



# Clinical significance of common-stem lenticulostriate arteries in patients with internal watershed infarction

Wen -huo Chen<sup>1</sup> · Ting-yu Yi<sup>1</sup> · A-lai Zhan<sup>2</sup> · Yan-min Wu<sup>1</sup> · Mei-fang Zhang<sup>1</sup> · Yi-min Li<sup>2</sup> · Yan-yu Lu<sup>2</sup> · Ding-lai Lin<sup>1</sup> · Xiao-hui Lin<sup>1</sup> · Zhi-nan Pan<sup>1</sup>

Received: 18 January 2019 / Accepted: 27 May 2019 / Published online: 16 June 2019  
© Fondazione Società Italiana di Neurologia 2019

## Abstract

**Background** A common-stem origin of lenticulostriate arteries (CS-LSAs) is an anatomical variation that supplies a moderate to large section of the basal ganglia. We hypothesized that CS-LSAs with a patent orifice are located at distal positions of the acute-occluded middle cerebral artery (MCA) and that the blood flow of CS-LSAs is supplied by pial arterial anastomoses and results in hypoperfusion of CS-LSAs, similar to a deep watershed (DWS) infarction.

**Objective** Our study evaluated the possibility of CS-LSAs in patients with DWS infarction and MCA occlusion and also assessed the safety of endovascular therapy (ET) in these patients.

**Methods** A cohort of consecutive patients with DWS infarction and MCA occlusion and in whom full recanalization via ET was achieved were identified. Patients were divided into two groups based on the presence of CS-LSAs observed during ET. In addition, radiological and clinical data were retrospectively analyzed.

**Results** Thirty-three patients were included, and CS-LSAs were observed in 48.5% (16/33) of patients. The possibility (72.2%, 13/18) of CS-LSAs was high in patients with DWS infarction accompanied with basal ganglia infarction. A good clinical outcome was similar in patients with CS-LSAs and basal ganglia infarction and in patients without CS-LSAs and basal ganglia infarction (69.2% vs. 81.8%,  $P = 0.649$ ).

**Conclusions** The possibility of CS-LSAs was 48.5% in patients with DWS infarction and MCA occlusion, and the revascularization procedure was safe and feasible in these patients despite the moderate-to-large basal ganglia infarction.

**Keywords** Lenticulostriate arteries · Common stem · Deep watershed infarction · Middle cerebral artery occlusion · Hypoperfusion

## Implication of patient care

1. There is a moderate chance of common-stem LSAs (CS-LSAs) in progressive ischemic stroke patients with deep watershed (DWS) infarction and middle cerebral artery (MCA) occlusion.
2. The mechanism of basal ganglia infarction was hypoperfusion in these types of patients.
3. The recanalization procedure was feasible in these patients.

**Electronic supplementary material** The online version of this article (<https://doi.org/10.1007/s10072-019-03953-w>) contains supplementary material, which is available to authorized users.

✉ Ting-yu Yi  
siyuyufen@163.com

<sup>1</sup> Department of Neurology, Zhangzhou affiliated Hospital of Fujian Medical University, Zhangzhou, Fujian, China

<sup>2</sup> Department of Radiology, Zhangzhou affiliated Hospital of Fujian Medical University, Zhangzhou, Fujian, China

## Abbreviations

LSAs	Lenticulostriate arteries
CS-LSAs	Common-stem LSAs
MCA	Middle cerebral artery
ICA	Internal carotid artery
ET	Endovascular therapy
DWS	Deep watershed
NIHSS	National Institutes of Health Stroke Scale
mRS	Modified Rankin Scale
FVH	Fluid-attenuated inversion recovery vascular hyperintensities
CWS	Cortical watershed
mTICI	Modified Thrombolysis in Cerebral Infarction
ACG	American Society of Interventional and Therapeutic Neuroradiology collateral grading
TOAST	Trial of Org 10172 in Acute Stroke Treatment
sICH	Symptomatic intracranial hemorrhage

## Introduction

Lenticulostriate arteries (LSAs) are among the largest perforating arteries in the brain. These vessels originate from the middle cerebral artery (MCA) and range from 2 to 12 in number [1]. LSAs may arise as individual vessels and/or from a common stem [1]. LSAs supply most of the basal ganglia, including the upper part of the head and body of the caudate nucleus, the putamen, the lateral part of the pallidum, the superior part of the anterior and the posterior limbs, and the genu of the internal capsule as well as the lateral third of the anterior commissure [2]. The territory of LSAs is balanced with the territories of the other perforating arteries arising from the internal carotid, anterior cerebral, and anterior choroidal arteries [2]. Occlusion of common-stem origin LSAs (CS-LSAs) leads to a large basal ganglia infarction throughout the entire region of supply [3], which can lead to severe neurological deficits.

