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ABSTRACT

BACKGROUND AND PURPOSE: Perfusion-based collateral indices such as the perfusion collateral index and the hypoperfusion intensity ratio have shown promise in the assessment of collaterals in patients with acute ischemic stroke. We aimed to compare the diagnostic performance of the perfusion collateral index and the hypoperfusion intensity ratio in collateral assessment compared with angiographic collaterals and outcome measures, including final infarct volume, infarct growth, and functional independence.

MATERIALS AND METHODS: Consecutive patients with acute ischemic stroke with anterior circulation proximal arterial occlusion who underwent endovascular thrombectomy and had pre- and posttreatment MRI were included. Using pretreatment MR perfusion, we calculated the perfusion collateral index and the hypoperfusion intensity ratio for each patient. The angiographic collaterals obtained from DSA were dichotomized to sufficient (American Society of Interventional and Therapeutic Neuroradiology [ASITN] scale 3–4) versus insufficient (ASITN scale 0–2). The association of collateral status determined by the perfusion collateral index and the hypoperfusion intensity ratio was assessed against angiographic collaterals and outcome measures.

RESULTS: A total of 98 patients met the inclusion criteria. Perfusion collateral index values were significantly higher in patients with sufficient angiographic collaterals ($P < .001$), while there was no significant ($P = .46$) difference in hypoperfusion intensity ratio values. Among patients with good (mRS 0–2) versus poor (mRS 3–6) functional outcome, the perfusion collateral index of ≥ 62 was present in 72% versus 31% ($P = .003$), while the hypoperfusion intensity ratio of ≤ 0.4 was present in 69% versus 56% ($P = .52$). The perfusion collateral index and the hypoperfusion intensity ratio were both significantly predictive of final infarct volume, but only the perfusion collateral index was significantly ($P = .03$) associated with infarct growth.

CONCLUSIONS: Results show that the perfusion collateral index outperforms the hypoperfusion intensity ratio in the assessment of collateral status, infarct growth, and determination of functional outcomes.

ABBREVIATIONS: AIS = acute ischemic stroke; ASITN = American Society of Interventional and Therapeutic Neuroradiology; AUC = area under the curve; HIR = hypoperfusion intensity ratio; IQR = interquartile range; mTICI = modified TICI; PCI = perfusion collateral index; ROC = receiver operating characteristic; Tmax = time-to-maximum

In patients with acute ischemic stroke (AIS), collateral status is considered a critical variable in determination of infarction growth and the success of thrombectomy.[1-4] Determining collateral status has the potential to extend endovascular treatment beyond the current timeline of 24 hours.[5] The latest American Stroke Association guidelines state that collateral status may help to determine endovascular treatment eligibility in some candidates,[6] though no specific recommendations about the methodology to measure collaterals has been proposed.

DSA is the standard of reference for assessment of collaterals due to high spatial and temporal resolution. Noninvasive assessment of collaterals has been increasingly improving, though it remains a moving target with no concrete recommendations. Direct visualization of collateral vessels can be performed by CTA or MRA by taking into account the number, size, and density of these vessels,[7-10] with incremental added value of multiphasic-over-single arterial phase imaging.[11-13]

By means of perfusion imaging, a multitude of methods for automated assessment of collateral status have been proposed,

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including the hypoperfusion intensity ratio (HIR) and the perfusion collateral index (PCI).[14-16] The HIR was first investigated in the Diffusion-Weighted Imaging Evaluation for Understanding Stroke Evolution Study-2 (DEFUSE-2) cohort and is calculated by dividing the volume of tissue with a time-to-maximum (Tmax) delay of >10 seconds by the volume of tissue with Tmax of >6 seconds. The HIR has been used as a measure of collaterals, and HIR values > 0.4 have been associated with poor collaterals, infarction growth, and poor functional outcome.[14,17]

The PCI uses a multiparametric approach and is defined as the volume of moderately hypoperfused tissue (delay of 2–6 seconds) multiplied by its corresponding mean relative CBV.[15] Similar to the HIR, the PCI has been shown promising in the determination of collateral status. PCI values of >60 have been associated with good collaterals and outcome measures such as final infarct volume and functional outcomes.[15,16] The PCI was previously shown to have a diagnostic accuracy of 94% compared with DSA in the prediction of collateral status.[15]

The diagnostic accuracy of the HIR or PCI for the determination of collateral status remains to be investigated against each other in a single study. The central premise of this exploratory study was to evaluate the diagnostic performance of the HIR and PCI in the assessment of collateral status. The aims of this study were the following: 1) to assess the agreement of collateral status obtained from the HIR and PCI against DSA as the standard of reference; and 2) to perform a comparative analysis between the HIR and PCI in association with outcome measures, including final infarct volume, infarct growth, and functional outcome in patients with AIS.

MATERIALS AND METHODS

Patients

This retrospective study was approved by University of California Los Angeles (UCLA) institutional review board, and informed consent was waived. Consecutive patients with AIS were identified between January 1, 2010, and August 31, 2019, and they were included if they met the following inclusion criteria: 1) anterior circulation proximal arterial occlusion including the intracranial ICA or proximal MCA (M1), 2) pretreatment MRI with inclusion of MR perfusion, 3) DSA and endovascular treatment, and 4) the presence of follow-up MRI for the determination of final infarct volume.

