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ABSTRACT

BACKGROUND AND PURPOSE: Parenchymal hemorrhage is a severe complication following mechanical recanalization in patients with acute ischemic stroke with large-vessel occlusion. This study aimed to assess whether the metallic hyperdensity sign on noncontrast CT performed immediately after mechanical thrombectomy can predict parenchymal hemorrhage at 24 hours.

MATERIALS AND METHODS: We included consecutive patients with acute ischemic stroke with large-vessel occlusion who underwent noncontrast CT immediately after mechanical thrombectomy between January 2014 and September 2018. The metallic hyperdensity sign was defined as a nonpetechial intracerebral hyperdense lesion (diameter, ≥ 1 cm) in the basal ganglia and a maximum CT density of >90 HU. The sensitivity, specificity, and positive and negative predictive values of the metallic hyperdensity sign in predicting parenchymal hemorrhage were calculated.

RESULTS: A total of 198 patients were included. The metallic hyperdensity sign was found in 59 (29.7%) patients, and 51 (25.7%) patients had parenchymal hemorrhage at 24 hours. Patients with the metallic hyperdensity sign are more likely to have parenchymal hemorrhage than those without it (76.3% versus 4.3%, $P < .001$). The sensitivity, specificity, positive predictive value, and negative predictive value of the metallic hyperdensity sign in predicting parenchymal hemorrhage were 88.2%, 90.5%, 76.3%, and 95.7%, respectively.

CONCLUSIONS: The presence of the metallic hyperdensity sign on noncontrast CT performed immediately after mechanical thrombectomy in patients with large-vessel occlusion could predict the occurrence of parenchymal hemorrhage at 24 hours, which might be helpful in postinterventional management within 24 hours after mechanical thrombectomy.

ABBREVIATIONS: HT = hemorrhagic transformation; MT = mechanical thrombectomy; PH = parenchymal hemorrhage

Reperfusion therapy with mechanical thrombectomy (MT) improves functional outcome in patients with acute ischemic stroke with large-vessel occlusion.^{1–5} However, $>50\%$ of patients still experience unfavorable outcome.^{6,7} One of the most common reasons is hemorrhagic transformation (HT), especially parenchymal hemorrhage (PH), which is closely related to deterioration of neurologic symptoms.^{8,9} Early prediction of

HT, therefore, is important for postinterventional therapy adjustment—that is, whether to start treatment with the glycoprotein IIb/IIIa inhibitor tirofiban to prevent early re-occlusion due to endothelial damage.¹⁰

In some hospitals, immediate postinterventional non-contrast-enhanced CT has been introduced to estimate bleeding complications. Hyperdense areas may frequently be detected on NCCT after intervention.^{11–14} However, such hyperdense areas on NCCT have not been well understood, and different interpretations have been reported in various publications.

For instance, Yoon et al¹³ demonstrated that intracerebral hyperdense lesions with a maximum Hounsfield unit measurement of >90 were highly associated with symptomatic hemorrhage after intra-arterial thrombolysis. On the other hand, Jang et al¹⁴ reported that the possibility of HT depended on different morphologic features of intracerebral hyperdense lesions after intra-arterial thrombolysis, and they proposed the concept of metallic hyperdensity lesions, which were closely related to a high proportion of HT after intra-arterial throm-

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Table 1: Predictive value of characteristics of metallic hyperdensity for parenchymal hemorrhage

	AUC	95% CI	P Value	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
Presence of metallic hyperdensity sign	0.894	0.835–0.952	<.001	0.882	0.905	0.763	0.957

Note:—AUC indicates area under the curve.