However, in our clinical setting, we determined that CS-LSAs with patent orifices originated from the distal location of the acute-occluded MCA in acute progressive ischemic stroke patients with moderate-to-larger basal ganglia infarction, and the neurological deficit became good when recanalization of the acute occluded MCA was achieved despite the moderate-to-larger basal ganglia infarction. Therefore, we hypothesized that the blood flow of CS-LSAs arises from pial arterial anastomoses, similar to the deep watershed zone [4] in these types of patients. We also hypothesized that endovascular therapy was safe in these patients despite the moderate-to-larger basal ganglia infarction. Therefore, this study evaluated the possibility of CS-LSAs in patients with progressive DWS infarction and MCA occlusion and also assessed the safety of endovascular therapy (ET) in these patients.

## Materials and methods

We performed a retrospective review of prospectively collected data from a registry of patients with acute ischemic stroke undergoing endovascular treatment at a single, high-volume, stroke center between January 2015 and February 2017. Data for this consecutive series of patients were collected and then analyzed. Our Institutional Review Board approved this study, and informed consent was not obtained because of the retrospective nature of the study.

### Patient selection

The study inclusion criteria were as follows: (1) acute anterior circulatory ischemic stroke secondary to acute MCA occlusion; (2) acute and progressive onset of neurological signs with an increase in the National Institutes of Health Stroke Scale (NIHSS) score of  $\geq 4$  points [5] within 72 h [6]; (3) a

pretreatment NIHSS score of 6–19; (4) a preoperative MRI was performed, and MCA occlusion without internal carotid artery (ICA) stenosis and DWS with/without basal ganglia infarction was observed on the MRI; (4) age older than 18 years; (5) the prestroke modified Rankin Scale (mRS) score was 0–2; and (6) full recanalization of MCA was achieved by ET.

Patients were excluded from the study for the following reasons: (1) their acute ischemic stroke was the result of Moyamoya disease or vasculitis; (2) there were more than five micro-bleed lesions on SWI [7]; (3) the massy infarction and the infarct volume were  $\geq 70$  ml [8]; and (4) there was a contraindication to undergoing endovascular therapy, including a contrast-agent allergy.

### Radiological assessment

Four authors who were blinded to the patient information independently and retrospectively reviewed the pretreatment DWI data and procedure DSA data. Each author recorded the presence of basal ganglia, anterior cortical watershed (CWS), posterior CWS, or DWS infarction. Anterior CWS infarction was defined as the cortical border zone between the middle cerebral artery and the anterior cerebral artery; posterior CWS was defined as the cortical border zone between the middle cerebral artery and posterior cerebral artery; and DWS infarct was defined as rosary-like, confluent, striated, or solitary and located in the supraventricular or paraventricular areas (corona radiata or centrum semiovale) [9]. The presence of CS-LSAs with a patent orifice located at the distal position of the acute occluded MCA on DSA was also documented. CS-LSAs were defined as at least three LSAs originating from the same stem. Brain tissue reperfusion was radiologically assessed immediately after the operation using the modified Thrombolysis in Cerebral Infarction (mTICI) Scale. Successful reperfusion was defined as a mTICI score  $\geq 2b$ . The American Society of Interventional and Therapeutic Neuroradiology collateral grading system was used to assess the extent and rate of retrograde collateral flow to the target downstream territory in the anterior circulation (ASTIRN) [10]. There was excellent intraobserver agreement ( $\kappa = 1.0$  for basal ganglia lesions,  $\kappa = 0.9$  for CWS lesions,  $\kappa = 0.8$  for DWS lesions,  $\kappa = 0.95$  for CS-LSAs, and  $\kappa = 0.9$  for both mTICI and ACG grade). The final adjudication was completed by consensus.

### Clinical outcome assessment

The follow-up clinical examinations included an NIHSS assessment of patients' neurological functions and mRS assessments of the outcomes at 90 days, with a good outcome defined as an mRS score  $\leq 2$ . The stroke subtypes were determined according to the classification of the Trial of Org 10172 in Acute Stroke Treatment (TOAST) [11]. TOAST diagnosis was made by three stroke neurologists, and discrepancies were settled by a consensus

discussion. Symptomatic intracranial hemorrhage (sICH) was defined as an increase of >4 points in the NIHSS score [12].

