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Original Paper

Branch Atheromatous Plaque: A Major Cause of Lacunar Infarction (High-Resolution MRI Study)

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Key Words
High-resolution MRI · Lacunar infarction · Branch atheromatous disease

Abstract
Background: Lacunar infarctions account for up to 25% of all ischemic strokes and, thus, constitute a numerically important subgroup. It is important that the two pathogeneses of lacunar infarction, that is, small-vessel occlusion and branch atheromatous disease, be differentiated because prognoses and treatment strategies differ. The authors evaluated the presence of branch atheromatous plaque in parent arteries that supply lacunar infarcts by high-resolution magnetic resonance imaging (HR-MRI). ***Methods:*** HR-MRI was performed in 15 patients with (1) a clinical presentation consistent with classical lacunar syndromes; (2) an acute lacunar infarction by diffusion-weighted imaging, measuring ≤20 mm in maximal diameter; (3) a magnetic resonance angiography showing a normal middle cerebral artery or basilar artery supplying the ischemic lesion, and (4) no other obvious etiology for small-vessel distribution ischemic stroke. ***Results:*** The median time of vessel wall imaging after index events was 4 days (range, 2–15 days). Six of the 15 patients had a lacunar infarction in the middle cerebral artery territory, and 9 had a lesion in the basilar artery territory. HR-MRI detected underlying atheromatous plaques in 9 patients (60%) with a lacunar infarction. In these 9 patients, asymptomatic intracranial atherosclerotic stenosis was more frequent compared to patients without branch atheromatous plaque (55.6 vs. 16.7%). In pontine infarctions, ischemic lesions that extended to the pial base of the pons were more frequent in patients with branch atheromatous plaques (83.3 vs. 33.3%), and all the ischemic lesions and atheromatous plaques were on the same side (right, n = 2; left,

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n = 4). All plaques responsible for acute symptomatic lacunar infarction were enhanced in contrast-enhanced T1-weighted HR-MR images. ***Conclusions:*** HR-MRI results enabled underlying symptomatic branch atheromatous disease to be detected in lacunar infarction patients. The experience gained during this study indicates that HR-MRI better delineates intracranial arterial lesions, suggesting that its use will lead to a further understanding of the mechanisms involved in stroke.

Lacunar infarcts are small lesions located in basal ganglia, deep hemispheric white matter, or the brain stem, and are referred to as 'small deep infarcts'. These lesions are ≤20 mm in maximal diameter and are attributable to occlusion of a single perforating artery [1]. The presence of an occluded perforating artery related to a small deep infarct was first described by Fisher [2]. Furthermore, lacunar infarction is being increasingly recognized as an important stroke subtype due to its clinical implications regarding management and prognosis [3, 4].

Most perforating artery occlusions are considered to be caused by a process of arterial disorganization similar to that of lipohyalinosis, or in the acute stage to fibrinoid necrosis [5]. Few occluded perforating arteries have ever been observed pathologically. However, patients rarely expire soon after lacunar stroke and, thus, occluded arteries may recanalize before necropsy [3]. Small subcortical infarcts can be caused by in situ thrombosis of parent arteries that block penetrating branches, by parent artery atheromas that encroach on the orifice of the perforating artery [6], or proximal sources of embolism, such as cardioembolism or carotid artery atherosclerosis [7]. The relative importance of these mechanisms has been much debated, largely in view of the difficulties of identifying perforating artery occlusions during life. Nevertheless, to determine optimal strategies for secondary stroke prevention, the precise differentiation of underlying pathologies of lacunar infarction is an important issue.

Recent studies have reported that high-resolution magnetic resonance imaging (HR-MRI) can be used to study the morphology of intracranial artery vessels, including atherosclerotic plaque [8], irregular wall thickening [9], arterial remodeling [10], and intraplaque hemorrhage [11]. However, it has not been determined whether HR-MRI can identify early-stage atherosclerotic plaque occluding the orifice of a perforating artery in patients with normal magnetic resonance angiography (MRA) findings. Accordingly, our aim was to investigate the wall imaging findings of middle cerebral artery (MCA) and basilar artery (BA) in patients with a lacunar infarction suspected of having small-vessel occlusions.

