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Bright and dark vessels on stroke imaging: different sides of the same coin?

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PURPOSE
Prominent hypointense cerebral vessels on susceptibility-weighted imaging (SWI) and the hyperintense vessel sign (HVS) on fluid-attenuated inversion recovery (FLAIR) imaging are considered as markers of compromised tissue perfusion in cerebral ischemia. In this study, we aimed to identify the correlation between HVS on FLAIR and hypointense vessels on SWI, and to determine whether these imaging features provide independent prognostic information in patients with ischemic stroke.

METHODS
We retrospectively analyzed consecutive ischemic stroke patients with proximal middle cerebral artery (MCA) occlusion who underwent SWI and FLAIR within 24 h of symptom onset. The presence of hypointense vessels on SWI and hyperintense vessels on FLAIR in >4 of 10 slices encompassing the MCA territory were considered to represent prominent hypoperfusion.

RESULTS
Among 50 patients, 62% had a prominent HVS on FLAIR and 68% had prominent hypointense vessels on SWI. There was a moderate but significant correlation between the number of slices with HVS on FLAIR and prominent hypointense vessels on SWI (r=0.425, $P = 0.002$). In multivariate analyses, the prominence of hypointense vessels on SWI, but not HVS on FLAIR, was significantly associated with a higher discharge NIHSS score ($P = 0.027$), mRS score ($P = 0.021$), and lesion growth ($P = 0.050$).

CONCLUSION
The significant, albeit moderate, correlation between markers of compromised tissue perfusion on FLAIR and SWI suggests that these imaging features reflect different but interrelated aspects of cerebral hemodynamics during ischemic stroke. Our findings highlight that while HVS on FLAIR denotes the presence of leptomeningeal collaterals, hypointense vessels on SWI signify the sufficiency of cerebral blood flow at the tissue level and are therefore more critical in terms of prognosis.

The determination of the extent of brain tissue that is ischemic, but still viable and therefore under risk for irreversible injury (i.e., penumbra), is one of the major goals in acute stroke imaging (1). Advances in technology and the availability of perfusion imaging, either by computed tomography (CT) or magnetic resonance imaging (MRI), are major steps in this regard, and multi-modal imaging by CT or MRI is gaining importance as a tool in the development of both prognostic and therapeutic algorithms in acute stroke (2). Despite these developments, there are certain roadblocks in the utilization of perfusion imaging, with MRI in particular, in acute stroke patients. These include the unavailability of resources, primarily MRI scanners, in emergency departments; the need for technical and medical personnel for the postprocessing and interpretation of perfusion images, which can partially be overcome by automated imaging softwares; and the use of intravenous contrast agents, which are highly critical in patients with renal failure.

Disregarding perfusion imaging by arterial spin labeling, other imaging signatures of compromised tissue perfusion that can be detected by MRI sequences that do not necessitate the use of contrast administration might provide clinicians with alternative and more practical tools in this setting. One of these signatures is the hyperintense vessel sign (HVS) observed on fluid-attenuated inversion recovery (FLAIR) imaging, which is considered to reflect slow and compensatory blood flow within leptomeningeal collaterals distal to the proximal occlusion (3). Prior studies have shown that patients with HVS evident on FLAIR are more like-

