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Posterior Cerebral Artery Infarction: Diffusion-Weighted MRI Analysis of 205 Patients

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Key Words
Posterior cerebral artery · Brain infarction · Diffusion-weighted MRI · Magnetic resonance angiography · Etiology

Abstract
Background and Purpose: Although cardiac embolism has been shown to be the leading stroke mechanism of posterior cerebral artery (PCA) territory infarction, intrinsic PCA atherosclerotic disease may play a more important role in regions where intracranial diseases are prevalent. We aimed to assess the etiologies and stroke pattern of PCA territory infarction in a Korean population. ***Method:*** We reviewed consecutive patients with acute PCA territory infarction who underwent diffusion-weighted MRI (DWI) and magnetic resonance angiography (MRA) within 7 days after onset. ***Results:*** A total of 205 patients (male 56%, mean age 65.4 ± 12.4 years) were recruited. 'Superficial', 'deep' and 'superficial plus deep' infarcts accounted for 26.3, 47.8 and 25.9% of infarcts, respectively. There were 126 patients who had infarcts limited to the PCA territory, whereas 79 patients had concomitant infarcts in other territories. Large artery atherosclerosis (LAA, 42.4%) was the most frequent etiology, followed by cardiogenic embolism (20%), small-vessel occlusion (20%), undetermined (18%) and other determined (3%) etiology. Among the 87 patients with LAA, 38 had intrinsic PCA disease without atherosclerosis in the proximal vessels. In these patients, stroke mechanisms included atheromatous branch occlusion (n = 19), in situ thrombotic occlusion (n = 11) and artery-to-artery embolism (n = 8). While the occipital area was the most frequently involved in general, the ventrolateral thalamic area was more frequently involved than the occipital area in patients with intrinsic PCA atherosclerotic disease. ***Conclusion:*** The patterns of PCA territory infarction are different according to underlying etiologies. In our population, intrinsic atherosclerotic disease is a relatively important cause of PCA territory infarction that produces strokes through a variety of mechanisms.

Posterior cerebral artery (PCA) territory infarction is not uncommon. However, most previous reports concentrated on either superficial [1–4] or deep [5, 6] infarction, while comprehensive studies encompassing both superficial and deep infarction have been rare. Moreover, the etiologies and pathogenic mechanisms of PCA territory infarction remain less clear. Prior studies have shown that the leading etiology is embolism from the heart or proximal vertebrobasilar atherosclerotic disease, while intrinsic atherosclerosis of the PCA was considered uncommon [1–3, 7–9]. However, in the majority of previous studies, angiographic evaluation was done only in a lim-

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ited number of patients. Thorough angiographic evaluation along with modern imaging tools such as diffusion-weighted MRI (DWI) may allow us to examine more precisely the etiology and stroke pattern in a PCA territory infarction.

Furthermore, most of the previous studies were done in western countries, and information from Asians has been scarce. Since intracranial diseases are more common in Asians than in Caucasians [10–14], PCA atherosclerotic disease may play an important role in Asian patients. However, the incidence and detailed stroke mechanisms of PCA atherosclerosis remain yet to be investigated. The purpose of our study was to assess the etiologies and stroke pattern of PCA territory infarction in a Korean population using DWI and magnetic resonance angiography (MRA). In particular, we wished to examine the prevalence and the stroke mechanism of PCA atherosclerosis in these patients.

Patients and Methods

Patient Selection

We retrospectively reviewed the data from prospectively maintained registries of patients with acute stroke who were admitted to the Asan Medical Center between January 2005 and July 2007. We chose patients who (1) had acute PCA territory infarcts according to previously published anatomic studies [15–17] and (2) underwent DWI and MRA within 7 days after the onset of acute symptom. We included patients who had concomitant acute infarcts identified by DWI in other vascular territories, such as the middle cerebral artery (MCA), basilar artery (BA) and posterior inferior cerebellar arteries (PICA).

We used the following definitions when examining the risk factor prevalence. The presence of hypertension was defined as the prior use of anti-hypertensives or systolic blood pressure >140 mm Hg and/or diastolic blood pressure >90 mm Hg on repeated examinations. Diabetes was defined as the use of anti-diabetic treatments or fasting (≥126 mg/dl) and postprandial 2-hour (≥200 mg/dl) blood glucose. Hyperlipidemia was defined as present when total cholesterol was >200 mg/dl or low-density lipoprotein cholesterol was >130 mg/dl. Current smoking and heavy alcohol drinking (consumption of >60 g alcohol/day) were considered as risk factors [18]. We also recorded 'other potential' risk factors including cancer, coagulopathy, migraine, recent dehydration and head or neck trauma.

An electrocardiogram (ECG) was performed in all patients. Trans-thoracic echocardiography (TTE), trans-esophageal echocardiography (TEE), Holter monitoring and thallium single photon emission computed tomography were performed in selected patients: (1) who had a history of, or clinical or ECG evidence of, cardiac abnormalities, (2) in whom infective or non-bacterial endocarditis was suspected, (3) who were young (aged <50 years) without risk factors for atherosclerosis, (4) who were suspected to have an embolism clinically but with an unclear source.