Materials and Methods

The medical records of patients admitted to our stroke unit, a comprehensive stroke center in a tertiary hospital, who underwent HR-MRI were reviewed. These files contained data on 50 patients that registered from March 22, 2011, to August 24, 2011. Inclusion criteria for the present study were (1) a clinical presentation consistent with one of the classical lacunar syndromes described by Fisher [12]; (2) an acute lacunar infarction by diffusion-weighted (DW) imaging, measuring ≤20 mm in maximal diameter, in the corona radiata, basal ganglia, internal capsule, or pons; (3) an MRA showing a normal MCA or BA supplying the ischemic lesion, and (4) no other obvious etiology for small-vessel distribution ischemic stroke (e.g. no primary vasculitis, active amphetamine use, or cardiac disease with strong embolic potential). This study was approved by our institutional review board, and all diagnostic modalities were performed after obtaining informed consent.

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Table 1. Baseline characteristics

Patient No.	Age/ sex	HTN	DM	Hypelip- idemia	Heart disease	Smoking status	Previous stroke or TIA	Concomitant medical condition	Clinical syndrome
1	61/M	Yes	No	Yes	No	Current	No	No	DCHS
2	44/M	Yes	No	No	No	Former	No	No	SMS
3	46/M	No	Yes	Yes	No	No	No	No	SMS
4	85/M	No	No	No	No	No	No	Liver cirrhosis	PMS
5	63/M	Yes	No	Yes	No	Former	No	No	PMS
6	69/F	Yes	Yes	Yes	Old MI	No	No	No	SMS
7	79/M	Yes	No	No	No	Former	No	No	AH
8	38/M	No	No	Yes	No	Current	Yes	No	PMS
9	78/F	No	No	No	No	No	No	No	PMS
10	85/F	No	Yes	No	No	No	Yes	No	PMS
11	69/F	Yes	No	No	No	No	No	No	PMS
12	75/F	Yes	Yes	No	No	No	Yes	No	PMS
13	53/M	Yes	No	Yes	No	Former	No	Alcohol abuse	SMS
14	54/M	Yes	Yes	No	No	No	Yes	No	PMS
15	79/F	Yes	No	No	No	No	No	No	SMS

HTN = Hypertension; DM = diabetes mellitus; TIA = transient ischemic attack; DCHS = dysarthria clumsy hand syndrome; SMS = sensory motor stroke; PMS = pure motor stroke; MI = myocardial infarction; AH = ataxic hemiparesis.

All 15 patients were imaged using a 3.0-tesla MR scanner (Verio; Siemens Medical Solution, Erlangen, Germany) equipped with a 32-channel head coil. Conventional MRI protocols included a complete set of DW images, T1- and T2-weighted images, fluid-attenuated inversion recovery images, T2* gradient-echo images, and contrast-enhanced MRA images of intracranial and extracranial arteries. HR-MRI was performed on the parent artery supplying lacunar infarction lesions. Maximum intensity projection images and source images of 3D time-of-flight MRA (used as topograms) were used to ensure that cross-section images were perpendicular to infarction locations. Four different types of contrast-weighted images [T1-weighted, T2-weighted, proton density (PD), and contrast-enhanced T1 (T1E)-weighted] were obtained. The parameters of the imaging sequences were as follows: (1) T1-weighted, turbo spin-echo [repetition time (TR)/echo time (TE), 600/12 ms; field of view (FOV), 12 cm; matrix size, 384/219; slice thickness, 2 mm; number of excitations (NEX), 4]; (2) turbo spin-echo PD-weighted imaging (TR/TE, 2,910/23 ms; FOV, 12 cm; matrix size, 384/269; slice thickness, 2 mm; NEX, 4), and (3) turbo spin-echo T2-weighted images (TR/TE, 2,910/70 ms; FOV, 12 cm; matrix size, 384/269; slice thickness, 2 mm; NEX, 4). The non-phase-wrap technique was used to avoid aliasing. Images were interpreted by consensus between board-certified neuroradiologists (C.H. Sohn) and experienced neurologists specializing in stroke (J.-W. Chung and S.-H. Lee) based on HR-MRI findings and medical records.

