However, the studies vary widely regarding timing of post-procedural imaging, the imaging technique (FDCT, CT, dual-energy CT, or MRI), and the precise location of hyperdensities (parenchymal, subarachnoid, or ventricular). In addition, the terminology is inconsistent and poorly defined, with terms ranging from contrast extravasation, hyperdensity, contrast enhancement, metallic hyperdensity, and contrast staining, to hemorrhage.[9-29]

Previous studies have mainly focused on parenchymal hyperdensities only[14,22,25] or included hyperdensities in all compartments[11,16,20] and reported frequencies ranging from 28–84%.[11,14,16,20,22,25] Compared to the study by Parrilla et al. from 2012 we found a higher incidence of SH in our cohort (12.5% versus 45%).[20] This may be explained by evolving indications in MT targeting more distal occlusion sites, as well as improved imaging quality with new-generation FDCT technology. A more recent study reported a prevalence of SH on FDCT of 37.1%, which is similar to what was observed in our cohort.[29]

Performed either peri-interventionally or immediately post-interventionally, FDCT serves a similar purpose to post-procedural multidetector CT. However, FDCT has the advantage of being performed directly in the angiosuite, eliminating the need to transfer the patient to a CT suite. While multidetector CT and FDCT may provide comparable diagnostic performance,[33] identification of incomplete reperfusion or peri-interventional complications on FDCT directly in the angiosuite may prompt consideration of additional treatments such as secondary mechanical thrombectomy, intra-arterial lytics, or timely rescue maneuvers.[37-39] Immediate identification of cerebral intraparenchymal hyperdensities after thrombectomy provides valuable prognostic information about the eventual infarct volume and may aid in early prediction of patient clinical outcome and acute management after thrombectomy.[40,41] Accurate and reliable differentiation between hemorrhage and contrast enhancement is essential to guide subsequent antithrombotic therapy. However, despite its advantages, FDCT obtained peri- or immediately post-interventionally faces challenges in reliably distinguishing between blood and extravascular contrast medium. Because contrast usually resolves within 24 hours, accurate differentiation is typically possible at 24-hour follow-up imaging. Although it is not yet a standardized method, dual-energy CT has the ability to accurately differentiate between blood and contrast.[29] It offers a promising avenue for accurate blood/contrast differentiation. Emerging technologies such as photon-counting computed tomography also show promise for improving blood/contrast differentiation in the future.[42]

In line with the study by Zidan et al.,[29] we also found that SHs were more prevalent in MVO/DVOs than LVOs. In addition, distal device placement during MT increased the likelihood of presenting with SH grade I–IV. The advent of dedicated devices designed for distal MT has expanded the range of treatment options. The anatomical characteristics of smaller vessels, however, may render the procedure more challenging requiring navigation of microcatheters and guidewires through numerous bifurcations. Also, vessel tortuosity may lead to displacement, straightening and stretching of smaller vessels.[15,43] These complexities of device handling increase the likelihood of vessel damage, rupture of adjacent arterioles and venules, disruption of the BBB, and subsequent SH (Figure 5).[15,29,43]

A greater number of passes was found to be associated with a higher risk of presenting with SH grade I–IV. This observation is consistent with previous research.[29] The amount of contrast administered can serve as a surrogate for the duration and complexity of the procedure and the number of device passes; it is not surprising that administration of a higher volume of contrast was also linked to an increased risk of SH grade I–IV.[16] The increase in incidence of SH with more thrombectomy attempts is likely due to a cumulative effect of the inherent risk of vascular injury with each revascularization attempt. Initially, there may be a minimal microperforation of the vessel wall or endothelial damage that compromises the BBB. However, with repeated passes and/or subsequent reperfusion, complications may be exacerbated.[15,29]

In addition, our analysis showed that lower eTICI scores were related to an increased likelihood of developing SH. This association is likely indicative of more complex procedures with a greater number of passes. A potential mechanism other than the number of maneuvers may be at play, such that residual clots associated with lower eTICI could also cause more damage to the vessel wall when interacting with stent retrievers or aspiration catheters.

