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Lenticulostriate Artery Infarction Involving the Striatocapsular Region: A High-resolution MRI Study

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Abstract

Background To study the effectiveness of three-dimensional high-resolution magnetic resonance vessel wall imaging (3D-HR-VWI) in diagnosing ischemic stroke caused by perforating artery infarction. Methods We retrospectively studied 38 patients who were admitted to two different centers in China between 2016 and 2018. We examined the structure of the lenticulostriate arteries by 3D-HR-VWI and studied the characteristics of the perforating arterial lesions. Results There were no statistically significant differences between the number of lenticulostriate arteries on the lesion side and the normal side (P=0.2512), but there was a significant difference in the ratio of lenticulostriate arteries between patients with a striatum infarction and those with single perforating artery infarction (P=0.020). The depths of the lenticulostriate arteries on the lesion sides were also significantly different between the two groups (P=0.032). Differences among risk factors including hypertension, diabetes, hyperlipidemia, and smoking were not statistically significant. The differences in vascular involvement, length of M1 lesions, etiological type, number of lenticulostriate arteries, and depth of the lenticulostriate arteries were also not significant. Conclusion 3D-HR-VWI can show lesions of the lenticulostriate arteries and perforating arteries clearly, which has diagnostic value for judging the nature of ischemic stroke caused by perforating artery disease.

Background

Stroke is a leading cause of death and morbidity worldwide, and the mortality rate of stroke in China has been increasing in recent years[1]. Xinjiang is one of the large provinces in northwest China, and has higher prevalence, morbidity and mortality rates of stroke than the eastern coastal area[1-3].

The etiology of ischemic stroke is complex and diverse. In China, arterial lesions, including aortic atherosclerosis, small vessel disease, arterial dissection, vasculitis, and Moyamoya disease, constitute the main causes of ischemic stroke. Basal ganglial infarctions and lacunar infarctions caused by perforating lesions have a high incidence in stroke patients in China. These are of great importance in clinical practice because the striatum is easily affected by vascular abnormalities, these structures are sensitive to ischemia, and the severity of infarction in these areas is closely related to the clinical outcome.

Perforating lesions refer to the infarction of the corresponding blood supply area due to occlusion of the lenticulostriate artery, generally involving the basal ganglia and the internal capsule. Large striatum infarcts usually appear as comma-shaped areas encompassing the putamen, caudate nucleus and internal forelimbs. The affected blood supply corresponds to the lateral and medial group of Beans, the Heubner's return artery, and/or the choroidal artery. Small subcortical lacunar infarctions are usually located in the same blood supply area, but have different clinical manifestations and risk factors. There are two types: large lenticulostriate nucleus infarctions or striatum infarctions and terminal end infarctions; the major diagnostic feature is that the maximum diameter of the lesion is larger than 20 mm, and other brain tissues (especially adjacent cortical areas) are normal.

The identification of the cause of ischemic stroke is vital, and is the basis for subsequent treatments. However, the existing cerebral angiography technologies provide limited information, and while the methods used to diagnose cerebral infarctions are improving, identifying the cause of infarction remains difficult. Cerebral arterial magnetic resonance angiography permits the accurate diagnosis of intracranial plaques, thrombosis, and wall abnormalities, and can help to identify the etiology of ischemic stroke. Different research groups have developed two-dimensional black blood imaging techniques for intracranial vascular wall imaging research. Most have used Turbo Spin Echo (TSE) to acquire images to ensure a good signal-to-noise ratio. Region Saturation (RSAT) is used to suppress blood flow signals, and Interleaved MultiSlice is used to improve coverage. While 3T magnetic resonance systems have rarely been reported, 3T high-field magnetic resonance imaging should be able to provide the required signal-to-noise ratio, allowing for higher intra-layer resolution.

In patients with striatum stenosis or lacunar infarction caused by perforating arterial disease, the changes in perforating vessels have not been well investigated. It has been speculated that in these diseases, the number of perforating arteries will decrease. However, to the best of our knowledge, it has not been reported whether the number of arteries is decreased, and whether the decrease is related to the type of lesion.

We hypothesized that in patients with ischemic stroke caused by perforating lesions, the changes in the perforating artery are related to the type of ischemic stroke. In order to investigate this hypothesis, we developed a 3D high resolution arterial vessel wall joint imaging method to evaluate intracranial arterial plaques and the involved vessels. We performed a retrospective study to evaluate the effectiveness of this imaging technique in diagnosing ischemic stroke and assessing combined structural changes.