Results

The ages of the 15 patients ranged from 38 to 85 years (median 69 years), and 9 were men (table 1). Hypertension was the most common risk factor; 4 patients had a stroke history. In all patients, MRA showed a normal MCA or BA proximal to the ischemic lesion. The median time of vessel wall imaging after index events was 4 days (range, 2–15 days). Six of the 15 patients

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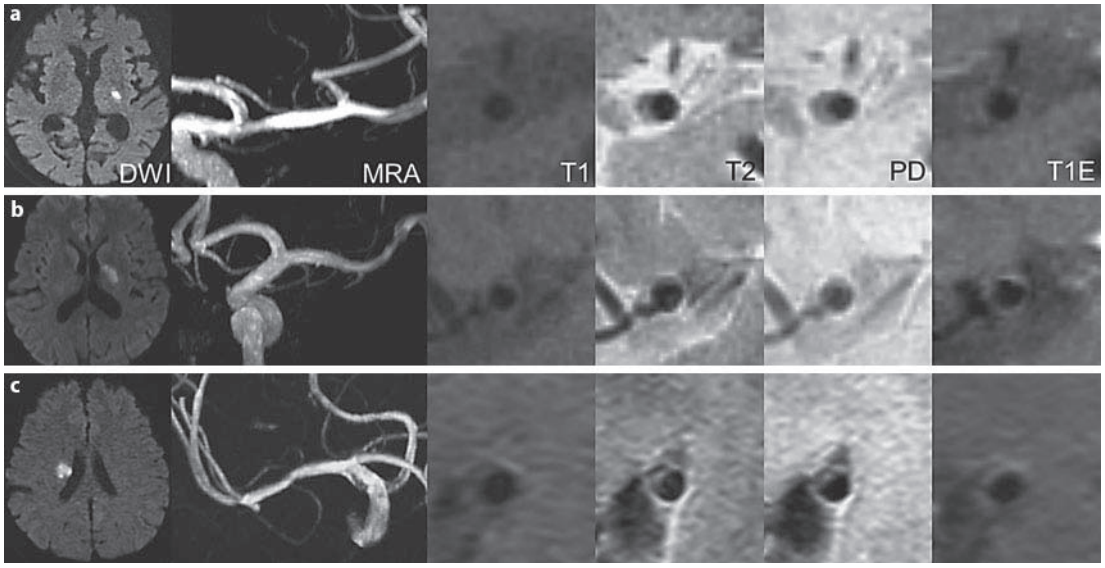

Fig. 1. Imaging findings of 3 patients with branch atheromatous MCA plaques. **a** Patient 4: DW image with a focal high signal intensity lesion in the internal capsule, MRA without significant stenosis in the MCA, and T1, T2, PD, and T1E images demonstrating atheromatous plaque at ventral MCA with subtle enhancement. **b** Patient 5: DW image with a focal lesion in basal ganglia, normal MCA by MRA, and T1, T2, PD, and T1E images showing atheromatous plaque at the superior MCA with enhancement. **c** Patient 6: DW image with a focal corona radiata lesion, normal MCA by MRA, and T1, T2, PD, and T1E images showing atheromatous plaque at the ventrosuperior portion of the MCA with enhancement.

(patients 1–6) had a lacunar infarction in the MCA territory, and 9 (patients 7–15) had a lesion in the BA territory. Three patients had a corona radiata lesion (patients 1, 2, and 6), 2 involved basal ganglia (patients 3 and 5), 1 the internal capsule (patient 4) and 9 the pons (patients 7–15). Among the 15 patients, 8 had a history of a pure motor stroke, 5 of a sensory motor stroke, 1 of dysarthria clumsy hand syndrome, and 1 of ataxic hemiparesis. Of the 6 patients with MCA territory lacunar infarction, HR-MRI showed atherosclerotic plaques in the parent artery in 3 patients, which were located at the ventral (n = 2) and superior (n = 1) aspects of the vessel wall (fig. 1). In 9 patients with a BA territory lacunar infarction, 6 had atherosclerotic plaques in the parent artery, 3 in the ventrolateral, and 3 in the lateral aspect of the vessel wall (fig. 2). For BA territory lacunar infarctions, ischemic lesions extending to the pial surface of the pontine base were more frequent in patients with branch atheromatous plaques (83.3 vs. 33.3%), and all lesions and atheromatous plaques were on the same side (right, n = 2; left, n = 4). Enhancement of underlying atherosclerotic plaques was observed in all 9 patients with parent artery plaques. Asymptomatic intracranial atherosclerotic stenosis was found in 5 patients (55.6%) with branch atheromatous plaque and in 1 patient (16.7%) with no evidence of branch atheromatous plaque. The imaging findings of patients without branch atheromatous disease are shown in figure 3. Imaging study results are described in table 2.