## Data availability

We declare that the individual de-identified participant data in our study are available.

## Statistical analyses

Patients were divided into the CS-LSAs or no CS-LSAs group, and the study variables were compared between the two groups. Differences in continuous variables were examined using Student's *t* test or the Mann-Whitney *U* test, as appropriate. Differences in categorical variables were examined by the  $\chi^2$  test, and  $P \leq 0.05$  was considered significant. All of the statistical analyses were performed using the Statistical Package for the Social Sciences Version 23.0 (SPSS, Chicago, IL, USA).

## Results

A total of 33 consecutive patients with progressive DWS ischemic stroke and MCA occlusion who were admitted to our stroke center between January 2015 and February 2017 were included in this study.

### Comparison of patients with and without common-stem LSAs

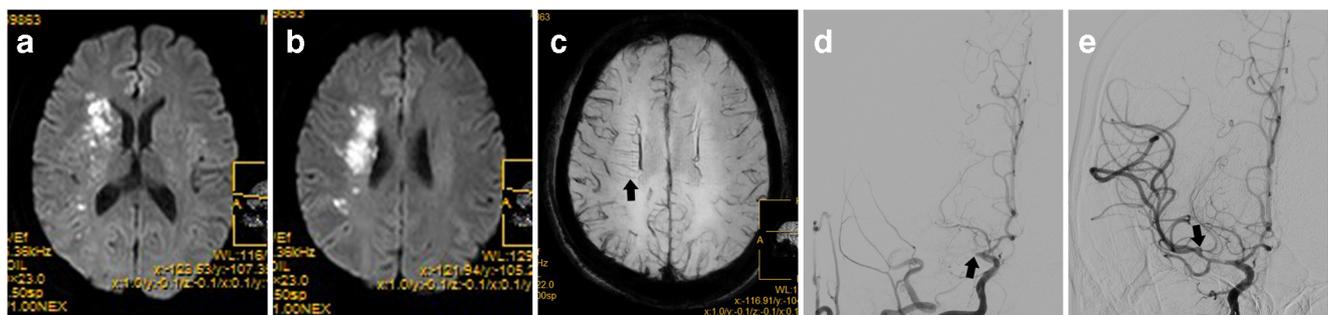
The median age of patients was 60 years, and 69.7% (23/33) of patients were men. Sixteen patients were included in the CS-LSAs group (a typical case is shown in Fig. 1) and 17 were included in the no CS-LSAs group (a typical case is shown in Fig. 2). Patients' baseline characteristics and clinical outcomes

are shown in Table 1; there were no significant differences in baseline characteristics between the two groups. The rates of hypertension (50.0% versus 60.7%), smoking (62.5% versus 47.1%), diabetes mellitus (18.8% versus 29.4%), and heart disease (6.3% versus 5.9%) were similar in the CS-LSAs group compared with the no CS-LSAs group. The age of patients in the CS-LSAs versus no CS-LSAs groups (62 years versus 57 years, respectively) and the pretreatment NIHSS score (12 points versus 13 points, respectively) were also similar between groups. Large-artery arteriosclerosis was the stroke subtype in 93.8% of patients with CS-LSAs and 94.1% of patients with no CS-LSAs. We found no significant difference in clinical outcomes between the groups; the rate of good prognosis was 72.7% (24/33), and the rate of sICH and mortality was 3.0% for both outcomes (1/33).

The angiographic findings and procedural techniques are shown in Table 2. Basal ganglia infarction on the pretreatment DWI was more common in patients with CS-LSAs than in those without CS-LSAs (81.3% versus 29.4%, respectively;  $P = 0.002$ ). All patients had (33/33) DWS infarction, 54.5% (18/33) had anterior CWS, and 63.6% (21/33) had posterior CWS. There was no significance difference in the procedure DSA data. A good clinical outcome was observed in 9 of 13 (69.2%) patients with CS-LCSAs and basal ganglia infarction and in 9 of 11 (81.8%) patients without CS-LSAs and basal ganglia infarction ( $P = 0.649$ ). All patients achieved successful reperfusion, and 66.7% (22/33) of patients had an ACG score  $\geq 3$ . Pure clot retrieval was performed in 30.3% (10/33) of patients, while emergent angioplasty was performed in 69.7% (23/33) of patients.

