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Journal of Clinical Imaging Science

Original Research *Neuroradiology/Head and Neck Imaging*

Intraluminal arterial transit artifact as a predictor of intracranial large artery stenosis on 3D time of flight MR angiography: Expanding the application of arterial spin labeling MRI in ischemic stroke

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Received : 12 March 2023
Accepted : 24 May 2023
Published : 17 June 2023

DOI
10.25259/JCIS_27_2023

Quick Response Code:

ABSTRACT

Objectives: The objective of this study was to evaluate the diagnostic value of "intraluminal arterial transit artifact" in the prediction of intracranial large artery stenosis and to determine if this finding is predictive of ischemic stroke in the territory of the involved artery.

Material and Methods: The presence of arterial transit artifact (ATA) within the lumen of an intracranial large vessel was noted on three-dimensional time of flight (3D-TOF) magnetic resonance angiography (MRA) (ATA group). The patients with stenosis but with no ATA (no-ATA group), patients with total occlusion (total occlusion group), and patients with no stenosis/occlusion (normal group) were included in the analysis.

Results: There were four groups of patients included in the final analysis, the ATA group (n = 22), the no-ATA group (n = 23), the normal group (n = 25), and the total occlusion group (n = 9). Among patients with any demonstrable stenosis (n = 45), the presence of ATA within the stenotic segment was predictive of stenosis of ≥56% (Sensitivity of 100% [85.2–100, 95% CI], specificity of 100% [86.4–100, 95% CI]), with area under curve of 1.0 (0.92–.0, 95% CI). The presence of intra-arterial ATA signal was significantly associated with ischemic stroke as compared with the no-ATA group (86.36% vs. 26.08%, P = 0.0003). Intraluminal ATA was found to be an independent predictor of infarction in the territory of the involved artery.

Conclusion: Intraluminal ATA is predictive of stenosis of at least 56% in the involved artery on 3D-TOF MRA. Intraluminal ATA sign may be an independent predictor of infarction in the territory of the involved artery.

Keywords: Arterial transit artifact, Stroke, Stenosis, Infarct, Arterial spin labeling

INTRODUCTION

Stenosis of the intracranial arteries refers to a focal narrowing of the lumen, which may occur due to various etiologies. Intracranial atherosclerosis is a common cause of stroke among the Asian population with as high as 50% of cases of ischemic stroke attributable to intracranial atherosclerotic disease (ICAD).[1] However, not all cases of ICAD are symptomatic. Vasculitides, post-radiation arteriopathy, intracranial tumors (such as meningioma), angioinvasive infections (such as angioinvasive aspergillosis and angioinvasive mucormycosis), and intracranial dissection

are among the other causes of stenosis of the intracranial arteries.[2-6] Post-varicella infection arteriopathy, moyamoya disease, sickle cell disease, and neurofibromatosis type I feature among etiologies of intracranial stenosis in the pediatric age group.[7-10] Substance abuse, particularly Cannabis and Cocaine abuse, may also lead to multifocal intracranial stenosis.[11,12]

Intracranial stenosis may lead to ischemic stroke by various mechanisms. Initially, stenosis leads to compensatory vasodilatation in the distal vasculature which maintains the cerebral perfusion pressure.[13] As the severity of stenosis increases, the compensatory vasodilation reaches its limit, and the cerebrovascular reserve is exhausted.[13] With increasing severity and chronicity of stenosis, there is an opening of collateral arteries to augment the perfusion in the ischemic vascular bed.[13] However, collateral perfusion may fail in situations that challenge the cerebral blood flow (CBF) (such as hypotension) and this may lead to ischemia and subsequent infarction of the cerebral tissue within the territory of the ischemic vascular bed.[14] This is the hemodynamic mechanism of stroke in intracranial stenosis. Other mechanisms of stroke include the formation of a thrombus at the site of stenosis which may worsen the distal perfusion (which is already compromised) or may lead to thromboembolism in the distal territory. In cases of ICAD, there may be intraplaque hemorrhage and plaque rupture which may lead to atheroembolism, in addition to thromboembolism.[13,14]

Arterial spin labeling (ASL) is a technique of measuring CBF non-invasively using magnetic resonance imaging (MRI). During ASL acquisition, blood is tagged using an inversion pulse (in a continuous, pulsed, or pseudo-continuous manner, depending on the mode of acquisition), and after a post-labeling delay (PLD), the images are acquired using echo-planar imaging, gradient echo-spin echo, or fast spin echo-based readout scheme.[15,16] In patients with stenosis of the intracranial arteries, there may be a slowing of blood flow through the stenotic segment, and this may lead to

