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Role of conventional MRI rain for basilar artery plaque detection in solitary pontine infarct

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Abstract

Background: Solitary pontine infarct is divided into paramedian pontine infarct (PPI) and small deep pontine infarct (SDPI). High-resolution MRI is currently the most useful imaging method to characterize vessel walls and detect atherosclerotic plaques of the intracranial arteries. However, high-resolution MRI is not included in the routine imaging protocol for patient with acute stroke. We intend to determine the role of conventional MRI and MRA of the brain in solitary pontine infarct. *Methods:* Fifty patients with solitary pontine infarct underwent a MRI study of the brain on T1-weighted image, T2-weighted image and post-gadolinium T1-weighted image to assess the presence of atherosclerotic plaque, and MRA of the brain using 3-dimensional time-of-flight MRA (3D TOF MRA) to assess the basilar artery flow. The basilar artery assesses by axial T2-weighted image was scored as "presence of plaque" or "absence of plaque" and the 3D TOF MRA of the basilar artery is scored as "normal", "irregular" or "stenosis" ≥ 30%. Pontine infarct is divided into paramedian pontine infarct (PPI) and small deep pontine infarct (SDPI) groups. *Results:* Thirty-five patients had PPI and 15 had SDPI. Axial T2WI MRI of the brain detected basilar artery plaques in 50% of patients with pontine infarction: 51.4% (18 of 35 cases) in PPI and 46.7% (7 of 15 cases) in SDPI. No plaque was found in all cases of normal-appearing 3D TOF MRA and the plaque was identified in all patients with basilar artery stenosis on the 3D TOF MRA.
Conclusion: The basilar artery atherosclerotic branch disease is frequently detected in both groups of solitary pontine infarct (PPI and SDPI). Conventional MRI and MRA of the brain are useful imaging methods that help characterize basilar wall abnormalities.

INTRODUCTION

A solitary pontine infarct is usually classified as paramedian pontine infarct (PPI) and small deep pontine infarct (SDPI) according to shapes of the lesion and its location.[1-3] The PPI is defined by the infarct extension until basal surface of the pons, while lesion of SDPI, (also called island infarct) is located deep and is separated from surface of basis pontis.[4] It is difficult to differentiate PPI from SDPI by using only clinical features.[5,6] However, patients with PPI often report motor fluctuation, so-called pontine warning syndrome, or progression of weakness.[7-10] In contrast, pontine warning syndrome and progressive weakness are rarely reported in patients with SDPI.[11]

The mechanism of PPI is considered to be due to basilar branch disease.[1] The postulated mechanism is an atheromatous branch occlusive disease in which basilar artery atherosclerotic plaques protrude into the orifice of the perforators, occluding the lumen and causing infarction.[12] On the other hand, the postulated mechanism of SDPI is lipohyalinosis which is related to small vessel disease.[13] Magnetic resonance imaging (MRI) is the most sensitive method to easily detect the lesion, particularly in acute phase.[14]

The detection of basilar plaque in a patient with PPI by the high resolution MRI is firstly reported in 2005, supporting the role of basilar branch occlusive disease in pontine infarction.[1,3] However, further study demonstrates that basilar plaque is also detected by high resolution MRI in patients with SDPI.[15,16] Moreover, the prevalence of basilar plaque in patients with SDPI is higher than the prevalence in patients with PPI.[15] The authors propose the hypothesis that basilar branch occlusion can be the important mechanism in SDPI.[15] Another study also reports that basilar plaque was detected by high resolution MRI in patients with both PPI and SDPI.[17]

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High resolution vessel wall imaging or luminography using MRI may be better than MR angiograms for detection of basilar plaque.[3,15,18] Initial case reports described basilar plaque detection by high resolution MRI in patients who had normal magnetic resonance angiography (MRA).[3] The rate of basilar plaque detection by MRA is lower than the high resolution luminography MRI by about 40% in one study.[15] The most recent study confirms similar findings that the high resolution luminography with the 3.0-Tesla MRI is better than MRA for basilar plaque detection.[16,18]

However, most routine MRI studies do not include high-resolution vessel wall imaging, and there are few descriptions of basilar plaque features in the conventional MRI evaluation in patients with solitary pontine infarct. We believe that conventional MRI may able to detect basilar artery plaque in routine clinical practice. Therefore, we report here conventional MRI and MRA of the brain for evaluation of basilar artery atherosclerotic plaques in solitary pontine infarct.

METHODS

Study Population

We studied 50 patients who were admitted in the stroke unit of Thammasat University Hospital between July 2010 and February 2014 with solitary pontine infarct and underwent a MRI study of the brain on a T1-weighted image (T1WI), a T2-weighted image (T2WI) and a post-gadolinium T1-weighted image (T1WI/Gd, if available), and a MRA of the brain using the 3-dimensional time-of-flight MRA (3D TOF MRA). The patients with a solitary pontine infarct who did not undergo a MRI and a MRA study of the brain were excluded from the study.

