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The anterior one third of the posterior limb of the internal capsule is also supplied by the anterior choroidal artery

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ABSTRACT

Purpose: It is universally recognized that the anterior choroidal artery (AChA) supplies the posterior two-third of the posterior limb of internal capsule (PLIC). On the other hand, the blood supply to the anterior one third of the PLIC has remained undetermined. We posit the anterior one third of the PLIC is also supplied by the AChA referring the previous microsurgical descriptions.

Methods: Ninety consecutive patients with isolated acute infarction in the PLIC were studied. We classified patients into 4 groups. 1. The anterior type that involved the anterior one-third part of the PLIC. 2. The posterior type that involved the caudal two-third part of the PLIC, 3. The combined type that located in the full length of the PLIC, 4. The dot type that restricted within PLIC up to10mm in diameter.

Results: Patient numbers in groups 1 through 4 were 7 (7.7%), 46 (51.1%), 9 (10.6%) and 28 (31.1%). The anterior type involved the medial part of pallidum (MPP) in 5 patients (71.4%) and none in the lateral thalamus (LT), while the posterior type involved MPP only in 6 patients (13.0%) and LT in 33 patients (71.7%).

Conclusion: Corresponding to previous microsurgical descriptions, an occlusion of the proximal branches may cause anterior type infarct and that of the distal branches may cause posterior type infarcts. The anterior one third of the PLIC is also supplied by the branches of the AChA, albeit the low prevalence.

1. Introduction

It is universally recognized that the anterior choroidal artery (AChA) supplies the inferior part of the posterior two-third of the posterior limb of internal capsule (PLIC) [1–6]. On the other hand, the blood supply to the inferior part of the anterior one-third of the PLIC remains uncertain [7,8]. Various perforators such as those arising from the internal carotid artery and the anterior cerebral artery are suggested to possibly supply the genu of the capsule and the proximal part of the PLIC. Furthermore, the AChA perforators were also indicated to be one of the candidates for supplying the anterior part of the PLIC. Precise radiologic determination of perforators involved in ischemic events may help to clarify the stroke mechanism in patients who show deep small infarcts and even differentiate from other neurological diseases such as inflammatory or demyelinating diseases [8].

In the late 20th century, the AChA domain had been well characterized especially by the studies on microsurgical anatomy [9–12]. The superior branches arising from the cisternal segment of the AChA are suggested to have two different branches comprising of the proximal and the distal branches. Researchers described that the proximal branch supplies the genu of the internal capsule, the medial segment of the globus pallidus or the anterior part of the PLIC, while the distal branch supplies the posterior two-third of PLIC, retro-lenticular part of the internal capsule and the lateral thalamic nuclei [10,11]. We then posit the PLIC may be perfused by two different types of branches arising from the cisternal segment of the AChA. We studied ninety acute consecutive patients with PLIC infarction focusing on the vascular supply to the anterior PLIC using a multi-planar reconstruction algorithm [13].

2. Methods

2.1. Patient selection

Among the prospective registration of consecutive patients with acute ischemic strokes in the Kyoto Second Red Cross Hospital, we

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Table 1
Characteristics of patients. Ages are expressed as means ± SD. Values are n (%). Abbreviations.

Group	Total	Anterior	Posterior	Combined	Dot	*p* values
n (%)	90	7 (7.7)	46 (51.1)	9 (10.0)	28 (31.1)	
Age	70.8 ± 12.5	75.1 ± 9.2	69.5 ± 11.4	73.3 ± 15.4	70.3 ± 14.2	ns
Male	58 (64.4)	5 (71.4)	30 (65.2)	3 (33.3)	20 (71.4)	ns
Hypertension	78 (86.6)	7 (100.0)	37 (80.4)	8 (88.8)	26 (92.8)	ns
Diabetes mellitus	27 (30.0)	3 (42.8)	15 (32.6)	2 (22.2)	7 (25.0)	ns
Dyslipidemia	47 (52.2)	4 (44.4)	23 (50.0)	4 (44.4)	16 (57.1)	ns
Smoking	51 (56.6)	2 (28.5)	29 (63.0)	4 (44.4)	16 (57.1)	ns
Alcohol	21 (23.3)	2 (28.5)	10 (21.7)	2 (22.2)	7 (25.0)	ns
Atrial fibrillation	12 (13.3)	2 (28.5)	5 (10.8)	1 (11.1)	3 (10.7)	ns
WIA						
Normal	64 (71.1)	5 (83.3)	31 (67.3)	3 (33.3)	23 (82.1)	< 0.0001
> 50% stenosis	9 (10.0)	0	5 (10.8)	4 (44.4)	2 (7.1)	ns
ICA						
Normal	73 (82.0)	5 (83.3)	36 (78.2)	5 (55.5)	27 (96.4)	ns
> 50% stenosis	5 (5.5)	0	2 (4.3)	2 (22.2)	1 (3.5)	ns
Stroke mechanism						
PAD	80 (88.8)	5 (71.4)	44 (95.6)	4 (44.4)	27 (96.4)	< 0.0001
ATD	5 (5.5)	0	2 (4.3)	2 (22.2)	1 (3.5)	ns
ES	5 (5.5)	2 (28.5)	0	3 (33.3)	0	ns