Methods

Subjects

A total of 38 consecutive patients admitted to two different centers between 2016 and 2018 were enrolled in this study. The study was approved by the local institutional review board. Informed consent was obtained from all participants. Patients were divided into two groups, a striatum infarction group and a single perforating infarction group, based on the MRI findings and clinical features. The patients classified into the striatum infarction group had an infarction due to the occlusion of the branching veins from the main artery of the MCA, and there was infarction of the forelimb, putamen and caudate nucleus of the internal capsule[4]. Patients in the single perforating infarction group had an infarction most commonly in the basal ganglia, corona radiate, thalamus or pons that was presumed to be due to occlusion of a single perforating artery, such as a lenticulostriate artery (LSA), thalamic artery or anterior pontine artery, which originate from the middle cerebral artery, posterior cerebral artery and basilar artery, respectively. This study mainly concentrated on the LSA[5].

Inclusion and exclusion criteria

The inclusion criteria were: 1. Acute or subacute unilateral lenticulostriate artery blood supply area infarction (within 14 days from onset); 2. Age > 18 years; 3. Had cranial plain scan, MR angiography scan and 3D-HR-VWI images obtained during admission (no longer than 20 days from onset); 4. Image quality meets the diagnostic requirements.

The exclusion criteria were: 1. Chronic lenticulostriate artery infarction; 2. Bilateral lenticulostriate artery blood supply area infarction; 3. Infarction relevant to the anterior choroidal artery or Heubner's artery; 4. Thalamic infarction; 5. A lack of high-resolution magnetic resonance vessel wall Images; 6. Poor image quality that did not meet the diagnostic requirements; 7. Comorbid hemorrhage of the basal ganglia; 8. Infraction involving the non-lenticulostriate artery blood supply area.

MR Scanning Strategy

A 32-channel skull coil and 3D fast/turbo spin echo acquisition (VISTA) was performed using a Philips 3.0T superconducting MR scanner at one center (Achieva scanner; Philips Healthcare, Best, The Netherlands); head and neck combined coils and a different type of 3D fast/turbo spin echo acquisition (SPACE) was performed using a Siemens 3.0T superconducting MR scanner at the other center (Trio scanner; Siemens Healthcare, Germany). If a contrast agent was used, the dose was 0.2 ml/kg, and was injected intravenously using a high-pressure syringe at a rate of 2.5 ml/s. The scan sequence and parameters are shown in Table 1.

Image Processing

Images were reviewed and analyzed using the commercially-available VesselMass software (Leiden University Medical Center, Leiden, the Netherlands). Multi-planar reconstruction of the 3D-HRVWI images was conducted using minimum density projection on the coronal plane, after adjusting the coronal plane angle so that the lenticulostriate artery was displayed on the same plane as much as possible (Fig. 1). Then, the blood vessel parameters were measured, and the narrowest peripheral area of the vessel and the lumen area of the reference peripheral vessel were calculated, followed by calculating the plaque load and vascular remodeling index. Other relevant parameters of the plaque were also measured. Tracing of the MCAs and LSAs was manually performed by a radiologist with two years of experience in MRI image interpretation. The LSA was traced from the MCA along the contrast difference between tissues and vessels around in the inner capsule.

The length of the lesion was evaluated semi-quantitatively according to the original image obtained during high-resolution T1WI vessel wall imaging. The length of the M1 segment of the middle cerebral artery was evaluated first, followed by an evaluation of the length of the lesion. The length of the lesion

was then accounted for via the proportion of the M1 segment of the middle cerebral artery and divided into groups of <1/3, 1/3-2/3, and >2/3.

Based on the T1WI image of the 3D-HRVWI obtained after the minimum density projection and post-processing, the coronal plane was used to observe the relevant layer of the basal ganglia to identify the lenticulostriate arteries. The criterion for identifying the lenticulostriate arteries was a linear low-signal shadow stretching from the middle cerebral artery wall. The longitudinal arteries were regarded as lenticulostriate arteries. If a lenticulostriate artery passed a short distance and divided into more than one artery, those arteries were all counted to give the number of lenticulostriate arteries. According to the minimum density projection, the depth of the lenticulostriate artery entering into the basal nucleus was divided into the upper part, the middle part and the lower part, and the depth was judged as the area reached by the longest lenticulostriate artery (Fig. 2).