Contrast staining/enhancement occurs when the BBB becomes more permeable due to endothelial cell damage, resulting in the leakage

of the contrast agent from the blood vessels into the extracellular spaces.[16] Hemorrhagic lesions, on the other hand, are thought to be caused by an additional degradation of the basal lamina. This disruption results in the extravasation of cellular blood elements from the microvessels.[45] Various studies have consistently reported that blood and contrast agents can be differentiated according to the time they take to disappear.[9,23] Hyperdensities that disappear within 24 hours are considered to be contrast agent, as extracellular contrast is mostly cleared within 24–48 hours.[9,23] Blood, on the other hand, degrades and remains visible on imaging for several days to weeks.[9,23] In our cohort, evolution of the SH on follow-up imaging differed significantly between groups, with the predominant trend indicating that milder grades of SH were more likely to resolve on follow-up imaging within 24 hours. We hypothesize that complete resolution of SH on follow-up imaging within this 24-hour-window may be indicative of the presence of contrast media in the subarachnoid space, whereas comparable or visibly increased SH may correspond to the presence of additional blood components. Furthermore, cases with a significant reduction of SH could potentially involve minimal blood components, or the follow-up imaging performed within 24 hours may have captured the clearing process of the contrast agent while it was still in progress. It is noteworthy that only very few patients with SH (8/100, 8%) demonstrated a clear progression of SH on the follow-up imaging. A significant proportion (37/100, 37%) of all SH had completely resolved on follow-up imaging within 24 hours, a similar percentage (39.4%) to that reported in previous research.[29]

In the analysis of outcome, we observed that those of the SH 0 and SH I groups were comparable, whereas patients with SH II–IV had generally worse outcomes. A small amount of hyperdensities (SH I) appears to have a minor impact on outcome. The course of smaller intracranial vessels (M2 and beyond) tends to straighten more than proximal vessels during microcatheter/wire navigation. The displacement becomes even more pronounced during retrieval. This leads to a straightening of the loops of the peripheral cortical branches, resulting in excessive forces on the perforators, which may be sheared off during MT due to stretching (Figure 5). During this procedure, only the endothelial cells of the vessels are potentially affected, resulting in leakage of the contrast agent but not extravasation of cellular blood elements from the blood vessels. However, as the procedure becomes more complex, with more thrombectomy passes and more contrast used, the extent of hyperdensities increases, leading to worse outcomes. A plausible hypothesis is that the basal membrane of the vessels may be compromised in these cases, potentially contributing to the incorporation of hemorrhagic components.

The SH IV group appears to have some unique features. Overall, MVO/DVO occurred more frequently than LVO in the group consisting of all patients with SH I–IV. However, when analyzing the baseline intracranial occlusion site of the SH IV group only, this group was characterized by a higher proportion of LVO compared to all other SH groups (Figure 3). In addition, in 8 out of 11 cases (73%) an active extravasation of contrast was observed on DSA, meaning that this group experienced peri-interventional complications, usually requiring a rescue maneuver.

Although this study was not designed to draw conclusions about therapeutic implications, the results suggest that patients in the SH II–IV groups might potentially benefit from more intensive post-interventional monitoring and treatment regimens, such as stricter control of arterial blood pressure and adjustment of antithrombotic regimens.

This study has several limitations. First, its retrospective and monocentric design introduces inherent biases related to study design. Second, there was a selection bias towards those receiving FDCT and patients receiving FDCT do not represent a random sample from all patients undergoing MT. Third, the lack of histopathologic correlates presents a challenge in accurately distinguishing between contrast medium and blood; however, we tried to mitigate this with spatial correlation between FDCT findings and scheduled follow-up imaging. Finally, the study included only patients with anterior circulation stroke, which may limit the generalizability of the findings.

Table 1: Baseline characteristics stratified by subarachnoid hyperdensities seen on flat-panel detector computed tomography

	Total N*	SH 0-IV	Total N*	SH 0	Total N*	SH I-IV	*P*-Value
Baseline characteristics							
Age – median [lq, uq]	223	75.5 [63.3, 83.1]	123	77.6 [63.7, 85.0]	100	74.5 [63.3, 82.1]	0.24
Sex (male) – n (%)	223	110 (49.3%)	123	65 (52.8%)	100	45 (45.0%)	0.24
Hypertension – n (%)	223	164 (73.5%)	123	93 (75.6%)	100	71 (71.0%)	0.44
Diabetes mellitus – n (%)	223	50 (22.4%)	123	31 (25.2%)	100	19 (19.0%)	0.27
Coronary heart disease – n (%)	223	30 (13.5%)	123	18 (14.6%)	100	12 (12.0%)	0.57
Smoking (current) – n (%)	223	49 (22.0%)	123	30 (24.4%)	100	19 (19.0%)	0.33
Hyperlipidemia – n (%)	223	131 (58.7%)	123	71 (57.7%)	100	60 (60.0%)	0.73

