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Intracranial Plaque Imaging Using High-Resolution Magnetic Resonance Imaging: A Pictorial Review

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1. Introduction

Intracranial atherosclerotic disease is defined as the development, progression, and complication of atherosclerotic lesions on intracranial large arteries. It has been considered to be the most common cause of ischemic stroke worldwide (Gorelick et al., 2008; Kim et al, 2006; Won et al., 1998).

Intracranial atherosclerotic disease is normally detected by hemodynamically relevant intracranial stenosis using luminography-based methods. However, from the extracranial study, it is well known that lumen diameter can be maintained due to remodeling of the arteries in spite of wall thickening (Glagov et al., 1987), suggesting conventional luminography-based methods might underestimate atherosclerotic disease. Arenillas (Arenillas, 2001) summarized limitations of conventional approach. First, it only detects advanced stage of intracranial atherosclerotic disease with luminal narrowing. Second, it cannot provide the histopathologic information of the intracranial atherosclerotic plaque. Third, it is unable to differentiate atherostenosis from stenosis caused by other entities.

Intracranial small infarctions result from atherosclerosis of the parent arteries, which may occlude the orifice of the perforating artery as branch atheromatous disease. Small infarctions also result from lipohyalinosis or microatheromatous, or embolic occlusion of a perforating artery (Adams et al., 1993; Cho et al., 2007; Donnan et al., 1991; Fisher, 1965, 1982). Since branch atheromatous disease is more aggressive than typical lacunar infarctions, intensive antiplatelette or anti-thrombic therapy may be needed for the treatment of branch atheromatous disease. However, the diagnosis of these small infarctions is still difficult because conventional techniques are limited to detect the plaque.

Studies regarding intracranial plaque image have achieved to reveal the mechanism of small infarctions or to diagnose early stage of intracranial atherosclerotic disease. In this report, we address such new magnetic resonance imaging techniques to detect intracranial plaques.

2. Intracranial plaque image

Recently, high-resolution magnetic resonance imaging has been reported to visualize intracranial atherosclerotic plaques (Klein et al., 2005, 2006; Niizuma et al., 2008). It can

detect not only stenotic but nonstenotic intracranial atheromatous plaques or vessel wall thickning. It might provide the information regarding well-known determinants of plaque instability such as richer content in lipid, intraplaque hemorrhage and inflammatory cell infiltration (Chen et al., 2008)

For high-resolution magnetic resonance imaging, pulse sequences were varied. T1-weighted, T2-weighted, moderate T2-weighted, proton density-weighted, and/or postcontrast T1-weighted images by gadolinium-diethylenetriaminepenta-acetic acid with/without miuticontrast black blood sequences were used.