Imaging Analysis

MRI examinations were obtained using either a 1.5 T or 3.0 T MR imaging unit. We performed DWI, 3D-time-of-flight (TOF)-MRA, and 3D-contrast-enhanced MRA. T1-, T2-weighted and fluid attenuated inversion recovery (FLAIR) were also performed. The common MRI parameters for DWI were a slice of thickness of 5 mm, an interslice gap of 2 mm, 20 axial slices and a field-of-view of 250 mm. DWI parameters included a repetition time (TR) of 7,500 ms, an echo time (TE) of 84 ms, a matrix number of 128 × 128, and 2 b-values of 0 and 1,000 s/mm^2. Common MRA parameters included a flip angle of 20°, a matrix number of 512 × 512, and a field-of-view of 250 mm. A TOF-MRA of the circle of Willis was performed with a TR of 25 ms and TE of 2 ms. A contrast enhanced-MRA from the aortic arch to the level of the central skull base was obtained using an intravenous bolus injection of 20 ml (3–4 ml/s) gadopentetate dimeglumine (Magnevist; Schering, Berlin, Germany) with a TR of 6 ms and TE of 1 ms. Two hundred patients underwent TOF-MRA for the visualization of intracranial vessels and contrast enhanced-MRA for the visualization of extracranial vessels.

Follow-up MRI and MRA examinations (at 4–6 days after the initial MRI) were done in selected patients with presumed etiologies of large-artery atherosclerosis (LAA) or cardiogenic embolism (CE) (for definition, see below) to evaluate the status (persistence or recanalization) of stenotic or occlusive vessels. The follow-up MRA results were used as a reinforcing database for the subtype classification. The MRA data were reviewed by a neurologist (E.L.) and a neuroradiologist, separately. Discrepancies between those diagnoses were settled by consensus discussion with another stroke neurologist (J.S.K.).

Assessment of the Pattern and Etiologies of the PCA Territory Infarct

Evaluation of Arterial Stenoses Based on MRA

We examined the PCA at 3 segments [15, 17]: P1, between the basilar bifurcation and the posterior communicating artery (PCoA), P2, between the PCoA and the posterior aspect of the midbrain, and P3, from the pulvinar to the anterior limit of the calcarine fissure. We did not divide the P4 segment (segment distal to P3) from P3 because it was difficult to evaluate the P4 using MRA. The vertebral artery (VA) was examined at extracranial (V1, V2, and V3) and intracranial (V4) sites [19].

The degree of arterial stenosis was categorized into mild (<50% diameter reduction), moderate (≥50% diameter reduction with complete distal flow), severe (segmental nonvisualization of artery), occlusion, or aplasia/hypoplasia (defined in vertebrobasilar arteries and P1 segment). We interpreted a nonvisualization of the entire VA as 'aplasia', and a diffuse homogeneous narrowing of the entire VA as 'hypoplasia' [20]. Arbitrarily, we interpreted a non-visualization of the P1 segment with a fetal type PCA as aplasia rather than occlusion. However, an asymmetrical or irregular narrowing of a P1 segment was considered as a P1 stenosis. If there was any possibility of pulsation or motion artifacts in the V1 [20], we did not consider the lesion as abnormal. We considered aortic arch atherosclerosis as a cause of infarction only when TEE showed a 'complex' protruding atheroma more than 4-mm thick, mobile debris or plaque ulceration [21].