Atrial fibrillation – n (%)	223	77 (34.5%)	123	48 (39.0%)	100	29 (29.0%)	0.12
NIHSS at baseline – median [lq, uq]	219	12.0 [5.0, 20.0]	121	13.0 [6.0, 19.0]	98	11.0 [5.0, 20.0]	0.37
modified Rankin scale (pre-stroke) – median [lq, uq]	212	0.0 [0.0, 1.0]	118	0.0 [0.0, 1.0]	94	0.0 [0.0, 1.0]	0.66
Baseline intracranial occlusion site – n (%)	223		123		100		
Large vessel occlusion		114 (51%)		76 (62%)		38 (38%)	**<0.001**
Medium and distal vessel occlusion		109 (49%)		47 (38%)		62 (62%)	
ASPECTS – median [lq, uq]	220	7.0 [6.0, 9.0]	122	7.0 [5.0, 9.0]	98	7.0 [6.0, 9.0]	0.45
Most distal device position – n (%)	223		123		100		**<0.001**
Large vessel		34 (15%)		30 (24%)		4 (4.0%)	
Medium vessel		140 (63%)		72 (59%)		68 (68%)	
Distal vessel		49 (22%)		21 (17%)		28 (28%)	
Intravenous thrombolysis – n (%)	223	101 (45.3%)	123	49 (39.8%)	100	52 (52.0%)	0.07
Time of symptom onset known – n (%)	223		123		100		0.44
no		59 (26.5%)		34 (27.6%)		25 (25.0%)	
wake up		40 (17.9%)		25 (20.3%)		15 (15.0%)	
yes		124 (55.6%)		64 (52.0%)		60 (60.0%)	
Onset to groin puncture (min.) – median [lq, uq]	223	192.0 [155.0, 266.0]	123	186.0 [156.5, 266.0]	100	202.5 [150.0, 264.0]	0.99
Medications (pre-stroke)							
Antihypertensives – n (%)	220	130 (58.3%)	120	76 (61.8%)	100	54 (54.0%)	0.16
Lipid-lowering drugs – n (%)	223	70 (31.4%)	123	40 (32.5%)	100	30 (30.0%)	0.69
Anticoagulation – n (%)	223	44 (19.7%)	123	28 (22.8%)	100	16 (16.0%)	0.21
Antiplatelet – n (%)	223	47 (21.1%)	123	24 (19.5%)	100	23 (23.0%)	0.53

*N: number of patients without missing data.lq, lower quartile; uq, upper quartile; ASPECTS, Alberta stroke program early CT score; NIHSS, National Institutes of Health Stroke Scale; SH, subarachnoid hyperdensities

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Table 2: Procedural characteristics and outcome measures stratified by subarachnoid hyperdensities seen on flat-panel detector computed tomography

	Total N*	SH 0-IV	Total N*	SH 0	Total N*	SH I-IV	*P*-Value
Procedural characteristics							
Number of passes - median [lq, uq]	218	2.0 [1.0, 3.0]	121	1.0 [1.0, 2.0]	97	2.0 [1.0, 4.0]	**<0.001**
Most distal device position – n (%)	223		123		100		
Large vessel		34 (15%)		30 (24%)		4 (4.0%)	**<0.001**
Medium vessel		140 (63%)		72 (59%)		68 (68%)	
Distal vessel		49 (22%)		21 (17%)		28 (28%)	
Amount of contrast medium (ml) – median [lq, uq]	217	120 [100, 180]	119	110.0 [90, 160]	98	133 [100, 190]	**0.035**
Active extravasation seen on DSA – n (%)	223	13 (5.8%)	123	0 (0.0%)	100	13 (13.0%)	**<0.001**
Parenchymal hyperdensities on FDCT– n (%)	223	110 (49%)	123	67 (55%)	100	43 (43%)	**0.09**
eTICI score – n (%)	221		123		98		**0.014**
0		14 (6.3%)		3 (2.4%)		11 (11.0%)	
1		4 (1.8%)		1 (0.8%)		3 (3.0%)	
2a		8 (3.6%)		6 (4.9%)		2 (2.0%)	
2b50		28 (12.6%)		13 (10.6%)		15 (15.0%)	
2b67		36 (16.1%)		16 (13.0%)		20 (20.0%)	
2c		50 (22.4%)		32 (26.0%)		18 (18.0%)	
3		81 (36.3%)		52 (42.3%)		29 (29.0%)	
Outcome measures							