Table 2: Comparison of characteristics between patients with and without the metallic hyperdensity sign

Characteristics	Presence of Metallic Hyperdensity Sign (n = 59)	Absence of Metallic Hyperdensity Sign (n = 139)	P Value
Age (mean) (yr)	70.5 ± 10.5	69.4 ± 12.2	.568
Female (No.) (%)	26 (44.1)	56 (40.3)	.639
Comorbid conditions			
Hypertension (No.) (%)	37 (62.7)	84 (60.4)	.874
Diabetes mellitus (No.) (%)	14 (23.7)	24 (17.3)	.326
Atrial fibrillation (No.) (%)	35 (59.3)	50 (35.9)	.003
Clinical variables			
Baseline NIHSS (median) (IQR)	15 (12–18)	14 (10–17)	.116
Onset-to-puncture time (median) (IQR) (min)	334 (237–425)	306 (213–415)	.289
Retrieval attempts (median) (IQR)	2 (1–3)	1 (1–3)	.039
Radiologic data			
Baseline infarct volume (median) (IQR) (mL)	59.9 (34.7–80.9)	46.3 (27.5–80.0)	.096
Baseline hypoperfusion volume (median) (IQR) (mL)	108.9 (79.2–153.6)	107.1 (68.0–160.0)	.639
Recanalization (No.) (%)	48 (81.4)	123 (88.4)	.181
24-Hour hemorrhagic transformation (No.) (%)	58 (98.3)	54 (38.8)	<.001
24-Hour parenchymal hemorrhage (No.) (%)	45 (76.3)	6 (4.3)	<.001

Note:—IQR indicates interquartile range.

bolysis. At present, thrombectomy has been demonstrated as an effective therapy for patients with acute ischemic stroke with large-vessel occlusion.¹⁵ However, the relationship between the occurrence of parenchymal hemorrhage at 24 hours and hyperdense lesions on NCCT immediately after thrombectomy is still unclear.

Here, we propose a new kind of metallic hyperdensity sign combining maximum Hounsfield units and morphologic features to predict PH at 24 hours. The aim of our study was to evaluate the value of the newly defined metallic hyperdensity sign following MT for predicting PH at 24 hours in patients with acute ischemic stroke with large-vessel occlusion.

MATERIALS AND METHODS

Ethics Statement

Written informed consent was obtained from each patient or an appropriate family member. The human ethics committee of the Second Affiliated Hospital of Zhejiang University approved the protocol of this study. All clinical investigations were conducted according to the principles expressed in the Declaration of Helsinki.

Patient Selection

We analyzed our prospectively collected data base for consecutive patients with acute ischemic stroke who received mechanical thrombectomy between January 2014 and September 2018, as NCCT examination immediately after mechanical thrombectomy was performed since January 2014. We enrolled patients with the following qualifications: 1) They underwent MT due to large-vessel occlusion of the anterior circulation within 24 hours post-symptom onset based on multimodal imaging, 2) they underwent postinterventional

NCCT immediately after MT, and 3) they had a follow-up CT or MR imaging at 24 hours.

Definition of the Metallic Hyperdensity Sign

All CT examinations were performed on a multidetector CT scanner with axial 5-mm section thickness. “Hyperdense lesion” was defined as the presence of high density within the brain parenchymal area. First, the hyperdense lesions on each layer were manually segmented, respectively, by 2 experienced neurologists (C.X. and Y.Z.), with the open-source software RadiAnt DICOM Viewer (<https://www.radiantviewer.com>). Second, we measured the density value (Hounsfield unit) of the segmented area on each layer and recorded the maximum value. The metallic hyperdensity sign was defined when a maximum CT density of >90 HU was identified within the non-petechial intracerebral hyperdense lesion (diameter, ≥1 cm) in the basal ganglia.

Two experienced neurologists (X.C. and Z.Y.) blinded to clinical data reviewed images for assessing the presence or absence of the metallic hyperdensity sign on NCCT independently, and the inconsistent data were reviewed by another experienced neurologist and settled by consensus discussion afterward.