WIA: Whole intracranial artery disease, ICA: Relevant intracranial internal carotid artery disease, PAD: Penetrating artery disease, ATD: Atherothrombotic disease, ES: Embolic stroke.

Table 2
Neurological manifestations. NIHSS and mRS are expressed as median value (range).

Group	Total	Anterior	Posterior	Combined	Dot	p values
n (%)	90	7 (7.7)	46 (51.1)	9 (10.0)	28 (31.1)	
NIHSS	2 (0–24)	2 (1 − 21)	2.5 (0–24)	5.5 (4–22)	2 (0–5)	0.0002
mRS	1 (0–6)	2 (0–3)	1 (0–5)	1 (0–5)	1 (0–6)	ns
Dysarthria	63 (67.0)	5 (71.4)	30 (65.2)	9 (100)	17 (60.7)	ns
Facial palsy	39 (41.9)	4 (57.1)	17 (36.9)	7 (77.7)	8 (28.5)	0.047
Hemiparesis	43 (47.7)	3 (42.8)	20 (43.4)	8 (88.8)	12 (42.8)	ns
Monoparesis (U)	14 (15.5)	2 (28.5)	7 (15.2)	1 (11.1)	4 (14.2)	ns
Monoparesis (L)	13 (14.4)	0	9 (19.5)	0	4 (14.3)	ns
Sensory disturbance	20 (22.2)	0	12 (25.5)	5 (55.5)	3 (10.7)	0.016
Ataxia	9 (10.0)	0	6 (13.0)	0	3 (10.7)	ns
Visual disturbance	1 (1.0)	0	1 (2.1)	0	0	ns
Abulia	9 (14.2)	2 (28.5)	1 (2.2)	6 (66.6)	0	< 0.0001

Values are n (%). Abbreviations; NIHSS: National Institutes of Health Stroke Scale, mRS: modified Rankin Scale, U: upper limb, L: lower limb.

selected the patients with AChA territory infarctions. A total of 2095 stroke patients were admitted from January 2007 to March 2017 and there were 827 patients with single small subcortical infarctions. We first selected 97 patients with AChA territory infarctions who at least had infarcts in the PLIC and/or the retrolenticular part of internal capsule. Seven patients who showed extensive infarction involving the temporal lobe or the midbrain, were excluded remaining 90 patients.

We considered that the AchA supply the medial temporal lobe, the midbrain, the PLIC, the retro-lenticular part of the PLIC, the lateral geniculate body, the genu of the internal capsule, the medial segment of the globus pallidus, the retro-lenticular part of the internal capsule and the lateral thalamic nuclei [1–6]. Moreover, we carefully excluded infarcts that were located in the structures presumed to be supplied by the lenticurostriate arteries such as the entire putamen, the head of the caudate nucleus, the anterior limb of internal capsule and the corona radiata. Moreover, a single infarct restricted within the genu of the internal capsule and/or antero-ventral thalamus that may be supplied by the tuberothalamic artery was also excluded. As the lateral thalamus is partly supplied by the AChA, thalamic infarcts caused by thalamogenuclate artery are carefully differentiate using the coronal and sagittal section. The institutional review board of Kyoto Second Red Cross Hospital approved this study and informed consent was obtained from all patients.

2.2. Clinical information

Hypertension was defined as the previous or present use of antihypertensive agents or a systolic blood pressure (SBP) > 140 mmHg and/or a diastolic BP (DBP) > 90 mmHg. Diabetes mellitus was defined as the previous or present use of an anti-diabetic medication or either a fasting glucose level ≥ 126 mg/dl or HbA1c is > 6.5%. Dyslipidemia was defined as the previous or present use of a lipid-lowering agent, fasting total cholesterol level > 220 mg/dl or LDL cholesterol level > 140 mg/dl. Cigarette smoking was defined as regular smoking at the time of stroke or had quit smoking less than two years prior. Systolic blood pressures (SBP) and diastolic blood pressures (DBP) were measured and recorded soon after admission.