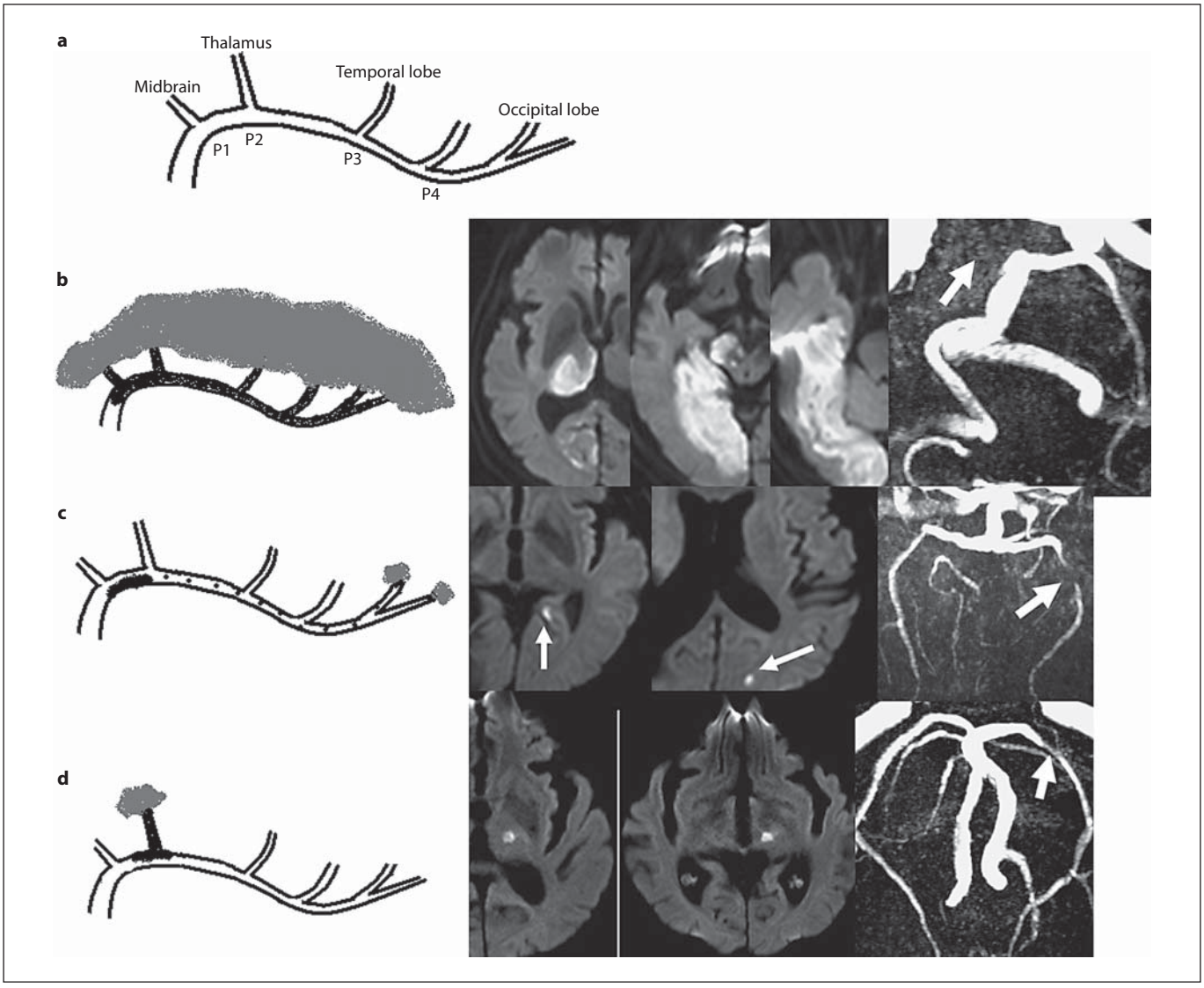

Fig. 1. Schematic drawing and illustrative examples of the diverse patterns of PCA territory infarction in LAA, as assessed by DWI and MRA. The stenosis or occlusion of PCA shown in these patients was persistent on follow-up MRA. **a** Schematic drawing illustrating PCA branches supplying midbrain, thalamus and temporo-occipital area. **b** In situ thrombotic occlusion; the whole right PCA territory infarction was produced by the right P1 occlusion (arrow). **c** AA embolism; the left PCA P2 severe stenosis (thick arrow) presumably produced the multiple small embolic infarcts (thin arrows) scattered in occipital lobe. **d** AB occlusion; the left P2 stenosis (arrow) caused the left lateral thalamic infarction probably by occluding the orifice of the thalamogenicular perforating artery.

Distribution of Infarcts Based on DWI Findings

Based on the DWI findings, we categorized the anatomical distribution of infarcts into 'superficial' (pial), 'deep' (perforating), or 'superficial plus deep' territories [16, 17]. Anterior temporal, posterior temporal branches, calcarine and parieto-occipital artery territories were included in the 'superficial' territories, while the 'deep' territories included the areas supplied by the thalamoperforating and thalamogeniculate branches, polar artery branches, and the posterior choroidal artery.

Presumed Etiologies of Infarction

Using a modified version of the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification [22], the presumed etiologies of infarction were categorized by 2 neurologists (E.L. and J.S.K.) as follows:

(1) LAA etiology: defined when there was definite stenosis or occlusion of the PCA, BA or VA that was considered responsible for the referent ischemic lesion. We divided the LAA into the following categories (fig. 1): (i) in situ thrombotic occlusion, when

there was an infarct just distal to the steno-occluded site involving the whole or most of the involved territory (e.g. a persistent P1 occlusion with infarcts involving the midbrain, ventrolateral thalamus, lateral geniculate body and temporo-occipital lobe, fig. 1b), (ii) artery-to-artery (AA) embolism, when there was a moderate to severe stenosis or occlusion in the VA (in the V1 segment, only severe stenosis or occlusion was considered), BA, PCA, or 'complex' aortic atheroma with infarcts occurring in the remote area not adjacent to the diseased vessel (e.g., a persistent P2 stenosis with small scattered infarcts in the occipital area, fig. 1c), (iii) atheromatous branch (AB) occlusion [6, 23, 24], when the infarcts were on the territory of one or a few perforating branches arising from the stenosed (of any degree) PCA that presumably occluded the orifice of perforators (e.g. a persistent P2 stenosis with ventrolateral thalamic infarction, fig. 1d), and (iv) AA+AB, when infarcts were located in deep perforator territory adjacent to the stenosed PCA and also in the remote area.