Clinical data, including patient age, sex, time from stroke symptoms, NIHSS score, time of initial (pretreatment) imaging, and site of large-vessel occlusion, were collected. In addition, treatment type, including intravenous tPA, endovascular therapy, degree of reperfusion using the modified TICI (mTICI) scale,[18] and the mRS at 90 days were recorded when available.

Image Analysis

Collateral Assessment on DSA. An interventional neuroradiologist with 8 years of postfellowship experience graded the collaterals using the American Society of Interventional and Therapeutic Neuroradiology (ASITN)/Society of Interventional Radiology collateral flow grading system (grades 0–4)[19] on baseline pretreatment DSA images. The interventional neuroradiologist was blinded to the clinical information and MRI findings. Patients were dichotomized to those with sufficient collaterals (grades 3 and 4) and insufficient collaterals (grades 0, 1, and 2). Patients were excluded if the baseline DSA was deemed inadequate to provide collateral assessment (ie, lack of an adequate number of phases, injections, or coverage).

MR Imaging and Analysis

DWI was acquired using a single-shot spin-echo EPI sequence (TR/TE, 4300/78 ms; flip angle, 90°; FOV, 22 × 22 cm; matrix, 128 mm^2; 26 slices × 5 mm). Diffusion gradients were applied along 6 noncollinear directions with b-values of 0 and 1000 s/mm^2.

DSC perfusion was performed using a single-shot gradient-echo EPI sequence (TR/TE, 1450/30 ms; flip angle, 90°; FOV, 22 × 22 cm; matrix, 128 mm^2; 24 slices × 5 mm). Sixty dynamic frames were obtained during a 90-second acquisition time.

From the perfusion data, the HIR (*Volume of Tmax >10 Seconds/Volume of Tmax >6 Seconds*) was calculated using RAPID software (Version 5.0.4; iSchemaView). Subsequently, MR perfusion data were processed using Olea Sphere (SP.23; Olea Medical) by applying a Bayesian probabilistic method.[20] The PCI ($Volume\ of\ Delay^{2\text{-}6\ seconds} \times Relative\ CBV$) was calculated for each patient.

The baseline infarct volume was calculated automatically using ADC $<600 \times 10^{-6}$ mm2/s from the pretreatment MRI. Final infarct volume was calculated from the follow-up MRI (obtained within 1–2 days from the pretreatment MRI). Due to the increase in ADC values following reperfusion,[21] final infarction volumes were segmented using hyperintensity on DWI (b=1000) by 1 neuroradiologist with >10 years of experience. The difference in infarction volumes between the second and first MRI was recorded as infarct growth.

Statistical Analysis

Demographic characteristics and neuroimaging variables were presented as mean (SD) for continuous data and as median and interquartile range for categoric data. Statistical tests were performed using the Fisher t test or Wilcoxon rank-sum test as appropriate.

Receiver operating characteristic (ROC) curve analysis was performed, and the area under the curve (AUC) was calculated for the prediction of collateral status for both the HIR and PCI. The optimal cutoff point to identify collateral status was determined by the Youden index. Summary measures such as sensitivity and specificity were calculated on the basis of the optimal threshold to determine collateral status (sufficient-versus-insufficient). Furthermore, to test the effect of collaterals on measured outcomes with continuous values, including final infarct volume and infarct growth, a Mood median test was used. A χ^2 test was performed to compare the proportion of patients with good functional outcomes (mRS 0–2) among patients with sufficient and insufficient collaterals. All tests were 2-tailed and assumed significance at $P < .05$.

RESULTS

Among 141 patients initially evaluated, a total of 98 met the study entry criteria. Fifteen patients were excluded due to nondiagnostic MR imaging (eg, severe motion artifacts, susceptibility distortion) and 28 patients due to baseline DSA deemed inadequate to provide collateral assessment (lack of an adequate number of phases and injections or coverage). Among the 98 patients included, 44.8% were women, mean age = 70.4 (SD, 13.6) years,

Baseline and clinical data in patients with insufficient-versus-sufficient collateral flow

Variable	Overall (n = 98)	Sufficient Collaterals (n = 43)	Insufficient Collaterals (n = 55)	P Value
Age (mean) (SD)	70.4 (13.6)	69.6 (12.6)	71.2 (14.3)	.58
Sex, female (No.) (%)	44 (44.8%)	24 (55.8%)	20 (36.3%)	.55
Baseline NIHSS (median) (IQR)	15 (9–19)	14 (9–19)	15 (9–20)	.55
IV tPA (No.) (%)	10 (10%)	2 (5%)	8 (15%)	.11
Time from stroke onset (median) (IQR) (hr)	2.48 (1.27–4.98)	2.00 (1.38–4.57)	2.50 (1.24–5.31)	.90
Location of LVO (ICA/MCA) (No.)/(No.)	14/84	4/39	10/45	.80
Successful reperfusion (mTICI 2b–3) (No.) (%)	79 (80.6%)	38 (88.4%)	41 (74.5%)	.86

Note:—LVO indicates large-vessel occlusion; IQR, interquartile range.