2.3. Magnetic resonance imaging

All patients underwent MRI and MRA. MRI scans were performed using 1.5-Tesla superconducting magnets (Gyroscan Intera Achieva 1.5 Pulsar, Philips). Each MRI was performed within three days after ictus. Diffusion-weighted scans (Repetition Time: 1861 ms, Echo Time: 69 msec, Echo-planar imaging factor: 37) and T2-weighted scans (Repetition Time: 3500 ms, Echo Time: 100 ms) were obtained at a slice thickness of 5 mm. The three-dimensional time-of-flight images (Repetition time: 25 ms, Echo Time: 6.9 ms, flip angle: 18°, 210-mm field of view, the partition of 64, 219 × 512 acquisition matrix, and one signal average, for a total imaging time of 4 min 33 s) were acquired in

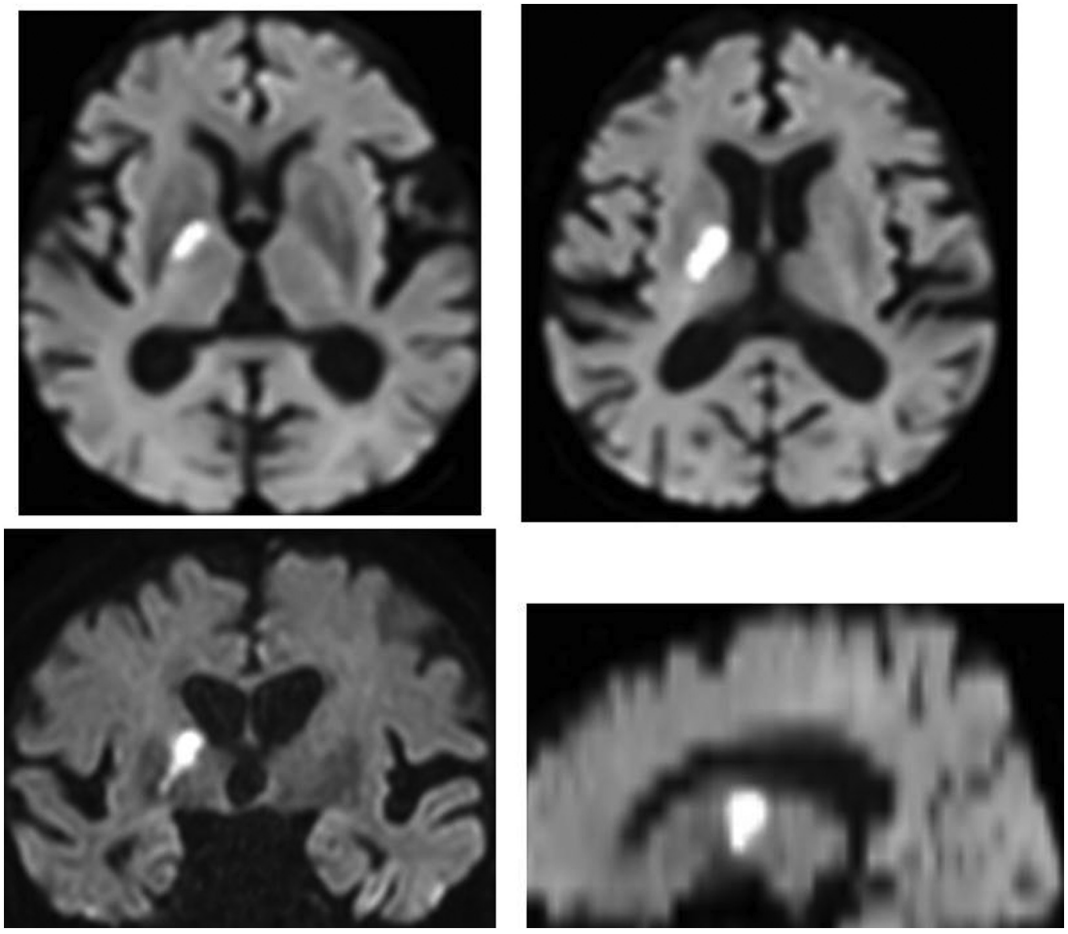

Fig. 1. An infarct of the anterior type. Upper: axial view. Lower left: coronal view. Lower right: sagittal view.

the axial plane. Diffusion-weighted scans were generally performed for axial and coronal slices. We then obtained sagittal reformatted images by using a multi-planar reconstruction (MPR) algorithm that is a method of displaying three-dimensional datasets for all patients [13].

2.4. The topography of infarcts of the AChA

We identified anatomical structures that are considered to be supplied by AChA referring to the template developed by Hamoir et al. [3]: the genu of internal capsule (GIC), the lateral thalamus (LT), the medial segment of pallidum (MSP), the posterior part of putamen (PPP), the lateral geniculate body (LGB), the retro-lenticular part of internal capsule (RLIC), and the posterior corona radiata (PCR).