modified Rankin scale at 3 months – median [lq, uq]	196	3.0 [1.0, 6.0]	107	2.0 [1.0, 5.0]	89	3.0 [1.0, 6.0]	0.21
NIHSS at 24 hours – median [lq, uq]	213	8.0 [3.0, 16.0]	120	8.0 [3.0, 16.0]	93	6.0 [3.0, 15.0]	0.76

*N: number of patients without missing data.

lq, lower quartile; uq, upper quartile; ASPECTS, Alberta stroke program early CT score; DSA; digital subtraction angiography; eTICI, expanded thrombolysis in cerebral infarction; FDCT, flat-panel detector computed tomography; NIHSS, National Institutes of Health Stroke Scale; SH, subarachnoid hyperdensities.

CONCLUSIONS

SH on immediate post-interventional FDCT appears to be a frequent finding, appearing in about half of MT patients. The following factors were associated with an increased SH risk: MVO or DVO as compared to a proximal LVO, a more distal device position, a higher number of device passes, a larger amount of applied contrast, worse final reperfusion scores, and after receiving intravenous thrombolysis. Patients with SH II–IV showed overall worse outcomes, indicating a potential need for more intensive post-interventional monitoring and tailored treatment strategies. Further research is necessary to optimize outcomes in this MT subpopulation by testing personalized preventive and recovery strategies.

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SUPPLEMENTAL FILES

Supplementary Table 1: Baseline characteristics, procedural characteristics, and outcome measures of the study group and the comparator group

	Total N*	Overall (study and comparator group	Total N*	Study group (MT)	Total N*	Comparator group (MT + FDCT)	*P*-Value
Baseline and procedural characteristics							
Age – median [lq, uq]	449	76.2 [65.3, 84.6]	223	75.5 [63.3, 83.1]	226	77.0 [67.2, 85.8]	0.14
Sex (male) – n (%)	449	229 (51.0%)	223	110 (49.3%)	226	119 (52.7%)	0.48
Hypertension – n (%)	449	330 (73.5%)	223	164 (73.5%)	226	166 (73.5%)	0.98
Diabetes mellitus – n (%)	449	91 (20.3%)	223	50 (22.4%)	226	41 (18.1%)	0.26
Coronary heart disease – n (%)	449	70 (15.6%)	223	30 (13.5%)	226	40 (17.7%)	0.21
Smoking (current) – n (%)	446	83 (18.5%)	223	49 (22.0%)	223	34 (15.0%)	0.07
Hyperlipidemia – n (%)	449	281 (62.6%)	223	131 (58.7%)	226	150 (66.4%)	0.09
Atrial fibrillation – n (%)	449	169 (37.6%)	223	77 (34.5%)	226	92 (40.7%)	0.18
NIHSS at baseline – median [lq, uq]	449	13.0 [6.0, 19.0]	219	12.0 [5.0, 20.0]	226	13.0 [7.0, 19.0]	0.44