Discussion

In a previous study performed in the USA, stenotic lesions in large arteries were found in 10% of patients with a lacunar infarction [13], and in a Korean study, 36% of patients with striatocapsular area lacunar infarctions had stenotic MCA lesions [14]. In these studies, MRA

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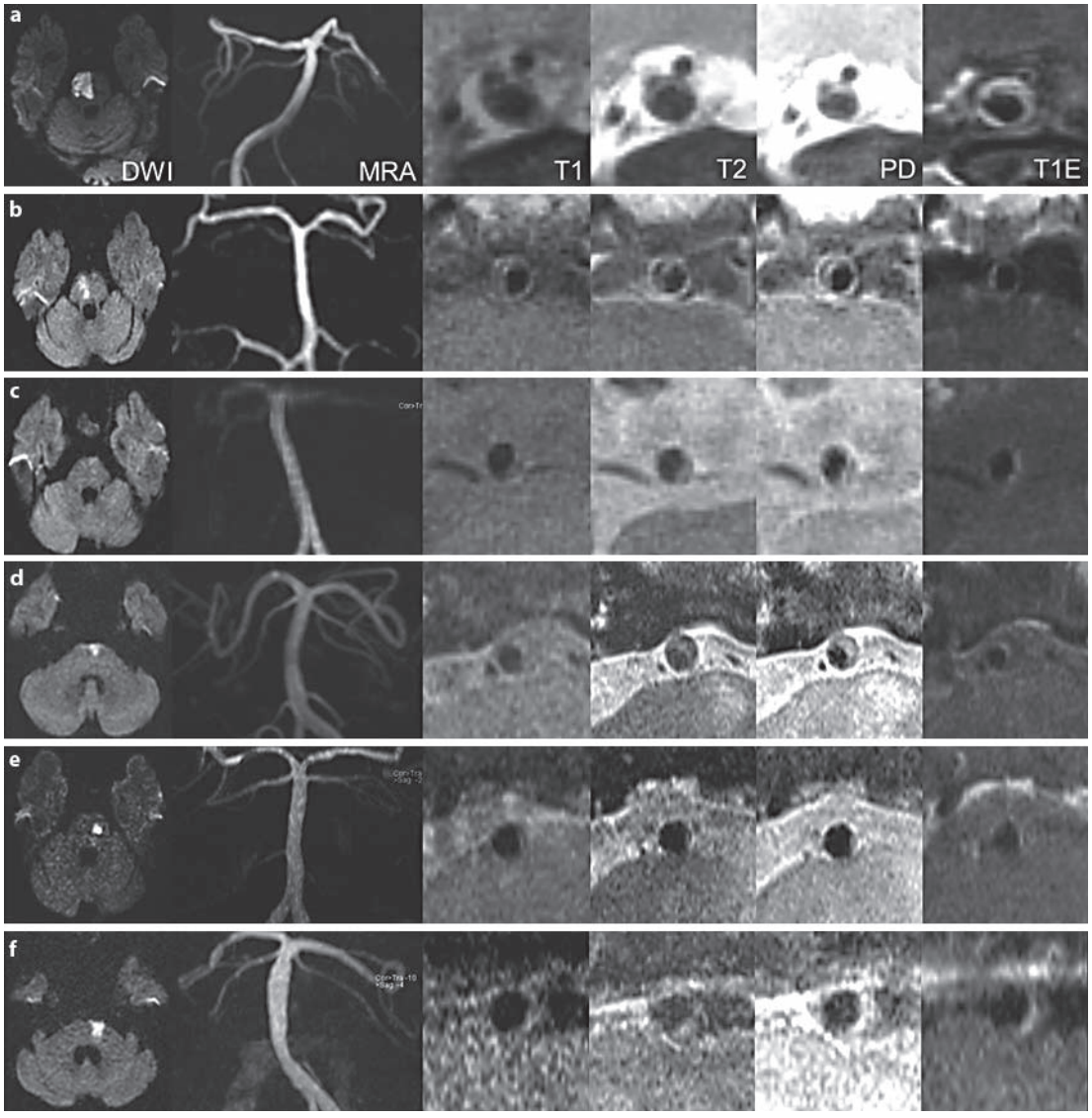