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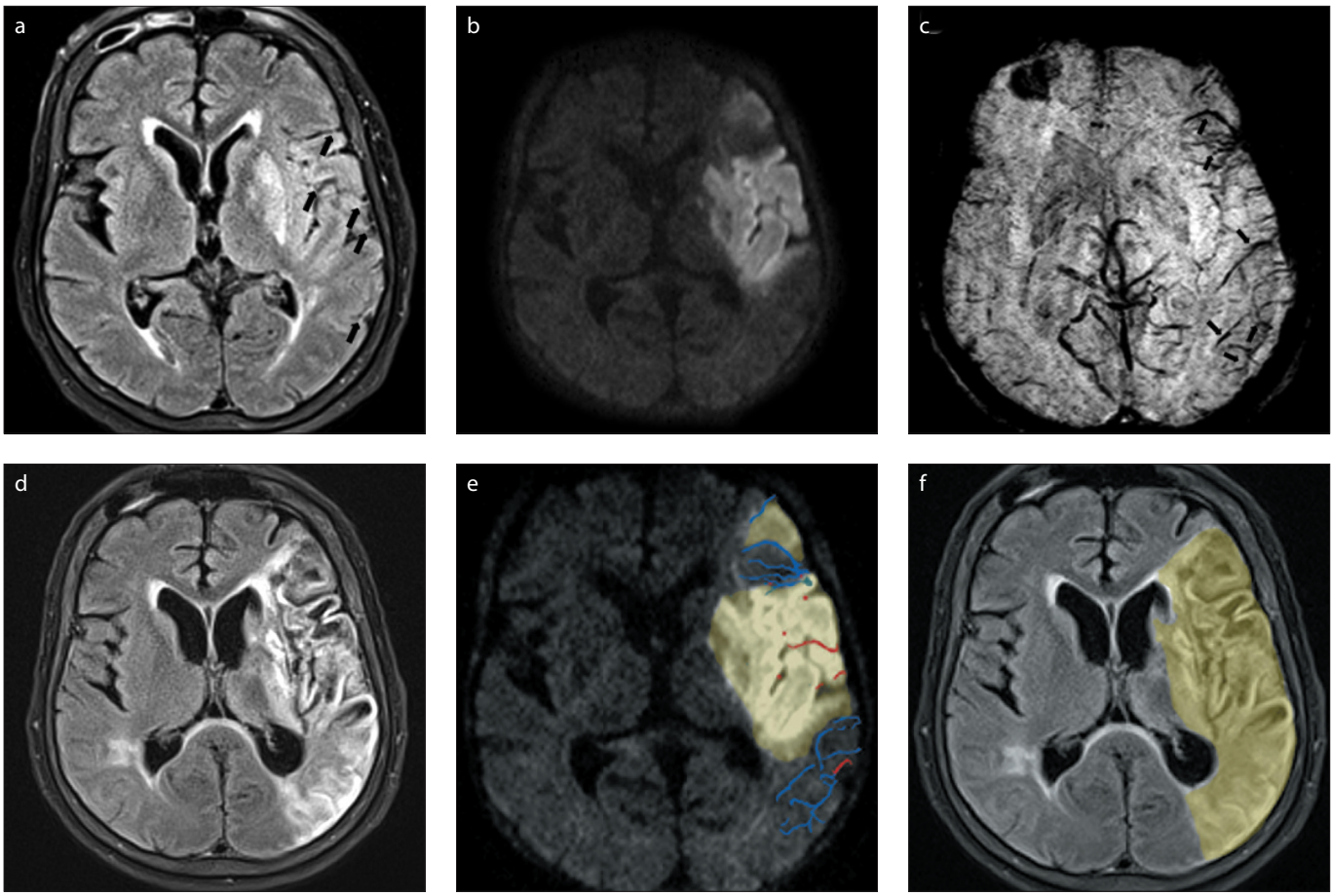

Figure 1. a–f. A patient with a high number of hypointense vessels on susceptibility-weighted imaging (SWI) and prominent infarct growth. On initial MRI, hyperintense vessel sign (HVS) on fluid-attenuated inversion recovery (FLAIR) **(a)** is observed to a greater extent, in particular, posterior to the diffusion-restricted area **(b)** (*arrows*). Hypointense vessels on SWI are seen in a larger territory than both the area with diffusion restriction and HVS on FLAIR **(c)** (*arrows*). Significant lesion growth is seen on follow-up FLAIR image **(d)**. For better illustration, these sequences were overlaid and color-coded as described in the Methods section **(e, f)**. A high number of hypointense vessels on SWI (**e**, *depicted in blue*) associated with significant lesion growth (**e, f,** *depicted in yellow*) can be seen (admission DWI lesion volume, 87 mL; follow-up lesion volume, 231 mL).

Main points

- Prominent hypointense cerebral vessels on susceptibility-weighted imaging (SWI) and hyperintense vessel sign (HVS) on fluid-attenuated inversion recovery (FLAIR) imaging are considered as markers of compromised tissue perfusion in the setting of cerebral ischemia.
- However, their relation to each other has not been studied before. Our results suggest that there is a moderate correlation between the extent of FLAIR-HVS and prominent hypointense vessels on SWI.
- Prominence of hypointense vessels on SWI, but not FLAIR-HVS was significantly associated with worse prognosis (higher discharge NIHSS score, modified Rankin scale and lesion growth).
- These imaging features reflect different, but interrelated aspects of cerebral hemodynamics during ischemic stroke.

ly to have a diffusion/perfusion mismatch, and although not consistently documented in all published literature, they are at risk of infarct progression (4, 5). Another imaging feature suggestive of impaired tissue perfusion is the presence of marked hypointense vessels on susceptibility-weighted imaging (SWI), which is believed to represent veins with increased deoxyhemoglobin concentration secondary to an increased oxygen extraction fraction within the ischemic tissue (6–8). Although various studies have separately evaluated the influence of these imaging features on clinical and tissue prognosis, none have formally analyzed their prognostic role in the same patient cohort and evaluated whether these imaging markers reflect similar aspects of impaired tissue perfusion or contain information independent of each other. In this study, we aimed to identify the correlation between the extent of HVS on FLAIR and hypointense vessels on SWI, and to determine whether these imaging features provide independent prognostic information in patients with ischemic stroke.