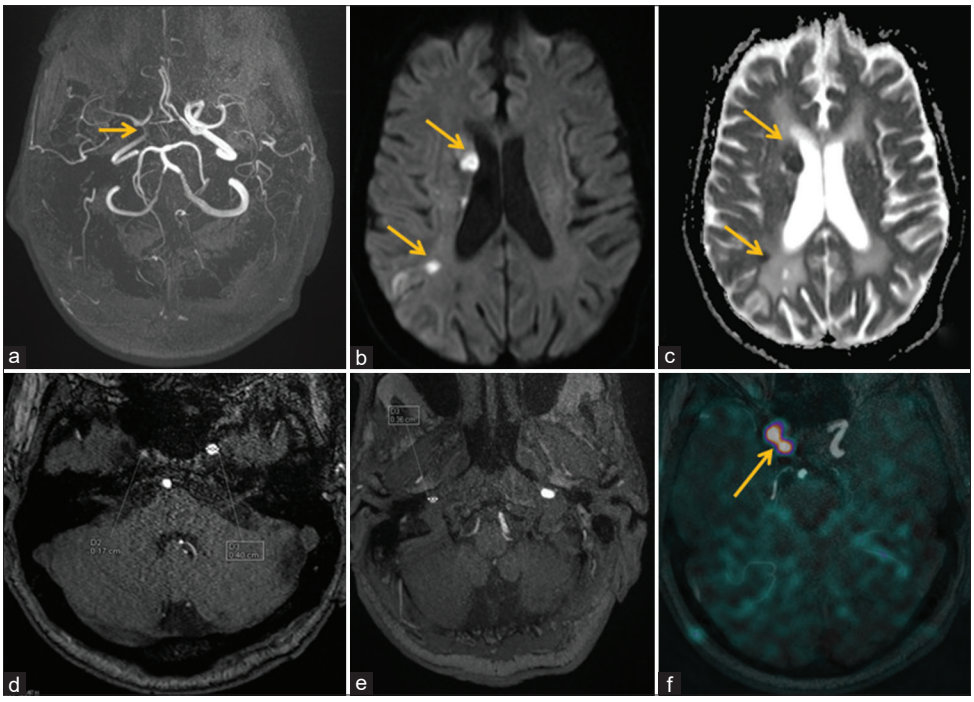

Figure 1: Representative image of a patient in ATA group. (a) 3D-TOF MRA with MIP reconstruction shows severe stenosis involving the cavernous segment of the right ICA with attenuation of the lumen (arrow). Diffusion-weighted images (b) and corresponding ADC images (c) show multiple acute infarcts are noted involving the head of right caudate nucleus and right parietal white matter (arrows in b and c). (d) Axial TOF MRA image shows the luminal diameter of right ICA at the level of stenosis measuring 1.7 mm (Ds) and the contralateral corresponding segment measures 4 mm (Do). (e) Axial TOF MRA image shows the diameter of the segment of the right ICA proximal to the stenosis measuring 3.6 mm (Dn). The percent stenosis in this case is 55.13%. (f) Fusion image of 3D pulsed ASL over 3D-TOF MRA shows intraluminal ATA within and across the stenotic segment of the right ICA (arrow). ATA: Arterial transit artifact, 3D-TOF: three-dimensional time of flight, MRA: magnetic resonance angiography, MIP: maximum intensity projection, ICA: internal carotid artery, and ADC: apparent diffusion coefficient.

"trapping" of the labeled spins within the lumen of the artery proximally and/or distally if the PLD is lesser than the arterial transit time through the involved artery.[17] This intraluminal arterial transit artifact (ATA) typically occurs in ASL acquisitions utilizing a single PLD and may lead to erroneous interpretation of reduced perfusion in the cerebral parenchyma perfused by the involved artery, thus necessitating a multi-delay acquisition technique.[17,18]

In this study, we hypothesize that the intraluminal ATA within a large intracranial artery may be predictive of significant stenosis, as seen in time-of-flight (TOF) magnetic resonance angiography (MRA), in a segment of that artery, when a single-PLD ASL acquisition is used. We further hypothesize that the presence of ATA within a large intracranial artery may be a significant predictor of ischemic stroke in the territory supplied by the involved artery. The findings of our study may be useful for radiologists interpreting MRI scans in cases of ischemic stroke, as the presence of intraluminal ATA may facilitate the diagnosis of significant intracranial stenosis.