Fig. 2. Image findings of 6 patients with branch atheromatous plaque in the BA. **a** Patient 10: DW image showing a high signal intensity lesion in the right pons extending to the base surface, MRA showing no significant stenosis in the BA, and T1, T2, PD, and T1E images demonstrating atheromatous plaque at the ventrolateral portion of the BA with enhancement. **b** Patient 11: DW image showing a focal lesion in the right pons extending to the base surface, MRA showing a normal BA, and T1, T2, PD, and T1E images demonstrating atheromatous plaque at the lateral BA with enhancement. **c** Patient 12: DW image showing a focal lesion in the left pons extending to the base surface, MRA showing a normal BA, and T1, T2, PD, and T1E images demonstrating atheromatous plaque at the dorsolateral BA encroaching a perforating artery orifice with enhancement. **d** Patient 13: DW image showing a high signal intensity lesion in the left pons extending to the base surface, MRA showing no significant stenosis in the BA, and T1, T2, PD, and T1E images demonstrating atheromatous plaque at the ventrolateral BA with enhancement. **e** Patient 14: DW image showing a focal isolated lesion in the left pons, MRA showing a normal BA, and T1, T2, PD, and T1E images demonstrating small atheromatous plaque at the lateral BA with enhancement. **f** Patient 15: DW image showing a focal lesion in left pons extending to the base surface, MRA showing a normal BA, and T1, T2, PD, and T1E images demonstrating atheromatous plaque at the dorsolateral BA with enhancement.