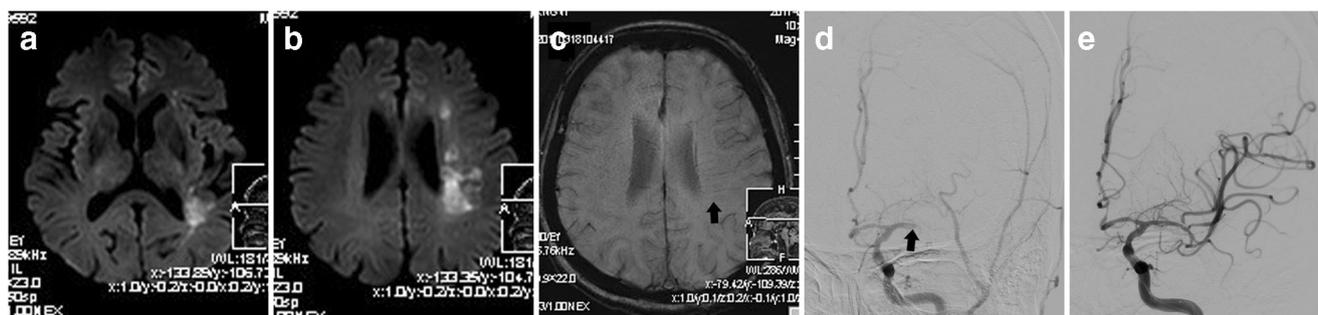
## Discussion

Most of lenticulostriate arteries originate from the middle cerebral artery (MCA). Some of the lenticulostriate arteries were



**Fig. 1** Illustrative case of patients with common-stem origin LSAs. A 33-year-old male smoker presented with left limb progressive weakness and an increased NIHSS score from 5 points to 16 points. **a–c** Pretreatment cranial MRI scan. **a**, **b** MR-DWI showing the right basal ganglia, right corona radiates, and posterior cortical watershed infarction. **c** MR-SWI showing prominent medullary veins within the right cerebral hemisphere (black arrow). **d** DSA showing a right MCA occlusion (black arrow). **e**

DSA showing the common-stem LSAs (black arrow) located at the distal MCA when the occluded MCA was recanalized. NIHSS, National Institutes of Health Stroke Scale; MRI, magnetic resonance imaging; MR-DWI, magnetic resonance-diffusion-weighted imaging; MR-SWI, magnetic resonance-susceptibility-weighted imaging; DSA, digital subtraction angiography; MCA, middle cerebral artery; LSAs, lenticulostriate arteries



**Fig. 2** An illustrative case of patients without common-stem LSAs. A 62-year-old male smoker presented with right limb progressive weakness and increased NIHSS from 3 points to 11 points. **a–c** Pretreatment cranial MR scan. **a, b** MR-DWI showing left posterior cortical watershed infarction and the left corona radiata with basal-ganglia-spare. **c** MR-SWI showing prominent medullary veins within the left cerebral hemisphere (black arrow). **d** DSA showing a left MCA

occlusion (black arrow). **e** DSA showing no common-stem LSAs at the distal MCA when the occluded MCA was recanalized. NIHSS, National Institutes of Health Stroke Scale; MR-DWI, magnetic resonance-diffusion-weighted imaging; SWI, susceptibility-weighted imaging; DSA, digital subtraction angiography; MCA, middle cerebral artery; LSAs, lenticulostriate arteries

occluded with the occurrence of an MCA acute occlusion and caused a part of a basal ganglia infarction. However, if LSAs arise from a common stem [1], which are called CS-LSAs, these CS-LSAs may be occluded or spared when MCA acute occlusion occurs. Occlusion of CS-LSAs leads to large basal ganglia infarction, which can rapidly lead to severe neurological deficits. If CS-LSAs with a patent orifice located at the distal occlusion site occur, the anterograde blood flow is suspended to CS-LSAs, which is located in the distal occluded MCA (Fig. 3). The blood flow of CS-LSAs is then compensated by the retrograde flow from the internal carotid artery and/or the pial branch of the posterior cerebral artery. Because CS-LSAs are mostly located distally, the perfusion pressure is likely to be the lowest for these vessels. Therefore, we considered that hypoperfusion is the underlying infarction

mechanism in the territory supplied by CS-LSAs and is similar to the mechanism underlying DWS infarction; this phenomenon was named CS-LSAs hypoperfusion. Unlike the neurological deficit caused by the occlusion of CS-LSAs, the neurological deficit caused by hypoperfusion of CS-LSAs may progress from mild to moderate and even to severe.