2.5. Patient classification

We classified patients into four different types: 1. The anterior type; the infarcts involved the anterior one-third or half of the PLIC with or without the genu of the internal capsule, 2. The posterior type; the infarcts involve the caudal two-third part of the PLIC with or without the retro-lenticular portion of the internal capsule, 3. The combined type; the infarcts of anterior type extended to the end point of the PLIC and 4. The dot type; the infarcts were restricted within PLIC and did not exceed 10 mm in diameter in axial slice. Baseline characteristics, symptom and/or sign, neurological functions and the topography of infarcts were compared between the four groups.

2.6. Functional evaluation

We evaluated the neurological manifestations on admission and assessed the National Institutes of Health Stroke Scale (NIHSS). Functional outcome was evaluated by modified Rankin Scale (mRS) on discharge.

2.7. Stroke mechanism

Intracranial atherosclerosis was evaluated by MRA. Relevant internal carotid artery diseases in the parasellar portion were classified into 4 grades as normal, mild stenosis ($\leq$50% stenosis) and severe stenosis ($>$ 50%) and occlusion. Whole intracranial atherosclerosis including anterior cerebral artery and middle cerebral artery was also assessed based on their maximum stenosis. Classification of grades was same as that in the relevant internal carotid artery. Atherothrombotic disease was defined when relevant internal carotid artery showed severe stenosis or occlusion. Penetrating artery disease was defined as patients in whom severe stenosis in the ipsilateral internal carotid artery nor other potential source of embolism were not identified. Embolic stroke was defined as abrupt onset with identified potential sources of embolism such as atrial fibrillation, aortic atheroma with $\geq$5 mm plaque in aortic arch or significant patent foramen ovale with leg venous thrombus.

2.8. Statistical analysis

One-way analysis of variance (ANOVA) for numerical variables such as ages and infarct sizes, Kruskal-Wallis test for ordinal variables such