Evaluation of HT and PH

HT (including hemorrhagic infarction and PH) was assessed on the basis of imaging characteristics at 24 hours after MT, referring to the European Cooperative Acute Stroke Study II (ECASS II) criteria.¹⁶ According to previous studies, intracerebral hyperdense lesions that were no longer discernible on the 24-hour follow-up CT were defined as contrast enhancement, while lesions persisting on follow-up CT were considered hemorrhagic lesions.¹⁷

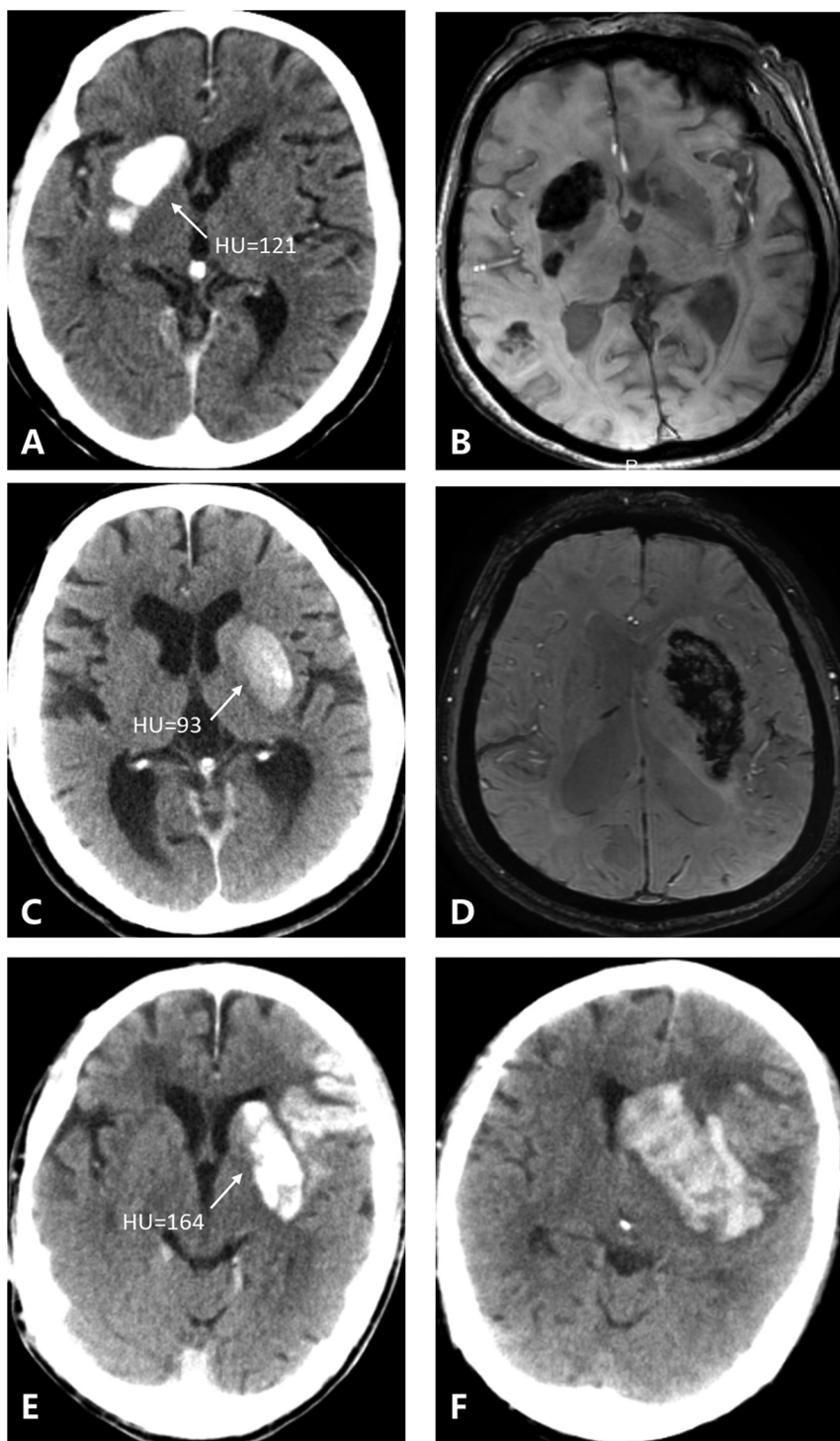


FIG 1. Three patients with 3 kinds of metallic hyperdensity signs. A, C, and E, NCCT images obtained immediately after mechanical thrombectomy. B and D, SWI at 24 hours after mechanical thrombectomy. A 77-year-old woman with a hyperdense lesion and a maximum CT density of 121 HU in the right caudate nucleus on NCCT images immediately after mechanical thrombectomy (A) has parenchymal hemorrhage on SWI at 24 hours (B). A 71-year-old man with a hyperdense lesion and a maximum CT density of 93 HU in the left lenticular nucleus on NCCT images immediately after mechanical thrombectomy (C) has parenchymal hemorrhage on SWI at 24 hours (D). An 81-year-old man with a hyperdense lesion and a maximum CT density of 164 HU in the left lenticular nucleus on NCCT images immediately after mechanical thrombectomy (E) has parenchymal hemorrhage on the NCCT image at 24 hours (F).

Statistical Analysis

All statistical analyses were performed with the SPSS package (Version 19.0; IBM, Armonk, New York). Metric and normally

distributed variables were reported as mean $\pm$ SD; non-normally distributed variables were reported as median and interquartile range. Categorical variables were presented as frequency (percentage). The Fisher exact test was used to compare the dichotomous variables, and the Mann-Whitney *U* test was used for the non-normally