Endovascular therapy is an effective treatment for acute intracranial artery occlusion, and the time window is limited to approximately 6 h [13]; patients whose time window is 6–24 h (selected via brain imaging) can also benefit from endovascular therapy [14–16]. DWS infarction is common in MCA occlusion [9] because of the compromised hemodynamics [9, 17, 18]. The neurological function quickly deteriorates in patients with DWS infarction because of the enlarged infarction volume [9, 19], which may be related to the

**Table 1** Baseline patient characteristics and clinical outcomes between the two groups

	All patients (n = 33)	LSAs common stem (n = 16)	No LSAs common stem (n = 17)	P value
Sex (male, n)	23 (69.7%)	14 (87.5%)	9 (52.9%)	0.057
Age (mean, years)	60 ± 12	62 ± 14	57 ± 11	0.194
Smoker (n [%])	18 (54.5%)	10 (62.5%)	8 (47.1%)	0.491
Hypertension (n [%])	19 (57.6%)	8 (50.0%)	11 (64.7%)	0.491
Diabetes mellitus (n [%])	8 (24.2%)	3 (18.8%)	5 (29.4%)	0.688
Hyperlipidemia (n [%])	11 (33.3%)	6 (40.0%)	5 (33.3%)	1.000
Atrial fibrillation and/or rheumatic heart disease	2 (6.10%)	1 (6.3%)	1 (5.9%)	1.000
Pretreatment NIHSS score (median, IQR)	12 (10, 15)	12 (11, 14)	13 (10, 15)	0.657
TOAST subtype				
LAA	31 (93.9%)	15 (93.8%)	16 (94.1%)	1.000
CE	1 (3.0%)	1 (5.9%)	0 (0%)	1.000
Undetermined	1 (3.0%)	0 (1.0%)	1 (6.3%)	0.485
SICH	1 (3.0%)	1 (6.3%)	0 (0%)	0.485
90-day mRS ≤ 2 (n [%])	24 (72.7%)	11 (68.8%)	13 (76.5%)	0.708
Mortality	1 (3.0%)	1 (6.3%)	0 (0%)	0.485

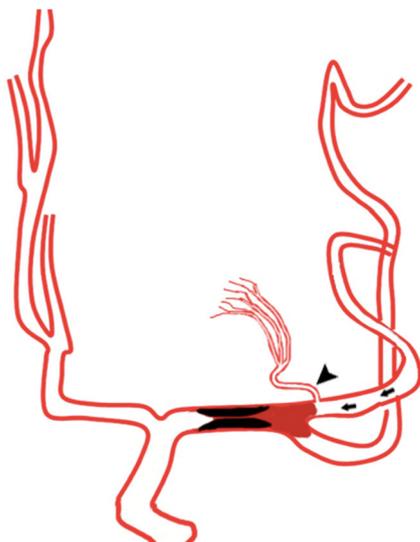
LSAs, lenticulostriate arteries; NIHSS, National Institutes of Health Stroke Scale; IQR, interquartile range; TOAST, Trial of Org 10172 in Acute Stroke Treatment; LAA, large-artery atherosclerosis; CE, cardioembolism; SICH, symptomatic intracranial hemorrhage; mRS, modified Rankin Scale

**Table 2** Radiological characteristics in the two groups

	All patients ( <i>n</i> = 33)	LSAs common stem ( <i>n</i> = 16)	No LSAs common stem ( <i>n</i> = 17)	<i>P</i> value
Pretreatment Infarction site				
Basal ganglia	18 (54.5%)	13 (81.3%)	5 (29.4%)	0.005
DWS infraction	25 (75.8%)	13 (81.3%)	12 (70.6%)	0.688
CWS infraction				
Anterior CWS	18 (54.5%)	10 (62.5%)	8 (47.1%)	0.491
Posterior CWS	21 (63.6%)	12 (75.0%)	9 (52.9%)	0.282
ACG grade $\geq 3$ ( <i>n</i> [%])	22 (66.7%)	10 (62.5%)	12 (70.6%)	0.721
mTICI grade $\geq 2b$	33 (100%)	16 (100%)	17 (100%)	
Treatment for intracranial occlusion				
Angioplasty directly	5 (15.2%)	2 (12.5%)	3 (17.6%)	1.000
Clot retrieval plus angioplasty	18 (54.5%)	7 (43.8%)	11 (64.7%)	0.303
Clot retrieval	10 (30.3%)	7 (43.8%)	3 (17.6%)	0.141

