Application of Magnetic Resonance Plaque Imaging Using Inversion-Recovery and Fat-Suppressed Three-Dimensional Steady-State Free Precession to Iliac Artery Stenosis

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Abstract

A 61-year-old Japanese man with right common iliac artery stenosis underwent magnetic resonance plaque imaging for an evaluation of the nature of the stenosis that used inversion-recovery and fat-suppressed three-dimensional steady-state free precession (IR-SSFP) and T1-weighted black blood imaging. The stenosis was depicted as diffuse high signal intensity on the IR-SSFP, and the deep layer was depicted as high signal intensity on the T1-weighted black blood image, suggesting a lipid-rich necrotic core in the shallow layer and intraplaque hemorrhage in the deep layer. After an evaluation by intravascular endoscopy and optical coherence tomography (OCT), stent placement was performed under balloon protection. Yellowish plaque was aspirated from a balloon guiding catheter, and a histopathological examination confirmed cholesterol crystals and a thrombus.

Key words: iliac artery stenosis, MR plaque imaging, IR-SSFP, vulnerable plaque

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Introduction

Vulnerable plaque consists of a large lipid-rich necrotic core, increased plaque inflammation, and intraplaque hemorrhage [1-3]. Such plaques can cause an ischemic event during or after percutaneous coronary intervention, carotid artery stenting, and carotid endarterectomy [4-6]. Several studies have demonstrated the efficacy of magnetic resonance (MR) plaque imaging for detecting vulnerable plaque on the carotid and coronary arteries [4-7], but there has been no report of MR plaque imaging for peripheral artery disease (PAD) of the iliac artery. Cholesterol crystal embolization (CCE) is one of the serious complications associated with endovascular therapy (EVT) for PAD. There is no established treatment for CCE other than supportive therapy [8], resulting in limb amputation [9]. Therefore, we believe that detecting vulnerable plaque before EVT for PAD will be helpful for predicting and preventing CCE.

Here, we report a case of iliac artery stenosis that was suspected to have a lipid-rich necrotic core and intraplaque hemorrhage on MR plaque imaging. We performed a successful stent placement under balloon protection.

Case Report

A 61-year-old Japanese man was referred to our hospital for the treatment of intermittent claudication on the bilateral calf. His medical history was significant for subarachnoid hemorrhage and dyslipidemia. His ankle brachial pressure index (ABPI) value was 0.74 on the right and 0.62 on the left. His renal function was normal (eGFR: 91.3). Computed tomography angiography (CTA) revealed stenosis of the bilateral common iliac artery (CIA) and occlusion of the right internal iliac artery (Fig. 1). There was no calcified lesion but a thick, low-density area of stenosis was observed, sug-
Figure 1. Computed tomography angiography (CTA) image showing the stenosis of the bilateral common iliac arteries and right external iliac artery. The left iliac artery stenosis was treated with stent placement on the first admission.

Figure 2. Coronal magnetic resonance (MR) plaque images of the right iliac artery stenosis. The entire plaque of the iliac artery is depicted as homogeneously high signal intensity on the IR-SSFP image.

Figure 3. Corresponding black blood T1-weighted images showing a low signal intensity area (arrow) in the shallow layer, suggesting a lipid-rich plaque and a high signal area (arrowhead) in the deep layer consistent with the intra plaque hemorrhage.

We therefore performed an evaluation using MR plaque imaging. A navigator-gated free-breathing inversion-recovery and fat-suppressed three-dimensional steady-state free precession (IR-SSFP) sequence and T1-weighted black blood imaging were performed. The scan parameters were as follows: 1) IR-SSFP gradient echo sequence: field of view (FOV), 350 mm; matrix, 256 × 256; slice thickness, 3.0 mm on coronal image; repetition time/echo time/inversion time, 4.4/2.2/1,000 msec; flip angle (FA), 90°; and SENSE factor, 2.5, and 2) black blood T1-weighted spin echo sequence: FOV, 350 mm; matrix, 288 × 288; slice thickness, 1.2 mm on coronal image; repetition time/echo time, 400/14 msec, FA, 80°, refocusing angle, 40°; and SENSE factor, 2.8.

The entire plaque of the right common iliac artery was depicted as high signal intensity on IR-SSFP images (Fig. 2). On the other hand, the shallow layer of the plaque was depicted as low signal intensity on T1-weighted black blood images, and the deep layer of the plaque was depicted as high signal intensity on T1-weighted black blood images (Fig. 3), suggesting that the stenosis of the shallow layer reflected a lipid-rich necrotic core and that the stenosis of the deep layer reflected the intraplaque hemorrhage. The stent placement of the left common iliac artery was performed without any complication on first admission.