modified Rankin scale (pre-stroke) – median [lq, uq]	429	0.0 [0.0, 2.0]	212	0.0 [0.0, 1.0]	217	0.0 [0.0, 2.0]	**<0.001**
Baseline intracranial occlusion site – n (%)	449		223		226	148 (65.5%)	**0.002**
Large vessel occlusion		262(58.4%)		114 (51.1%)		148 (65.5%)	
Medium and distal vessel occlusion		187 (41.6%)		109 (48.9%)		78 (34.5%)	
ASPECTS – median [lq, uq]	446	7.5 [6.0, 9.0]	220	7.0 [6.0, 9.0]	226	8.0 [6.0, 9.0]	**0.028**
Intravenous thrombolysis – n (%)	449	184 (41.0%)	223	101 (45.3%)	226	83 (36.7%)	0.06
Time of symptom onset known – n (%)	449		223		226		**0.019**
no		98 (21.8%)		59 (26.5%)		39 (17.3%)	
wake up		73 (16.3%)		40 (17.9%)		33 (14.6%)	
yes		278 (61.9%)		124 (55.6%)		154 (68.1%)	
Onset to groin puncture (min.) – median [lq, uq]	448	195.0 [151.0, 260.0]	223	192.0 [155.0, 266.0]	225	200.0 [150.0, 258.0]	0.89
Medications (pre-stroke)							
Antihypertensives – n (%)	443	278 (61.9%)	220	130 (58.3%)	223	148 (65.5%)	0.11
Lipid-lowering drugs – n (%)	447	142 (31.6%)	223	70 (31.4%)	224	72 (31.9%)	0.86
Anticoagulation – n (%)	449	79 (17.6%)	223	44 (19.7%)	226	35 (15.5%)	0.24
Antiplatelet – n (%)	449	102 (22.7%)	223	47 (21.1%)	226	55 (24.3%))	0.41
eTICI score – n (%)	447		221		226		0.07
0		19 (4.3%)		14 (6.3%)		5 (2.2%)	
1		4 (0.9%)		4 (1.8%)		0 (0.0%)	
2a		16 (3.6%)		8 (3.6%)		8 (3.5%)	
2b50		51 (11.4%)		28 (12.6%)		23 (10.2%)	
2b67		77 (17.2%)		36 (16.1%)		41 (18.1%)	

2c		117 (26.2%)		50 (22.4%)		67 (29.6%)	
3		163 (36.5%)		81 (36.3%)		82 (36.3%)	
Outcome measures							
modified Rankin scale at 3 months – median [lq, uq]	419	3.0 [1.0, 6.0]	196	3.0 [1.0, 6.0]	223	3.0 [1.0, 6.0]	0.50
NIHSS at 24 hours – median [lq, uq]	383	7.0 [2.0, 15.0]	214	8.0 [3.0, 16.0]	169	5.0 [2.0, 14.0]	**0.03**

*N: number of patients without missing data.

lq, lower quartile; uq, upper quartile; ASPECTS, Alberta stroke program early CT score; eTICI, expanded thrombolysis in cerebral infarction; FDCT, flat-panel detector computed tomography; MT, mechanical thrombectomy; NIHSS, National Institutes of Health Stroke Scale; SH, subarachnoid hyperdensities.

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Supplementary Table 2: Evolution of subarachnoid hyperdensities (SH) on follow-up imaging within 24 hours

	SH I-IV (N = 100)	SH I (N = 40)	SH II (N = 26)	SH III (N = 23)	SH IV (N = 11)	*P*-Value
SH on follow-up – n (%)						**0.004**
Complete resolution	37 (37%)	23 (57%)	6 (23%)	6 (26%)	2 (18%)	
Similar expansion	35 (35%)	12 (30%)	9 (35%)	7 (30%)	7 (64%)	
Markedly reduced expansion	20 (20%)	2 (5.0%)	10 (38%)	6 (26%)	2 (18%)	
Markedly increased expansion	8 (8.0%)	3 (7.5%)	1 (3.8%)	4 (17%)	0 (0.00%)	

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Supplementary Table 3: Baseline and procedural characteristics and outcome measures stratified by subarachnoid hyperdensities

	Total N*	SH 0 (N = 123)	Total N*	SH I (N = 40)	Total N*	SH II (N = 26)	Total N*
Baseline characteristics							
Age – median [lq, uq]	123	77.6 [63.7, 85.0]	40	74.6 [63.5, 80.0]	26	76.4 [67.1, 83.5]	23
Sex (male) – n (%)	123	65 (52.8%)	40	20 (50.0%)	26	15 (57.7%)	23
Hypertension – n (%)	123	93 (75.6%)	40	25 (62.5%)	26	22 (84.6%)	23
Diabetes mellitus – n (%)	123	31 (25.2%)	40	7 (17.5%)	26	7 (26.9%)	23
Coronary heart disease – n (%)	123	18 (14.6%)	40	6 (15.0%)	26	4 (15.4%)	23