LSAs, lenticulostriate arteries; DWS, deep watershed; CWS, cortical watershed; ACG, American Society of Interventional and Therapeutic Neuroradiology collateral grading system; mTICI, modified Thrombolysis in Cerebral Infarction

hemodynamic compromise in the acute stage [20]. Endovascular therapy can quickly open the acute occluded intracranial artery and restore anterograde blood flow, and the neurological deficit of these patients may become good due to solving of the hemodynamic compromise [9]. Normally, moderate-to-large basal ganglia infarction indicates that all or part of the orifices of the lenticulostriate artery is occluded during acute MCA occlusion, which also indicates that the basal ganglia infarct was not reversible even with full recanalization of the MCA. Therefore, the neurological deficit would not change due to moderate-to-large basal ganglia infarction. It is very possible that CS-LSAs hypoperfusion can occur in patients with a progressive onset and MCA occlusion accompanied with moderate-to-large basal ganglia infarction.



**Fig. 3** Hypoperfusion of common-stem LSAs. The common-stem LSAs (arrow) are located in the most distal area supplied by retrograde blood flow from the meningeal arteries

Once the revascularization of MCA is achieved, the antero-grade blood flow to CS-LSAs is restored and the hemodynamic compromise of CS-LSAs might also improve. Then, the neurological status may concurrently improve despite the moderate-to-large basal infarction. Our study showed that a good clinical outcome rate was similar in CS-LSAs patients with basal ganglia and DWS infarction and no CS-LSAs patients with pure DWS infarction (69.2% vs. 81.8%,  $P = 0.649$ ). This result supports the hypothesis of CS-LSAs hypoperfusion.

We analyzed data from the pretreatment MRI, which showed that basal ganglia infarction was more common in patients with CS-LSAs than in those without CS-LSAs (81.3% versus 29.4%, respectively;  $P = 0.005$ ). CS-LSAs were observed in 72.2% (13/18) of patients with DWS plus basal ganglia infarction and MCA occlusion. In reverse, the occurrence of basal ganglia infarction often indicates hypoperfusion of CS-LSAs in progressive DWS infarction with symptomatic MCA occlusion.

Revascularization procedures that improve hemodynamic compromise might also improve the prognosis of patients by preventing cortical neuronal damage and stroke recurrence [9]. In our study, DWS infarction was seen in all patients and all of the patients underwent emergent revascularization procedures. In addition, successful reperfusion was achieved in all patients, and good prognosis was observed in 72.7% of patients. Our findings support the feasibility of performing endovascular therapy during the acute phase in progressive ischemic stroke patients because of the concurrent hemodynamic compromise.

In our study, the prevalence rate of CS-LSAs in progressive DWS ischemic stroke patients with symptomatic MCA occlusion was 48.4%. This rate greatly varies based on the different examination tools and the definition of CS-LSAs. In two studies with data obtained from brain autopsies, the prevalence rate

of CS-LSAs was high (61.76% [1] and 70.8% [3]), whereas in two other studies with data obtained from a brain MRI, the rate was much lower (38.8% [21] and 39.0% [22]). The observation of LSA branches may be more precise via brain autopsy because some LSA branches may be too small to be visualized during brain imaging. Compared with data obtained from a brain MRI, the prevalence rate of CS-LSAs was higher in our study despite the stricter definition. We defined CS-LSAs as at least three branches arising from the same trunk compared with the definition in another study of two or more branches arising from the same trunk. The high proportion of CS-LSAs in our study may reflect our particular study population; our study focused on patients with progressive DWS infarction and symptomatic MCA occlusion, while the focus in the other study was a healthy population or patients with only basal ganglia infarction. Furthermore, the diagnostic accuracy of DSA adopted in our study was higher than that of MRA adopted in the other study. In addition, the DSA image was not as clear as the brain autopsy. The diagnosis difficulty of CS-LSAs via DSA may increase if we define it as at least two branches arising from the same trunk as CS-LSAs. For the diagnosis of CS-LSAs to be clearer and more consistent, we use the definition that requires at least three branches arising from the same trunk as CS-LSAs.