MATERIAL AND METHODS

This was a retrospective analysis of a prospectively maintained database of patients referred for MRI examination for various indications. We identified patients with intracranial stenosis based on findings of TOF MRA. The patients with ATA within the lumen of a large intracranial artery (ATA group) were identified [Figure 1]. We included the intracranial segments of internal carotid artery (ICA) (cavernous segment and above), V4 segment of the vertebral artery (VA), basilar artery (B), A1 and A2 segment of the anterior cerebral artery, M1 and M2 segments of the middle cerebral artery (MCA), and P1 and P2 segments of the posterior cerebral artery, as large arteries for analysis of the ATA. The patients with intracranial stenosis, but without intraluminal ATA, were grouped into the no-ATA group [Figure 2]. The findings in these groups of patients were compared with those patients with no intracranial stenosis and no ATA (normal group) and patients with complete occlusion of an intracranial artery (Total occlusion group), as depicted in [Figures 3 and 4], respectively.

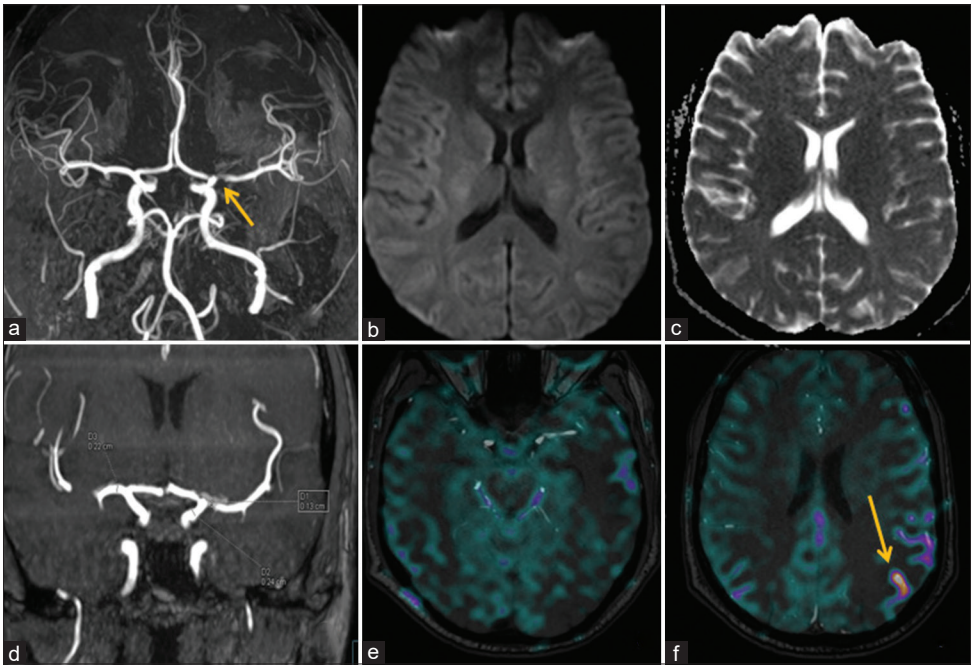

Figure 2: Representative image of a patient in no-ATA group. (a) 3D TOF MRA maximum intensity projection image shows stenosis at the proximal left MCA (arrow). No evidence of any infarct seen on diffusion weighted (b) and ADC (c) images. (d) TOF MRA image with coronal reconstruction shows diameter of stenotic segment (Ds) as 1.3 mm, diameter of proximal non-stenotic terminal ICA measuring 2.4 mm (Dn) and diameter of corresponding segment of contralateral normal MCA measuring 2.2 mm (Do). The percent stenosis in this case is 43.3%. (e) No intraluminal ATA is seen on fused 3D-ASL and 3D-TOF MRA image. (f) Multiple curvilinear areas of ATA noted in left parietal lobe (arrow) on fused 3D-ASL and 3D-TOF MRA image, suggestive of slow flow in the leptomeningeal collateral arteries as a consequence of left MCA stenosis. ATA: Arterial transit artifact, 3D-TOF: three-dimensional time of flight, MRA: magnetic resonance angiography, ICA: internal carotid artery, MCA: middle carotid artery, ASL: arterial spin labeling, and ADC: apparent diffusion coefficient.