Smoking (current) – n (%)	123	30 (24.4%)	40	9 (22.5%)	26	5 (19.2%)	23
Hyperlipidemia – n (%)	123	71 (57.7%)	40	25 (62.5%)	26	17 (65.4%)	23
Atrial fibrillation – n (%)	123	48 (39.0%)	40	13 (32.5%)	26	7 (26.9%)	23
NIHSS at baseline – median [lq, uq]	121	13.0 [6.0, 19.0]	39	10.0 [5.0, 15.0]	26	8.0 [4.0, 21.0]	22
mRS (pre-stroke) – median [lq, uq]	118	0.0 [0.0, 1.0]	36	0.0 [0.0, 0.5]	25	0.0 [0.0, 1.0]	22
Occlusion site(s) – n (%)	123		40		26		23
Large vessel occlusion		76 (62%)		13 (32%)		9 (35%)	
Medium and distal vessel occlusion		47 (38%)		27 (68%)		17 (65%)	
ASPECTS – median [lq, uq]	122	7.0 [5.0, 9.0]	40	7.5 [6.0, 9.0]	24	7.0 [6.5, 9.0]	23
Most distal device site – n (%)	123		40		40		23
Large vessel occlusion		30 (24%)		2 (5.0%)		2 (7.7%)	
Medium vessel occlusion		72 (59%)		33 (82%)		15 (58%)	
Distal vessel occlusion		21 (17%)		5 (13%)		9 (35%)	
Intravenous thrombolysis with rt-PA - n (%)	123	49 (39.8%)	39	14 (35.0%)	26	15 (57.7%)	23
Time of symptom onset known – n (%)	123		40		26		23
no		34 (27.6%)		12 (30.0%)		3 (11.5%)	
wake up		25 (20.3%)		8 (20.0%)		3 (11.5%)	
yes		64 (52.0%)		20 (50.0%)		20 (76.9%)	
Onset to groin puncture (min.) – median [lq, uq]	123	186.0 [156.5, 266.0]	40	202.5 [136.5, 310.0]	26	216.0 [166.5, 262.5]	23
Medications (pre-stroke)							
Hypertension drugs – n (%)	120	76 (61.8%)	40	20 (50.0%)	26	17 (65.4%)	23
Lipid-lowering drugs – n (%)	123	40 (32.5%)	40	11 (27.5%)	26	11 (42.3%)	23
Anticoagulation – n (%)	123	28 (22.8%)	40	9 (22.5%)	26	3 (11.5%)	23
Antiplatelets – n (%)	123	24 (19.5%)	40	10 (25.0%)	26	8 (30.8%)	23
Procedural characteristics							
Maneuver count – median [lq, uq]	121	1.0 [1.0, 2.0]		2.0 [1.0, 4.0]	26	2.0 [2.0, 3.0]	21
Amount of contrast medium (ml) – median [lq, uq]	119	110.0 [90.0, 160.0]	38	110.0 [100.0, 160.0]	26	135.0 [100.0, 190.0]	23
Active extravasation seen on DSA – n (%)	123	0 (0.0%)	40	0 (0.0%)	26	1 (3.8%)	23

Parenchymal hyperdensities on FDCT– n (%)	123	67 (54.5%)	40	15 (37.5%)	26	8 (30.8%)	23
eTICI score – n (%)	123		39		26		23
0		3 (2.4%)		4 (10.0%)		2 (7.7%)	
1		1 (0.8%)		1 (2.5%)		1 (3.8%)	
2a		6 (4.9%)		1 (2.5%)		0 (0.0%)	
2b50		13 (10.6%)		3 (7.5%)		5 (19.2%)	
2b67		16 (13.0%)		10 (25.0%)		5 (19.2%)	
2c		32 (26.0%)		9 (22.5%)		4 (15.4%)	
3		52 (42.3%)		11 (27.5%)		9 (34.6%)	
Outcome measures							
mRS at 90 days – median [lq, uq]	107	2.0 [1.0, 5.0]	37	2.0 [1.0, 4.0]	23	3.0 [2.0, 6.0]	19
NIHSS at 24 hours – median [lq, uq]	120	8.0 [3.0, 16.0]	38	5.0 [3.0, 11.0]	23	9.0 [3.0, 19.0]	22

*N: number of patients without missing data.

lq, lower quartile; uq, upper quartile; ASPECTS, Alberta stroke program early CT score; DSA; digital subtraction angiography; eTICI, expan
panel detector computed tomography; IV tPA, intravenous tissue plasminogen activator; mRS, modified Rankin scale; NIHSS, National
hyperdensities.