distributed continuous variables. Normally distributed continuous variables were assessed with the Student *t* test. Statistical significance was set at $P < .05$. κ statistics were used to test inter- and intrarater reliability for detecting the presence of the metallic hyperdensity sign. Receiver operating characteristic curve analysis was used to determine the predictive value.

RESULTS

A total of 198 patients were included. The mean age was 69.8 ± 11.7 years, and 82 (41.4%) were women. The median baseline NIHSS was 14 (interquartile range = 11–18). One hundred twelve (56.5%) patients had HT, and 51 (25.7%) patients had PH at 24 hours.

Intracerebral hyperdense lesions were found in 155 (78.3%) patients, among whom 59 (38.1%) patients had the metallic hyperdensity sign and the remaining 96 (61.9%) patients had a nonmetallic hyperdensity sign. Moreover, 14 (14.6%) patients had hyperdense lesions in the cortical area; 10 (10.4%), in the subcortical area; 64 (66.7%), in the basal ganglia; and 8 (8.3%), in both cortical and subcortical areas and basal ganglia. The inter- and intrarater observer agreement for assessing the presence of intracerebral hyperdense lesions ($\kappa = 0.924$, $\kappa = 0.947$) and the presence of the metallic hyperdensity sign ($\kappa = 0.874$, $\kappa = 0.912$) was good. Patients with the metallic hyperdensity sign were more likely to have PH than those without the sign (76.3% versus 4.3%; odds ratio, 71.250; odds ratio, 75.553 after adjusting for atrial fibrillation, onset to puncture time, and recanalization; $P < .001$). The sensitivity, specificity, positive predictive value, and negative predictive value of the metallic hyperdensity sign in predicting PH were 88.2% (95% CI, 79.1%–97.4%), 90.5% (95% CI, 85.7%–95.3%), 76.3% (95% CI, 65.1%–87.4%), and 95.7% (95% CI, 92.3%–99.1%), respectively (Table 1).