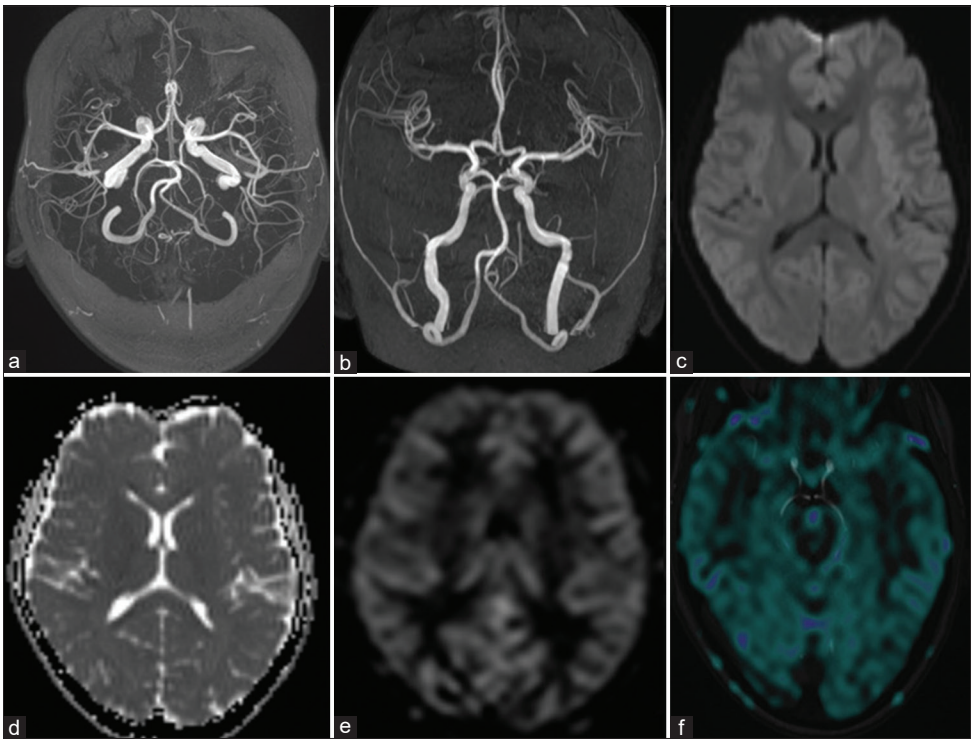

Figure 3: Representative image of a patient in normal group. 3D TOF MRA maximum intensity projection image in basal (a) and frontal (b) projection shows no evidence of intracranial stenosis. No evidence of any infarcts seen in the brain parenchyma on diffusion weighted (c) and ADC (d) images. (e) No evidence of perfusion deficits or ATA noted in bilateral cerebral hemisphere on gray-scale ASL map. (f) Fused ASL-TOF MRA image shows no intraluminal ATA. ATA: Arterial transit artifact, 3D-TOF: three-dimensional time of flight, MRA: magnetic resonance angiography, ICA: internal carotid artery, ASL: arterial spin labeling, and ADC: apparent diffusion coefficient.

The percentage of stenosis of the involved arterial segment was measured using the formula: $(1-[D_s/D_n+D_s/D_o]/2)\times100$, where D_s denotes the diameter of the cross-section of the artery at the site of maximum stenosis and D_n denotes the diameter of the normal proximal artery segment and D_o represents the diameter of the contralateral arterial segment corresponding to the site of stenosis. The purpose of including an additional metric using D_o is to control for flow-related effects which might influence the luminal diameter of the ipsilateral artery on TOF MRA, thus underestimating the degree of stenosis. However, in the case of the basilar trunk, the maximum diameter of the dominant VA only was considered. We excluded any patient with stenosis of the extra-cranial arterial segments, which may confound the assessment of intracranial intraluminal ATA. In addition, we also excluded patients with multiple intracranial stenoses (stenosis of multiple arteries or multiple segments of a single artery).

The ASL sequence used in this study was a 3D pulsed ASL with turbo gradient and spin echo (TGSE) readout. The various MRI sequences (3T, 32-Channel head coil, Magnetom Skyra, Siemens Healthineers, Germany) are summarized below in [Table 1].

Statistical analysis

The statistical analysis was performed using the MedCalc software for biomedical research (MedCalc software Ltd., Belgium). The continuous variables were expressed as mean and standard deviation, while the categorical variables were dichotomized. Differences between the groups were evaluated using the *t*-tests, analysis of variance, and Fischer's exact test, depending on the distribution of the data within the groups. Receiver operator characteristics (ROC) analysis was carried out to determine the cutoff value of the degree of stenosis predictive of intraluminal ATA along with the sensitivity and specificity of the determined value. Multivariate logistic regression (stepwise) was conducted to determine the independent variables which could predict the occurrence of stroke in the territory of the involved intracranial artery. The results were expressed along with their confidence intervals (95% CI). $P < 0.05$ was considered statistically significant.