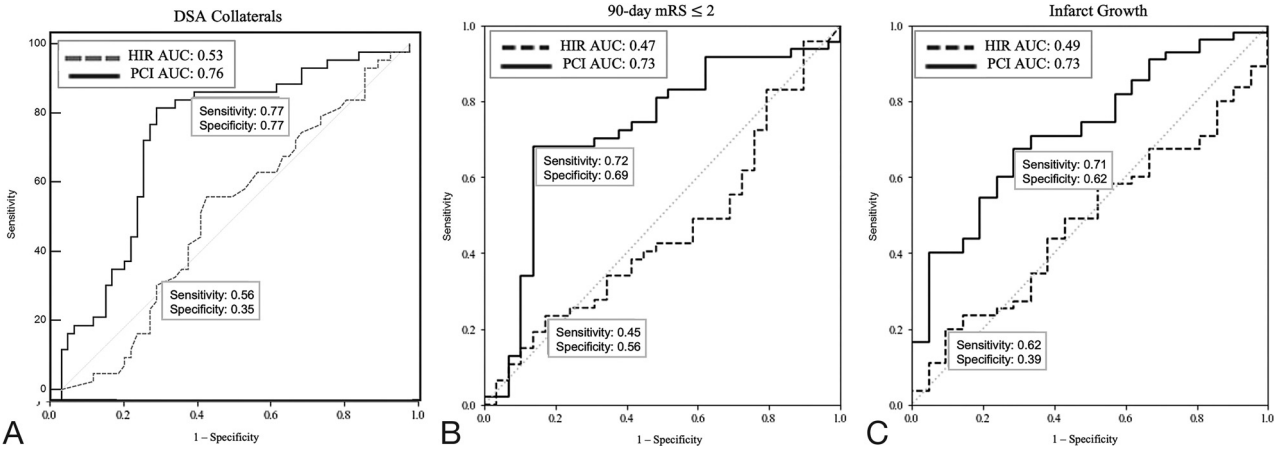

FIG 1. ROC curve analysis of the PCI and HIR in the prediction of DSA-based angiographic collaterals (*A*). In addition, ROC curves for PCI and HIR in the prediction of functional outcome (*B*) and infarct growth (*C*) are shown.

with NIHSS median = 15 (IQR, 9–19). Successful reperfusion (mTICI 2b–3) following mechanical thrombectomy was achieved in 79 patients (80.6%).

Collateral Assessment

Using pretreatment DSA as the standard of reference, 55 patients (56%) had insufficient collaterals (ASITN < 3), and 43 patients (44%) had sufficient collaterals (ASITN ≥ 3). The baseline demographic and clinical data for the entire cohort and among patients with sufficient-versus-insufficient angiographic collaterals are summarized in the Table.

The HIR values were not significantly different between patients with sufficient-versus-insufficient collaterals, mean = 0.47 (SD, 0.44) versus 0.41 (SD, 0.21), $P = 0.46$. The mean PCI values were significantly higher in patients with sufficient collaterals versus insufficient collaterals, 106.1 (SD, 56.6) versus 58.3 (SD, 40.4), $P < .001$. ROC analysis for the HIR in determination of DSA-based collaterals resulted in AUC/sensitivity/specificity of 0.53/0.56/0.35 ($P = .86$) at a 0.4 cutoff. ROC analysis for the PCI in determination of DSA-based collaterals showed an AUC/sensitivity/specificity of 0.76/0.77/0.77 ($P < .001$) at a cutoff of 62. The ROC curves for the HIR and PCI are shown in Fig 1.

Outcome Assessment

The 90-day mRS was used as the primary outcome measure to determine functional independence. Among 77 patients with 90-day mRS values available, 29 patients (38%) had good functional outcomes (mRS 0–2).

Comparative analysis of patients with good-versus-poor functional outcome showed a significantly younger age in patients with good outcome (mean = 63.9 [SD, 15.6] years versus 74.5 [SD, 11.1] years, $P = .001$); significantly higher PCI values in patients with good outcome (mean = 106.3 [SD, 56.4] versus 68.8 [SD, 53.4], $P = .005$); significantly smaller final infarct volumes in patients with good outcome (mean = 20.4 [SD, 20.1] mL versus 48.5 [SD, 43.1] mL, $P = .002$); and significantly smaller mean values of infarct growth in patients with good outcomes (6.25 [SD, 8.7] mL versus 20.3 [SD, 28.9] mL, $P = .001$). There was no significant difference between HIR values in patients with good-versus-poor outcome (mean = 0.45 [SD, 0.10] versus 0.49 [SD, 0.43], $P = .70$).

Figure 1 shows the comparative ROC analysis between the PCI and HIR in relation to outcome measures, including 90-day mRS and infarct growth.

The diagnostic performance of collateral assessment on each method using dichotomized scores (ie, ASITN ≥ 3 on DSA, PCI ≥ 62, and HIR ≤ 0.4) are summarized in the Online Supplemental Data. Patients with good collaterals had statistically significantly smaller infarction volumes. Patients with PCI ≥ 62 had statistically significant lower infarct growth than patients with PCI < 62. PCI ≥ 62 was associated with better functional outcomes with an OR of 2.83 (Online Supplemental Data).

In 2 patients with good angiographic collaterals, an example of concordant PCI and HIR is shown in Fig 2, while Fig 3 demonstrates an example of discordant PCI and HIR. Figure 4 demonstrates an example of poor angiographic collaterals with a poor PCI and a discordant HIR.