Methods

Patients

We retrospectively analyzed a consecutive series of patients with proximal middle cerebral artery (MCA) occlusion who were admitted to our center between 2010 and 2013. We included patients who underwent MRI, including both SWI and FLAIR imaging, within 24 h of symptom onset. The presence of MCA occlusion was verified either by magnetic resonance angiography concomitantly performed with SWI and FLAIR imaging or by a CT angiography study performed after MRI. The study was approved by the local institutional review board.

Clinical and demographic information including age, gender, stroke risk factors, admission National Institute of Health Stroke

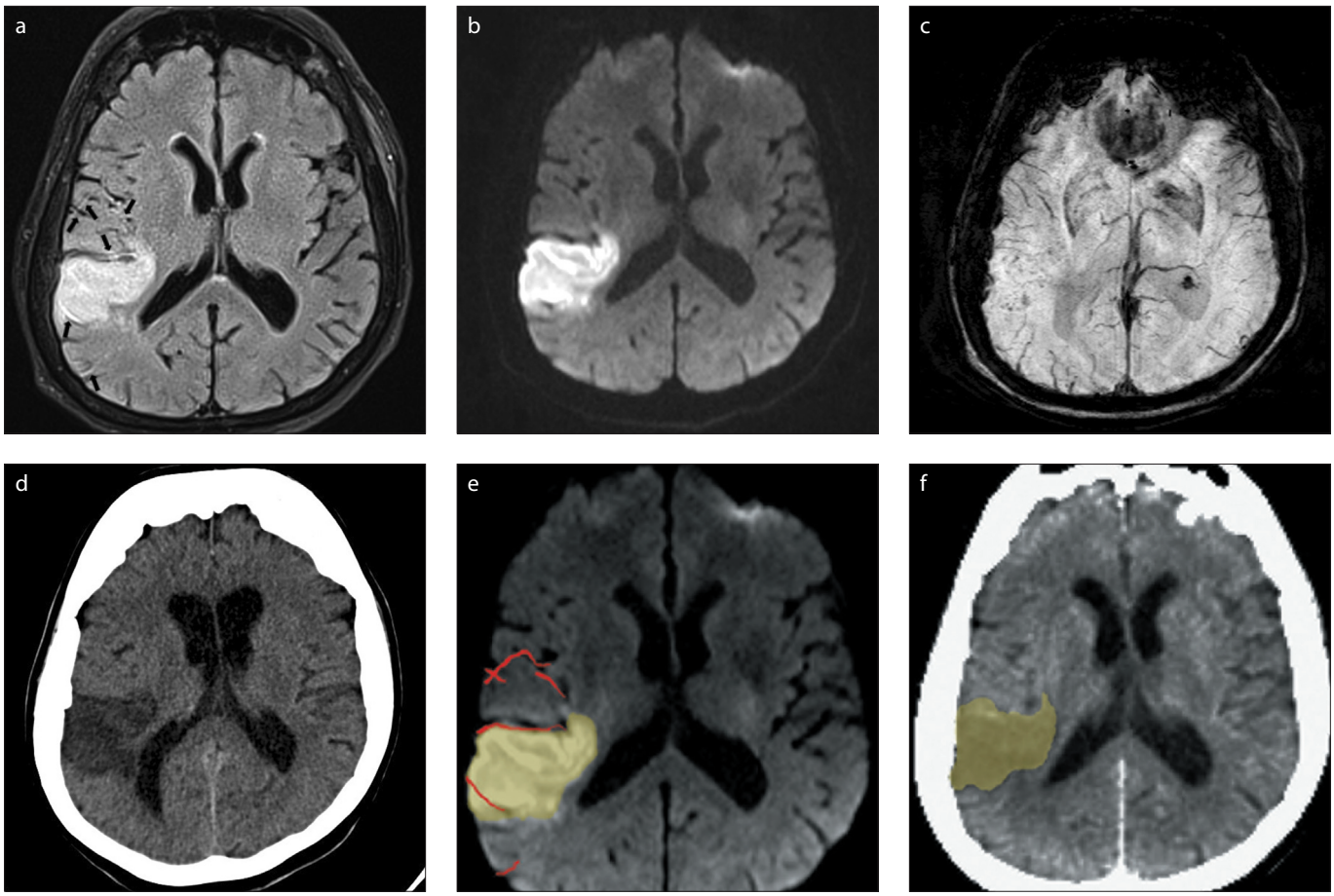

Figure 2. a–f. A patient with a high number of hyperintense vessels on FLAIR and no infarct growth. A prominent hyperintense vessel on FLAIR **(a)** is seen in a larger territory than the diffusion-restricted area (**b**, *arrows*). No apparent increase is observed in hypointense vessels on SWI in the diffusion-restricted area **(c)**. The follow-up CT **(d)** showed no lesion growth (admission DWI lesion volume, 57 mL; follow-up lesion volume, 52 mL). Colored illustrations of the admission and follow-up MRI are provided in **(e)** and **(f)**, respectively.

Scale (NIHSS) score, thrombolytic therapy use, stroke etiology according to the Causative Classification of Stroke (9), discharge NIHSS score, and modified Rankin scale (mRS) score were collected for all patients both by the use of our prospectively gathered departmental stroke database and by the review of retrospective charts. In addition, time to MRI, occlusion site (M1 or M2), admission lesion volume on diffusion-weighted imaging (DWI), follow-up lesion volume on FLAIR or CT obtained on day 5 or later, and time to follow-up imaging together with imaging features suggestive of compromised tissue perfusion on FLAIR and SWI (as described in detail below) were determined in all patients.

Imaging procedures

All MRI studies were performed by using a 1.5 T scanner (Magnetom TIM, Siemens). DWI together with apparent diffusion coefficient (ADC) maps and FLAIR and SWI sequences were obtained with the following acquisition parameters. FLAIR (TR/TE/TI, 8150/130/2200 ms; slice thickness, 5 mm; FOV, 220 mm; and matrix, 192×256), DWI (b values, 0–1000 s/mm^2), SWI (TR/TE, 50/40; flip angle, 15; slice thickness, 2 mm with 0 mm spacing; FOV, 230 mm; matrix, 221×320; and NEX, 1; each transverse slice constituted of four images: a magnitude image, a phase image, SWI data, and a minimum intensity projection image). Lesion volumes at admission and follow-up were calculated by semi-automated