(2) CE etiology: defined when there was emboligenic heart disease without significant atherosclerosis such as atrial fibrillation, valvular heart disease, dilated cardiomyopathy, endocarditis, recent myocardial infarction, and severe heart failure (ejection fraction <30%). Patent foramen ovale with right-to-left shunt was considered a potential cause of embolism by consensus after considering the patient's age, risk factors and clinical findings.

(3) Small-vessel occlusion (SVO) etiology: defined when the size of the lesion was <1.5 cm without evidence of clinically relevant atherosclerosis, CE or other determined etiologies.

(4) Undetermined etiology (presence of 2 or more causes, or unknown causes).

(5) Other determined etiology – arterial dissection, etc.

Statistical Analysis

Statistical analysis for risk factors and stroke etiologies was performed using SPSS 12.0 for Windows Descriptive Statistics. The categorical values and differences in characteristics between groups were examined with the χ^2 test; the Fisher's exact test was used when the number was small. A t test was used for comparing the mean age between two groups. Differences were considered significant when $p < 0.05$.

Results

Patient Characteristics and Risk Factors

Between January 2005 and July 2007, there were 229 consecutive patients who developed PCA territory infarction. This corresponded to 12.8% of the admitted ischemic stroke patients. 223 patients underwent DWI, of which 210 also underwent MRA. Five patients were further excluded as the orifice of VA was not properly evaluated. Thus, 205 (89.5%) patients became the subjects of this study in whom both the intracranial and extracranial vessels were appropriately evaluated. In addition, T1, T2, FLAIR, perfusion weighted MRI were performed in 9.3%, 47.3%, 73.7% and 39.0% of the 205 patients, respectively. Follow-up DWI and MRA were performed in 121 patients (59%) and in 72 patients (35%), respectively.

There were 114 men and 91 women with an age ranging from 23 to 89 years (mean 65.1 ± 12.4 years). Hypertension (n = 144, 70.2%) was the most prevalent risk factor, followed by diabetes (n = 74, 36.1%), smoking (n = 60, 29.3%), hyperlipidemia (n = 46, 36.1%), previous stroke (n = 35, 17.1%), heavy alcohol drinking (n = 29, 14.1%), and 'other potential' risk factors (n = 21, 10.2%). Sixty-nine patients (33.7%) had heart disease, the most common being atrial fibrillation (n = 19), followed by coronary heart disease (n = 13), patent foramen ovale with right to left shunt (n = 10), valvular heart disease (n = 2), congestive heart failure (n = 2) and others (n = 7). Sixteen patients had 2 or more heart diseases diagnosed. Among the 69 cardiac diseases, 56 were considered emboligenic. An ECG was performed in all patients, and TTE, TEE and Holter monitoring were performed in 92 (44.9%), 59 (28.8%) and 55 patients (26.8%), respectively. Thirty-six patients had atherosclerosis of the aortic arch, of which 8 had additionally a 'complex' atheroma on TEE.

Presumed Etiologies and Stroke Mechanism

As presented in table 1, LAA (42.4%) was the most frequent etiology, followed by CE, SVO, undetermined etiology and other determined etiologies. Other determined etiologies (n = 6) included moyamoya disease (n = 1), hypereosinophilia (n = 2), VA dissection (n = 1), thrombotic thrombocytopenic purpura (n = 1), and adenocarcinoma related coagulopathy (n = 1). If we add the patients with 2 or more causes (undetermined etiology), the LAA etiology would reach to 100 patients (48.8%) and CE to 56 patients (27.3%).

In the patients with LAA (n = 87), stroke mechanisms included AA embolism (n = 47, 54%), AB occlusion (n = 22, 25.3%), in situ thrombotic occlusion (n = 11, 12.6%), and AA+AB (n = 7, 8%). Among these patients, 38 had pure intrinsic PCA disease without vertebrobasilar or aortic arch disease (pure PCA disease), whereas 49 patients had vertebrobasilar or aortic arch disease with or without PCA stenosis (other LAA). Among the patients with pure PCA disease, AB (n = 19) was the most prevalent mechanism, followed by in situ thrombotic occlusion (n = 11) and AA embolism (n = 8) (table 1; fig. 1). If we include PCA disease with concomitant vertebrobasilar and/or aortic arch diseases, the prevalence of PCA disease reached to 37.1% (76 patients, table 1). Compared with the pure PCA disease group, the other LAA group had a higher proportion of males, and more often had a superficial infarction, especially in the parieto-occipital area (table 2).