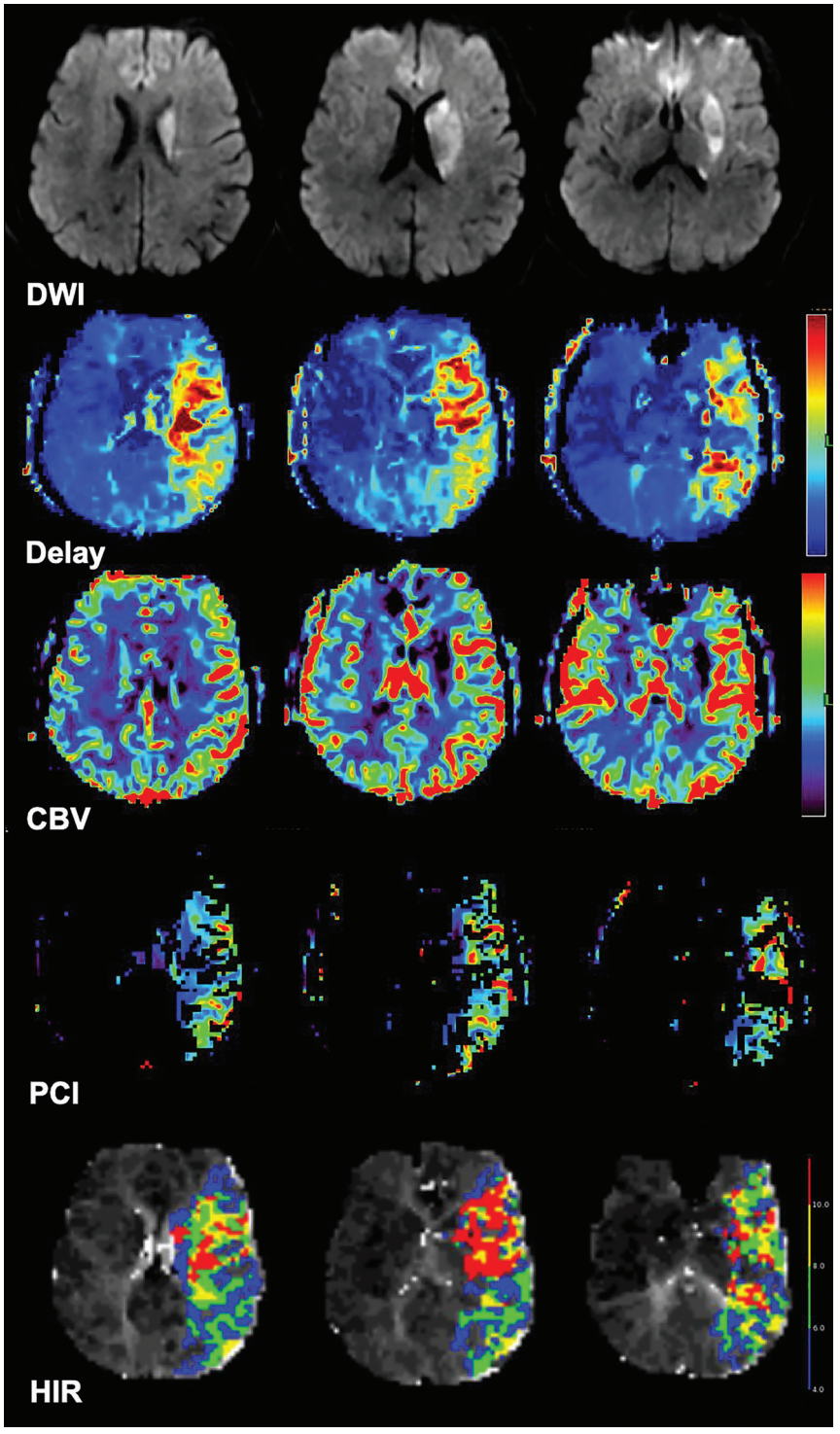

FIG 2. Example of concordance between the PCI and HIR. Adult patient with right hemiparesis who underwent MR imaging approximately 10 hours after symptom onset. The patient has a left M1 occlusion (not shown). Aligned axial DWI, delay, CBV, PCI, and HIR maps from MR perfusion are shown. There is an ischemic core involving the left basal ganglia. The PCI value is estimated at 132, suggestive of sufficient collaterals. The estimated HIR is 0.36, also suggesting sufficient collaterals. Baseline catheter angiography (not shown) revealed good collaterals (ASITN 3).

DISCUSSION

This study, to our knowledge, is the first to compare the diagnostic performance of HIR and PCI in predicting collateral status defined by DSA as the standard of reference. Results showed superior performance of PCI over HIR in assessment of collateral status. We specifically highlight the following 2 findings:

First, the PCI provides a more accurate representation of true collaterals as measured by DSA compared with the HIR. At the threshold of 62, similar to what was previously reported,[15] the PCI provided a sensitivity and specificity of 77% and 77% in predicting sufficient angiographic collaterals. On the other hand, in our study, the HIR was not a significant predictor of DSA collaterals (AUC = 0.53, $P = .86$).

The PCI was developed on the basis of and in relation to DSA collaterals from its introduction.[15] This feature may explain the higher performance and correlation to DSA over HIR, which was developed initially as a surrogate of collateral status based on defining infarction core and growth rather than a direct correlation to DSA.[14,22] Recent studies have shown good correlation between HIR and CTA collaterals, including the work of Lyndon et al,[23] which showed an AUC of 0.86 for detection of good CTA collaterals using a CTP-estimated HIR $< .45$, and the work of Wang et al[24] with an AUC of 0.82 using an HIR $< .68$.