segmentation algorithms (using MRIcro software; www.mricro.com), as previously described (10). The burden of vessels showing characteristics of HVS on FLAIR was determined via a previously defined scoring algorithm, in which the presence or absence of HVS on FLAIR was evaluated on 10 consecutive slices encompassing the MCA territory (11). In line with this scoring algorithm, patients with HVS on FLAIR in ≤4 slices were categorized to have a low number of hyperintense vessels, in 5-6 slices to have a medium number of hyperintense vessels, and in ≥7 to have a high number of hyperintense vessels; in the current study, the latter two categories were combined into a single group and comprised the group of patients with prominent HVS on FLAIR. As a similar scoring algorithm has not been defined for determining the burden of hypointense vessels on SWI, we developed a scoring system analogous to the principles of HVS on FLAIR assessment. To provide consistency between FLAIR and SWI evaluations, we first reformatted SWI data in 5 mm thick slices (as in FLAIR images). We then determined the number of slices with marked hypointense vessels in comparison to the contralateral hemisphere and considered patients with hypointense vessels of >4 slices to have prominent findings on SWI. All assessments were performed while blinded to clinical and outcome data; two raters (R.G., a neuroradiologist with three years of experience, and E.M.A., a stroke neurologist with four years of experience) evaluated the FLAIR and SWI findings by a consensus reading.

Images with colored overlay (Figs. 1e, 1f, 2e, and 2f) were created using Adobe Photoshop CS4 Version 11.0 (Adobe Systems Inc.).

Table 1. Clinical and imaging characteristics of the study population (n=50)

Age (years), median (IQR)	72 (62–78)
Female, n (%)	25 (50)
Risk factors, n (%)	
Hypertension	41 (82)*
Diabetes	11 (22)
Coronary heart disease	21 (42)
Atrial fibrillation	15 (30)
Admission NIHSS score, median (IQR)	15 (8–20)
CCS subtype, n (%)	
Large artery atherosclerosis	8 (16)
Cardio-aortic embolism	29 (58)
Small artery occlusion	0 (0)
Other causes	4 (8)
Undetermined/unclassified	9 (18)
Thrombolytic therapy, n (%)	10 (20)
Time to MRI (h), median (IQR)	12 (8–16)
Site of occlusion, n (%)	
M1	36 (72)
M2	14 (28)
Admission DWI lesion volume (mL), median (IQR)	40 (10–102)
Type of follow-up imaging,* n (%)	
CT	23 (68)
MRI	11 (32)
Time to follow-up imaging* (days), median (IQR)	17 (7–33)
Follow-up lesion volume* (mL), median (IQR)	90 (19–205)
Time to discharge (days), median (IQR)	15 (9–30)
Discharge NIHSS score, median (IQR)	13 (3–19)
Discharge mRS, median (IQR)	4 (2–5)

*Analyses were performed in 34 patients.