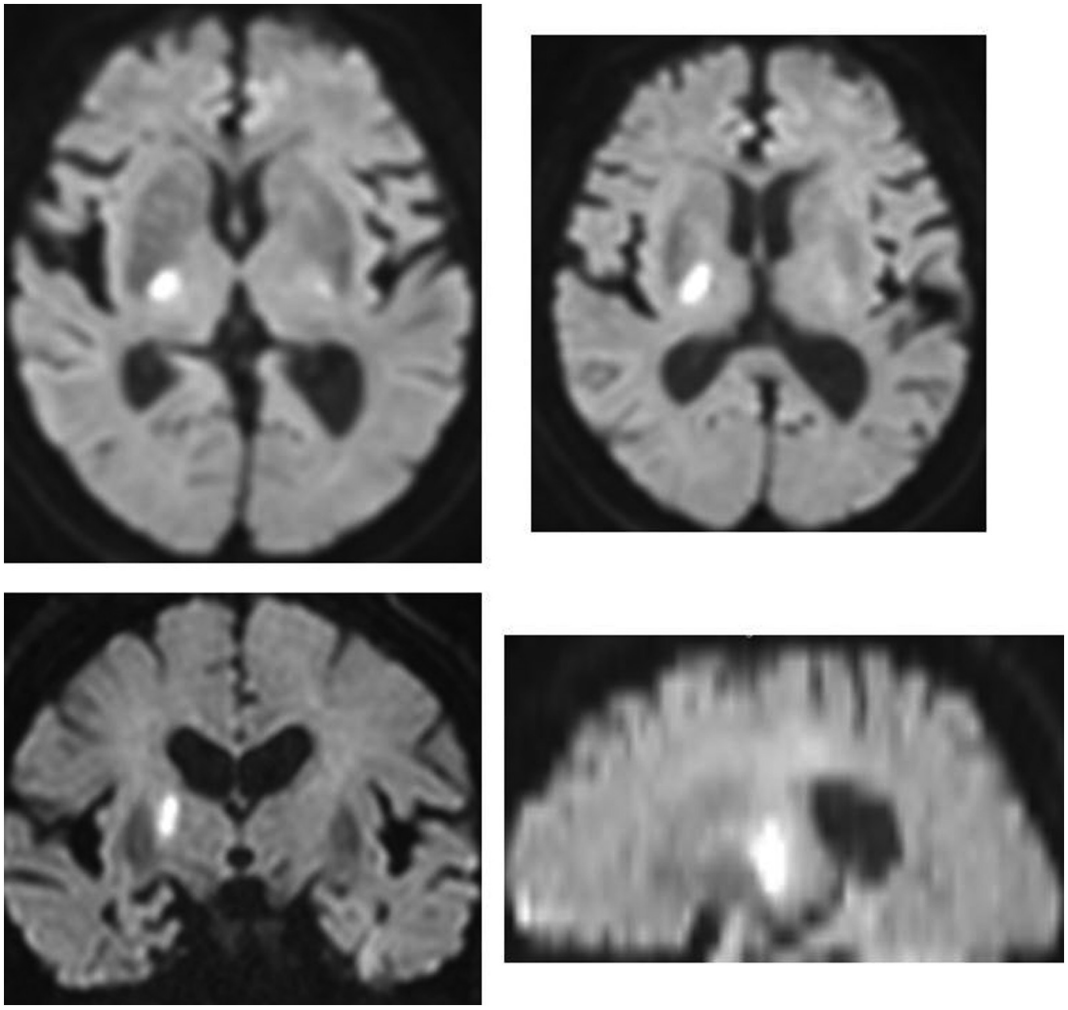

Fig. 2. An infarct of the posterior type. Upper: axial view. Lower left: coronal view. Lower right: sagittal view.

as NIHSS and mRS and Pearson's chi-square test for nominal variables were performed to compare between five different groups. The *p*-value of < 0.05 was considered statistically significant. All statistical analysis was performed with JMP12 (SAS Institute, Cary NC).

3. Results

3.1. Characteristics of patients

Patients with the posterior type were most prevalent accounting for 46 patients (51.1%), succeeded by 28 patients with the dot type (31.1%), 9 with the combined type (10%) and 7 with the anterior type (7.7%). The prevalence and degree of intracranial atherosclerosis were generally low and minor. Even so, the combined type tended to have atherosclerosis to a certain degree and the stroke mechanism of penetrating artery disease was significantly low in the combined type. (Table 1).

3.2. Neurological manifestations

Motor paresis frequently occurred for 77.6% including hemiparesis (47.7%) and mono-paresis of upper limb (15.5%) and lower limb (14.4%). Dysarthria was the second most frequent symptom accounting for 67%, succeeded by facial paresis for 41.9% and sensory deficits for 22.3%. (Table 2).

3.3. The topography of infarcts of the AChA

The representative patients with three types were shown in the Figs. 1 through 3. The anterior type located in the inferior and rostral portion of the PLIC involving the genu and the globus pallidus (Fig. 1A, B). In the coronal section, the infarct located upward in the medial part of pallidocapsular area (Fig. 1B). In the sagittal section, the infarct located in rostro-caudally oblique direction in the anterior or middle of the lentiform nucleus (Fig. 1C). The all 7 patients with anterior type are displayed in the Fig. 4. The posterior group located in the posterior two third of the PLIC involving the lateral thalamus (Fig. 2A, B). In sagittal section, the infarct located upright in the posterior part of the lentiform nucleus (Fig. 2C). The combined type located in the long area of the PLIC involving the medial part of the globus pallidus and the posterior part of the putamen (Fig. 3).

The topography of infarcts in the 4 groups was given in Table 3. The involvement of the lentiform nucleus including the MSP and the PPP in the anterior type was 71.4%, while that in the posterior group were 13.0%. In contrast, the involvement of the LT in the posterior group was 71.7%, while there was no involvement in the anterior type. In the combined type, the lentiform nucleuses were prevalently involved accounting for 77.5% in the MSP and 55.5% in the PPP as well as the LT (55.6%).