2.1 Basillar artery plaque imaging (Fig. 1)

The infarction mechanism of basilar perforating artery territory can be divided into branch atheromatous disease, cardiogenic embolism, and small vessel disease. Especially, basilar branch atheromatous disease is the most frequent cause of basilar perforating artery territory infarctions (Bassetti et al., 1996; Kumral et al., 2002). Basilar artery atheomatous plaque can block at the orifice of the branch, or can extend into the perforating branch, which results in parapontine or deep pontine infarctions (Fisher & Caplan, 1971; Fisher, 1977).
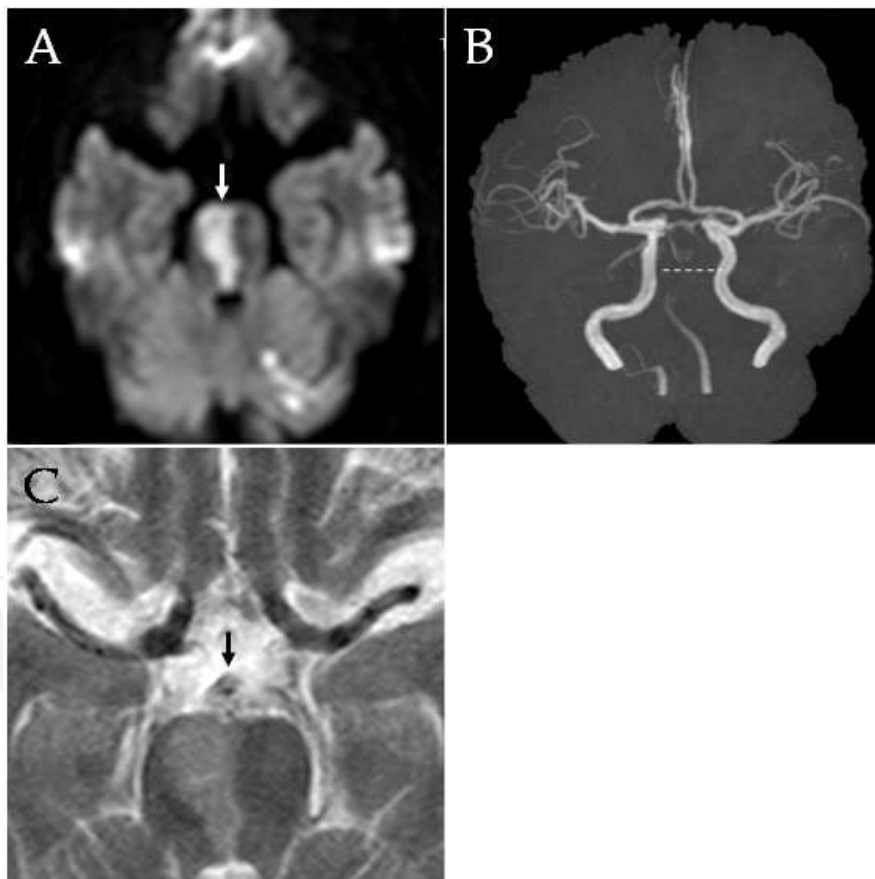

Fig. 1. Basilar artery plaque image using 1.5 tesla magnetic resonance imaging scanner. A: Axial diffusion-weighted image demonstrating a right paramedian pontine high-intensity lesion (arrow). B: Magnetic resonance angiography showing severe stenosis of the basilar artery. C: T2-weighted image at the level of the dotted line on B (TR/TE 3500/100, matrix size 254 × 320), presenting plaque on the vessel wall (arrow).

To detect basilar artery plaques of the basilar artery, Klein et al used high-resolution magnetic resonance imaging with a 1.5-tesla system (Klein et al., 2005, 2010). Twenty-four consecutive patients with the paramedian pontine infarctions underwent high-resolution magnetic resonance imaging, which revealed basilar atherosclerotic plaques in up to 70% patients. Plaque was identified in all patients with severe or moderate stenosis as well as in some patients with normal findings on magnetic resonance angiography (Klein et al., 2005).

Basilar artery plaque could also be a prevalent mechanism not only in the paramedianpontine infarctions but also lacunar or small deep pontine infarctions (Klein et al., 2010). High-resolution magnetic resonance imaging was performed on 43 consecutive patients of medial pontine infarct, including lacunar or small pontine infarction. Basilar artery plaque was detected in 73% of lacunar or small deep pontine infarction cases. This pattern was similar to the cases or paramedian pontine infarctions.

2.2 Middle cerebral artery plaque imaging (Fig. 2-4)

The infarct mechanism in the middle cerebral perforating arterial territory can be divided into four subtypes regardless of lesion size (Cho et al., 2007): (1) middle cerebral artery disease, if there is a corresponding ipsilateral atherosclerotic middle cerebral artery lesion without cardiogenic embolism or atherosclerotic lesion proximal to the middle cerebral artery lesion; (2) internal carotid artery disease, if there is significant ipsilateral internal carotid artery stenosis (> 50%) without evidence of middle cerebral artery disease or cardiogenic embolism; (3) cardiogenic embolism, 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 (Adams et al., 1993) in the absence of atherosclerotic diseases in cerebral vessels; and (4) small vessel disease, if there is no cardiogenic embolism, middle cerebral artery disease, or internal carotid artery disease. People with symptomatic middle cerebral artery disease have overall stroke risk of 12.5% per year (Kern et al., 2005). Since the outcome of middle cerebral artery disease was poor, diagnosis of middle cerebral artery disease is clinically important.