RESULTS

A total of 79 patients were included in the analysis. The patients could be divided into four groups based on the

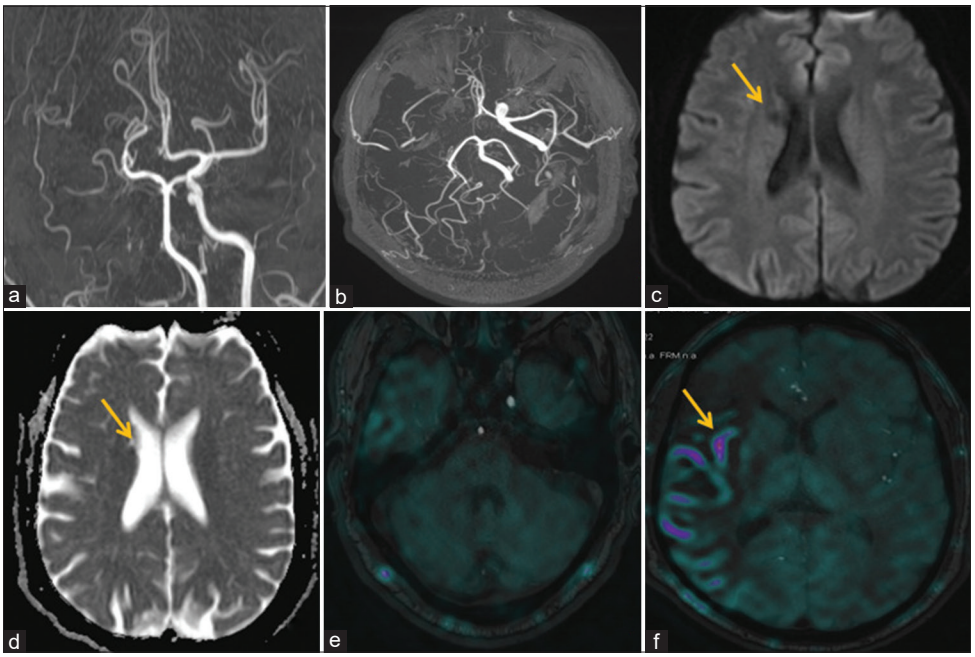

Figure 4: Representative image of a patient in total occlusion group. 3D TOF MRA images with maximum intensity projection reconstruction in frontal (a) and basal (b) projection, shows complete occlusion of the right ICA. Chronic lacunar infarct is noted in the head of the right caudate nucleus on diffusion weighted image (c) and the corresponding ADC (d) image. On fused ASL-MRA image (e), no evidence of intraluminal ATA is seen. (f) On ASL-MRA fusion image, multiple curvilinear areas of ATA are noted along the right cerebral hemisphere (arrow), suggestive of slow flow in the leptomeningeal collaterals, secondary to complete right ICA occlusion. ATA: Arterial transit artifact, 3D-TOF: three-dimensional time of flight, MRA: magnetic resonance angiography, ICA: internal carotid artery, ASL: arterial spin labeling, and ADC: apparent diffusion coefficient.

findings on imaging: Patients with intraluminal ATA with stenosis identified on TOF MRA (ATA group, n = 22), patients with no intraluminal ATA, but with identifiable stenosis on TOF MRA (no-ATA group, n = 23), patients with no ATA and no stenosis identified on TOF MRA (normal group, n = 25), and patients with complete occlusion of the intracranial artery (total occlusion group, n = 9). The demographic characteristics of patients in all the groups, identifiable risk factors, vessels involved, and symptoms are summarized in [Table 2].

Three patients in the ATA group had a paracavernous meningioma causing significant stenosis of the ipsilateral cavernous ICA. Two patients in the ATA group had a diagnosis of angioinvasive rhinocerebral mucormycosis. In the rest of the cases, the exact etiology of intracranial stenosis could not be established conclusively and it was presumed to be secondary to intracranial atherosclerosis.

Intergroup comparison between the ATA and no-ATA group of patients (among those with identifiable intracranial stenosis) is summarized in [Table 3].

No significant difference was noted among the patients in ATA and no-ATA group as far as demographics and various risk factors (such as smoking, diabetes mellitus, dyslipidemia, hypertension, and alcohol consumption) are concerned. No statistically significant difference was noted between the two groups concerning the intracranial vascular segment involved in the stenosis. Patients in the ATA group had a significantly higher degree of percent stenosis. The patients with ATA were more likely to have stroke whereas the patients without ATA were more likely to be asymptomatic. None of the patients in the total occlusion group and the normal group showed ATA. In patients with any demonstrable stenosis of TOF MRA, the presence of ATA within the lumen of an intracranial artery was predictive of stenosis of ≥56% within that artery, by ROC analysis, with a sensitivity of 100% and specificity of 100%. The results of the ROC analysis are shown in [Table 4].

Among patients with stenosis seen on TOF MRA (n = 45), the presence of an infarct (acute and/or chronic) within the territory of the involved artery was noted in 25 cases (55.56%). Using stepwise forward logistic regression analysis, only positive intraluminal ATA was found to be an independent predictor of infarct (acute and/or chronic) within the territory of the stenosed artery. Other variables including percent stenosis, age, and gender were not found to be significant predictors of infarction. The logistic regression

Table 1: Specific parameters of MRI sequences.