The infarctions were divided into a striatum infarction group and a single perforating infarction group. According to the location of the infarction on DWI, the infarctions were further divided into three groups: lateral lenticulostriate artery lesions, medial lenticulostriate artery lesions and internal + lateral lenticulostriate artery lesions.

Statistical Analysis

Continuous variables were expressed as the means ± standard deviations, and categorical variables were expressed as medians with interquartile ranges. The *t*-test or non-parametric rank sum test was used to compare the continuous variable groups. The X^2 test or Fisher's exact probability method was used to compare the categorical variables. Differences with values of $P<0.05$ were considered statistically significant. All statistical analyses were performed using SPSS 17.0 (IBM Corp., USA).

Results

A total of 38 patients with infarctions in the lenticulostriate artery blood supply area were included in this study. One patient was excluded due to high-resolution image motion artifacts, and 2 patients were excluded due to softening of the contralateral basal ganglia. Finally, 35 patients were included, encompassing 30 males and 5 females, with a mean age of 50.7±10.9 years. Of the 35 patients, 20 had hypertension, 12 had diabetes, 19 had hyperlipidemia, and 17 had a history of smoking. However, there were no statistically significant differences in these risk factors between the striatum infarction group and the single perforating artery group (Table 2).

There were no obvious motion artifacts in the high-resolution images of the 35 patients, and there were no pleat artifacts in the field of view. The images of the lenticulostriate arteries after the minimum-density

projection reconstruction was satisfactory, and the image quality grades were all above level 2.

The average number of lenticulostriate arteries on the lesion side was 3.23±1.54, and the average on the normal side was 3.6±1.12. There was no significant difference in the absolute number of lenticulostriate arteries between the lesion side and the normal side (t=1.157, df=68, *P*=0.2512). Among the 21 patients with striatum cystic infarction diagnosed according to the DWI lesion pattern analysis, 13 had lateral lenticulostriate artery lesions, 4 had medial lenticulostriate artery lesions, and 4 had internal + lateral lenticulostriate artery lesions. Among the 14 patients with single perforating infarctions, 8 had lateral lenticulostriate artery lesions and 6 had medial lenticulostriate artery lesions, while internal or lateral lenticulostriate arteries were not involved in any of the patients. There were no significant differences between the striatum infarction group and the single perforating artery group (X^2=4.365, df=2, *P*=0.113).

The mean number of lenticulostriate arteries on the lesion side was 3±1.73, and was 3.38±1.12 on the normal side. The number of lenticulostriate arteries on the lesion side of the single perforating infarction group was 3.57± 1.16, while the normal side showed a mean number of 3.93 ± 1.01. The number of lenticulostriate arteries was significantly different between the two sides (*P*=0.02). In the single perforating artery infarction group, the pattern of lenticulostriate arteries was most often the same as on the normal side, that is, there was no decrease in the number of lenticulostriate arteries. In the striatum, the infarction group had all three types of lesions. The depth of the diseased side of the lenticulostriate arteries was also significantly different between the groups (P=0.032). Among them, the lenticulostriate arteries in the striatum cyst group showed the greatest depth, while the lenticulostriate arteries showed a similar depth to that of the normal side in the single perforating infarction group (Table 2).

When patients were divided into lateral lenticulostriate artery, internal lenticulostriate artery, and internal + lateral lenticulostriate artery groups, the vascular condition, M1 segment lesion length, etiological type, number of lenticulostriate arteries, and the depth of the lenticulostriate arteries did not show any statistically significant differences. Thus, this study indicates that the involvement of different types of lenticulostriate arteries is not associated with vascular lesions, the etiology, or the morphological features of the lenticulostriate arteries (Tables 2 and 3).

Discussion

The blood supply to the striatum comes primarily from the internal carotid artery, middle cerebral artery (MCA), anterior cerebral artery, and anterior choroidal artery, followed by posterior communicating arteries and posterior choroidal arteries, which are constantly distributed within a small area. Heubner's artery is located in the anterior cerebral artery region. The lateral small branch supplies the forearm of the inner capsule and the lateral part of the caudate nucleus, the medial small branch supplies the anterior nucleus and the caudate nucleus, and the short central artery branch of the anterior cerebral artery supplies the caudate nucleus. In the medial anterior aspect of the head, the medial branch of the middle cerebral artery, within 10 mm of the MCA, is emitted at a right angle. About 2-3 short and thin protrusions pass through the sac nucleus through the inner sac to the caudate nucleus. The lateral group comprises about