The patients are divided into two groups according to the type of a solitary pontine infarct: (1) PPI, when the lesion extends to ventral pontine surface and (2) SDPI, when the lesion does not extend to ventral pontine surface.

Demographic characteristics and risk factors

Demographic features (age, sex) and risk factors (hypertension, diabetes mellitus, hyperlipidemia, atrial fibrillation, chronic kidney disease, smoking, alcoholic drinking) were collected.

Imaging Protocol

Images were performed using a 1.5-Tesla Philips Achieva XR Scanner with a neurovascular-16 channel radiofrequency head coil.

To examine the basilar arterial wall, an axial T2WI with an axial T1WI (pre-and post gadolinium injection) were performed with the following parameters; (1) T2WI: TR/TE (ms) = 4500/120 multi-slice with spin echo and use fast imaging mode, field of view (FOV) (mm^2) = 230x230, acquisition matrix = 384x240, slice thickness = 5 mm with gap = 1 mm, voxel size AP/RL (mm) = 0.6/0.75 and number of excitations (NEX, NSA) = 2, and (2) T1WI (pre-and post gadolinium injection): TR/TE (ms) = 650/15 multi-slice with spin echo technique, field of view (FOV) (mm^2) = 230x230, acquisition matrix = 244x196, slice thickness = 5 mm with gap = 1 mm, voxel size AP/RL (mm) = 0.94/1.17 and number of excitations (NEX, NSA) = 2.

To study the basilar artery luminography, imaging is performed using the 3D TOF sequence: TR/TE (ms) = 15/5.2, field of view (FOV) (mm^2) = 210x210, thickness = 1.2 mm with 0.6 mm overlapping, matrix = 368x168, number of excitations (NEX, NSA) = 1. No smoothing filter is applied. Sense program to reduce scan time is used.

Data Analysis

The demographic data between SDPI and PPI was measured by Pearson's chi-square test.

The MRI and the 3D TOF MRA of two groups of pontine infarctions were reviewed independently by two experienced neuroradiologists (U.C. and A.W.). Readers were blinded to clinical and etiologic data. Discrepancies between the two readers are solved by visual consensus.

Presence of a basilar artery plaque is assessed using an axial T2WI. Wall abnormalities were scored as: (1) presence of plaque: when a wall thickening (either eccentric or concentric) is observed and (2) absence of plaque: when normal thin arterial wall was shown. Basilar artery wall enhancement was assessed using axial T1WI/Gd (if available) and scored as presence or absence of wall enhancement.

The basilar artery luminograph is analyzed on 3D TOF MRA for the presence of luminal stenosis. Stenoses are classified in three stages: (1) normal: when the lumen is regular, (2) irregular: when the outer boundaries of basilar artery are not strictly parallel but no significant lumen reduction, and (3) stenotic: when we observe a lumen with a significant stenosis ≥ 30%.

Table 1: Demographic data of the study population

Factors	Infarction type				p-value
	SDPI (n=15)		PPI (n=35)		
	No.	(%)	No.	(%)	
Male	12	34/3	23	65.7	0.825
Hypertension	8	28.6	20	71.4	1.000
Diabetes mellitus	5	31.3	11	68.7	1.000
Hyperlipidemia	7	35.0	13	65.0	0.753
Atrial fibrillation	0	0.0	2	100.0	0.875
Chronic kidney disease	1	25.0	3	75.0	1.000
Smoking	7	36.8	12	63.2	0.611
Alcohol drinking	10	38.5	16	61.5	0.294

SDPI, small deep pontine infarct; PPI, paramedian pontine infarct

RESULTS

We studied 35 men and 15 women (mean age 60.5 years; range 35 to 86 years). Among the 50 patients with solitary pontine infarct, 35 (70%) have PPI and 15 (30%) had SDPI. Among 35 men, 23 (65.7%) had PPI and 12 (34.3%) had SDPI and among 15 women, 12 (80%) had PPI and 3 (20%) have SDPI. The demographic data is showed in Table 1.

Patients with PPI

On the 3D TOF MRA, among 35 patients with PPI, 3 (8.6%) have normal basilar luminography, 19 (54.3%) had irregular lumen, and 13 (37.1%) display basilar stenosis (Table 2).

All three patients with normal basilar luminography on 3D TOF MRA, axial T2WI MRI did not find a basilar artery plaque.

Among 19 patients with irregular lumen on 3D TOF MRA, axial T2WI MRI found a basilar artery plaque in 5 cases. Two patients in this group who had basilar artery plaque did not receive gadolinium injection during the imaging study. Two of the three patients who received gadolinium injection showed plaque enhancement and one had no plaque enhancement.