It was not until recently that the HIR relationship with DSA was shown in a study by Guenego et al.[17] In this study, HIR values < 0.4 were associated with good angiographic collaterals with a specificity of 56% (compared with 35% in our study) and a sensitivity of 79% (compared with 56% in our study). Although the exact reason for the more modest results of the HIR in our study is unclear, our study included both ICA and M1 occlusions, while Guenego et al focused on patients with only M1 occlusions. It is plausible that inclusion of patients with ICA occlusion could have affected the sensitivity of the HIR in our study due to more proximal occlusion and its effect on upstream flow.

The second finding is that while both the PCI and HIR were significant in predicting the final infarction volume, only the PCI was predictive of infarct growth and functional independence. Collateral information in the HIR is obtained using Tmax only. The focus, therefore, is on arterial delay and the severity of hypoperfusion. Tissue with severe hypoperfusion

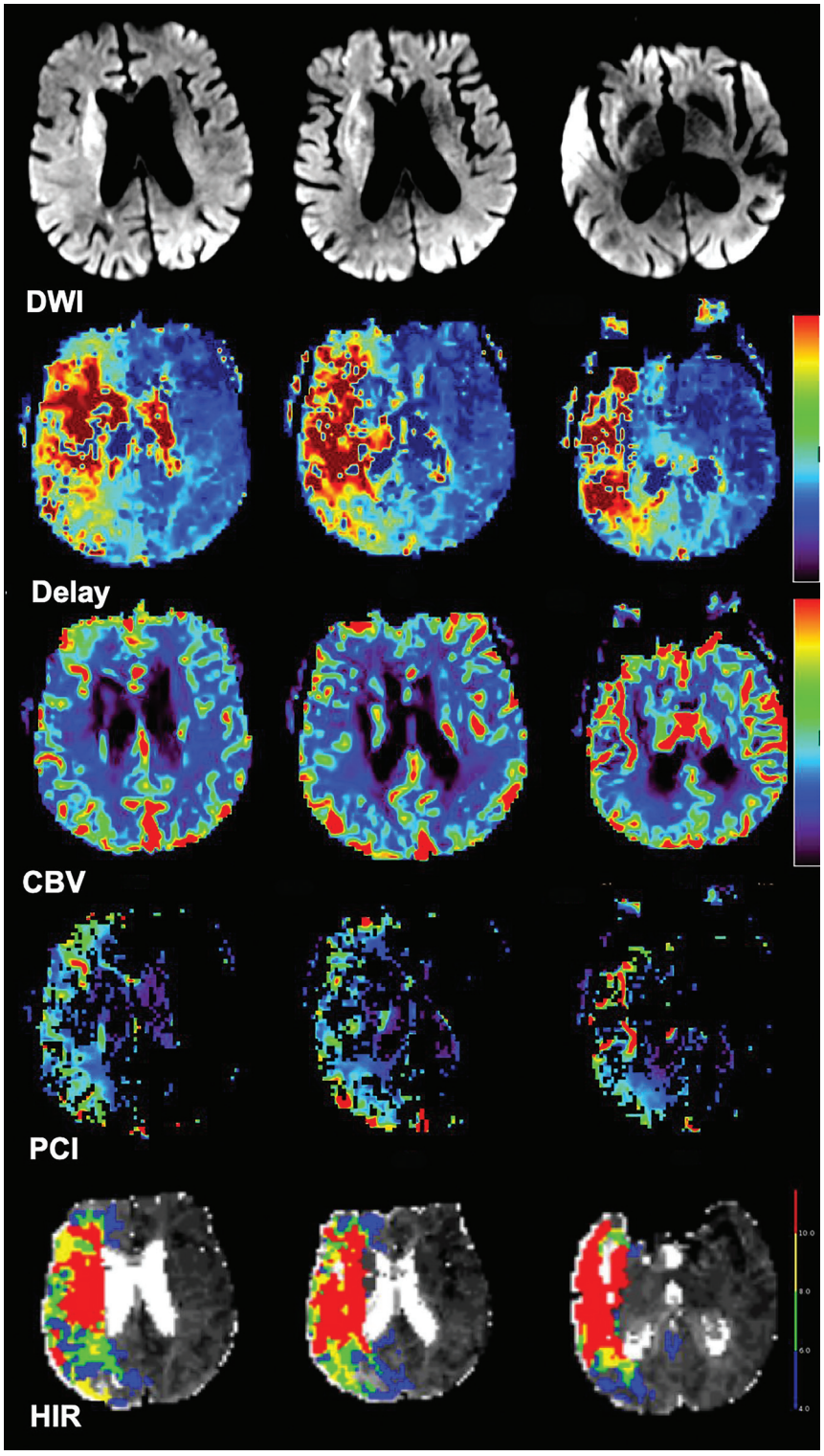

FIG 3. Example of discordance between the PCI and HIR in a patient with sufficient angiographic collaterals. Adult patient with left hemiparesis who underwent MR imaging approximately 4.5 hours after symptom onset. The patient has a right carotid terminus occlusion (not shown). Aligned axial DWI, delay, CBV, PCI, and HIR maps from MR perfusion are shown. There is an ischemic core involving the right basal ganglia, insula, and right temporal lobe. The PCI value is estimated at 130, suggestive of sufficient collaterals. The estimated HIR is 0.49, suggestive of poor collaterals. Baseline catheter angiography (not shown) revealed good collaterals (ASITN 3).

measured by Tmax of >10 seconds is more likely to undergo infarction; hence, the HIR is a significant predictor of final infarct volume as shown by our study and other previous investigations.