Middle cerebral artery plaque imaging was first reported by on 6 patients of severe middle cerebral artery stenosis using 1.5-tesla magnetic resonance imaging system (Klein et al., 2006). Plaque was clearly detected on the middle cerebral artery wall in all cases. Quantitative measurements presented that lumen area was significantly larger in normal middle cerebral artery segments compared with atherosclerotic ones.

Then several authors reported high-resolution magnetic resonance imaging with a 3-tesla system, by which they detected plaques on the middle cerebral artery and/or wall thickening (Li et al., 2009; Niizuma et al., 2008; Ryu et al., 2009; Swartz et al., 2009; Turan et al., 2011; Xu et al., 2010). Niizuma et al (Niizuma et al., 2008) demonstrated moderate T2-weighted high-resolution magnetic resonance imaging showed plaques on the middle cerebral artery in cases of corona radiate infarction, which indicated the etiology of the infarction (Fig. 2-4). Swartz et al (Swartz et al., 2009) presented that high-resolution magnetic resonance imaging may distinguish the enhancement patterns of different pathologies such as atherosclerosis, inflammation, and other pathologies of the intracranial vessel wall. Ryu et al (Ryu et al., 2009) found multicontrast-weighted black blood imaging have the potential to characterize atherosclerotic plaques. Li et al (Li et al., 2009) and Xu et al (Xu et al., 2010) demonstrated that T2-weighted high-resolution magnetic resonance
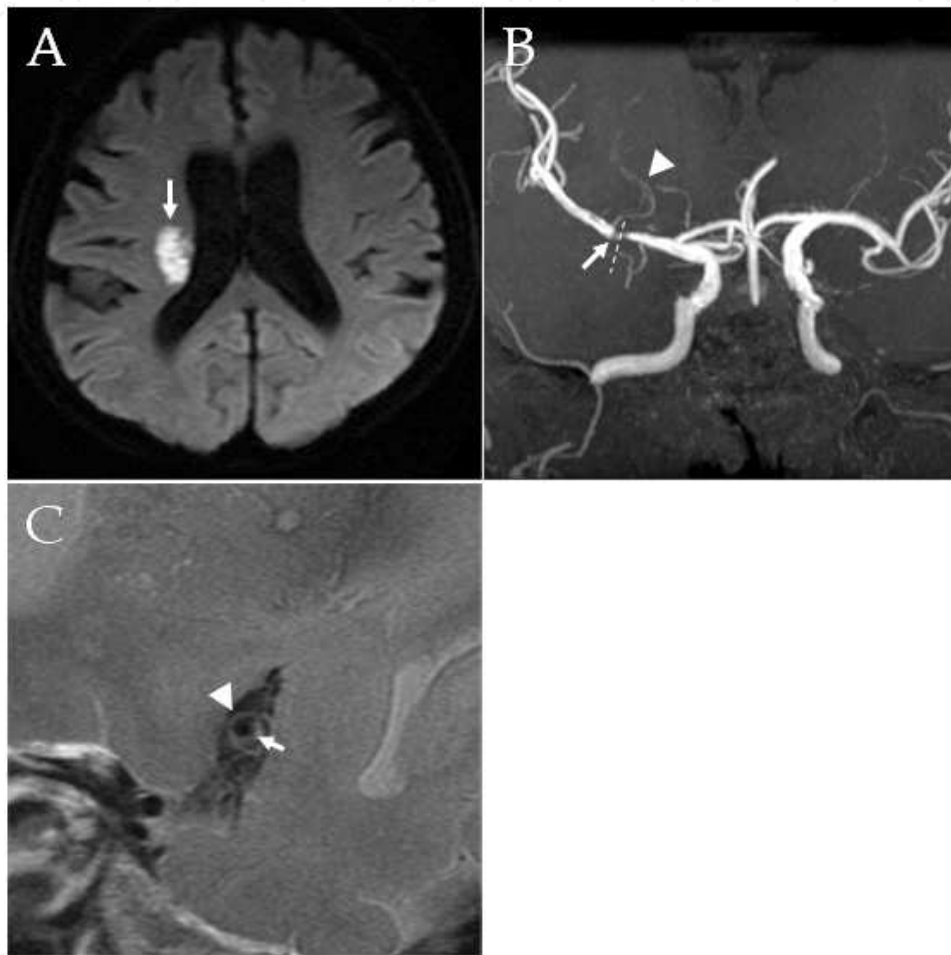
Fig. 2. Middle cerebral artery plaque which is also detected by magnetic resonance angiography (modified from Niizuma et al., 2008). A: Axial diffusion-weighted image demonstrating a high-intensity lesion, 20 mm in diameter, in the right corona radiata (arrow). B: Magnetic resonance angiography processed by partial maximum intensity projection indicating the relationship between the stenotic area (arrow) and lateral striate arteries (arrowhead). C: High-resolution magnetic resonance image perpendicular to the right MCA, at the level of the dotted line on B (TR/TE 2800/50.8, FOV 12 cm × 12 cm, matrix size 512 × 256, slice thickness 2 mm, interslice gap 0.3 mm, NEX 5), presenting the lumen of the right middle cerebral artery (arrow) and plaque on the vessel wall (arrowhead).
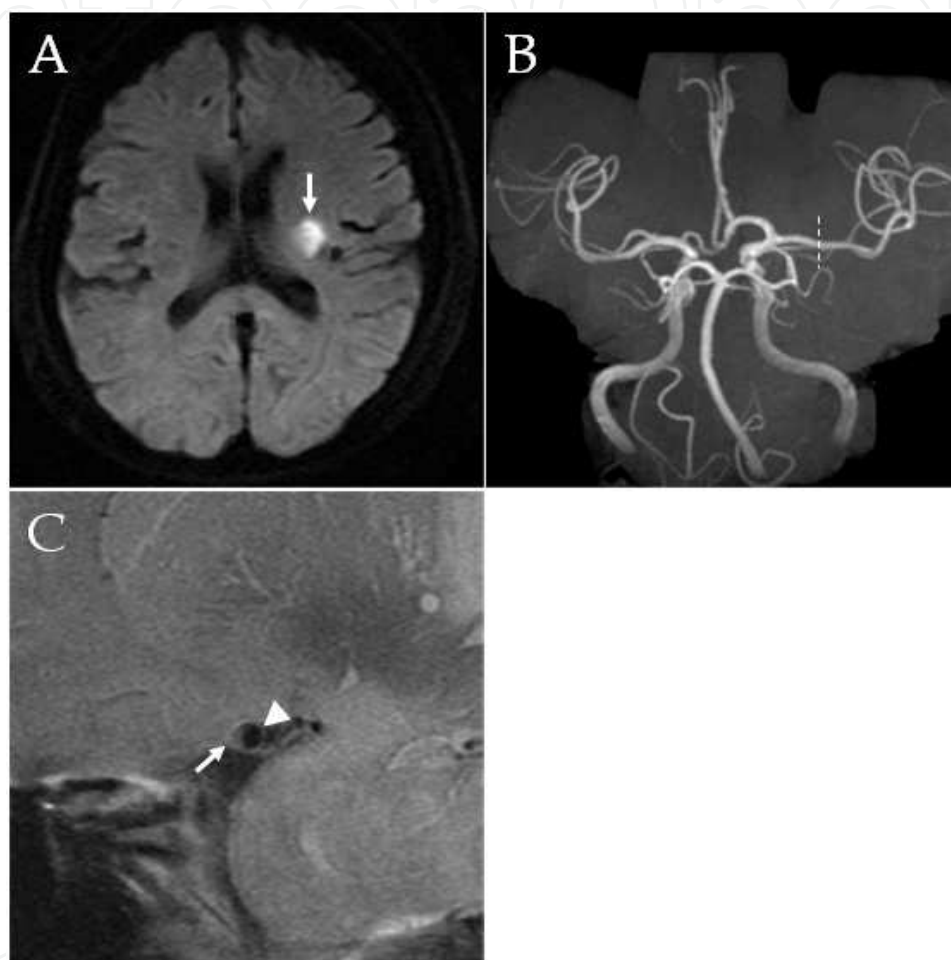