In all 13 patients with basilar artery stenosis, axial T2WI MRI confirmed the presence of plaque. Three patients in this group did not receive gadolinium injection during the imaging study. Nine out of ten patients with gadolinium injection showed plaque enhancement.

Patients with SDPI

On the 3D TOF MRA, in the group of 15 patients with SDPI, 5 (33.3%) had normal basilar luminography, 6 (40.0%) have irregular lumen, and 4 (26.7%) displayed basilar stenosis (Table 3).

Table 2: Distribution of basilar artery atherosclerosis according to imaging modality (3D TOF MRA versus axial T2WI MRI) among patients with paramedian pontine infarct (PPI)

	T2WI MRI		
3D TOF MRA	Normal	Plaque (+)	**Total**
Normal	3	0	3
Irregular	14	5	19
Stenosis	0	13	13
Total	17	18	35

Table 3: Distribution of basilar artery atherosclerosis according to imaging modality (3D TOF MRA versus axial T2WI MRI) among patients with small deep pontine infarct (SDPI)

	T2WI MRI		
3D TOF MRA	Normal	Plaque (+)	**Total**
Normal	5	0	5
Irregular	3	3	6
Stenosis	0	4	4
Total	8	7	15

No basilar artery plaque was found in all 5 patients with normal basilar luminography on 3D TOF MRA.

Among the 6 patients with irregular lumen on 3D TOF MRA, axial T2WI MRI found a basilar artery plaque in 3 cases. One of these 3 patients did not receive gadolinium injection during the imaging study. The other 2 with gadolinium injection showed plaque enhancement.

In all 4 patients with basilar artery stenosis, axial T2WI MRI confirmed the presence of plaque. One patient in this group did not receive gadolinium injection during the imaging study. Two out of 3 patients with gadolinium injection showed plaque enhancement and one have no plaque enhancement.

Examples of axial T2WI MRI of the brain and 3D TOF MRA in patients with PPI and SDPI are illustrated respectively in Figure 1 and Figure 2.

DISCUSSION

The mechanism of PPI is thought to be basilar branch occlusion from atherosclerotic plaque while that of SDPI is postulated to be small vessel disease from lipohyalinosis.[1,2] This hypothesis is based on the autopsy study which has been reported since 1971.[1] In contrast, the novel studies based on MRI demonstrate that PPI and SDPI share the mechanism of basilar branch occlusion.[15,17,18] In our study, a conventional axial T2WI MRI detected basilar artery atherosclerotic plaque in 50% of patients with solitary pontine infarct (25 of 50 cases) and detection rate was nearly the same in both PPI and SDPI groups: 51.4% (18 of 35 cases) in PPI and 46.7% (7 of 15 cases) in SDPI. The result supports the hypothesis from previous novel studies that basilar artery atheromatous branch disease can be detected in patients with both PPI and SDPI.[15-18]

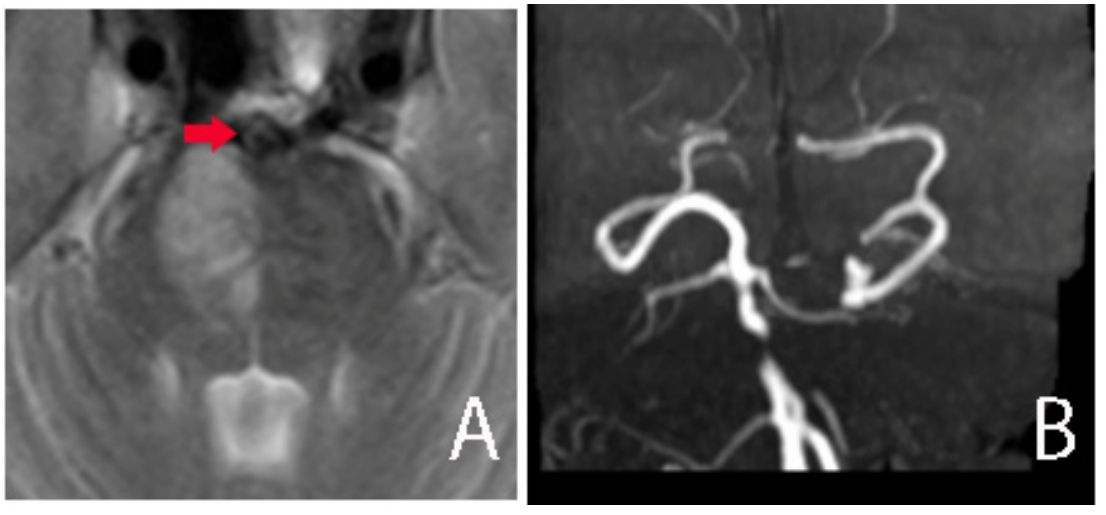

Figure 1. Paramedian pontine infarct (PPI); A: Axial T2WI MRI of the brain shows a hyperintense lesion of infarct at right-sided pons abutting the ventral pontine surface. At this level, a hyperintense arterial wall thickening of basilar artery (red arrow) is also seen, representing atherosclerotic plaque. B: 3D TOF MRA demonstrates severe stenosis of basilar artery.