However, in our study, the HIR was not a significant predictor of infarct growth or functional outcome, contrary to initial studies of the HIR.[14,22] In a follow-up study by Arenillas et al,[25] similar to our study, the HIR was not associated with infarct growth. Instead, the authors showed an association between infarct growth and the relative CBV index defined as the mean of normalized CBV values within the volume of tissue with a perfusion delay of Tmax > 6 seconds.[25,26] This association is similar to what is incorporated into the PCI.

Evaluation of collaterals using perfusion imaging should consider both delay and dispersion.[27] By means of Tmax in the HIR and delay in the PCI, both measures likely evaluate the early arterial phase of collaterals, representing the delay component. The addition of CBV in the PCI, however, provides information regarding the amount of blood flow beyond the point of arterial occlusion (via dispersion), which is not evaluated in the HIR. Therefore, the PCI provides a more comprehensive assessment of collateral phases (both delay and dispersion components), similar to what has been shown by angiographic studies.[28] In our study, the PCI was able to predict infarct growth similar to DSA collaterals, in which combined arterial and venous phases of collaterals are considered,[29] reinforcing the importance of considering collaterals as a whole and not just focusing on the delay component.

The association between functional outcome and final infarct volume has been mixed in the literature.[30-32] Moreover, infarct growth rather than final infarct volume has been reported as a better predictor of functional outcomes.[33] In our study, the HIR was only associated with final infarct volume, while the PCI was a significant determinant of both infarct growth and functional outcome. Previous studies have shown that the extent of venous drainage can predict outcomes,[28] and again, it is plausible