4-6 long and thick, fan-shaped branches that pass through the lenticular nucleus through the inner capsule to the caudate nucleus. The internal choroidal artery of the internal carotid artery is divided into sections 1-4, three of which are located 2 mm from the distal end of the posterior communicating artery, emitting 1-3 cortical branches, each of which has 2-3 branches, which eventually generate the striate cystic artery. One reaches the globus pallidus, and the other runs into the posterior limb of the internal capsule, reaching the radiation. In the striatum, the body of the caudate nucleus is supplied by the lenticulostriate artery. The anterior and lateral parts of the caudate nucleus are supplied by Heubner's artery, the inner side is supplied by the short central artery, and the caudate nucleus is supplied by the anterior and posterior choroid. The anterior part of the nucleus returns to the artery, the middle part becomes the lenticulostriate artery, and the posterior part is supplied by the choroidal artery.

The concept of perforating lesions was first proposed by Caplan et al. in 1989[2]. They believed that deep and small infarcts may be caused by small perforating arteries due to occlusion by tiny atherosclerotic plaques. These tiny arteries are prone to hypertension, which presents as glassy degeneration. These perforating arteries, such as the lenticulostriate artery or the para-aortic artery, usually branch vertically from the main artery. Damage to the vascular endothelium caused by rapid blood flow can lead to the formation of local atherosclerotic plaques. Therefore, effective imaging methods can help identify relevant pathophysiological changes and provide valuable information for the clinician. In their studies, von Morze et al. and Cho et al.[3,4] used 7T MR imaging systems to reveal the lenticulostriate artery by MRA and found that non-invasive evaluation of the lenticulostriate artery can be achieved, but the 7T MR system has not yet been applied in clinical practice. However, the images of lenticulostriate arteries based on the 3T MR imaging system and the 3D-HRVWI sequence obtained in this study were satisfactory, suggesting a potential for clinical application.

The overall incidence of striatum infarction was 5%, and most patients in the acute phase developed severe neurological deficits. Acute large striatum cystic infarction usually presents with similar symptoms and signs as infarcts in the cortex, which may manifest as aphasia and go unnoticed. There are many speculations about the development of cortical symptoms in these subcortical deep gray area infarctions. The cortical region has a connecting loop in the deep gray matter nucleus, and this part of the infarction leads to cortical disorders, which may be caused by cortical damage itself, or the functional activity of the corresponding cortical region may be weakened due to the distance effect.

There are four main mechanisms leading to striatum sac infarction[2,5]: 1. occlusion of the proximal middle cerebral artery; 2. the presence of a T-shaped thrombus at the end of the internal carotid artery; 3. occlusion of the external carotid artery and deep nucleus infarction caused by embolic or hemodynamic damage at the proximal end of the middle cerebral artery; or 4. atherosclerotic lesions at the middle cerebral artery, complicated by *in situ* thrombosis or other abnormal middle cerebral artery conditions, such as a dissection or vasculitis. There may be good collateral circulation in the cortical region, and due to the lack of collateral circulation in the basal ganglia, the embolus can reach the middle cerebral artery and cause an embolic event.

Although the lacunar infarction caused by a single perforating artery is small, the resulting neurological deficit can be severe. In addition, while a single lacunar infarction rarely causes serious consequences such as coma, a new infarct can continue to develop due to diffuse cerebral arteriolar lesions, resulting in multiple lacunar infarctions. The accumulation and superposition of such lacunar brain damage is bound to cause a wider range of brain dysfunction, and may even lead to vascular dementia. Recent studies[6] suggest that not all lacunar infarctions that were confirmed clinically or via imaging are caused by cerebral small vessel disease, and the etiology also includes atherosclerotic lesions.

According to the available literature[3, 7-9], a reduction in the number of lenticulostriate arteries is a common manifestation of an infarction of the lenticulostriate artery blood supply area. On the one hand, this conforms to the pathophysiological changes, and confirms the blood supply changes caused by the occlusion of the lenticulostriate arteries. The pathophysiological process of infarction in the area provides strong evidence for the etiology of the infarct involving the lenticulostriate arteries. On the other hand, it has also been confirmed that the HRVWI sequence can reach the lenticulostriate area through the lenticulostriate arteries, as shown by the minimum density projection method (which meets the requirements of vascular imaging), but this is limited by technical conditions such as the imaging resolution of the wall, which may also cause low sensitivity or poor image quality for imaging of the lenticulostriate arteries.