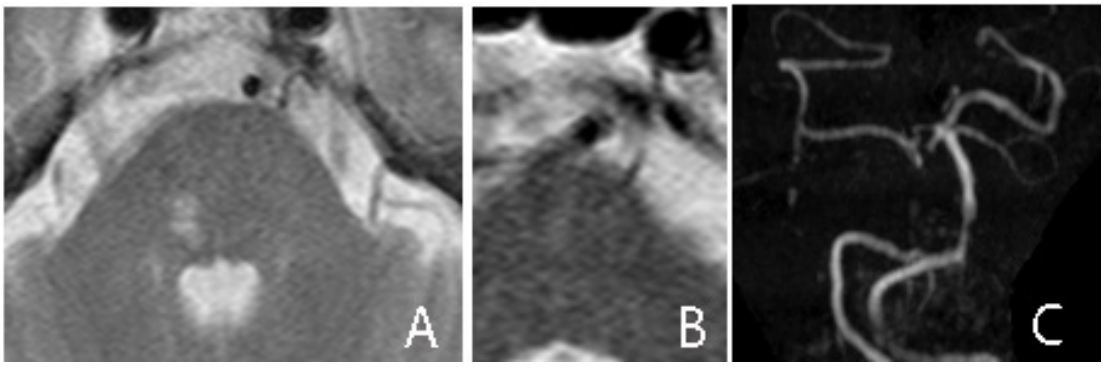

Figure 2. Small deep pontine infarct (SDPI); A: Axial T2WI MRI of the brain shows a small hyperintense lesion of infarct at right-sided pons, which does not extend to the ventral pontine surface. B: Axial T2WI MRI of the brain shows a hyperintense arterial wall thickening of basilar artery, representing atherosclerotic plaque. C: 3D TOF MRA demonstrates severe stenosis of basilar artery.

In both PPI and SDPI, no plaque is found in all cases of normal-appearing basilar luminography on 3D TOF MRA. Compared with previous studies, luminography with high-resolution MRI can identify atherosclerotic plaques even in patients with normal-appearing MRA.[3,15,17,18] This may be important because high-resolution MRI displays high-spatial resolution giving better quality of images as compared with images from conventional MRI.[18]

In patients with irregular basilar luminography on 3D TOF MRA, axial T2WI MRI could detect plaques in about 32% (8 of 25 cases): 26.3% (5 of 19 cases) in PPI and 50% (3 of 6 cases) in SDPI. Therefore, the conventional axial T2WI MRI could detect atherosclerotic plaques even in patients with just irregular basilar luminography (not fulfilling the criteria for basilar artery stenosis) on 3D TOF MRA. Plaque was identified in all patients with basilar artery stenosis on 3D TOF MRA. All detected plaques in our study showed high signal intensity on axial T2WI.

There are some limitations that need to be addressed in our study. Although identifying a basilar artery atherosclerotic plaque is highly suggestive of a causative mechanism for correlated solitary pontine infarct, we cannot deny the chance that they are just the co-existing lesions. We possibly underestimated the prevalence of basilar artery plaques in both PPI and SDPI groups because 19 of 50 cases (38%) did not receive gadolinium injection during the MR study. The use of contrast agent may likely enhance detection of wall lesions and may accurately detect plaque in questionable cases. In our study, however, we believe that this does not alter the direction of our conclusion because all cases without basilar artery plaque on axial T2WI MRI, who were administered gadolinium, also showed no abnormal gadolinium enhancement along basilar artery wall.

The novel, high resolution, MRI may be a useful method in basilar artery plaque detection[15-18] and shows clinical potential in detection of middle cerebral artery plaque.[19,20] However, this complicated and high-cost technique is still not included in the routine protocol for stroke evaluation. On the other hand, conventional MRI and MRA are feasible and available in most stroke centers today. Our study shows that the conventional MRI and MRA should have a role in evaluation of basilar artery plaque in patients with solitary pontine infarct.

In conclusion, our study suggests that basilar artery atherosclerotic branch disease is frequently detected in patients with both PPI and SDPI. Conventional MRI and MRA of the brain are the useful imaging methods that may characterize intracranial vessel wall abnormalities. These MRI and MRA techniques may be helpful in further study of intracranial arterial diseases and in comprehension of stroke mechanisms. Radiologist should pay attention on basilar artery plaque as a routine evaluation of MRI and MRA in patient with solitary pontine infarct.

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