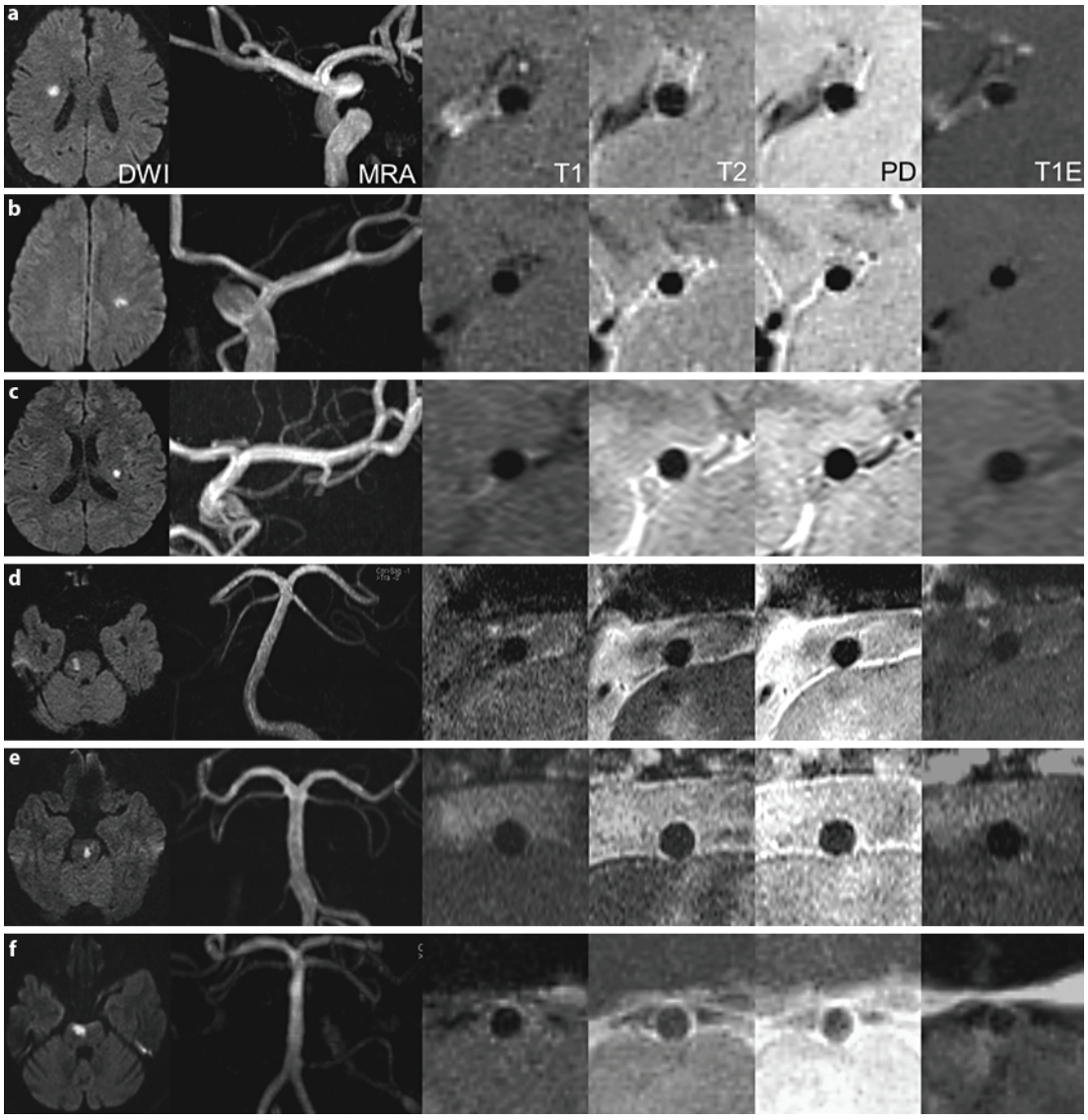

Fig. 3. Imaging findings of 6 patients without branch atheromatous plaque in the MCA or BA. **a–c** Patients 1–3: DW images showing a focal high signal intensity lesion in the right corona radiata, left corona radiate, and left basal ganglia, and MRA showing a normal MCA. **d–f** Patients 7–9: DW image showing a focal isolated lesion in the right pons, left pons, and right pons extending to the basal surface, and an MRA showing no significant stenosis in the BA. All HR-MRI sequence, T1, T2, PD, and T1E images demonstrating normal vessel walls without enhancement.

and digital subtraction angiography were used to evaluate the presence of parent artery stenosis. However, MRA and digital subtraction angiography cannot identify branch atheromatous disease, occlusion of the perforating artery by an artery-to-artery embolism or a microembolism from atherosclerotic parent artery plaques. Microatheromas, which have been reported to be the most common underlying mechanism of symptomatic lacunar infarction in autopsy studies [2, 6], may not be accompanied by significant stenosis in classic luminographic angiography studies. In the present study, HR-MRI performed during the acute period of lacunar infarction showed a surprisingly high prevalence of parent artery branch atheromatous plaques.

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Table 2. Imaging studies results

Patient No.	DWI infarction location	HR-MRI results (time of scanning after stroke onset)	Other atherosclerotic vascular lesion
1	R corona radiata	No evidence of atheromatous plaque (4 days)	No
2	L corona radiata	No evidence of atheromatous plaque (2 days)	No
3	L basal ganglia	No evidence of atheromatous plaque (5 days)	No
4	L internal capsule	Atherosclerotic plaque at ventral aspect of L MCA, with enhancement (2 days)	No
5	L basal ganglia	Atherosclerotic plaque at ventral portion of L MCA, with enhancement (7 days)	Moderate narrowing at BA
6	R corona radiata	Atherosclerotic plaque at superior portion of R MCA, with enhancement (3 days)	No
7	R pons, isolated	No evidence of atheromatous plaque (5 days)	No
8	L pons, isolated	No evidence of atheromatous plaque (2 days)	No
9	R pons, extended to basal surface	No evidence of atheromatous plaque (2 days)	Focal stenosis in L ACA, R superior division of MCA
10	R pons, extended to basal surface	Atherosclerotic plaque at ventrolateral aspect of BA, with enhancement (15 days)	No
11	R pons, extended to basal surface	Atherosclerotic plaque at ventrolateral aspect of BA, with enhancement (5 days)	No
12	L pons, extended to basal surface	Atherosclerotic plaque at lateral aspect of BA, with enhancement (2 days)	Focal stenosis in both distal ICA
13	L pons, extended to basal surface	Atherosclerotic plaque at ventrolateral aspect of BA, with enhancement (3 days)	Focal severe stenosis of both ophthalmic segments of ICA, focal stenosis of R PCA orifice and L distal PCA
14	L pons, extended to basal surface	Atherosclerotic plaque at lateral aspect of BA, with enhancement (4 days)	Atherosclerotic luminal narrowing in R ICA, R PCA
15	L pons, extended to basal surface	Atherosclerotic plaque at lateral aspect of BA, with enhancement (4 days)	Focal luminal narrowing in R proximal MCA, focal luminal narrowing in the distal PCA