Our findings have certain clinical implications. First, CS-LSAs with the patent orifice located at the distal portion of occluded MCA in progressive DWS ischemic stroke patients with basal ganglia infarction was possible. Second, the underlying mechanism of basal ganglia infarction was hypoperfusion of CS-LSAs in these types of patients. The recanalization procedure can improve the hemodynamic compromise, and the neurological status may improve in these patients despite the presence of large basal ganglia infarction. Finally, ET was safe and feasible in progressive DWS infarction and MCA occlusion.

The limitations of our study include the retrospective, single-stroke-center design and the relatively small number of patients involved. Another limitation is that conventional MRI perfusion or computed tomographic perfusion was not evaluated in our study. However, the mechanism of DWS infarction was hemodynamic compromise, the prognosis was poor despite the standard medical therapy, and the revascularization procedure could improve the prognosis.

## Conclusion

Common-stem LSAs are common in progressive DWS ischemic stroke patients with symptomatic MCA occlusion. DWS infarction accompanied by basal ganglia infarction is common in patients with CS-LSAs. Revascularization is safe and feasible in these types of patients despite the basal ganglia infarction.

**Acknowledgments** We thank teams from American Journal Expert (<https://secure.aje.com/cn/researcher/>), for editing the English text of a draft of this manuscript.

**Sources of funding** This research received grant from Joint Funds for the innovation of science and Technology, Fujian province (Grant number: 2018J01207).

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

## References

- Djulejić V, Marinković S, Maliković A, Jovanović I, Djordjević D, Četković M, Todorović V, Milisavljević M (2012) Morphometric analysis, region of supply and microanatomy of the lenticulostriate arteries and their clinical significance. *J Clin Neurosci* 19:1416–1421. <https://doi.org/10.1016/j.jocn.2011.10.025>
- Decavel P, Vuillier F, Moulin T (2012) Lenticulostriate infarction. *Front Neurol Neurosci* 30:115–119. <https://doi.org/10.1159/000333606>
- Marinković S, Gibo H, Milisavljević M, Četković M (2001) Anatomic and clinical correlations of the lenticulostriate arteries. *Clin Anat* 14:190–195. <https://doi.org/10.1002/ca.1032>
- Moustafa RR, Momjian-Mayor I, Jones PS, Morbelli S, Day DJ, Aigbirhio FI, Fryer TD, Warburton EA, Baron JC (2011) Microembolism versus hemodynamic impairment in rosary-like deep watershed infarcts: a combined positron emission tomography and transcranial doppler study. *Stroke* 42:3138–3143. <https://doi.org/10.1161/STROKEAHA.111.616334>
- Alawneh JA, Moustafa RR, Baron J-C (2009) Hemodynamic factors and perfusion abnormalities in early neurological deterioration. *Stroke* 40:e443–e450. <https://doi.org/10.1161/STROKEAHA.108.532465>
- Chen W-H, Yi T-Y, Wu Y-M, Zhang MF, Lin DL, Lin XH (2018) Safety of endovascular treatment in progressive ischemic stroke and anterior circulation large artery occlusion. *World Neurosurg* 122:e383–e389. <https://doi.org/10.1016/j.wneu.2018.10.059>
- Wang Z, Soo YO, Mok VC (2015) Cerebral microbleeds is anti-thrombotic therapy safe to administer? *Stroke* 45:2811–2817. <https://doi.org/10.1161/STROKEAHA.114.004286>
- Natarajan SK, Snyder KV, Siddiqui AH, Ionita CC, Hopkins LN, Levy EI (2009) Safety and effectiveness of endovascular therapy after 8 hours of acute ischemic stroke onset and wake-up strokes. *Stroke* 40:3269–3274. <https://doi.org/10.1161/STROKEAHA.109.555102>
- Seok WY, Oh YB, Phil HL, Wen YL (2006) Internal and cortical border-zone infarction: clinical and diffusion-weighted imaging features. *Stroke* 37:841–846. <https://doi.org/10.1161/01.STR.0000202590.75972.39>
- Zaidat OO, Yoo AJ, Khatri P, Tomsick TA, von Kummer R, Saver JL, Marks MP, Prabhakaran S, Kallmes DF, Fitzsimmons BF, Mocco J, Wardlaw JM, Barnwell SL, Jovin TG, Linfante I, Siddiqui AH, Alexander MJ, Hirsch JA, Wintermark M, Albers G, Woo HH, Heck DV, Lev M, Aviv R, Hacke W, Warach S, Broderick J, Derdeyn CP, Furlan A, Nogueira RG, Yavagal DR, Goyal M, Demchuk AM, Bendszus M, Liebeskind DS, Cerebral Angiographic Revascularization Grading (CARG) Collaborators, STIR Revascularization working group., STIR Thrombolysis in Cerebral Infarction (TICI) Task Force (2013) Recommendations on angiographic revascularization grading standards for acute ischemic stroke: a consensus statement. *Stroke* 44:2650–2663. <https://doi.org/10.1161/STROKEAHA.113.001972>