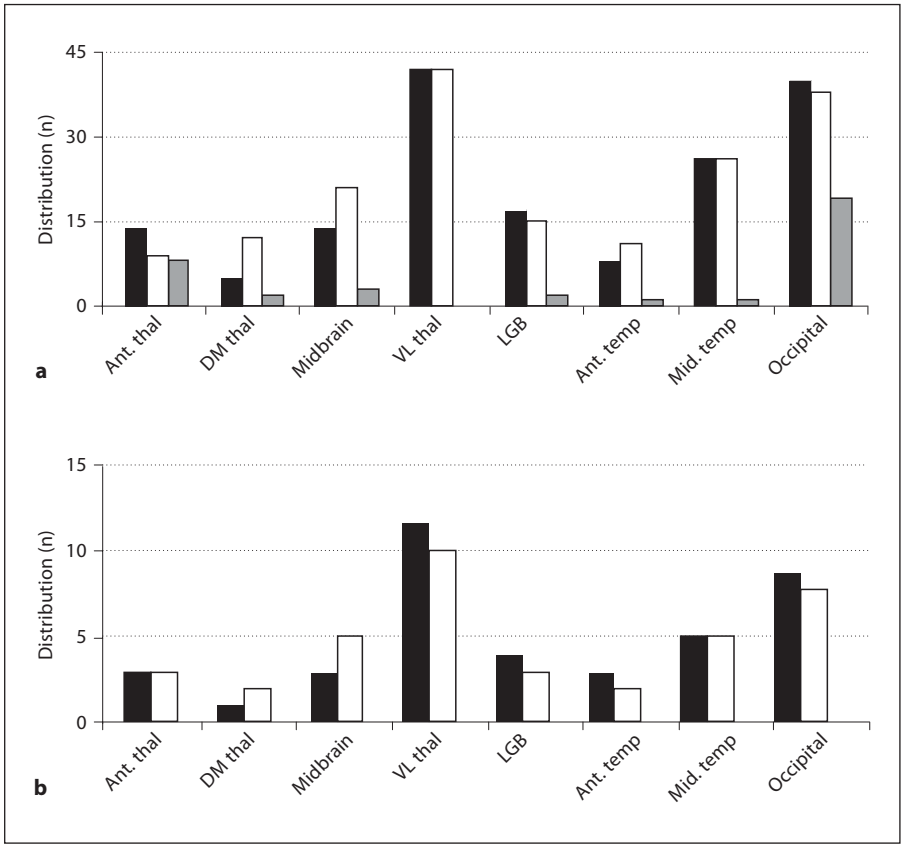

Fig. 2. Anatomical distribution of PCA territory infarcts. **a** The occipital lobe was the most frequently involved site, followed by ventrolateral thalamic infarct. **b** In 38 patients who had intrinsic PCA disease, ventrolateral thalamic infarct was the most frequent, followed by occipital lobe infarct. Black bar: Right; white bar: left; gray bar: both sides. Ant. thal = Anterior thalamus; DM thal = dorsomedial thalamus; VL thal = ventrolateral thalamus; LGB = lateral geniculate body; Ant. temp = anterior temporal lobe; Mid. temp = mid-temporal lobe; Occiptal = parieto-occipital area.

Table 1. Etiologies of 205 PCA territory infarcts

Etiologies	n = 205
LAA	87 (42.4%)
PCA disease only	38
In situ thrombotic occlusion	11
AA embolism	8
AB occlusion	19
Vertebrobasilar (VB) disease only	8
Aortic arch disease only	2
PCA + VB	36
PCA + aortic arch	1
VB + aortic arch	1
PCA + VB + aortic arch	1
CE	41 (20%)
SVO	41 (20%)
Undetermined etiology	30 (14.6%)
Two or more causes	15
LAA + CE	13
CE + other determined	2
Cryptogenic	13
Incomplete evaluation	2
Other determined etiology	6 (3%)

Table 2. Comparison between pure PCA disease and other LAA

	Pure PCA disease only (n = 38)	Other LAA (n = 49)	p value
Mean age ± SD, years	68.6 ± 1.0	67.8 ± 9.5	0.609
Males, %	44.7	75.5	0.006
Infarct pattern, %			0.046
Superficial	13.2	36.7	
Deep	52.6	36.7	
Superficial plus deep	34.2	26.5	
DWI lesions, %			
Anterior thalamus	15.8	22.5	0.446
Dorsomedial thalamus	7.9	10.2	0.377
Midbrain	21.1	24.5	0.637
Ventrolateral thalamus	57.9	24.5	0.006
Lateral geniculate body	18.4	10.2	0.383
Anterior thalamus	13.2	4.1	0.297
Mid-temporal lobe	26.3	22.5	0.575
Parieto-occipital area	44.7	59.1	0.020

Distribution of Infarcts Based on DWI Findings

The anatomical distributions of PCA territory infarction included the parieto-occipital area (n = 97), ventrolateral thalamic area (n = 84), mid-temporal area (n = 53), anterolateral midbrain (n = 38), lateral geniculate body (n = 34), anterior thalamus (n = 31), anterior temporal area (n = 20) and dorsomedial thalamus (n = 19) (fig. 2). The pattern of DWI lesions was different ($p < 0.001$) among the underlying etiologies. Among the 54 superficial, 98 deep and 53 superficial plus deep infarcts, SVO was the most common cause of deep infarcts (41.8%), while CE was the most common cause of superficial (29.6%) and superficial plus deep (30.2%) infarcts. Embolic 'superficial' and 'superficial plus deep' infarcts (39.0%) originated from an artery (AA embolism, 18.0%), the heart (CE, 15.6%) or both (5.4%).