Sequence	TR (ms)	TE (ms)	Slice thickness (mm)	Field of view (mm×mm)	Voxel size (mm×mm×mm)	Matrix (mm×mm)	Flip angle (degree)	Time of acquisition (Min: S)	b-values	Post- labelling Delay (ms)	Read out
ASL	3000	15.9	3.5	240×240	2.0×2.0×3.5	63×56	180	3:18	NA	1800	TGSE
DWI	6400	98	4	220×220		192×192	90	1:36	0 and 90	NA	EPI
3D-TOF MRA	21.0	3.4	0.5	181×200	0.3×0.3×0.5	313×384	18	4:02	NA	NA	NA

ASL: Arterial spin labeling, DWI: Diffusion-weighted imaging, EPI: Echo planar imaging, NA: Not applicable, TGSE: Turbo gradient and spin echo, 3D-TOF MRA: Three-dimensional time of flight magnetic resonance angiography, MRI: Magnetic resonance imaging

model had an accuracy of 80% in the prediction of infarct while considering the presence of intraluminal ATA as an independent variable. The summary of the logistic regression analysis is presented in [Table 5].

DISCUSSION

ASL was introduced in the 1990s as a potential non-invasive tool for the assessment of CBF.[19] Since then, it has largely been a part of neuroscience research.[20] In recent years, due to improvements in the clinical MR systems, ASL has broken through into clinical MRI protocols as well.[21] Fundamentally, ASL deals with labeling the blood flowing into the brain (or for that matter, any part of the body), using inversion magnetization pulses in a particular region of the proximal vasculature. Following this, the labeled blood is allowed to flow into the brain, and after an interval of time (known as post-labeling delay), images are acquired. A control image (without labeling) is also acquired, and the labeled and control images are subtracted, to generate ASL maps.[22] During the PLD, magnetization is exchanged between the flowing blood and the protons within the cerebral tissue through the capillary blood.[23] The images thus obtained are essentially CBF weighted. In addition, there is a loss of signal within the labeled blood due to the T1 effect. This leads to a relatively low signal-to-noise ratio (SNR) if we consider a single ASL acquisition.[23] To improve the SNR, multiple acquisitions are done and an average of all the acquisitions is done during post-processing.[21-23]

ASL has been used as a non-invasive tool to evaluate cerebral parenchymal perfusion in various neurological disorders. In patients with acute ischemic stroke, ASL shows a reduction in the CBF in the affected territory.[24] Many studies have shown the usefulness of ASL in the assessment of ischemic penumbra and have found ASL comparable to CT perfusion (CTP) and dynamic susceptibility contrast MR perfusion.[25,26] However, in cases of stenosis of an artery, there is a significant slowing of blood flow, which may lead to the trapping of labeled spins within the lumen of the artery, giving a high intraluminal signal on ASL. In contrast, the distal territory supplied by the artery may show falsely low perfusion.[24,26,27] This leads to ATA within the lumen of the involved artery. In chronic steno-occlusive diseases, collaterals develop in the territory, which perfuses the ischemic territory. The blood flow within these collaterals is also slow, and this also leads to ATA on ASL.[28] Many studies have found ATA around the ischemic territory to be predictive of good prognosis in cases of ischemic stroke.[29]

The problem of ATA may be mitigated using bipolar crushing gradients, or by increasing PLD, or using a multi-delay ASL protocol.[18,20] However, ATA may have diagnostic potential in the assessment of significant intracranial stenosis. The present study was driven by this hypothesis that the presence of intraluminal ATA within a large intracranial artery is

Table 2: Summary of various characteristic features, risk factors, and demographic variables among different groups of patients.

	ATA group (*n*=22)	No-ATA group (*n*=23)	Normal group (*n*=25)	Total occlusion group (*n*=9)
Age (years)	52.7±12.4	54±10.12	42.72±9.28	45.6±10.24
Gender (Male: Female)	14:8	16:7	13:12	6:3
Percent stenosis (mean)	69.7±43.1	47.3±4.36	0	~100
Current smoking (%)	72.7	56.5	32	66.7
Past smoking (%)	4.5	8.7	12	22.2
No smoking (%)	22.8	26.8	56	11.1
Alcohol intake (%)	45.4	39.13	24	28.57
Hypertension (%)	63.6	60.86	40	66.7
Type 2 diabetes mellitus (%)	50	43.5	16	44.4
Dyslipidemia (%)	31.8	26.08	12	22.2
Symptoms (%)				
Transient ischemic attack	13.6	17.39	0	44.4
Stroke	72.7	13.04	0	55.6
Amaurosis fugax	9.09	17.39	0	0
Asymptomatic	4.6	52.17	100	0
Arterial segment involved (%)				
Right ICA	22.7	39.13	0	11.1
Left ICA	27.3	17.39	0	22.2
Right ACA	0	0	0	0
Left ACA	4.5	4.34	0	0
Right MCA	9.09	8.69	0	0
Left MCA	9.09	8.69	0	22.2
Right VA	9.09	4.34	0	11.1
Left VA	9.09	8.69	0	22.2
Basilar trunk	9.09	8.69	0	11.1
Acute and/or chronic infarct in the territory of the stenosed artery (%)	86.36	26.08	0	77.8

