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# Neuroradiology report

# Middle cerebral artery plaque imaging using 3-Tesla high-resolution MRI

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#### Abstract

Diagnosis of deep subcortical infarcts based on atherosclerosis of the middle cerebral artery (MCA) is important because this type of infarct is usually more aggressive than typical lacunar infarcts. However, current imaging techniques are of limited utility in the diagnosis of MCA plaques. Here, we report the use of 3-Tesla (3T) high-resolution moderate T<sub>2</sub>-weighted imaging (HRT<sub>2</sub>WI) to detect MCA plaques in three patients with acute MCA perforator territory infarcts. MCA plaques were seen with HRT<sub>2</sub>WI in a patient with MCA stenosis, which was observed by magnetic resonance angiography (MRA). Of the two patients without MCA stenosis (also confirmed by MRA), one had thin MCA plaques and the other had normal walls based on HRT<sub>2</sub>WI. Progression of symptoms occurred in the patients with plaques. We conclude that 3T HRT<sub>2</sub>WI can identify plaque on MCA walls and has the potential to identify patients at risk for stroke progression or recurrence.

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## 1. Introduction

Typical lacunar infarcts are usually caused by lipohyalinosis, or microatheromatous or embolic occlusion of a perforating artery, whereas larger infarcts result from atherosclerosis of the parent arteries, which may occlude the orifice of the perforating artery as branch atheromatous disease. <sup>1–5</sup> The infarct mechanisms in the middle cerebral artery (MCA) perforator territory can be divided into four subtypes regardless of lesion size: (i) MCA disease (MCAD), if there is a corresponding ipsilateral atherosclerotic MCA lesion without cardiogenic embolism (CE) or an atherosclerotic lesion proximal to the MCA lesion; (ii) internal carotid artery disease, if there is significant ipsilateral internal carotid artery stenosis (>50%) without evidence of MCAD or CE; (iii) CE, if

there is emboligenic heart disease based on stroke classification criteria developed by the investigators of the Trial of ORG 10172 in Acute Stroke Treatment<sup>7</sup> in the absence of atherosclerotic diseases in cerebral vessels; and (iv) small vessel disease, if there is no CE, MCAD, or internal carotid artery disease.

MCAD may be particularly important among these infarct mechanisms, because patients with symptomatic MCAD have an overall stroke risk of 12.5% per year and a poor outcome. Current imaging techniques such as CT angiography, digital subtraction angiography, and magnetic resonance angiography (MRA) are of limited utility in the diagnosis of MCAD, because they show only the arterial lumen, but do not provide information on the vessel wall. High-resolution MRI has been used for the detection of aortic, internal carotid artery, basilar artery, and MCA<sup>13</sup> plaques in vivo. Here we report three patients with infarction in the corona radiata, for whom a combination of 3-Tesla (3T) high-resolution moderate T<sub>2</sub>-weighted imaging (HRT<sub>2</sub>WI) and MRA was more useful than

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conventional MRI for detecting MCA plaques and evaluating the pathogenesis of the infarction.

#### 2. Materials and methods

### 2.1. MRI protocol

Examination using a 3T MRI unit (Signa EXCITE HD; GE Healthcare, Piscataway, NJ, USA) was performed in the subacute stage (within 1 week after stroke onset). The same imaging protocol was used for each patient: diffusion-weighted imaging (DWI) (time to repetition [TR]/time to echo [TE] 10000/70.4, field of view [FOV] 22 cm × 22 cm, matrix size 128 × 256, slice thickness 6 mm, interslice gap 2 mm, number of excitations [NEX] 1, with parallel imaging), three-dimensional time-of-flight MRA (TR/TE 25/3.5, field angle [FA] 15°, FOV 16 cm × 19 cm, matrix size 512 × 224, slice thickness 1 mm, NEX 1, with parallel imaging), and HRT<sub>2</sub>WI (TR/TE 2800/50.8, FOV 12 cm  $\times$  12 cm, matrix size 512  $\times$  256, slice thickness 2 mm, interslice gap 0.3 mm, NEX 5, acquisition time 6 min 52 sec). HRT<sub>2</sub>WIs were acquired perpendicular to the MCA axis, using MRA for localization. The black blood and cardiac gating techniques were not used for HRT<sub>2</sub>WI.

#### 3. Case reports

#### 3.1. Case 1

A 71-year-old woman was admitted with progressive left hemiparesis. She had no history of smoking and had received no major medical care. A neurological examination revealed slight left hemiparesis and dysarthria. DWI revealed a high-intensity infarct lesion of 20 mm in diameter, which was bigger than a typical lacunar stroke lesion, in the right corona radiata (Fig. 1A). MRA revealed relatively severe stenosis in the M1 portion of the right MCA, which appeared to have affected the lateral striate arteries in partial maximum intensity projection reconstruction images (Fig. 1B). No steno-occlusive lesion was found in the cervical carotid arteries. HRT2WI indicated the presence of plaques in the right MCA, at the level of the stenotic area seen with MRA (Fig. 1D), but there were no plaques in the right MCA at the level of the non-stenotic area (Fig. 1C,E). Despite aggressive drug treatment, including administration of edarabon, argatroban, aspirin and ticlopidine, the symptoms progressed and the patient required a lengthy rehabilitation period.

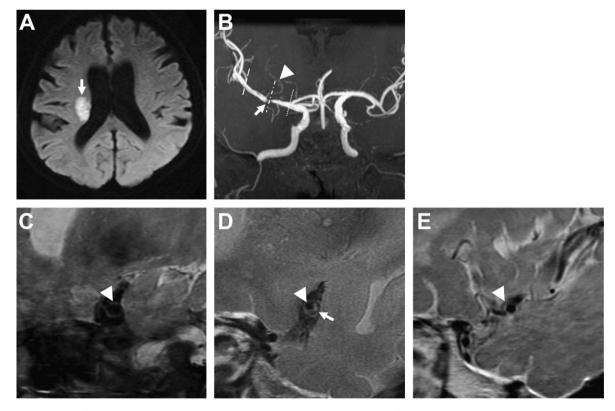


Fig. 1. Case 1. (A) Axial diffusion-weighted MRI showing a high-intensity lesion of 20 mm in diameter in the right corona radiata (arrow). (B) Magnetic resonance angiography (MRA) image, processed by partial maximum intensity projection, indicating the relationship between the stenotic area (arrow) and lateral striate arteries (arrowhead). (C-E) High-resolution moderate  $T_2$ -weighted images obtained perpendicular to the right middle cerebral artery (MCA), at the level of the dotted line (C), the dash-dotted line (D), and the broken line (E) in B, showing the lumen of the right MCA (arrowheads). There were plaques on the vessel wall lumen of the right MCA at the level of the stenotic area observed with MRA (D, arrow), but no plaques at the level of the non-stenotic area (C, E).

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