4. Discussion

The present study revealed that the infarct domain of the AChA in

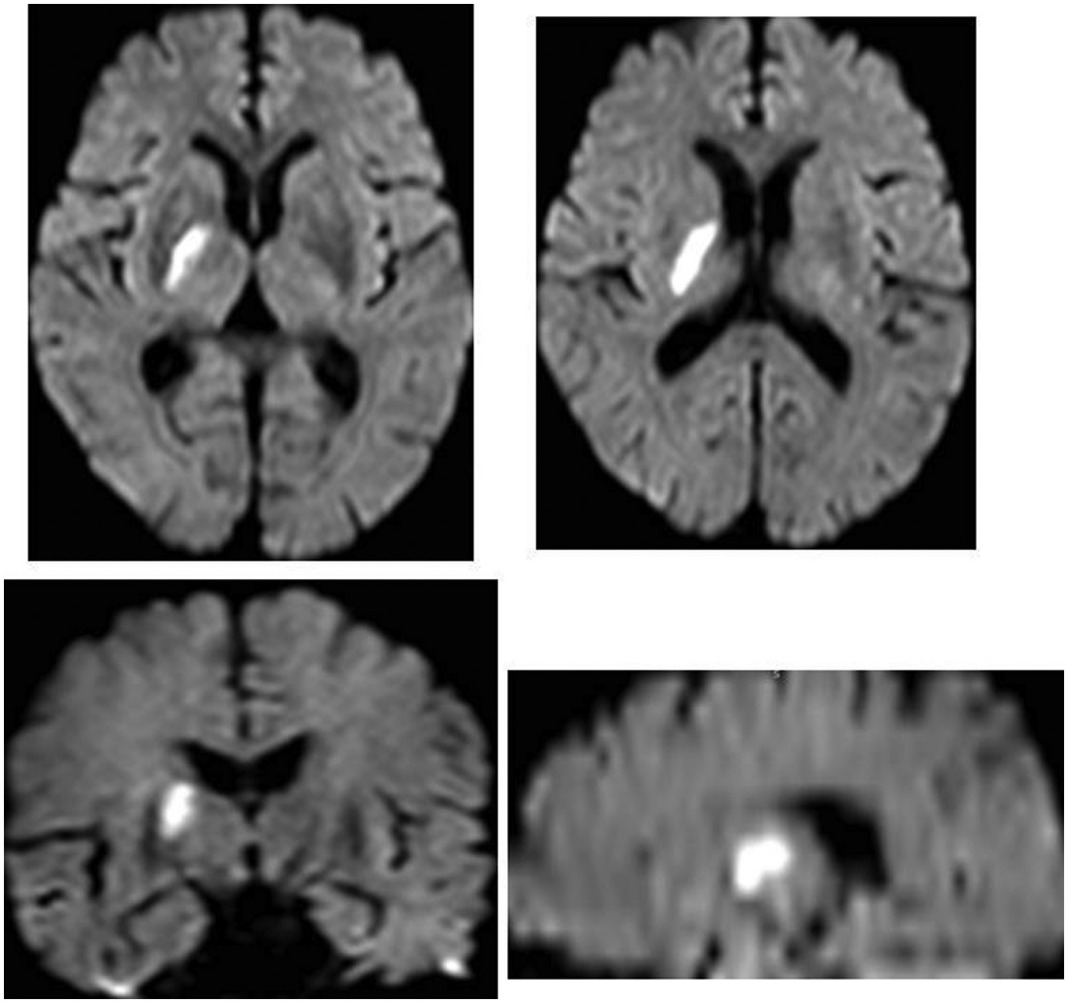

Fig. 3. An infarct of the combined type. Upper: axial view. Lower left: coronal view. Lower right: sagittal view.

the PLIC can be differentiated into two groups with the anterior and the posterior types. The anterior type preferentially involved the medial part of the globus pallidus for 71.4% and no patients involved the lateral thalamus. On the contrary, the posterior group involved the medial part of the globus pallidus only for 13.4% and the lateral thalamus for 71.4%. These two types of infarct domains are almost mutually exclusive albeit certain overlapping. Anatomical structures that were affected by the infarcts in the present study can mostly be explained by the descriptions by Rhoton et al. [10] and Marinković et al. [11]. The anterior type may be caused by the occlusion of the proximal branches and the pPLIC type caused by the distal branches, both of which arise from the cisternal segment of the AChA.

The cisternal segment of the AChA gives off three main groups of branches including the lateral branches that supply the medial temporal lobe, the medial branches that supply the cerebral peduncle or some other parts of the midbrain and the superior branches that supply the internal capsule and the basal ganglia. Rhoton et al. further discriminated superior branches into anterior branches that supply the genu of the internal capsule and/or the medial segment of the globus pallidus and distal branches that supply the posterior two-third of PLIC, retro-lenticular part of the internal capsule and the lateral thalamic nuclei [10]. Marinković et al. subsequently confirmed such discrimination of two types of superior branches as the proximal perforators and distal perforators under the stereoscopic microscope. They coined distal perforators as the capsulothalamic artery [11]. Takahashi et al. studied the distribution of the AChA by performing micro-angiography of cadaver brain on multi-planar MR images [12]. They observed more than two different branches irrigated the PLIC and illustrated in their cartoon. Aforementioned three researchers appear to be on the same page for two different branches supplying the PLIC.

The infarct of the anterior branch of AChA can be discriminated from that of the lenticulostriate arteries or the perforators of the internal carotid artery, because they are located along the PLIC in axial view and run upward and caudally in the middle of the lentiform nucleus in sagittal view. The medial branches of the lenticulostriate arteries course upward and rostrally usually supplying the areas of anterior limb of the internal capsule and the caudate head [8]. The perforators from the internal carotid artery and anterior cerebral artery may course differently.

The prevalence of the posterior type was 51.1% and that of the anterior type was 7.7%.