'PCA Territory Only' Infarct versus 'PCA Territory Plus' Infarct

Among the 205 patients, 126 (61.5%) had infarcts limited to the PCA territory ('PCA territory only'), while 79 patients (38.5%) had concomitant infarcts in other territories ('PCA territory plus') including anterior (n = 29) and posterior (n = 39) circulation or both (n = 11). MCA (17.6%) territory infarcts were the most frequent, followed by the superior cerebellar artery (SCA, 13.2%), PICA (13.2%), BA (8.8%), anterior cerebral artery (ACA, 6.3%), anterior inferior cerebellar artery (4.5%) and anterior choroidal artery (1%) territory infarcts. As shown in table 3, the PCA territory plus group had a higher proportion of males, a higher prevalence of previous stroke history, and a higher proportion of CE and parieto-occipital infarcts than did the 'PCA territory only' group. Patients with concomitant anterior circulation stroke (n = 29) mostly had CE (n = 17) or unknown cause (n = 11), while simultaneous embolism from the internal carotid artery and VA occurred in one patient.

Table 3. Comparison of clinical characteristics in patients with PCA territory only and PCA territory plus infarct

	PCA territory only (n = 126)	PCA territory plus (n = 79)	p value
Mean age ± SD, years	63.8 ± 12.8	67.1 ± 11.7	0.163
Males, %	46.0	70.9	<0.001
Risk factors, %			
Hypertension	66.7	75.9	0.157
Diabetes mellitus	38.9	31.6	0.293
Smoking	25.4	35.4	0.124
Alcohol	12.7	16.5	0.452
Hyperlipidemia	32.6	20.0	0.348
Previous stroke	10.3	27.8	0.001
Heart disease	24.6	45.6	0.002
Etiology, %			<0.001
LAA	40.5	45.6	
CE	15.9	26.6	
SVO	32.5	0	
Undetermined etiology	11.1	20.3	
Other determined etiology	0	7.6	
Infarct pattern, %			<0.001
Superficial	14.3	45.6	
Deep	63.5	22.8	
Superficial plus deep	22.2	31.6	
DWI lesions, %			
Anterior thalamus (31)	13.5	17.7	0.528
Dorsomedial thalamus (19)	7.1	12.7	0.591
Midbrain (38)	14.3	25.3	0.071
Ventrolateral thalamus (84)	53.2	21.5	<0.001
Lateral geniculate body (34)	15.1	19.0	0.342
Anterior temporal (31)	9.5	10.1	0.836
Mid-temporal lobe (53)	22.2	31.6	0.327
Parieto-occipital area (97)	33.3	69.6	<0.001

Figures in parentheses denote numbers of infarcts.

Discussion

We examined etiologies and stroke mechanisms of PCA territory infarction. The merits of our study are that we included both superficial and deep PCA territory infarction and that DWI and angiographic evaluation were performed in all the patients. We found that LAA (42.4%) was the most common etiology of PCA territory infarct, and 18.5% of the patients had intrinsic PCA disease without embolic sources. Adding together PCA disease 'with either vertebrobasilar or aortic arch disease', the frequency of PCA atherosclerotic disease reached 37.1% (76 of 205 patients, table 1). In previous studies, the frequencies of intrinsic PCA disease were reported to be 1.7% [1], 8% [2], 8.9% [7], and 16% [8, 9], respectively, where conventional or MR angiographic evaluation was performed in 43.6% [1], 30.0% [2], 50.6% [7], and 49.6% [8, 9], respectively. In one study that showed a relatively high frequency of PCA disease (26%) [3], MRA was performed in all 137 patients. Their results, however, are not comparable to ours because they were concerned only with 'superficial' PCA territory infarction. Therefore, the higher incidence of intrinsic PCA atherosclerotic disease in our study seems to reflect a more comprehensive evaluation of the vascular status as well as the higher prevalence of intracranial atherosclerosis in Asians compared with Caucasians [12, 13, 25].