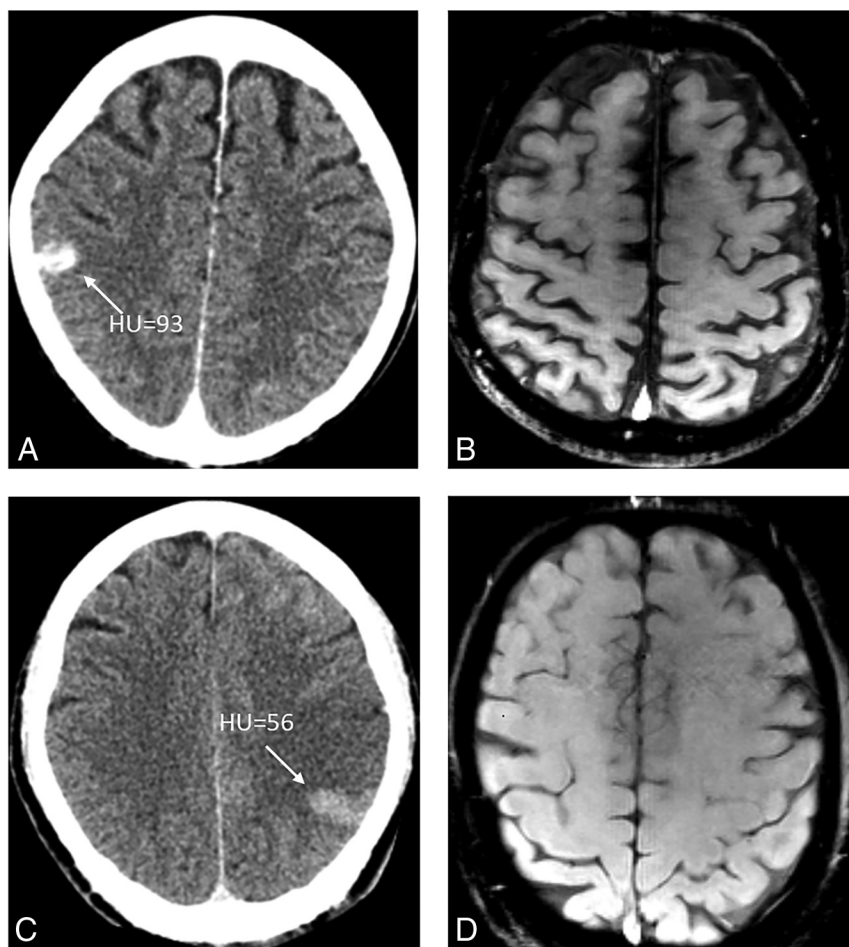


FIG 2. Two patients who show the typical cortical hyperdense lesion on NCCT immediately after MT (A and C) have no HT on follow-up SWI (B and D).

Patients with the metallic hyperdensity sign were more likely to have atrial fibrillation ($P = .003$) and underwent more retrieval attempts ($P = .039$) than those without the sign. Other demographic and clinical variables including age, sex, hypertension, diabetes mellitus, baseline NIHSS, baseline hypoperfusion volume, onset-to-puncture time, and recanalization did not significantly differ between patients with and without the metallic hyperdensity sign (Table 2).

Figure 1 shows 3 patients with 3 kinds of metallic hyperdensity signs.

DISCUSSION

Our study demonstrates that the metallic hyperdensity sign on NCCT performed immediately after MT can predict PH at 24 hours with high specificity and negative predictive value.

Intracerebral hyperdense lesions were first reported on NCCT immediately after intra-arterial thrombolysis in patients with acute ischemic stroke early in the 1990s.^{18,19} The rates of intracerebral hyperdense lesions shown on NCCT after intra-arterial reperfusion therapy varied greatly, from 32.9% to 84.2%.^{13,14,17,20} The reason for such a wide variation may be due to the differences in the definition of hyperdense lesions, number of patients, recanalization rate, and the quality of the imaging equipment.²¹ Compared with previous studies, the incidence of intracerebral hyper-

dense lesions in our center was relatively high (our center versus other centers, 83.3% versus 32.9%–84.2%); this finding may result from the relatively high recanalization rate (our center versus other centers, 86.3% versus 77.2%–80%).^{21,22} In addition, MT would increase the direct damage to the endothelium of cerebral arteries when pulling thrombus,²³ compared with intra-arterial thrombolysis, which might increase the incidence of intracerebral hyperdense lesions.