The present study found that the number of lenticulostriate arteries on the lesion side was increased. This may be related to three factors: 1. The bilateral distribution of the lenticulostriate artery itself is asymmetrical; 2. The striatum infarction area is larger than shown in the images, but the secondary pathophysiological changes are still unclear. Whether there are new or newly opened lenticulostriate arteries therefore still needs further study; 3. The original lenticulostriate arteries are dilated and developed as compensatory vessels, while the corresponding lenticulostriate arteries on the normal side are too small to see. Among the 35 patients, six also had deeper lenticulostriate arteries on the lesion side than on the normal side. A study with a larger sample size is needed to clarify the clinical and physiological significance of these changes in lenticulostriate arteries.

Conclusions

A true striatum infarction and morphological changes of the lenticulostriate artery caused by single perforating infarction are different. The detection of these changes can provide a basis for future in-depth studies of the evolution and outcomes of perforating artery infarction. Our data indicate that 3D-HRVWI technology can be used to show the status of the lenticulostriate artery, and therefore support further research on perforating artery infarction.

Although the 3D-HRVWI technique has made great progress, it is still difficult to obtain clear images of the lenticulostriate artery due to its small diameter. High-resolution wall imaging often requires long scanning times, and is sensitive to motion artifacts, which affects the minimum density projection post-processing method. Since the display of the lenticulostriate artery relies on the imaged minimum density

projection to display the lenticulostriate arterial lumen, the information of the tube wall is partially lost. This leads to the inability to judge the wall of the lenticulostriate artery during the evaluation. In addition, the present study was retrospective in nature, and because patients with lacunar infarction have mild clinical symptoms, these patients do not typically receive blood vessel imaging or high-resolution wall imaging, so there is a certain bias in the sample selection. Finally, the patients were not followed up, and the relationship between the characteristics of the lenticulostriate artery and prognosis could not be explored further.

Abbreviations

MRI: Magnetic resonance imaging.

3D-HR-VWI: Three-dimensional high-resolution magnetic resonance vessel wall imaging.

TSE: Turbo Spin Echo.

RSAT: Region Saturation.

MCA: Middle Cerebral Artery.

LSA: Lenticulostriate artery

DWI: Diffusion weighted imaging

Declarations

Ethics approval and consent to participate: The study was approved by the institutional review board of the Second Affiliated Hospital of Xinjiang Medical University, which belongs to the Second Affiliated Hospital of Xinjiang Medical University. Informed consent was obtained from all participants, the informed consent obtained from study participants was written.

Consent for publication: The patients agreed to the publication of images, and no identifying data are present on any of the images.

Availability of data and materials: The data used/analyzed for the current study are available upon request from the corresponding author.

Competing interests: There are no competing interests related to this manuscript.

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Authors' contributions: JL, JWX and LX designed the study. JL, LJH, ZN, ZL, CC and KH performed the experiments and collected data. JL, LJH, ZN and ZL analyzed the data. JL, JWX and LX prepared the manuscript. JWX and LX reviewed the manuscript and supervised the work. All authors approved the final manuscript for submission.

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Tables

Table 1. MR Scanning Parameters

Name	TR (ms)	TE (ms)	TI (ms)	Slice thickness (mm)	FOV (cm)	Gap (mm)	Nr. of layers (20)	b value (s/mm^2)	NEX	Matrix
T1WI	1750	24								
T2WI	5752	93		5	24	1				
T2FLAIR	6000	160	2250							
DWI	6800	90		5	24	1	20	0,1000	1	128X128
MRA	34	min								
3D-HR-VWI	800	8.6	650		16				2	256X320