We also found that PCA atherosclerotic disease produces strokes through various mechanisms such as AA embolism, AB occlusion, AA+AB, and in situ thrombotic occlusion as observed in patients with intrinsic MCA or ACA diseases [26, 27]. Nevertheless, considering our previous results that demonstrated intracranial atherosclerosis was the major cause of stroke in either MCA (34.1%) [26] or ACA (61%) territory infarctions [27], the role of intracranial atherosclerosis as a cause of PCA territory infarction appears to be relatively minor. In other words, embolism is still an important mechanism of PCA territory infarction in our population. When all embolic sources were added (VA, BA disease, aortic arch disease and CE), embolic PCA territory infarcts accounted for 48.8%, a figure slightly lower than previous reports of the frequency of embolism (46 to 82%) [1, 7, 8, 28, 29]. The relatively low incidence of CE (20%) as compared to previous study results (31–40.5%) [2, 7] seems to reflect a relatively high frequency of intracranial PCA disease in our population, but this may also be related to the performance of the TTE and TEE in a limited number of cases. However, it is noteworthy that the incidence of CE is still higher than that shown in other studies of Korean stroke patients (11.6% [30], 15.2% [31]).

Consistent with previous reports [28], the occipital lobe was most frequently involved, followed by the ventrolateral thalamus and the mid-temporal area. However, when we consider intrinsic PCA diseases only, the most commonly involved area was the ventrolateral thalamus, which was followed by the occipital lobe, illustrating that perforator occlusion from P2 segment stenosis is an important stroke mechanism in intrinsic PCA disease. These results agree with the previous observation that subcortical infarction due to perforator occlusion is the major stroke mechanism in patients with intracranial MCA [26] or ACA [27] atherosclerotic disease. Our study showed that 38.5% of the patients had concomitant, acute infarcts in other vascular territories ('PCA territory plus'), a finding similar to the result from New England Medical Center Registry (39%) [7]. As expected, CE and superficial infarcts were more common in the PCA territory plus group than in the PCA territory only group. In addition, the proportion of males was higher and the history of previous stroke was more prevalent in the former group. Being male was also a factor differentiating other LAA from pure PCA disease, among the LAA patients. Perhaps this result might be related to an atherosclerotic involvement of more proximal vessels in males than in women in our population [18].

Our study has limitations. First, vascular evaluation was performed by MRA, but not conventional angiography. TOF-MRA may exaggerate the arterial stenosis and is less accurate in assessing the V1 and distal PCA segments. To avoid over-interpretation, we considered only a 'severe' V1 stenosis as a source of embolism, and this attempt might have an influence on the frequency of AA embolism. Second, echocardiography and Holter monitoring were performed in a limited number of patients. As discussed earlier, this might have underestimated the frequency of CE. However, we attempted to document the source of embolism whenever an embolism was suspected, and reinforced the diagnosis of intrinsic PCA disease by confirming the persistent stenosis on the follow-up angiogram. Finally, we included infarcts occurring in the tuberothalamic (polar artery) territory since the artery often arises from the PCA or posterior choroidal branch of the PCA. However, it may also stem from the PCoA rather than the PCA.

Despite these limitations, our data showed that intrinsic PCA diseases are more prevalent than previously recognized and that patterns of PCA territory infarction are different according to underlying etiologies. Although secondary stroke prevention strategy should generally be based on etiology (i.e. anticoagulation for CE, antiplatelets for a stroke of arterial origin), specific infarct patterns may alert physicians to employ certain etiology work-ups and different treatment strategies in consideration with underlying stroke mechanisms.

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References

1 Cals N, Devuyst G, Afsar N, Karapanayiotides T, Bogousslavsky J: Pure superficial posterior cerebral artery territory infarction in the Lausanne stroke registry. J Neurol 2002;249:855–861.

2 Steinke W, Mangold J, Schwartz A, Hennerici M: Mechanisms of infarction in the superficial posterior cerebral artery territory. J Neurol 1997;244:571–578.

3 Kumral E, Bayulkem G, Atac C, Alper Y: Spectrum of superficial posterior cerebral artery territory infarcts. Eur J Neurol 2004; 11:237–246.