DWI = Diffusion-weighted imaging; R = right; L = left; ICA = internal carotid artery; PCA = posterior cerebral artery.

Age, hypertension, and diabetes mellitus have been reported to be independent risk factors of intracranial atherosclerotic disease [15]. Other studies have also suggested that intracranial atherosclerotic disease is more common in men, particularly in younger men [16, 17]. Furthermore, intracranial atherosclerosis is more common in Asians than Caucasians [18–20]. Considering risk factors and ethnic diversities, some individuals are more prone to intracranial atherosclerotic disease. Intracranial atherosclerosis is often progressive, and the degree of stenosis often increases and plaques can ulcerate and promote the formation of

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platelet-rich and red-cell-rich thrombi. In the present study, asymptomatic intracranial atherosclerotic stenosis was more common in patients with branch atheromatous plaques that caused lacunar infarction. In patients with asymptomatic intracranial atherostenosis and lacunar infarcts, branch atheromatous disease is the likely cause of perforating territory infarcts.

The location of infarction on DW MRI has been posited to separate branch atheromatous disease from intrinsic disease of penetrating arteries. Patients with MCA disease tend to have larger and lower lesion patterns beginning from the proximal territory of the lenticulostriate artery [21]. In BA territory lacunar infarction, paramedian pontine and small deep pontine lesions were found to be no different in terms of the prevalence of branch atheromatous plaques (61.5 and 73%, respectively) [21]. In the present study, pontine lesions that extended to the ventral basal surface were more often caused by branch atheromatous plaques than isolated lacunar infarcts within the pons that did not extend to the pial basal surface. This finding concurs with those reported by Fisher and Caplan [22] in 1971 in an autopsy study.

The results of the present study should be interpreted with caution. First, because all patients survived with few neurologic deficits, atherosclerotic plaques that occluded perforating arteries were not confirmed by histopathological study. We did try to visualize the orifices of occluded perforating arteries, but only in 1 patient (patient 12) did we observe plaque protruding toward the orifice of a perforating artery. However, in 6 patients with atherosclerotic BA plaque, all plaques were on the same side as ischemic lesions and at the same level of ischemic lesions visualized by PD HR-MRI. Second, HR-MRI resolution was not sufficient to provide information on plaque composition or whether or not plaques qualified as 'vulnerable'. However, all atherosclerotic plaques showed contrast medium enhancement. In a recent study, enhancement of MCA atherosclerosis was found to be associated with a history of a symptomatic recent ischemic stroke [23]. Therefore, enhancement of atherosclerotic plaque in our study, imaged during the acute period of ischemic stroke, may indicate that these plaques were vulnerable. Finally, this study was conducted in a retrospective manner based on reviews of electronic medical charts and only 15 patients were included. Future studies with a prospective, consecutive design will be able to further address issues discussed in this study.

In the present study, by using HR-MRI, we were able to visualize branch atheromatous plaques in patients suspected of having small-vessel occlusions. Branch atheromatous disease was frequently observed even in patients with normal parent arteries by MRA. Our experiences indicate that HR-MRI will aid in vivo investigations on intracranial arterial disease and on the elucidation of stroke mechanisms.

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