Fig. 3. Middle cerebral artery plaque which cannot be detected by magnetic resonance angiography (modified from Niizuma et al., 2008). A: Axial Diffusion-weighted image demonstrating a high-intensity lesion, 12 mm in diameter, in the left corona radiata (arrow). B: Magnetic resonance angiography showing no evidence of atherosclerosis. C: High-resolution magnetic resonance image perpendicular to the left MCA, at the level of the dotted line in B (TR/TE 2800/50.8, FOV 12 cm × 12 cm, matrix size 512 × 256, slice thickness 2 mm, interslice gap 0.3 mm, NEX 5), presenting the lumen of the left middle cerebral artery (arrow) and plaque on the vessel wall (arrowhead), despite the apparently normal magnetic resonance angiography.
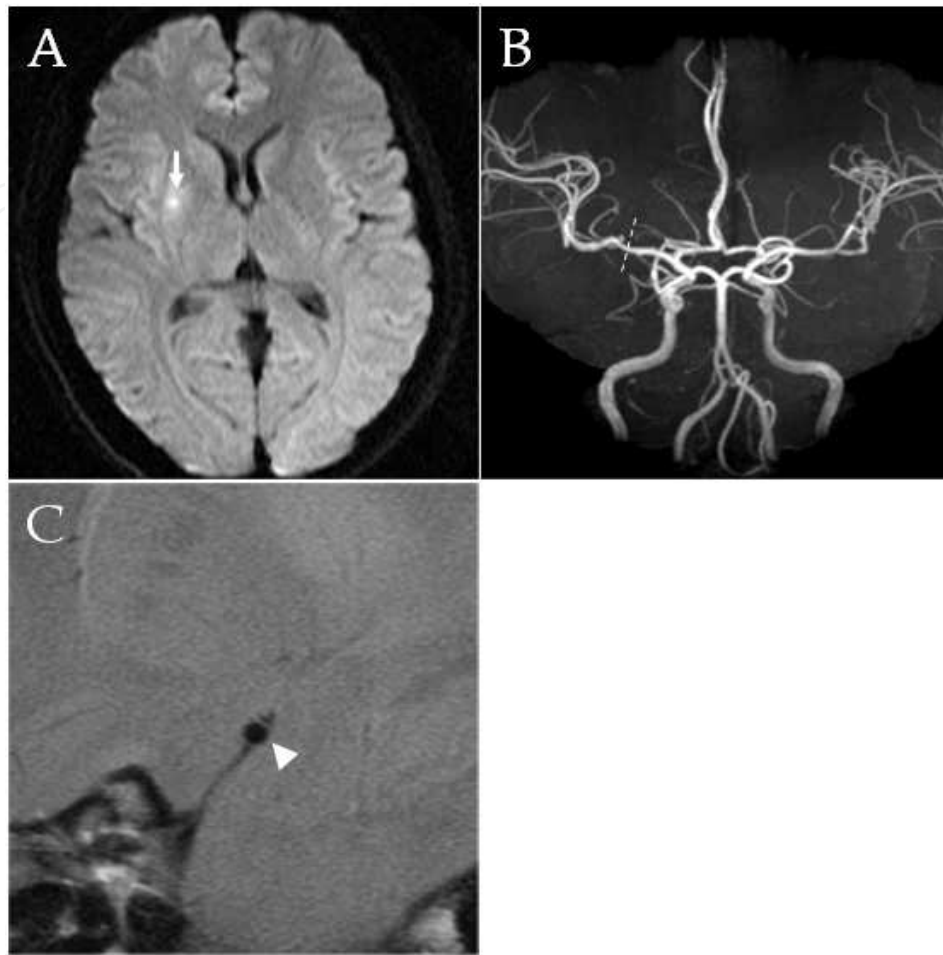
Fig. 4. No detection of middlee cerebral artery plaque on the right lacunar infarction (modified from Niizuma et al., 2008). Axial Diffusion-weighted image demonstrating a high-intensity lesion in the right putamen (arrow) and corona radiate. B: Magnetic resonance angiography showing no evidence of atherosclerosis. C: High-resolution magnetic resonance image perpendicular to the right middle cerebral artery, at the level of the dotted line in B (TR/TE 2800/50.8, FOV 12 cm × 12 cm, matrix size 512 × 256, slice thickness 2 mm, interslice gap 0.3 mm, NEX 5), presenting the lumen of the middle cerebral artery (arrowhead), though there were no plaques on the vessel wall.