4 Pessin MS, Lathi ES, Cohen MB, Kwan ES, Hedges TR 3rd, Caplan LR: Clinical features and mechanism of occipital infarction. Ann Neurol 1987;21:290–299.
5 Caplan LR, DeWitt LD, Pessin MS, Gorelick PB, Adelman LS: Lateral thalamic infarcts. Arch Neurol 1988;45:959–964.
6 Kim JS, Kim J: Pure midbrain infarction: Clinical, radiologic, and pathophysiologic findings. Neurology 2005;64:1227–1232.
7 Yamamoto Y, Georgiadis AL, Chang HM, Caplan LR: Posterior cerebral artery territory infarcts in the New England Medical Center Posterior Circulation Registry. Arch Neurol 1999;56:824–832.
8 Brandt T, Steinke W, Thie A, Pessin MS, Caplan LR: Posterior cerebral artery territory infarcts: clinical features, infarct topography, causes and outcome. Multicenter results and a review of the literature. Cerebrovasc Dis 2000;10:170–182.
9 Brandt T, Thie A, Caplan L, Hacke W: Infarcts in the brain areas supplied by the posterior cerebral artery. Clinical aspects, pathogenesis and prognosis. Nervenarzt 1995;66:267–274.
10 Kieffer SA, Takeya Y, Resch JA, Amplatz K: Racial differences in cerebrovascular disease. Angiographic evaluation of Japanese and American populations. Am J Roentgenol Radium Ther Nucl Med 1967;101:94–99.
11 Feldmann E, Daneault N, Kwan E, Ho KJ, Pessin MS, Langenberg P, Caplan LR: Chinese-white differences in the distribution of occlusive cerebrovascular disease. Neurology 1990;40:1541–1545.
12 Caplan LR, Gorelick PB, Hier DB: Race, sex and occlusive cerebrovascular disease: a review. Stroke 1986;17:648–655.
13 Gorelick P, Han J, Huang Y, Wong KS: Epidemiology; in Kim JS, Caplan LR, Wong KS (eds): Intracranial Atherosclerosis. Oxford, Wiley Blackwell, 2008, pp 33–44.
14 Klatsky AL, Armstrong MA, Friedman GD: Racial differences in cerebrovascular disease hospitalizations. Stroke 1991;22:299–304.
15 Haines DE: Fundamental neuroscience. Philadelphia, Churchill Livingstone, 2002.
16 Tatu L, Moulin T, Bogousslavsky J, Duvernoy H: Arterial territories of human brain: brainstem and cerebellum. Neurology 1996; 47:1125–1135.
17 Tatu L, Moulin T, Bogousslavsky J, Duvernoy H: Arterial territories of the human brain: cerebral hemispheres. Neurology 1998;50:1699–1708.
18 Kim JS, Choi-Kwon S: Risk factors for stroke in different levels of cerebral arterial disease. Eur Neurol 1999;42:150–156.
19 Gorelick PB, Caplan LR, Hier DB, Patel D, Langenberg P, Pessin MS, Biller J, Kornack D: Racial differences in the distribution of posterior circulation occlusive disease. Stroke 1985;16:785–790.
20 Kim SH, Lee JS, Kwon OK, Han MK, Kim JH: Prevalence study of proximal vertebral artery stenosis using high-resolution contrast-enhanced magnetic resonance angiography. Acta Radiol 2005;46:314–321.
21 Meissner I, Khandheria BK, Sheps SG, Schwartz GL, Wiebers DO, Whisnant JP, Covalt JL, Petterson TM, Christianson TJ, Agmon Y: Atherosclerosis of the aorta: risk factor, risk marker, or innocent bystander? A prospective population-based transesophageal echocardiography study. J Am Coll Cardiol 2004;44:1018–1024.
22 Adams HP, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, Marsh EE: Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke 1993;24:35–41.
23 Fisher CM, Caplan LR: Basilar artery branch occlusion: a cause of pontine infarction. Neurology 1971;21:900–905.
24 Caplan LR: Intracranial branch atheromatous disease: a neglected, understudied, and underused concept. Neurology 1989;39: 1246–1250.
25 Kim JS, Kang DW, Kwon SU: Intracranial atherosclerosis: incidence, diagnosis and treatment. J Clin Neurol 2005;1:1–7.
26 Lee DK, Kim JS, Kwon SU, Yoo SH, Kang DW: Lesion patterns and stroke mechanism in atherosclerotic middle cerebral artery disease: Early diffusion-weighted imaging study. Stroke 2005;36:2583–2588.
27 Kang SY, Kim JS: Anterior cerebral artery infarction: stroke mechanism and clinical imaging study in 100 patients. Neurology 2008; 70:2386–2393.
28 Kumral E, Bayulkem G, Atac C, Alper Y: Spectrum of superficial posterior cerebral artery territory infarcts. Eur J Neurol 2004; 11:237–246.
29 Caplan LR, Wityk RJ, Glass TA, Tapia J, Pazdera L, Chang HM, Teal P, Dashe JF, Chaves CJ, Breen JC, Vemmos K, Amarenco P, Tettenborn B, Leary M, Estol C, Dewitt LD, Pessin MS. New England Medical Center Posterior Circulation Registry. Ann Neurol 2004;56:389–398.
30 Yu KH, Bae HJ, Kwon SU, Kang DW, Hong KS, Lee YS, Rha JH, Koo JS, Kim JS, Kim JH, Lee JH, Cho SJ, Hwang SH, Jung S, Han MK, Cho KH, Kim BC, Shin DJ, Chang DI, Park JH, Kim EG, Jung DS, Ahn MY, Lee DH, Park KW, Chung CS, Cha JK, Lee JH, Uhm KY, Lee BC, Roh JK: Analysis of 10,811 cases with acute ischemic stroke from Korean Stroke Registry: hospital-based multicenter prospective registration study. J Korean Neurol Assoc 2006;24:535–543.
31 Kim JT, Yoo SH, Kwon JH, Kwon SU, Kim JS: Subtyping of ischemic stroke based on vascular imaging: analysis of 1,167 acute, consecutive patients. J Clin Neurol 2006;225–230.