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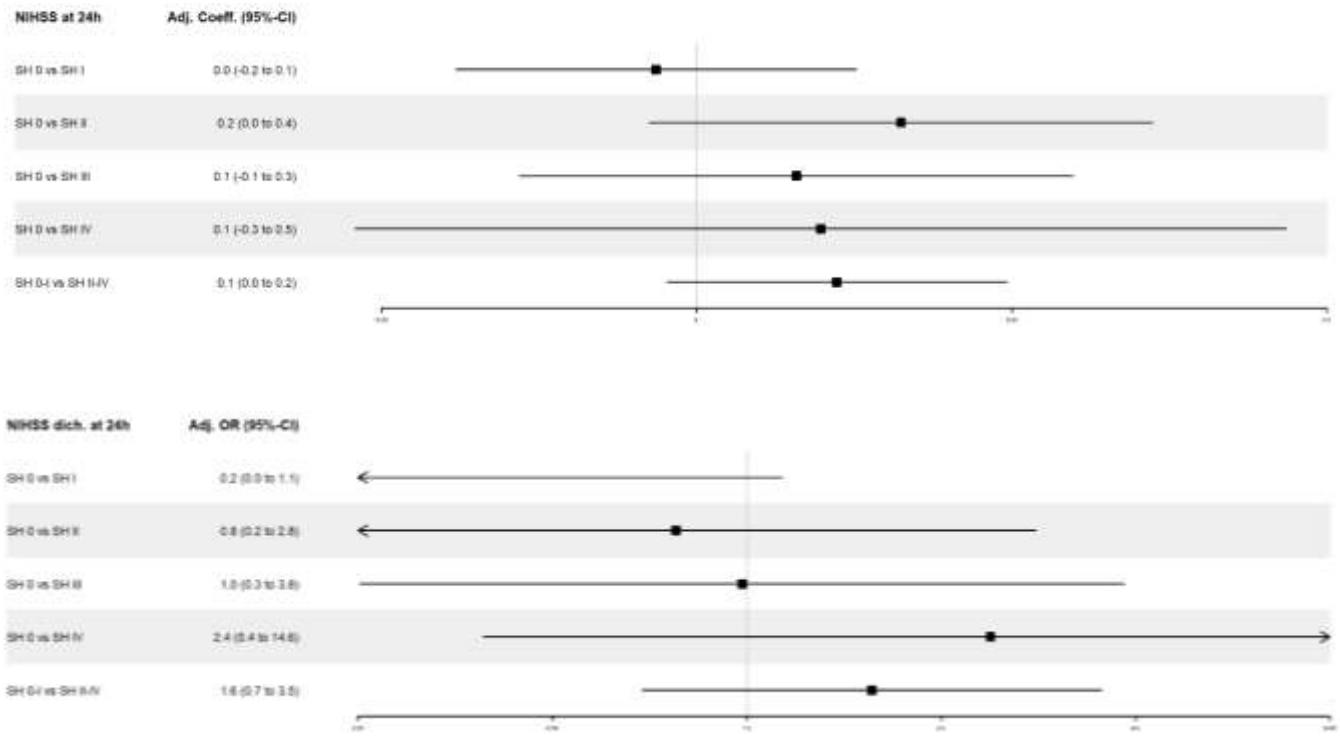
Supplementary Table 4: Outcome results of the unadjusted, adjusted and grouped comparisons			
Outcomes	OR (95% CI) or coefficient (95% CI)	Adj. OR (95% CI) or adj. coefficient (95% CI)	Adj. OR (95% CI) or adj. coefficient (95% CI) after multiple imputation
SH 0 vs SH I			
mRS (ordinal)	0.9 (0.5 to 1.7)	1.0 (0.5 to 2.0)	1.0 (0.5 to 1.9)
mRS (binary)	1.0 (0.5 to 2.1)	1.1 (0.5 to 2.3)	1.0 (0.5 to 2.1)
NIHSS at 24h (log10-transformed)	−0.1 (−0.3 to 0.1)	-0.03 (-0.2 to 0.1)	−0.04 (−0.2 to 0.1)
Worsening NIHSS (NIHSS score increase ≥4)	0.3 (0.1 to 1.2)	0.2 (0.1 to 1.1)	0.2 (0.04 to 1.0)
Mortality (mRS 6)	1.0 (0.4 to 2.5)	0.9 (0.3 to 2.5)	0.8 (0.3 to 2.0)
SH 0 vs SH II			