imaging differentiated wall thickening, arterial remodeling, and atherosclerotic plaque in symptomatic and asymptomatic patients with intracranial middle cerebral artery stenosis.

2.3 High-field magnetic resonance imaging

Although 3.0-tesla magnetic resonance imaging clearly demonstrated plaques and abnormal intracranial vessel walls, it seemed difficult to show the healthy vessel wall (Li et al., 2009; Niizuma et al., 2008; Ryu et al., 2009; Swartz et al., 2009; Turan et al., 2011; Xu et al., 2010). Van der Kolk et al (Van der Kolk et al., 2011) aimed to depict the vessel wall of intracranial

arteries, also in the absence of disease. They used a volumetric turbo spin-echo sequence with inversion recovery and magnetization preparation using 7.0-tesla system. They achieved high image resolution and sensitivity, thus walls of major arteries of the circle of Willis were identified in all cases. 7.0-tesla high-resolution magnetic resonance imaging depicted that 66% of the patients had more than one lesions in major intracranial arteries, whereas only 27% of the lesions caused stenosis on magnetic resonance angiography. They concluded that 7.0-tesla high-resolution magnetic resonance imaging has possibility to study the role of intracranial arterial wall pathology in more detail.

2.4 Plaque instability

Richer content in lipid, intraplaque hemorrhage and inflammatory cell infiltration are well-known determinants of plaque instability (Chen et al., 2008). Now, magnetic resonance imaging has the ability to differentiate such plaque components in addition to determining the degree of luminal narrowing of the carotid plaques (Fayad & Fuster, 2000). The use of gadolinium-diethylenetriaminepenta-acetic acid may enhance detection of wall lesions and determination of plaque instability. In the caroted artery, plaque enhancement may indicate neovascularized fibrous tissue that correlates with unstable plaque (Yuan et al., 2002a). Multisequence magnetic resonance imaging can identify different carotid plaque components with high sensitivity, specificity, and accuracy not only ex vivo but also in vivo (Shinnar et al., 1999; Yuan et al., 2001). High-resolution magnetic resonance imaging of the carotid plaque can distinguish advanced lesions from early and intermediate atherosclerotic plaque (Cai et al., 2002).

As intracranial plaque instability, Meyers et al (Meyers et al., 2009) reported that intraplaque hemorrhage was detected by intravascular ultrasound in symptomatic intracranial atherosclerotic disease, indicating that intracranial atherosclerotic plaques can become symptomatic after complication by intraplaque hemorrhage similer to coronary artery plaques. Moreover, Turan et al (Turan et al., 2011) observed intraplaque high signal intensity lesion in the symptomatic middle cerebral artery stenosis using high-resolution T1-weighted image. Its characteristics were similer to intraplaque hemorrhage on the caroted arteries. These findings suggest that high-resolution magnetic resonance imaging allows characterization of intraplaque hemorrhage in vivo.