Table 3 Comparison of Different Groups of Lenticulostriate Arteries

	Lateral	Internal	Lateral+ Internal	*P*
n	21	10	4	
Gender				0.215
Male	19	7	4	
Female	2	3	0	
Age(years)	53.62±9.18	51.3±10.56	38.75±13.38	0.036
Stenosis				0.791
No	9	5	1	
Light	1	0	0	
Medium	1	2	1	
Heave	8	2	1	
Occlusion	2	1	1	
Length of M1				0.256
0	9	5	1	
≤1/3	4	2	0	
1/3-2/3	1	2	2	
≥2/3	7	1	1	
Origin of Emboli				0.596
Atherosclerotic	12	5	3	
Small Vessel Diseases	1	2	0	
Cardiac embolism and other	8	3	1	
Lesion Side/Normal Side Ratio				0.139
Less	2	2	2	
Equal	10	7	1	
More	9	1	1	
Depth of lenticulostriate artery on both sides				
Shallower	7	1	2	0.501
Equal	10	7	1	
Deeper	4	2	1	
Risk factors				
Hypertension	12	5	3	0.694
Diabetes	7	4	1	0.858
Hyperlipidemia	12	3	4	0.055
Smoking	10	4	3	0.492

Table 2 Comparison Between Striatum and Single Perforator Infarction Group

	striatum infarction	single perforating infarction	p
n	21	14	
Gender			0.369[a]
Male	19	11	
Female	2	3	
Age(years)	47.57±11.09	56.79±7.82	0.011
Stenosis			0.392
No	8	7	
Light	1	0	
Medium	1	3	
Heave	8	3	
Occlusion	3	1	
Length of M1			0.634
0	8	7	
≤1/3	3	3	
1/3-2/3	3	2	
≥2/3	7	2	
Origin of Emboli			0.085
Atherosclerotic	13	7	
Small Vessel Diseases	0	3	
Cardiac embolism and other	8	4	
Lesion Side/Normal Side Ratio			0.020
Less	9	3	
Equal	7	11	
More	5	0	
Depth of lenticulostriate artery on both sides			0.032
Shallower	9	2	
Equal	7	11	
Deeper	5	1	
Risk factors			
Hypertension	13	7	0.486
Diabetes	5	7	0.153[a]
Hyperlipidemia	13	6	0.268
Smoking	13	4	0.053