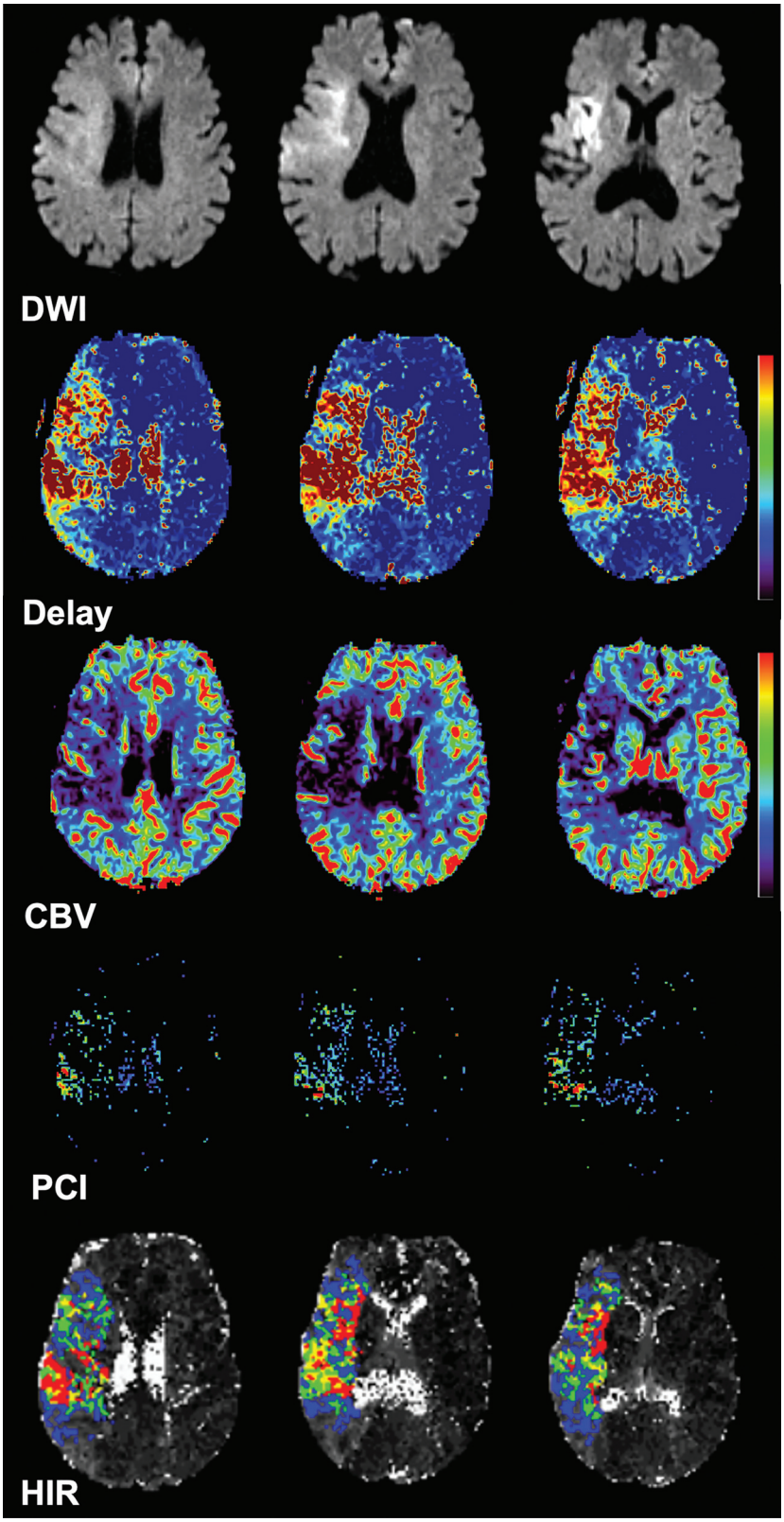

FIG 4. Example of discordance between the PCI and HIR in a patient with insufficient angiographic collaterals. Adult patient with left hemiparesis who underwent MR imaging approximately 1.3 hours after symptom onset. The patient has a right M1 occlusion (not shown). Aligned axial DWI, delay, CBV, PCI, and HIR maps from MR perfusion are shown. There is an ischemic core involving the right operculum, insula, and basal ganglia. There is low CBV in the region involved with PCI values estimated at 38, suggestive of insufficient collaterals. The estimated HIR is 0.30, suggestive of sufficient collaterals. Baseline catheter angiography (not shown) revealed poor collaterals (ASITN 1).

that when one incorporates relative CBV to evaluate late and venous phases of collaterals, the PCI captures some of this prognostic information that the Tmax alone does not provide. In our study, DSA-based collaterals were not a significant determinant of functional outcome, similar to the results of prior studies.[34,35]

Limitations

Our study has several limitations. First, the retrospective nature introduces unknown biases. Despite looking into 8 years of data, the sample size remains relatively small due to strict inclusion criteria from a single institution. DSA collaterals were scored by 1 observer only, which precludes assessment of interobserver agreement. Another limitation is that the diagnostic performance of the PCI in determination of angiographic collaterals was lower in comparison with a prior report (accuracy of 76% versus 94% in the previous study).[15] This result may be due to inclusion of a larger data set in the present study and further supports the need for a more comprehensive and multicenter study to further validate and establish the PCI thresholds for broader clinical use.

CONCLUSIONS

Results showed that the PCI outperforms the HIR in predicting of angiographic collaterals using DSA as the reference standard. In addition, while both the PCI and HIR are significant determinants of final infarct volume, only the PCI was significantly associated with infarct growth and functional independence. If its potential is realized in a larger study, the PCI, a quantitative index of collaterals derived from routinely performed perfusion imaging in patients with stroke, can be added as an extra imaging variable to provide a measure of baseline collateral status in patients with AIS. This information can be used for improved prognostication or treatment decision-making.

Disclosure forms provided by the authors are available with the full text and PDF of this article at www.ajnr.org.