11. Adams H, Adams H, Bendixen B et al (1993) Classification of subtype of acute ischemic stroke. *Stroke* 23:35–41. <https://doi.org/10.1161/01.STR.24.1.35>
12. Campbell BCV, Mitchell PJ, Kleinig TJ et al (2015) Endovascular therapy for ischemic stroke with perfusion-imaging selection. *N Engl J Med* 372:1009–1018. <https://doi.org/10.1056/NEJMoa1414792>
13. Powers WJ, Derdeyn CP, Biller J, Coffey CS, Hoh BL, Jauch EC, Johnston KC, Johnston SC, Khalessi AA, Kidwell CS, Meschia JF, Ovbiagele B, Yavagal DR, American Heart Association Stroke Council (2015) 2015 American Heart Association/American Stroke Association focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment: a guideline for healthcare professionals from the American. *Stroke* 46:3020–3035. <https://doi.org/10.1161/STR.0000000000000074>
14. Nogueira RG, Jadhav AP, Haussen DC et al (2017) Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct. *N Engl J Med*. <https://doi.org/10.1056/NEJMoa1706442>
15. Albers GW, Marks MP, Kemp S, Christensen S, Tsai JP, Ortega-Gutierrez S, McTaggart R, Torbey MT, Kim-Tenser M, Leslie-Mazwi T, Sarraj A, Kasner SE, Ansari SA, Yeatts SD, Hamilton S, Mlynash M, Heit JJ, Zaharchuk G, Kim S, Carrozzella J, Palesch YY, Demchuk AM, Bammer R, Lavori PW, Broderick JP, Lansberg MG, DEFUSE 3 Investigators (2018) Thrombectomy for stroke at 6 to 16 hours with selection by perfusion imaging. *N Engl J Med* 378:708–718. <https://doi.org/10.1056/NEJMoa1713973>
16. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, Biller J, Brown M, Demaerschalk BM, Hoh B, Jauch EC, Kidwell CS, Leslie-Mazwi TM, Ovbiagele B, Scott PA, Sheth KN, Southerland AM, Summers DV, Tirschwell DL, American Heart Association Stroke Council (2018) 2018 guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 49(3):e46–e110
17. Derdeyn CP, Khosla A, Videen TO et al (2001) Severe hemodynamic impairment and border zone–region infarction. *Radiology* 220:195–201. <https://doi.org/10.1148/radiology.220.1.r01j09195>
18. Yamauchi H, Nishii R, Higashi T, Kagawa S, Fukuyama H (2009) Hemodynamic compromise as a cause of internal border-zone infarction and cortical neuronal damage in atherosclerotic middle cerebral artery disease. *Stroke* 40:3730–3735. <https://doi.org/10.1161/STROKEAHA.109.560011>
19. van Heesewijk HPM, Vos JA, Louwerse ES, van den Berg JC, Overtom TTC, Ernst SMPG, Mauser HW, Moll FL, Ackerstaff RGA (2002) New brain lesions at MR imaging after carotid angioplasty and stent placement. *Radiology* 224:361–365. <https://doi.org/10.1148/radiol.2242011302>
20. Bladin CF, Chambers BR (1993) Clinical features, pathogenesis, and computed tomographic characteristics of internal watershed infarction. *Stroke* 24:1925–1932
21. Kang CK, Park CW, Han JY, Kim SH, Park CA, Kim KN, Hong SM, Kim YB, Lee KH, Cho ZH (2009) Imaging and analysis of lenticulostriate arteries using 7.0-tesla magnetic resonance angiography. *Magn Reson Med* 61:136–144. <https://doi.org/10.1002/mrm.21786>
22. Akashi T, Miyasaka T, Takewa M et al (2012) Branching pattern of lenticulostriate arteries observed by MR angiography at 3.0 T. *Jpn J Radiol* 30:331–335. <https://doi.org/10.1007/s11604-012-0058-7>

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.