IQR, interquartile range; NIHSS, National Institutes of Health Stroke Scale; CCS, Causative Classification System; MRI, magnetic resonance imaging; DWI, diffusion-weighted imaging; CT, computed tomography; mRS, modified Rankin Scale.

For the preparation of Figs. 1e and 2e, DWI sequences were used as background. Then, corresponding FLAIR and SWI images were overlaid as separate layers and were manually aligned to the DWI sequence. After that, regions corresponding to HVS on FLAIR were denoted with red and hypointense vessels on SWI were denoted with blue, and other areas were excluded. Lastly, the same DWI sequences were used again to mask the hyperintense region corresponding to the infarct area. The masked region was painted with yellow, and the opacity was set to 70%. For the preparation of Figs. 1f and 2f, infarct areas in follow-up FLAIR (Fig. 1f) and CT (Fig. 2f) images were manually selected, masked, and painted with yellow, and the opacity was set to 70%. These layers were overlaid on the original FLAIR and CT sequences. All layers were merged to compose the final image.

Statistical analysis

Categorical variables are expressed as n (%), and numerical variables are expressed as median and interquartile range (IQR). Spearman's correlation coefficient was used to determine the correlation between the number of slices with hyperintense vessels on FLAIR and hypointense vessels on SWI. Three outcome parameters were used to assess the prognostic influence of these imaging features: discharge NIHSS score (dichotomized according to the population median as <13 and ≥13), discharge mRS score (dichotomized as <2 and ≥2), and infarct growth (dichotomized as the ratio of follow-up to admission lesion volume <1.2 and ≥1.2). Group-wise comparisons for continuous variables were performed by the Mann–Whitney U test and for categorical variables by chi-square tests. Logistic regression models were used to determine the independent predictors of clinical and tissue outcome. Independent variables for these analyses were chosen from variables with a *P* value of <0.05 in bivariate analyses. Imaging features on SWI and FLAIR were incorporated into the multivariate models regardless of findings in bivariate analyses. Backward selection with inclusion criteria of ≤0.05 was used to prevent overfitting. All statistical analyses were performed using SPSS 16.0 (SPSS Inc.).

Results

There were a total of 50 patients who fulfilled the inclusion criteria during the study period. Table 1 summarizes clinical and imaging characteristics of the study population. Thrombolytic therapy was administered to 10 patients and preceded MRI in all cases. The median discharge NIHSS score and mRS after a follow-up duration of 15 days (range, 9–30 days) were 13 (IQR, 3–19) and 4 (IQR, 2–5), respectively. Follow-up imaging was available in 34 patients, and showed a median infarct growth rate of 19% with respect to baseline.

Throughout the MCA territory, a low number of HVS on FLAIR (present on ≤4 slices) was present in 19 patients (38%), while majority of the patients (n=31, 62%) had a medium to high number of HVS on FLAIR (present on >4 slices). On the other hand, marked hypointense vessels on SWI compared with vessels in the contralateral normal hemisphere were present in >4 slices in 34 patients (68%). There was a moderate, but significant, correlation between the number of slices with HVS on FLAIR and marked hypointense vessels on SWI (r=0.425, $P = 0.002$). Patients with M1 occlusion, rather than distal occlusion, were more likely to have prominent hypointense vessels on SWI or HVS on FLAIR ($P = 0.017$, for both). However, neither imaging parameters were significantly related to the admission NIHSS score or DWI lesion volume.