Figures

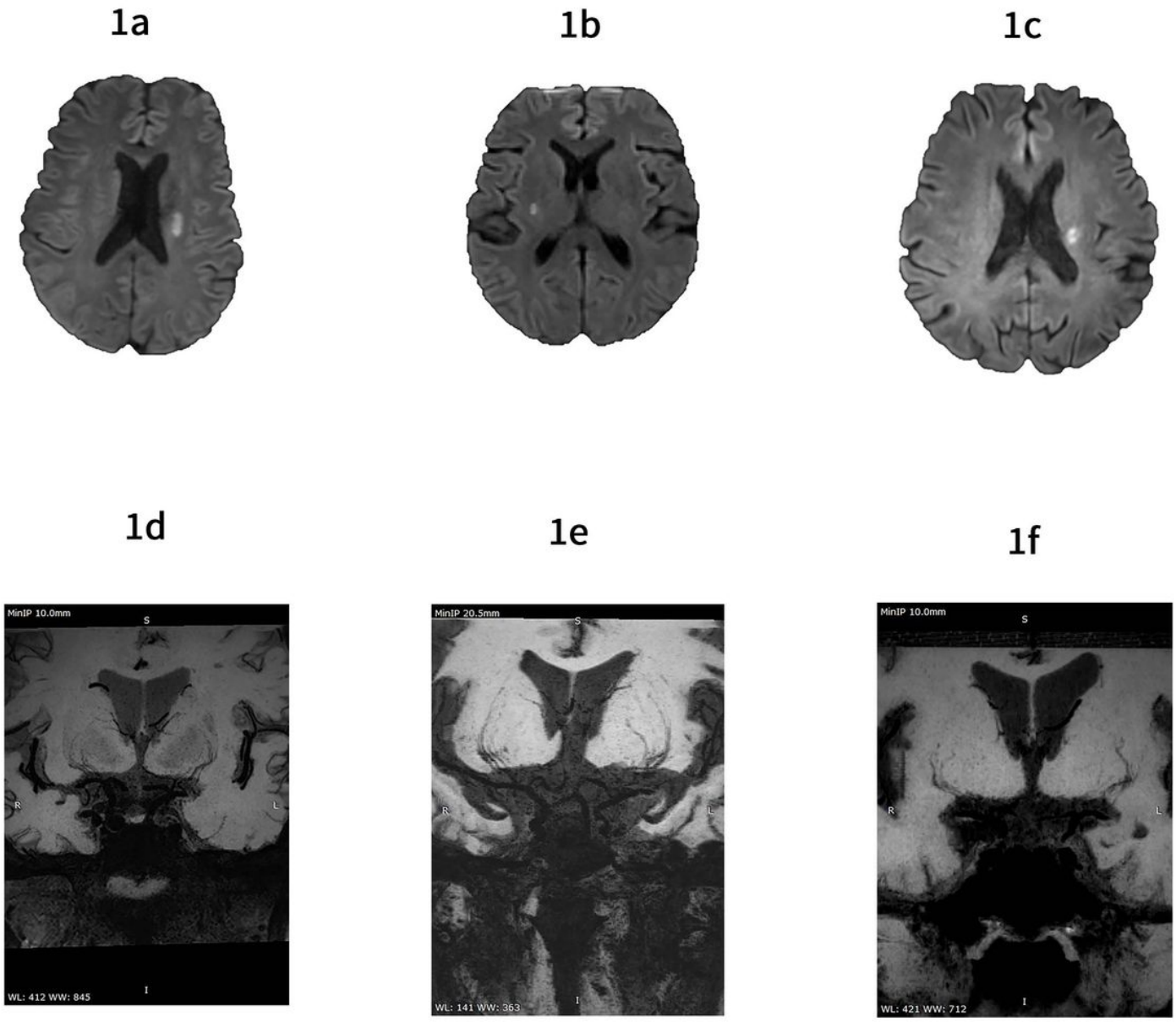

Figure 1

Representative images from three cases of infarction of the lenticulostriate arteries. a,d: a 48-year-old male; DWI showed a single perforating infarction in the left basal ganglia. b,e: a 48-year-old female; DWI showed a single perforating infarction in the right basal ganglia. c,f: a 60-year-old male; DWI showed a single perforating infarction near the left ventricle. All 3 high-resolution MIP images clearly showed that the number of bilateral lenticulostriate arteries was equal, and the depth of the lenticulostriate arteries was similar on both sides.

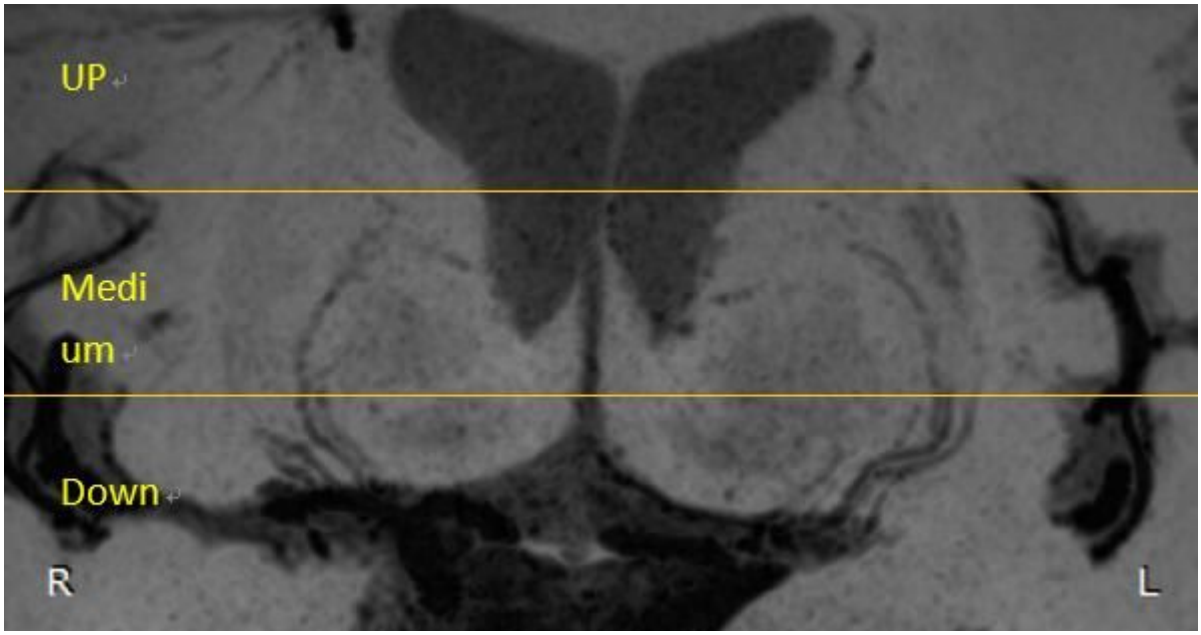

Figure 2

The depths of the lenticulostriate arteries.