Currently, the most widely accepted hypothesis is that intracerebral hyperdense lesions are caused by leakage of contrast medium into the extracellular spaces via vessels, as a result of microvascular damage and increased permeability of the BBB.^{17,21,22} Yoon et al¹³ documented that high-density areas (>90 HU) found on NCCT after intra-arterial thrombolysis are closely associated with PH, possibly due to pathologic changes of ischemic injury to the degradation of the basal lamina, a structural barrier. Moreover, PH is the result of severe microvascular damage extended to the basal lamina. In our study, 51 (25.7%) patients had PH at 24 hours, which is higher than that in published trials such as the Multicenter Randomized Clinical trial of Endovascular treatment for

Acute ischemic stroke in the Netherlands (MR CLEAN), Endovascular Treatment for Small Core and Proximal Occlusion Ischemic Stroke (ESCAPE), Extending the Time for Thrombolysis in Emergency Neurological Deficits–Intra-Arterial (EXTEND-IA), Solitaire with the Intention for Thrombectomy as Primary Endovascular Treatment (SWIFT PRIME), and Endovascular Revascularization With Solitaire Device Versus Best Medical Therapy in Anterior Circulation Stroke Within 8 Hours (REVASCAT) at 6%, 3.6%, 11%, 5%, and 5.8%, respectively.^{1–5} However, our rate is actually based on the real-world data for mechanical thrombectomy. The median time from stroke onset to groin puncture in these above studies was not >270 minutes (4.5 hours), which, to some extent, is difficult to achieve in the real world. Indeed, in previous retrospective studies, the occurrence of PH varied greatly, from 5.9% to 23.5%.^{17,24} Another reason for the relatively higher rate of PH is the use of SWI to detect hemorrhage because SWI accounted for 48% of the 24-hour follow-up examinations, which may result in a visual overestimation in hematoma size due to susceptibility effects.²⁵

In addition, we found that the metallic hyperdensity sign was more likely in patients with atrial fibrillation and a higher number of retrieval attempts. Actually, the extent of BBB disruption was more severe in cardiogenic embolism stroke than in other stroke

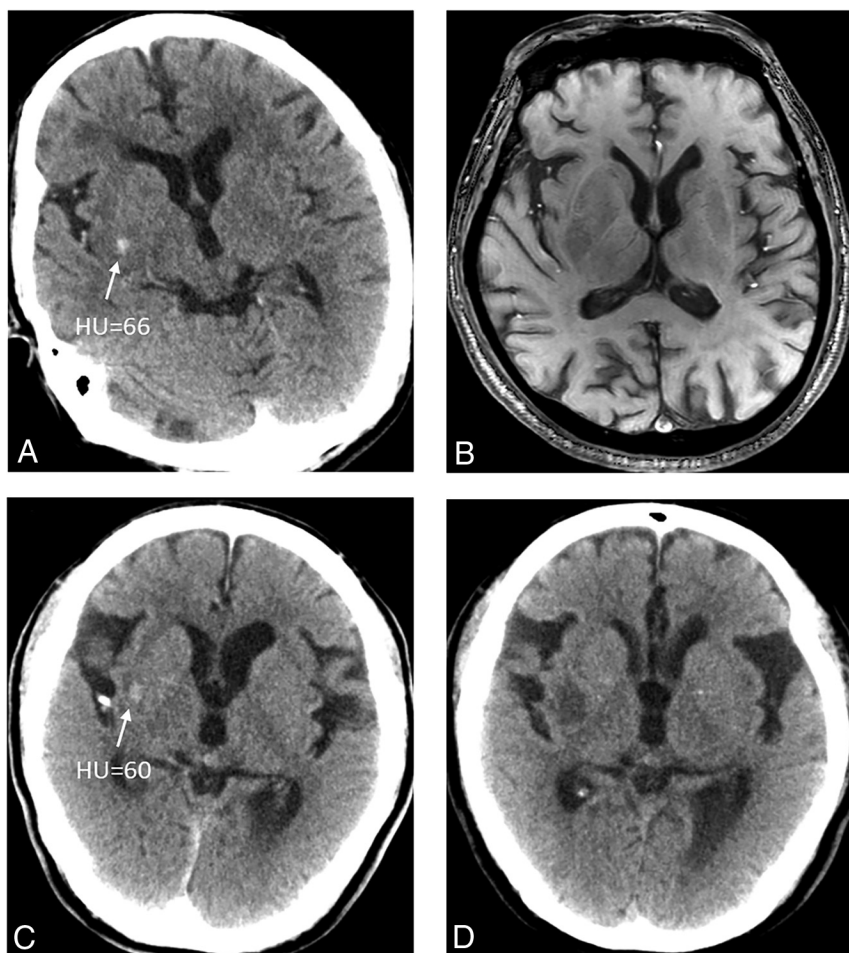


FIG 3. Two patients who show a petechial hyperdense lesion in the basal ganglia on NCCT immediately after MT (A and C) have no HT on follow-up SWI (B) and the remaining low densities on follow-up NCCT (D).