Table 2 summarizes the bivariate relationships between clinical and imaging variables and clinical outcome parameters. Patients with a history of hypertension, higher admission NIHSS score, and larger DWI lesion volume were more likely to have

Table 2. The effect of clinical and radiologic features of the study population on the discharge NIHSS score and mRS

	Discharge NIHSS score			Discharge mRS		
	0–12 (n=25)	13–38 (n=25)	*P*	0–2 (n=13)	3–6 (n=37)	*P*
Age (years), median (IQR)	65 (60–76)	74 (64–81)	0.125	70 (59–77)	72 (62–79)	0.370
Female, n (%)	11 (44)	14 (56)	0.396	6 (46)	19 (51)	0.747
Risk factors, n (%)						
Hypertension	17 (68)	24 (96)	0.023	8 (62)	33 (89)	0.040*
Diabetes	8 (32)	3 (12)	0.171	5 (38)	6 (16)	0.096
Coronary heart disease	9 (36)	12 (48)	0.390	3 (23)	18 (49)	0.191
Atrial fibrillation	12 (48)	11 (44)	0.777	7 (54)	16 (43)	0.509
Admission NIHSS score, median (IQR)	8 (4–14)	19 (17–21)	<0.001	4 (2–7)	17 (14–20)	<0.001*
CCS subtype, n (%)						
Large artery atherosclerosis	3 (12)	5 (20)		1 (8)	7 (19)	
Cardioaortic embolism	15 (60)	14 (56)		8 (62)	21 (57)	
Small artery occlusion	0 (0)	0 (0)		0 (0)	0 (0)	
Other causes	3 (12)	1 (4)		1 (8)	3 (8)	
Undetermined/unclassified	4 (16)	5 (20)	0.649	3 (23)	6 (16)	0.789
Thrombolytic therapy, n (%)	5 (20)	5 (20)	1.000	3 (23)	7 (19)	0.707
Time to MRI (h), median (IQR)	12 (9–16)	10 (8–16)	0.554	11 (8–13)	12 (9–18)	0.293
Site of occlusion, n (%)						
M1	13 (52)	23 (92)		7 (54)	29 (78)	
M2	12 (48)	2 (8)	0.004	6 (46)	8 (22)	0.090
Admission DWI lesion volume (mL), median (IQR)	21 (4–70)	73 (18–203)	0.009	9 (2–25)	73 (19–136)	<0.001*
Prominent HVS on FLAIR, n (%)	13 (52)	18 (72)	0.145	8 (62)	23 (62)	0.968
Prominent hypointense vessel on SWI, n (%)	12 (48)	22 (88)	0.005	5 (38)	29 (78)	0.008*

NIHSS, National Institutes of Health Stroke Scale; mRS, modified Rankin Scale; IQR, interquartile range; CCS, Causative Classification System; MRI, magnetic resonance imaging; DWI, diffusion-weighted imaging; HVS, hyperintense vessel sign; FLAIR, fluid-attenuated inversion recovery; SWI, susceptibility-weighted imaging.
*$P < 0.05$.

an unfavorable clinical prognosis at the time of discharge, regardless of the clinical parameter used, discharge NIHSS score, or mRS. In addition, a more proximal site of occlusion (i.e., M1 compared to M2) was associated with a higher discharge NIHSS score and mRS, but the statistical significance was borderline for the latter. When the subset of 34 patients with available follow-up imaging was analyzed, age, female gender, smaller admission DWI lesion volumes, and lesser time between admission and follow-up imaging were found to be significantly associated with ≥20% lesion growth (Table 3). Patients with prominent hypointense vessels on SWI were more likely to have unfavorable prognosis per discharge NIHSS score ($P = 0.005$) and mRS ($P = 0.008$). On the other hand, there was a trend for patients with a prominent HVS on FLAIR to more likely experience lesion growth per bivariate analyses ($P = 0.071$).

In multivariate analyses, the prominence of hypointense vessels on SWI, but not hyperintense vessels on FLAIR, was significantly associated with a higher discharge NIHSS score (odds ratio [OR], 10.84; 95% confidence interval [CI], 1.31–89.56; $P = 0.027$) and mRS (OR, 231.84; 95% CI, 2.28–23620.53; $P = 0.021$) (Table 4). Other significant predictors of unfavorable clinical outcome were admission NIHSS score (both for the NIHSS score and the mRS model) and DWI lesion volume (only in the mRS model). The multivariate model was exploratory for lesion growth due to the low number of patients and included only imaging parameters as independent variables; in this model, the presence of prominent hypointense vessels on SWI (OR, 6.51; 95% CI, 1.00–42.78; $P = 0.050$) together with the admission DWI lesion volume and shorter time from symptom onset to follow-up imaging were significantly associated with lesion growth (Figs. 1, 2).

Discussion

Our findings show that imaging features suggestive of compromised tissue perfusion on FLAIR and SWI are moderately correlated with each other and that prominent hypointense vessels on SWI, compared with hyperintense vessels on FLAIR, have a more obvious prognostic role in terms of clinical and tissue outcomes. This suggests that these imaging features reflect different, but interrelated, aspects of cerebrovascular hemodynamics during cerebral ischemia.