mRS (ordinal)	2.0 (0.9 to 4.6)	**3.2 (1.4 to 7.4)**	**2.7 (1.2 to 6.0)**
mRS (binary)	2.1 (0.8 to 5.5)	2.6 (1.0 to 6.7)	2.2 (0.9 to 5.5)
NIHSS at 24h (log10-transformed)	0.1 (−0.1 to 0.3)	0.2 (-0.04 to 0.4)	0.1 (−0.1 to 0.3)
Worsening NIHSS (NIHSS score increase ≥4)	1.0 (0.3 to 3.2)	0.8 (0.2 to 2.8)	0.7 (0.2 to 2.5)
Mortality (mRS 6)	2.4 (0.90 to 6.11)	**3.7 (1.4 to 9.9)**	3.0 (1.2 to 7.6)
SH 0 vs SH III			
mRS (ordinal)	2.2 (0.9 to 5.1)	1.8 (0.7 to 4.8)	2.1 (0.8 to 5.5)
mRS (binary)	2.5 (0.9 to 7.0)	2.3 (0.7 to 7.7)	2.6 (0.8 to 8.4)
NIHSS at 24h (log10-transformed)	0.05 (−0.2 to 0.3)	0.1 (−0.1 to 0.3)	0.1 (−0.1 to 0.3)
Worsening NIHSS (NIHSS score increase ≥4)	0.2 (0.03 to 1.8)	1.0 (0.3 to 3.8)	1.0 (0.3 to 3.7)
Mortality (mRS 6)	2.1 (0.8 to 6.0)	0.7 (0.1 to 3.7)	1.1 (0.3 to 4.8)
SH 0 vs SH IV			
mRS (ordinal)	1.3 (0.4 to 3.9)	0.9 (0.2 to 4.1)	0.9 (0.2 to 4.0)
mRS (binary)	1.1 (0.3 to 4.2)	0.7 (0.1 to 4.4)	0.7 (0.1 to 4.2)
NIHSS at 24h (log10- transformed)	0.2 (−0.1 to 0.4)	0.1 (−0.3 to 0.5)	0.1 (−0.3 to 0.5)
Worsening NIHSS (NIHSS score increase ≥4)	**3.9 (1.1 to 13.9)**	2.4 (0.4 to 14.6)	2.4 (0.4 to 14.7)
Mortality (mRS 6)	1.6 (0. 4 to 6.5)	1.3 (0.2 to 9.8)	1.2 (0.2 to 9.5)
SH 0–I vs SH II–IV			
mRS (ordinal)	**1.9 (1.1 to 3.4)**	**2.1 (1.2 to 3.8)**	**2.0 (1.2 to 3.6)**
mRS (binary)	**2.0 (1.0 to 3.8)**	**2.2 (1.1 to 4.3)**	**2.0 (1.1 to 3.9)**
NIHSS at 24h (log10-transformed)	0.0 (−0.03 to 0.2)	0.1 (-0.02 to 0.3)	0.1 (-0.02 to 0.2)
Worsening NIHSS (NIHSS score increase ≥4)	1.3 (0.6 to 2.9)	1.6 (0.7 to 3.5)	1.6 (0.7 to 3.6)
Mortality (mRS 6)	**2.1 (1.1 to 4.2)**	1.7 (0.8 to 3.6)	1.7 (0.9 to 3.5)

mRS, modified Rankin scale; NIHSS, National Institutes of Health Stroke Scale; SH, subarachnoid hyperdensities.



Supplementary FIG 1. The association of different grades of subarachnoid hyperdensities (SH 0 to IV) with National Institutes of Health Stroke Scale (NIHSS) at 24 hours, and worsening of NIHSS (change ≥4 between admission and 24-hour follow-up). Analyses were carried out using multivariable ordinal/logistic regression adjusting for prespecified confounders (see methods).