The low prevalence of the anterior type may be explained by the anatomical constellation of the proximal branches being smaller than the distal branches [12]. Moreover, there may be a variation of the vascular territory and a reciprocal relationship for supplying the anterior PLIC between the lenticulostriate arteries, the internal carotid artery and the AChA. Thus the prevalence of the anterior type was not so high as that of the posterior type accounting for 1/7.

The combined type extended obliquely in the rostro-caudal direction (Fig. 3C). The combined type preferentially involved the medial segment of the globus pallidus (77.7%) and the posterior part of the putamen (55.5%) rather than lateral thalamus (33.3%). Thus, this type appears to be closer to the aPLIC type rather than the pPLIC type. The proximal branches might be dominantly developed and thus irrigate the

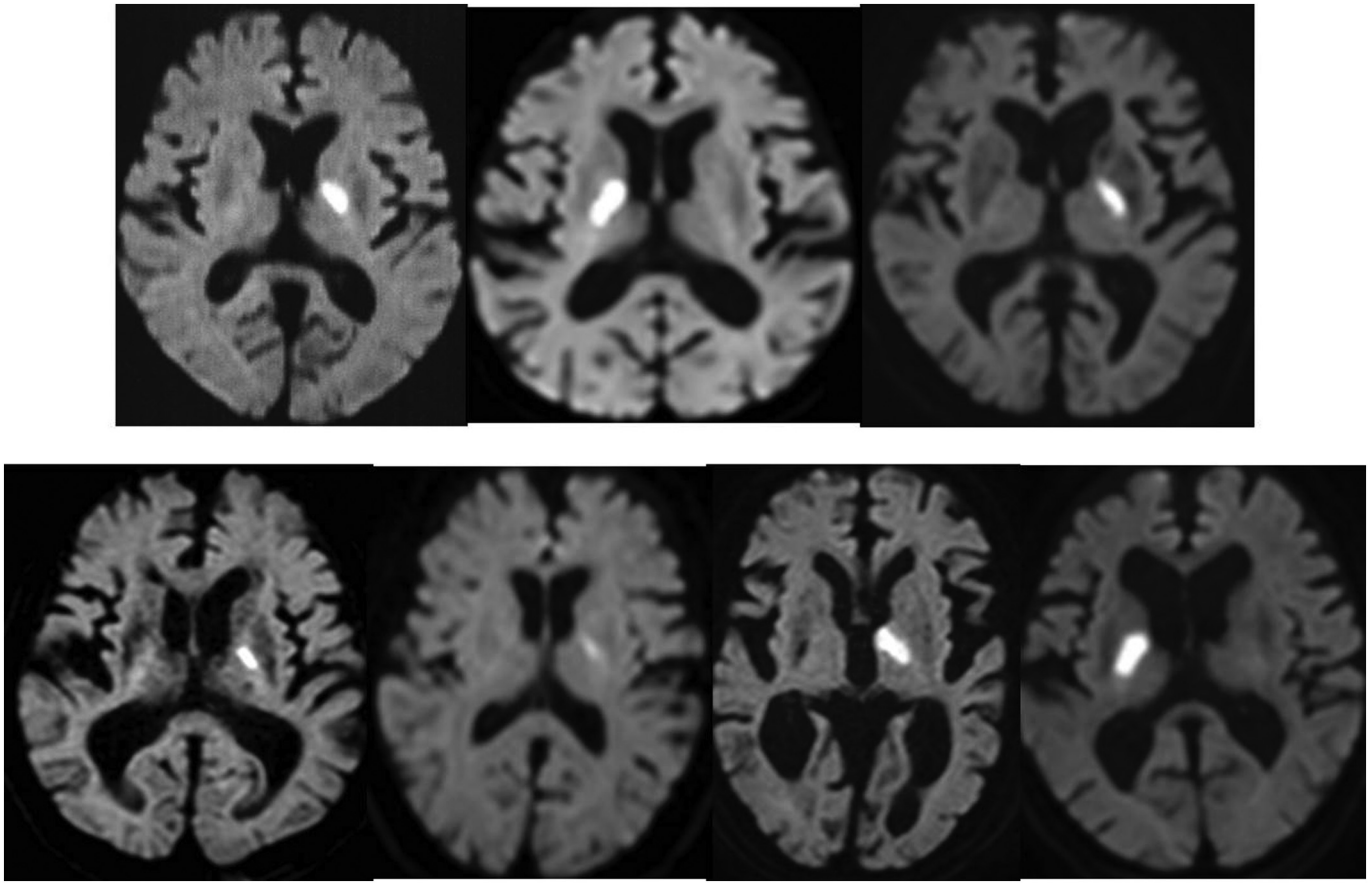

Fig. 4. Seven patients with anterior type infarction.