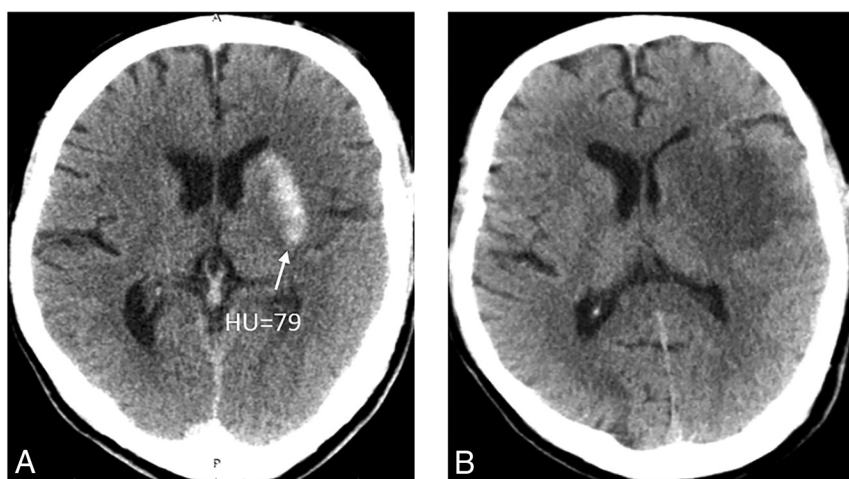


FIG 4. A patient who shows a nonpetechial hyperdense lesion on NCCT immediately after MT (A) has no HT on follow-up NCCT and the remaining low densities (B).

subtypes during the hyperacute stage²⁶; with the increased number of retrieval attempts, the damage to the endothelium of cerebral arteries would be more severe.²³ Besides, previous experimental research has shown that massive HT, with the distinguishing features of a rapid extravasation of blood leading to

the compression of contiguous tissues, occurs when the BBB integrity is lost.²⁷ Therefore, we propose that the presence of our defined metallic hyperdensity sign in the current study might reflect severe microvascular damage and extensive BBB disruption because the study focused on those subjects with relatively high contrast leakage, which leads to massive HT, especially PH.

To our knowledge, this is the first study focusing on both the density and extent of intracerebral hyperdense lesions on NCCT performed immediately after MT and its predictive value for PH at 24 hours. The metallic hyperdensity sign, a readily available and reliable NCCT-based imaging marker, might be helpful in identifying those patients with a higher risk of PH after MT and is easily applied in clinical practice. It may not only provide prognostic information for clinicians and patients but also be helpful in clinical management after MT. For instance, patients with the metallic hyperdensity sign should receive all possible care, including relatively strict blood pressure control and delayed or no antiplatelet and anticoagulation treatments.

Limitations included the retrospective nature in our single stroke center and the moderate number of cases, resulting in a potential risk of selection bias, though data were prospectively established using a stroke registry. Second, there was heterogeneity in the evaluation of PH, which was due to 2 methods of follow-up imaging (NCCT versus SWI, 52% versus 48%). Third, despite our effort to find the optimal Hounsfield unit threshold for the metallic hyperdensity sign, the selection of 90 HU was still based on previous literature. In addition, 6 patients in our study without the metallic hyperdensity sign on NCCT obtained immediately after MT still developed PH on follow-up images. Although the number of these patients is very small, the causes and mechanisms of this situation and the optimal HU threshold are worthy of further study.