ATA: Arterial transit artifact, ACA: Anterior cerebral artery, ICA: Internal carotid artery, MCA: Middle cerebral artery, VA: Vertebral artery

predictive of significant stenosis in that artery. We included four groups of patients in this study, to assess the utility of intraluminal ATA sign in the assessment of intracranial stenosis. The patients with total occlusion and those with no stenosis seen on TOF MRA did not show any intraluminal ATA. The occurrence of intraluminal ATA is indicative of a significant slowing of blood flow. This phenomenon will not occur if there is no flow within the artery (as in complete occlusion) and in the absence of any stenosis. The findings of our study are consistent with this hypothesis. Among patients with stenosis seen on TOF MRA, we sought to determine the threshold of stenosis beyond which intraluminal ATA may occur. Our data suggest that the presence of ATA within the lumen of an intracranial artery is predictive of ≥56% stenosis within the vessel (as evaluated on TOF MRA using the methodology of the present study). We also hypothesized that the presence of intraluminal ATA sign could predict significant ischemic events in the territory of the involved artery. Indeed, our data show that in a multivariate forward stepwise logistic regression model, the presence of intraluminal ATA is an independent predictor of an acute and or chronic infarct in the territory of the involved artery. This finding can be explained by both hypoperfusion as well as thromboembolic mechanisms of stroke in intracranial stenosis. Since intraluminal ATA suggested significant stenosis and significant slowing of blood flow in the distal territory, the territory of the involved artery is at a greater risk of ischemic events. Even if collaterals are robust, there is a chance of failure of collaterals in the event of challenges posed to the cerebral circulation, such as hypotension. Furthermore, slowing of blood flow may predispose to *de novo* thrombus formation at the site of stenosis or just proximal to it, thus increasing the risk of distal thromboembolism.

In a study by Di Napoli *et al*., the presence of ATA in the intracranial circulation was noted in patients with ≥ 70% stenosis in the cervical segment of the ICA, and the presence of ATA was the only factor associated with significant recent ischemic symptoms. In their study, contrast-enhanced MRA (CE MRA) was used to assess the stenosis of the extracranial ICA.[30] Another study by Ozpar *et al*. evaluated the relationship between the presence and localization of ATA and clinical symptoms. They utilized digital subtraction angiography as the reference standard for the quantification

Table 3: Comparison of various characteristics between ATA and no ATA groups.

	ATA group (*n*=22)	No-ATA group (*n*=23)	*P*-value
Age (years)	52.7±12.4	54±10.12	0.72
Gender (Male: Female)	14:8	16:7	0.67
Percent stenosis (mean)	69.7±43.1	47.3±4.36	0.0009
Current smoking (%)	72.7	56.5	0.52
Past smoking (%)	4.5	8.7	
No smoking (%)	22.8	26.8	
Alcohol intake (%)	45.4	39.13	0.67
Hypertension (%)	63.6	60.86	0.85
Type 2 diabetes mellitus (%)	50	43.5	0.66
Dyslipidemia (%)	31.8	26.08	0.67
Symptoms (%)			0.003
Transient ischemic attack	13.6	17.39	
Stroke	72.7	13.04	
Amaurosis fugax	9.09	17.39	
Asymptomatic	4.6	52.17	
Arterial segment involved (%)			0.33
Right ICA	22.7	39.13	
Left ICA	27.3	17.39	
Right ACA	0	0	
Left ACA	4.5	4.34	
Right MCA	9.09	8.69	
Left MCA	9.09	8.69	
Right VA	9.09	4.34	
Left VA	9.09	8.69	
Basilar trunk	9.09	8.69	
Acute and/or chronic infarct in the territory of the stenosed artery (%)	86.36	26.08	0.0003

ATA: Arterial transit artifact, ACA: Anterior cerebral artery, ICA: Internal carotid artery, MCA: Middle cerebral artery, VA: Vertebral artery

Table 4: Summary of results of ROC analysis.