The diagnostic value of different enhanced patterns of intracranial plaques by gadolinium-diethylenetriaminepenta-acetic acid is not determined, although plaque enhancement is commonly considered to be a marker of symptomatic or unstable plaque. Swartz et al (Swartz et al., 2009) described that enhanced patterns of plaques might differentiate pathologies such as atherosclerosis, inflammation, and other pathologies of the intracranial vessel wall (Swartz et al., 2009). However, Klein et al (Klein et al., 2006) described a case of asymptomatic middle cerebral artery stenosisi with strongly enhanced plaque by gadolinium-diethylenetriaminepenta-acetic acid, indicating plaque enhancement does not always indicate symptomatic plaque. Further studies are needed to determine this issue.

2.5 Limitations

For clinical use of high-resolution magnetic resonance imaging to detect intracranial plaques, following issues must be considered. First, whether the plaque is a true plaque must be more confirmed. Other modalities such as ultrasound, pathology, or interaoperative

observation should be compared with findings of high-resolution magnetic resonance image. Although previous studies of carotid artery plaques demonstrated a significant correlation between pathological findings and plaque characteristics detected by high-resolution magnetic resonance imaging (Honda et al., 2006; Toussaint et al., 1996; Yuan et al., 2002b), histological analyses are required to evaluate this technique in detail.

Second, high-resolution magnetic resonance imaging cannot demonstrate a direct causative relationship between plaque and infarctions, even if plaques are located at the level of the perforating arteries. Because of the small size of intracranial arteries, spatial resolution needs to be more optimized. Future technical improvements for imaging perforating arteries or high-field systems will reveal the relationship of plaque and infarctions.

Third, the relationships among signal pattern of high-resolution magnetic resonance imaging, findings of magnetic resonance angiography, clinical symptoms, and characteristics of the plaques including thickness and distributions, and vulnerability, remain unclear, although cumulative evidences indicate intracranial plaque imaging allows characterization of plaques *in vivo*.

3. Conclusions

High-resolution magnetic resonance imaging can identify plaques on the walls of basilar artery or middle cerebral artery. This technique compensates the limitation of magnetic resonance angiography. However, further studies are needed to evaluate the limitations as described above. Besides these limitations, we consider that high-resolution magnetic resonance imaging is promising for the direct identification of intracranial arterial plaques and for the accurate estimation of the pathogenesis of the infarction, which will have an impact on the treatment strategy.

Prospective studies or long term follow up studies are needed to evaluate the accuracy and reproducibility.

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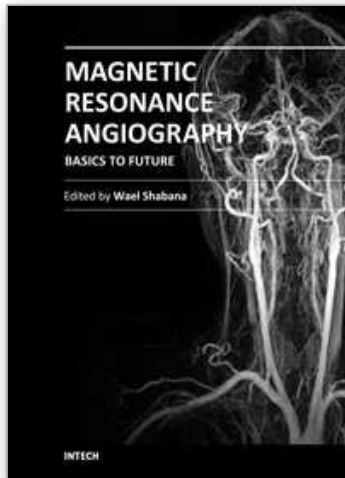
Magnetic Resonance Angiography Basics to Future

Edited by Prof. Wael Shabana

As MRI has paved its role in diagnostic angiography. MRA has the potential to provide more physiological and pathophysiological data over the disease in addition to the anatomical information. This book is divided into three sections. The first section discusses the basics of MRI angiography. It starts with focus on the contrast agents that are mainly used in MR angiography with detailed discussion of advantage and limitations of different types of contrast. The second chapter is oriented more towards the technical consideration that contribute to good quality examination, both the non contrast and contrast based sequences from black to bright blood imaging , contrast enhanced MRA, review of clinical application of MRA in different body systems and MR venography. The second section reviews the clinical application of MRI mainly in the head and neck and brain ischemia imaging. The new high resolution intracranial plaque imaging of the branch athermanous disease, to the hemodynamic of intracranial atherosclerotic stroke and quantitative MRA imaging in neurovascular imaging, are the topics in this section. Also this section covers the future prospective and the new frontiers MRI angiography is exploring. In the third section, MRA of aortic disease in children with emphasis on cardiac MRA.

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