REFERENCES

1. Sheth SA, Liebeskind DS. **Collaterals in endovascular therapy for stroke.** *Curr Opin Neurol* 2015;28:10–15 CrossRef Medline
2. Liebeskind DS. **Collateral circulation.** *Stroke* 2003;34:2279–84 CrossRef Medline
3. Christoforidis GA, Mohammad Y, Kehagias D, et al. **Angiographic assessment of pial collaterals as a prognostic indicator following intra-arterial thrombolysis for acute ischemic stroke.** *AJNR Am J Neuroradiol* 2005;26:1789–97 Medline
4. Menon BK, Smith EE, Modi J, et al. **Regional leptomeningeal score on CT angiography predicts clinical and imaging outcomes in patients with acute anterior circulation occlusions.** *AJNR Am J Neuroradiol* 2011;32:1640–45 CrossRef Medline
5. Jiang B, Ball RL, Michel P, et al. **Factors influencing infarct growth including collateral status assessed using computed tomography in acute stroke patients with large artery occlusion.** *Int J Stroke* 2019;14:603–12 CrossRef Medline
6. Powers WJ, Rabinstein AA, Ackerson T, et al. **Guidelines for the early management of patients with acute ischemic stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke—a Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association**. *Stroke* 2019;50:e344–418 CrossRef Medline
7. Menon BK, O'Brien B, Bivard A, et al. **Assessment of leptomeningeal collaterals using dynamic CT angiography in patients with acute ischemic stroke**. *J Cereb Blood Flow Metab* .2013;33:365–71 CrossRef Medline
8. Lima FO, Furie KL, Silva GS, et al. **The pattern of leptomeningeal collaterals on CT angiography is a strong predictor of long-term functional outcome in stroke patients with large-vessel intracranial occlusion.** *Stroke* 2010;41:23160–22 CrossRef Medline
9. Hernández-Pérez M, Puig J, Blasco G, et al. **Dynamic magnetic resonance angiography provides collateral circulation and hemodynamic information in acute ischemic stroke**. *Stroke* 2016;47:531–34 CrossRef Medline
10. Ernst M, Forkert ND, Brehmer L, et al. **Prediction of infarction and reperfusion in stroke by flow- and volume-weighted collateral signal in MR angiography.** *AJNR Am J Neuroradiol* 2015;36:275–82 CrossRef Medline
11. Menon BK, d'Esterre CD, Qazi EM, et al. **Multiphase CT angiography: a new tool for the imaging triage of patients with acute ischemic stroke**. *Radiology* 2015;275:510–20 CrossRef Medline
12. Tsui B, Nour M, Chen I, et al. **MR angiography in assessment of collaterals in patients with acute ischemic stroke: a comparative analysis with digital subtraction angiography.** *Brain Sci* 2022;12:1181 CrossRef Medline
13. Kim HJ, Lee SB, Choi JW, et al. **Multiphase MR angiography collateral map: functional outcome after acute anterior circulation ischemic stroke.** *Radiology* 2020;295:192–201 CrossRef Medline
14. Olivot JM, Mlynash M, Inoue M, et al. **Hypoperfusion intensity ratio predicts infarct progression and functional outcome in the DEFUSE 2 cohort.** *Stroke* 2014;45:1018–23 CrossRef Medline
15. Nael K, Doshi A, De Leacy R, et al. **MR perfusion to determine the status of collaterals in patients with acute ischemic stroke: a look beyond time maps.** *AJNR Am J Neuroradiol* 2018;39:219–25 CrossRef Medline
16. Nael K, Sakai Y, Larson J, et al. **CT perfusion collateral index in assessment of collaterals in acute ischemic stroke with delayed presentation: comparison to single phase CTA.** *J Neuroradiol* 2022;49:1988–204 CrossRef Medline
17. Guenego A, Fahed R, Albers GW, et al. **Hypoperfusion intensity ratio correlates with angiographic collaterals in acute ischaemic stroke with M1 occlusion.** *Eur J Neurol* 2020;27:864–70 CrossRef Medline
18. Tomsick T, Broderick J, Carrozella J, et al. **Revascularization results in the Interventional Management of Stroke II trial.** *AJNR Am J Neuroradiol*. Mar 2008;29:582–87 CrossRef Medline
19. Higashida RT, Furlan AJ, Roberts H, et al. **Trial design and reporting standards for intra-arterial cerebral thrombolysis for acute ischemic stroke.** *Stroke* 2003;34:e109–37 CrossRef Medline
20. Boutelier T, Kudo K, Pautot F, et al. **Bayesian hemodynamic parameter estimation by bolus tracking perfusion weighted imaging.** *IEEE Trans Med Imaging* 2012;31:1381–95 CrossRef Medline
21. An H, Ford AL, Vo K, et al. **Signal evolution and infarction risk for apparent diffusion coefficient lesions in acute ischemic stroke are both time- and perfusion-dependent.** *Stroke* 2011;42:1276–81 CrossRef Medline
22. Guenego A, Mlynash M, Christensen S, et al. **Hypoperfusion ratio predicts infarct growth during transfer for thrombectomy.** *Ann Neurol* 2018;84:616–20 CrossRef Medline
23. Lyndon D, van den Broek M, Niu B, et al. **Hypoperfusion index ratio correlates as a surrogate of collateral scoring on CT angiogram in large vessel stroke.** *AJNR Am J Neuroradiol* 2021;42:1380–86 CrossRef Medline
24. Wang CM, Chang YM, Sung PS, et al. **Hypoperfusion index ratio as a surrogate of collateral scoring on CT angiogram in large vessel stroke.** *J Clin Med* 2021;10:1296 CrossRef Medline
25. Arenillas JF, Cortijo E, García-Bermejo P, et al. **Relative cerebral blood volume is associated with collateral status and infarct growth in stroke patients in SWIFT PRIME**. *J Cereb Blood Flow Metab* 2018;38:1839–47 CrossRef Medline
26. Mlynash M, Lansberg MG, Kemp S, et al. **Combination of Tmax and relative CBV perfusion parameters more accurately predicts CTA collaterals than a single perfusion parameter in DEFUSE 3.** *Stroke* 2019;50(Suppl) CrossRef
27. Jerosch-Herold M, Hu X, Murthy NS, et al. **Time delay for arrival of MR contrast agent in collateral-dependent myocardium.** *IEEE Trans Med Imaging* 2004;23:881–90 CrossRef Medline
28. Parthasarathy R, Kate M, Rempel JL, et al. **Prognostic evaluation based on cortical vein score difference in stroke.** *Stroke* 2013;44:2748–54 CrossRef Medline
29. Parthasarathy R, Sohn SI, Jeerakathil T, et al. **A combined arterial and venous grading scale to predict outcome in anterior circulation ischemic stroke.** *J Neuroimaging* 2015;25:969–77 CrossRef Medline
30. Wardlaw JM, Keir SL, Bastin ME, et al. **Is diffusion imaging appearance an independent predictor of outcome after ischemic stroke?** *Neurology* 2002;59:1381–87 CrossRef Medline
31. Saver JL, Johnston KC, Homer D, et al. **Infarct volume as a surrogate or auxiliary outcome measure in ischemic stroke clinical trial: the RANTTAS Investigators.** *Stroke* 1999;30:293–98 CrossRef Medline
32. Ganesh A, Ospel JM, Menon BK, et al. **Assessment of discrepancies between follow-up infarct volume and 90-day outcomes among patients with ischemic stroke who received endovascular therapy.** *JAMA Netw Open* 2021;4:e2132376 CrossRef Medline
33. Deng W, Teng J, Liebeskind D, et al. **Predictors of infarct growth measured by apparent diffusion coefficient quantification in patients with acute ischemic stroke.** *World Neurosurg* 2019;123:e797–802 CrossRef Medline
34. Kauw F, Dankbaar JW, Martin BW, et al. **Collateral status in ischemic stroke: a comparison of computed tomography angiography, computed tomography perfusion, and digital subtraction angiography.** *J Comput Assist Tomogr* 2020;44:984–92 CrossRef Medline
35. Jansen IG, Berkhemer OA, Yoo AJ, et al. **Comparison of CTA- and DSA-based collateral flow assessment in patients with anterior circulation stroke.** *AJNR Am J Neuroradiol* 2016;37:2037–42 CrossRef Medline