ATA group (*n*)	22
No-ATA group (*n*)	23
Cut off value of percent stenosis	≥56%
Youden index J	1.00
Sensitivity	100%
Specificity	100%
AUC	1.00
95% CI	0.92–1.00
P-value	0.0006

ATA: Arterial transit artifact, ROC: Receiver operator characteristic, AUC: Area under curve, CI: Confidence interval

of stenosis. They concluded that the presence of ATA in distal MCA territory only corresponds to early-stage perfusion abnormalities, while ATA in ICA is related to the more advanced stage of stenosis of the ICA with higher rates of critical stenosis, collateral formation, and perfusion abnormalities.[31] In a study comparing the ASL with CTP in chronic stenosis of the MCA, Tian *et al*. found that pseudo-continuous ASL (pCASL) with single PLD overestimates regional CBF (rCBF) in cases of severe MCA stenosis while there is no difference between pCASL and CTP in the estimation of rCBF in mild-to-moderate MCA stenosis.[32] This finding may be explained by the significant delay in arterial transit of labeled blood through a severely stenotic artery, thus overestimating the reduction in CBF. However, the authors did not report intraluminal ATA in this study.

In the present study, we have reported our findings based on the evaluation of intracranial stenosis detected on TOF MRA. Although, TOF MRA has been reported to overestimate the degree of stenosis and is influenced by the flow conditions within a vessel, advances in MR acquisition technology with 3D acquisition techniques have improved the SNR in TOF MRA.[33,34] Many studies have shown that TOF MRA may be at least as accurate, if not more than contrast-enhanced MRA, in the assessment of stenosis and/or occlusion of intracranial and extracranial arteries in the evaluation of ischemic stroke.[35,36] Since we have included only TOF MRA in this study; thus, our absolute results may be limited to the assessment of intracranial stenosis by TOF MRA only; however, the principles and concept conveyed by our results may also apply to CE MRA, CT angiography, as well as DSA assessment of intracranial stenosis.

Table 5: Summary of stepwise forward logistic regression analysis.

Dependent variable				**Presence of infarct**
Enter variable if *P*<				0.05
Remove variable if *P*>				0.1
Infarct present				25 (55.56%)
Infarct absent				20 (44.44%)
Coefficients and Standard Errors				
Variable	**Coefficient**	**Standard error**	**Wald**	***P*-value**
Intraluminal ATA present	2.88	0.78	13.63	0.0002
Constant	−1.04145	0.474	4.81	0.0283
Odds Ratio and 95% CI				
Variable	**Odds ratio**			**95% CI**
Intraluminal ATA	17.944			3.87–83.08
Classification table				
Actual group	**Predicted group**			**Percent correct (%)**
	No infarct	**Infarct present**		
No infarct	17	3		85
Infarct present	6	19		76
Percent of cases correctly classified				80

ATA: Arterial transit artifact, CI: Confidence interval

"Arterial transit artifact" has been described as an "artifact" in the literature addressing the subject matter of ASL.[18] However, our study and other studies have consistently shown the usefulness of this artifact in addressing a very important question of intracranial stenosis.[30,32] The findings of our study may be useful in the detection of significant intracranial stenosis by radiologists in patients who may have got an MRI done for evaluation of ischemic stroke. The direct hemodynamic significance as well as the indirect implication of distal territorial thromboembolism conveyed by intraluminal ATA sign cannot be overemphasized. In this regard, there may be few false positives while interpreting intraluminal ATA, such as excessive tortuosity of intracranial arteries, dolichoectasias, large intracranial aneurysms, and spin trapping after flow diversion of intracranial aneurysms.[18,37] In our study, we did not find any of these confounders; however, we may advise caution in interpreting intraluminal ATA in the presence of any such confounders and correlation of the findings with other sequences such as CT angiography, DSA, CE MRA, and vessel-wall imaging.

As remarked by Zaharchuk, the "arterial transit artifact" may be "arterial transit awesomeness," since this particular finding has the potential to be a biomarker of various neurological conditions.[38] Herein, we have attempted to describe the utility of this arterial transit awesomeness as a biomarker of significant intracranial stenosis. Further studies using different angiographic modalities as well as high-resolution vessel wall imaging, including other disease conditions such as multifocal stenosis and intracranial as well as extracranial stenosis, may further our understanding of this arterial transit awesomeness.

CONCLUSION

Intraluminal ATA within large intracranial arteries on 3D Pulsed ASL using TGSE readout is predictive of 56% or greater stenosis of the segment of the artery where the artifact is noted as determined on 3D TOF MRA on 3T MRI scanner. The presence of intraluminal ATA is an independent predictor of an acute and/or chronic infarct within the territory of the involved artery based on the findings of our study.

Acknowledgments

The authors acknowledge the contributions of the technical and nursing staff for the acquisition of the data and care of the patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Peer S, Singh P. Intraluminal arterial transit artifact as a predictor of intracranial large artery stenosis on 3D time of flight MR angiography: Expanding the application of arterial spin labeling MRI in ischemic stroke. J Clin Imaging Sci 2023;13:17.