Table 3
The topography of infarcts in the 4 groups. Infarct sizes are expressed as means ± SD.

Group	Total	Anterior	Posterior	Combined	Dot	p values
n (%)	90	7 (7.7)	46 (51.1)	9 (10.0)	28 (31.1)	
GIC	21 (22.5)	7 (100)	0	9 (100)	0	< 0.0001
LT	39 (43.3)	0	33 (71.7)	5 (55.6)	1 (3.5)	< 0.0001
MSP	19 (21.1)	5 (71.4)	6 (13.0)	7 (77.7)	1 (3.5)	< 0.0001
PPP	14 (15.5)	3 (42.6)	6 (13.0)	5 (55.5)	0	0.0001
LGB	4 (4.4)	0	2 (4.2)	2 (22.2)	0	ns
RPIC	16 (17.7)	0	8 (17.3)	4 (44.4)	4 (14.2)	ns
PCR	11 (12.2)	0	9 (19.5)	2 (22.2)	0	0.045
Size (mm)						
Horizontal size	13.7 ± 6.8	16.9 ± 2.4	14.0 ± 3.9	26.0 ± 4.07	7.3 ± 2.2	< 0.0001
Vertical size	13.1 ± 5.4	15.4 ± 4.1	12.3 ± 4.3	19.4 ± 3.0	8.1 ± 2.3	< 0.0001

Values are n (%). Abbreviations; GIC: the genu of internal capsule, LT: the lateral thalamus, MSP: the medial segment of pallidum, PPP: the posterior part of putamen, LGB: the lateral geniculate body, RLIC: the retro-lenticular part of internal capsule, PCR: the posterior corona radiata.

total PLIC as a kind of variation. Another possible mechanism is that proximal and distal branches formed a common trunk when arising from the cisternal segment of the AChA and branch atheromatous disease may occur within the common trunk [14]. As the combined type involved anterior one third, total patients in whom the anterior one third of the PLIC was involved add up to 16 (17%).

Dysarthria, facial palsy and abulia were preferentially found in the anterior one third involved patients including the anterior and the combined types (87.5%, 68.7% and 50%, respectively). As the genu of the capsule and the adjacent part contain the corticobulbar bundle, facial palsy and dysarthria may occur [15]. Abulia was also known to be caused by the insult of the genu of the capsule.

Although embolic mechanism was highlighted in the previous reports [7] small artery disease was the main mechanism in the present study which is in line with recent studies [1–6]. Atherothrombotic mechanism was also rare. The dot type may be caused by lipohyalinotic disease in the small-sized arteries, while the combined type may be related to more atheromatous condition such as branch atheromatous disease [14].

The posterior corona radiata has been disputed as AChA-supplied anatomic structure [1]. In the present study, we found only 12 patients in whom the posterior corona radiate was involved (12.9%). Nine patients were found in the posterior type (19.5%) and two were in the combined type (22.2%). We identified infarcts located in the PLIC extended upward into the posterior corona radiata utilizing a multiplanar reconstruction that enables visualization of the axial, coronal and sagittal sections of DWI images. There may be a mutual entry relationship between the AChA and the lentiuclostriate arteries with wide variations

in the posterior corona radiata.

The limitation of the present study should be addressed. First, as the anatomical structures involved by infarcts were measured with our eye, boundary zone could be vaguely identified. Second, as a 1.5 T MRI was used, image resolutions are not so high, especially in sagittal section. Third, to correlate microsurgical anatomy described in previous reports with infarcts detected by the diffusion weighted images can in our study be subjective.

5. Conclusions

There were 7 patients with the anterior type (7.7%), 46 patients with the posterior type (51.1%) and 9 patients with the combined type (10%). Our hypothesis based on the previous descriptions on microsurgical anatomy was proved studying the consecutive patients with PLIC infarction. The anterior type may be caused by the occlusion of the proximal branches, while the posterior type may be by the distal branches, both of which arise from the cisternal segment of the AChA. Although the various perforators arising from middle cerebral artery, internal carotid artery and anterior artery can contribute to supply the anterior one third of the PLIC, the proximal branch of the AChA also supply the concerned territory, albeit the low prevalence. When a deep small lesion is found in the anterior part of the PLIC, the AChA territory infarction should be first cited as differential diagnosis.

Author contributions

Conception and study design: Takashi Koizumi, Yasumasa Yamamoto.

Acquisition and analysis of data: Takashi Koizumi, Yoshinari Nagakane.

Wrote the manuscript: Takashi Koizumi, Yasumasa Yamamoto, Yasuhiro Tomii.

All authors contributed to manuscript revisions and approve the submitted article.

Declaration of Competing Interest

There is no potential conflicts of interest for all authors.

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