Although a CT scan obtained at least

19–24 hours after endovascular therapy was reported as a reliable method to differentiate contrast from hemorrhage,²⁸ contrast enhancement could still be relatively dense 24 hours after thrombectomy, and the differentiation would be better with the extended time after an intervention. Finally, we did not measure BBB per-

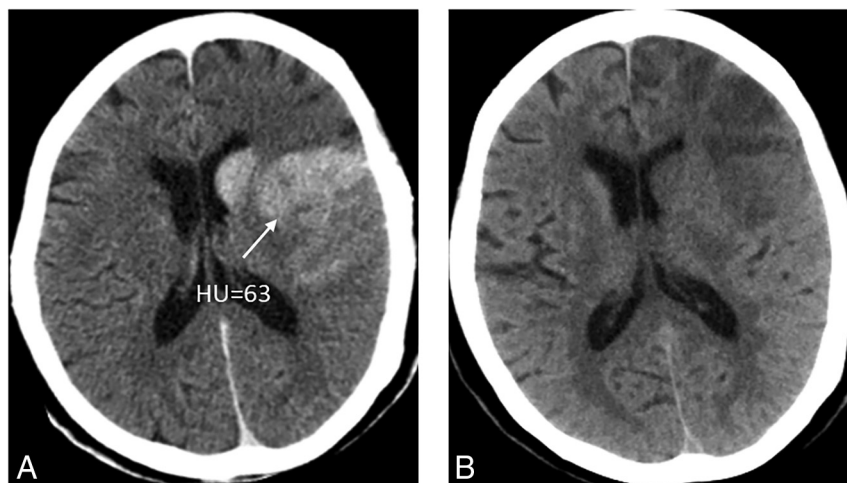


FIG 5. A patient who shows the subcortical hyperdense lesion involving both the basal ganglia and subcortical white matter on NCCT immediately after MT (A) has no HT on NCCT immediately after MT and the remaining low densities on follow-up NCCT (B).

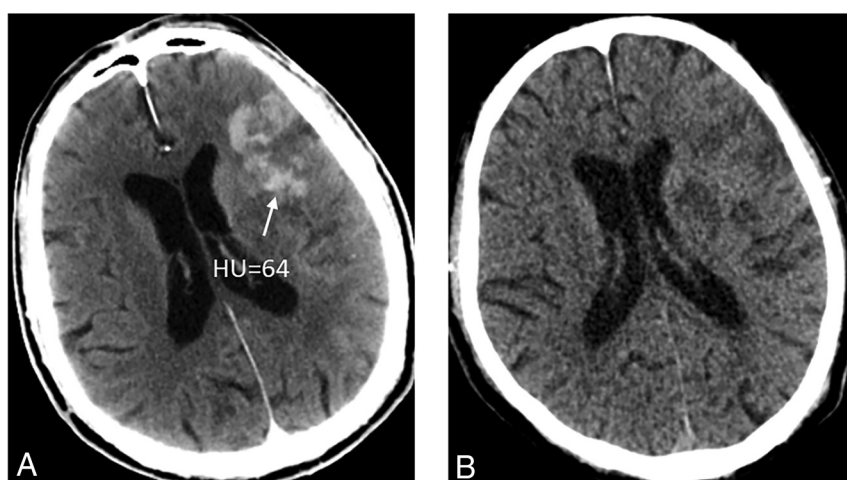


FIG 6. A patient who shows the subcortical hyperdense lesion on NCCT immediately after MT (A) but has no HT on follow-up NCCT and the remaining low densities (B).

meability on multimodal imaging, and the underlying mechanisms of hyperdense intracerebral lesions need to be clarified with further experimental and pathologic studies.

CONCLUSIONS

The NCCT-based metallic hyperdensity sign immediately after MT in patients with large-vessel occlusion may indicate a high risk of PH at 24 hours with a high specificity of 90.5% (odds ratio, 90.576; 95% CI, 85.7%–95.3%) and negative predictive value of 95.7% (odds ratio, 95.683; 95% CI, 92.3%–99.1%), which may be helpful in postinterventional management 24 hours after mechanical thrombectomy.

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