The prognostic role of hyperintense vessels on FLAIR has been subject to a number of studies in the literature. The presence of HVS on FLAIR has consistently been shown to be associated with proximal arterial occlusions (5, 12, 13) and large volumes of perfusion deficit (5, 14–17), thereby pointing to a subgroup of patients with a substantial amount of cerebral tissue subjected to ischemia and under risk for neurologic deterioration and lesion progression. Therefore, studies representative of an overall heterogeneous stroke population highlight a positive association between HVS on FLAIR and admission stroke severity and adverse clinical outcome on follow-up (13, 18, 19). On the other hand, when similar analyses are performed in homogeneous cohorts of stroke patients, such as patients with an evidence of proximal occlusion or measurable perfusion deficit, the presence of HVS on FLAIR either has no influence on clinical prognosis (5, 14) or might even be associated with favorable clinical or tissue outcomes (11, 14, 20–22). A recent study in patients subjected to acute thrombolytic therapy found a correlation of HVS on FLAIR with a poor baseline DWI lesion size and perfusion, but no association with prognosis in the third month, in agreement with our findings. These observations in the literature suggest that the presence of HVS on FLAIR in proximal arterial occlusions is accepted as a sign of severe ischemia. However, its prognostic role is probably subject to modification by a number of factors, most importantly, the presence or absence of recanalization (14, 17, 23). The low rate of thrombolytic treatment in our cohort could have contributed to the neutral clinical results observed.

In patients with collateral circulation, apart from the status of recanalization, another factor implemental in terms of prognosis

Table 3. The effect of clinical and radiologic features of the study population on lesion growth

Lesion growth	<20% (n=17)	≥20% (n=17)	*P*
Age (years), median (IQR)	62 (58–73)	77 (70–82)	0.003*
Female, n (%)	5 (30)	12 (71)	0.016*
Risk factors, n (%)			
Hypertension	13 (77)	16 (94)	0.335
Diabetes	5 (29)	2 (12)	0.398
Coronary heart disease	6 (35)	10 (59)	0.169
Atrial fibrillation	7 (41)	9 (53)	0.492
Admission NIHSS score (median, IQR)	15 (8–18)	19 (15–21)	0.045*
CCS subtype, n (%)			
Large artery atherosclerosis	2 (12)	3 (18)	
Cardioaortic embolism	9 (53)	12 (71)	
Small artery occlusion	0 (0)	0 (0)	0.348
Other causes	2 (12)	0 (4)	
Undetermined/unclassified	4 (24)	2 (12)	
Thrombolytic therapy, n (%)	3 (18)	5 (29)	0.688
Time to MRI (h), median (IQR)	12 (9–15)	10 (7–19)	1.000
Site of occlusion, n (%)			
M1	12 (71)	14 (82)	
M2	5 (29)	3 (18)	0.688
Admission DWI lesion volume (mL), median (IQR)	93 (26–278)	53 (9–90)	0.049*
Prominent HVS on FLAIR, n (%)	8 (47)	14 (82)	0.071
Prominent hypointense vessel on SWI, n (%)	11 (65)	13 (77)	0.708
Type of follow-up imaging, n (%)			
CT	13 (77)	10 (59)	
MRI	4 (24)	7 (41)	0.465
Time to follow-up imaging (days), median (IQR)	27 (11–55)	8 (5–20)	0.014*
Follow-up lesion volume (mL), median (IQR)	66 (17–240)	119 (25–178)	0.892

IQR, interquartile range; NIHSS, National Institutes of Health Stroke Scale; CCS, Causative Classification System; MRI, magnetic resonance imaging; DWI, diffusion-weighted imaging; HVS, hyperintense vessel sign; FLAIR, fluid-attenuated inversion recovery; SWI, susceptibility-weighted imaging; CT, computed tomography.
*$P < 0.05$.

is the level of sufficiency of collaterals, or in other terms, the level of hemodynamic compromise, in the brain tissue. Marked hypointense vessels on gradient-recalled echo or SWI sequences are considered to reflect veins with increased deoxyhemoglobin concentration that drain the ischemic territory with increased oxygen extraction fraction. The extent of cerebral tissue with marked hypointense vessels has been shown to harbor a close correlation with the area of perfusion deficit (24, 25) and hence can be used as a marker of hemodynamic compromise within the territory of arterial occlusion. Few studies in literature have analyzed the relationship between outcome and the presence of marked hypointense vessels on SWI, with discrepant results. Some have shown a significant association of hypointense vessels on SWI with an unfavorable clinical outcome together with improved prognosis after the normalization of this finding with recanalization (25–27). On the other hand, these observations could not be replicated by Huang et al. (20), yet their analyses did not take into account the admission DWI lesion volume, which is one of the major predictors of clinical outcome. In the present study, we have shown that patients with prominent changes on SWI were more likely to have unfavorable clinical (by an odds of approximately 10 for moderate/severe disability per NIHSS score and approximately 230 per mRS) and tissue (by a factor of approximately 6 for significant lesion growth) outcomes, when adjusted for lesion volume at admission, severity of stroke, or extent of hyperintense vessels on FLAIR.

Some limitations of our study merit consideration. The inherent selection bias due to the retrospective design led to the exclusion of patients who had not undergone SWI and FLAIR imaging within 24 hours of symptom onset according to the discretion of the treating physician. The status of recanalization and reperfusion, which are major determinants of outcome, were not formally evaluated and were therefore not included in our analyses. As both these imaging features on FLAIR and SWI are reversible with the restoration of blood flow, their presence is not always associated with infarct growth and their prognostic influence relies heavily on the level of reperfusion. Another limitation was that the cohort included a heterogeneous set of patients with respect to thrombolytic treatment, with some receiving intravenous tissue plasminogen activator therapy and others not. Nonetheless, the proportion of patients with prominent HVS on FLAIR or hypointense vessels on SWI was not significant among the thrombolytic treatment and no thrombolytic treatment cohorts. In addition, follow-up imaging was performed at various time points, included both CT and MRI follow-ups, and was restricted only to a subgroup of patients; therefore, analyses regarding lesion growth were only exploratory. Future studies with a higher number of patients with complete imaging follow-up information and long-term clinical outcome data will help us further understand the prognostic role of hypointense vessels on SWI and hyperintense vessels on FLAIR in acute stroke. Finally, our grading system used for estimating the extent of HVS on FLAIR and hypointense vessels on SWI was based on an algorithm that only took into account the craniocaudal extent of pathologic vessels; more sophisticated methods integrating the anteroposterior extent and intensity changes in these vessels might increase the accuracy of information obtained from these imaging modalities.

In conclusion, our results should not be considered as an implication that SWI and FLAIR sequences provide information similar to perfusion-weighted imaging and could replace this imaging modality. On the contrary, we believe that perfusion-weighted imaging provides clinicians with highly critical information regarding the hemodynamics of cerebral ischemia and should be an integral part of state-of-the-art stroke imaging. However, in emergency departments where this option

Table 4. Multivariate predictors of clinical and imaging outcome

	Discharge NIHSS≥13		Discharge mRS≥3		Lesion growth ≥20%	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Prominent hypointense vessel on SWI	10.84 (1.31–89.56)	0.027*	231.84 (2.28–23620.53)	0.021*	6.51 (1.00–42.78)	0.050*
Admission NIHSS score	1.54 (1.19–1.98)	0.001*	1.76 (1.08–2.87)	0.023*		
Admission DWI lesion volume			1.09 (1.01–1.17)	0.031*		
Time elapsed until follow-up imaging					0.93 (0.88–0.99)	0.019*
Other variables introduced into the model	History of hypertension Admission DWI lesion volume M1 occlusion Prominent HVS on FLAIR		History of hypertension Prominent HVS on FLAIR		Prominent HVS on FLAIR Admission DWI lesion volume	
P of overall model	<0.001*		<0.001*		0.003*	

Categorical variables (prominent hypointense vessel on SWI, prominent HVS on FLAIR, history of hypertension, M1 occlusion) were introduced as dummy variables (1/0) into the logistic regression model.

NIHSS, National Institutes of Health Stroke Scale; mRS, modified Rankin Scale; OR, odds ratio; CI, confidence interval; SWI, susceptibility-weighted imaging; DWI, diffusion-weighted imaging; HVS, hyperintense vessel sign; FLAIR, fluid-attenuated inversion recovery.

*$P < 0.05$.

is not available round the clock, alternative imaging tools such as SWI and FLAIR might guide clinicians in prognostic and therapeutic decision-making. Although both imaging modalities are closely linked to tissue hypoperfusion, the presence of only a moderate correlation among them and their discrepant influences on clinical and tissue outcomes when concurrently studied in the same patient population suggest that there are certain inherent differences with respect to the information they provide. These observations might be a reflection of the fact that HVS on FLAIR denotes the presence of leptomeningeal collateral circulation, while hypointense vessels on SWI signify the sufficiency of cerebral blood flow at the tissue level and are therefore more critical in terms of prognosis. Further information on the imaging correlates of these signs could be obtained from similar but more comprehensive stroke imaging studies including perfusion studies.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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