On the patient’s second admission, we planned stent placement for the right common iliac artery stenosis under balloon protection, following an evaluation using intravascular endoscopy and optical coherence tomography (OCT). We informed him that we would perform endovascular therapy under balloon protection for the right common iliac artery stenosis and investigate the stenotic lesions using various de-
Written informed consent was obtained from him and his family. A balloon-guiding catheter (OPTIMO PPI, Tokai Medical Products, Aichi, Japan) was inserted from the right femoral artery. Intravascular endoscopy was performed using a Smart-i (iHeart Medical Co., Tokyo), which revealed that the surface of the stenosis was whitish intima; no mural thrombus was identified, confirming that the stenosis was due to atheroma (Fig. 4).

The stenosis was negotiated with a 4-F angiographic catheter (RC-09, Medikit, Gifu, Japan) and a 0.035-inch guidewire (Radifocus, Terumo, Tokyo). The guidewire was then exchanged with a micro guidewire (Chevalier 14 floppy, Cordis, Miami Lakes, FL, USA) for evaluation by OCT using a Fast view coronary imaging catheter (Terumo, Tokyo). OCT revealed a decreased intensity signal area in the stenosis, compatible with a lipid-rich plaque with mild fibrosis (Fig. 5). An angioplasty catheter (Sterling, Boston Scientific, Marlborough, MA) was placed in the CIA for contralateral protection.

Both balloons were manually inflated for distal and contra-lateral protection during the stent placement with a self-expanding stent (Luminexx, C.R. Bard, Covington, GA), and post dilated with another angioplasty catheter (Sterling). Then, 80 mL of blood was aspirated from the balloon guiding catheter before deflation of the balloons. Yellowish plaques and a tiny thrombus were obtained from the aspirated blood. The histopathological examination revealed cholesterol crystals and a thrombus (Figs. 6, 7). The post-operative course of the patient was uneventful, and the
follow-up ABPI values were normalized (1.12 on the right, 1.06 on the left). The patient was discharged on the 6th day after admission with no recurrent symptoms over a 10-month follow-up.

Discussion

Distal embolization is one of the most serious complications during endovascular procedures. The incidence of distal embolization following routine angioplasty and EVT in patients with PAD has been reported to range from 0.8% to 18.8%, depending on the series [9]. Maezawa et al reported that large debris, which was defined as debris of >2 mm in maximal diameter, was confirmed in 33 of 69 patients (48%) and was trapped more frequently in the iliac artery than in the femoral artery. They concluded that distal protection would be considered when the lesion is ulcerative, located in the iliac artery, and found to contain a necrotic core on virtual histology intravascular ultrasound [9]. On the other hand, in order to increase cost-effectiveness, it is not necessary to use protection devices in all cases. Consequently, a clinical need exists to develop methods to identify vulnerable plaque before EVT. We are limited to performing evaluations using MR plaque imaging in patients who have a risk of CCE or thrombotic distal embolization, such as long occlusion of the iliac or superficial femoral artery. It is not always necessary to apply it to the stenotic lesions. MRI was performed in this patient as an exception because the risk of in-stent protrusion or distal embolization was higher since the stenosed CIA accompanied mild dilatation; therefore, the plaque was rich, and the ipsilateral internal iliac artery was occluded.

The utility of preoperative assessment of coronary or carotid artery stenosis using MR plaque imaging has been reported since 2008 [5,7]. The perioperative embolic complications tend to occur in lesions that show high signal intensity on a non-contrast black blood T1-weighted image, which probably reflects an intraplaque hemorrhage. Since intraplaque hemorrhages usually occur in the lipid-rich necrotic core [1] of an atheroma, the presence of an intraplaque hemorrhage suggests the presence of a lipid-rich necrotic core and that the atheroma is vulnerable on a carotid or coronary artery [2, 6, 7].

T1-weighted black blood imaging is the standard imaging modality used for the diagnosis of vulnerable plaques, but a lipid-rich necrotic core without an intraplaque hemorrhage cannot be detected by this imaging sequence because the main component of the necrotic core is cholesterol, which is usually depicted as a low signal on black blood T1-weighted images. There has been no report to date of an evaluation of plaque using only one sequence that can depict both a lipid-rich necrotic core and an intra-plaque hemorrhage simultaneously. In contrast, IR-SSFP imaging provides unique signal intensity; i.e. the signal intensity reflects the T2/T1 properties of the tissue [10], and the lipid-rich necrotic core, which contains free water, will be depicted as a high signal due to its T2-elongation effect. An intraplaque hemorrhage will also be depicted as a high signal due to its T1-reducing effect on this sequence. Thus, IR-SSFP imaging may be useful for more correct quantitative evaluations of vulnerable plaques with the use of only a single sequence.

The precise determination of the correct volume of vulnerable plaque may contribute to the prediction of the severity of thromboembolic complications during various endovascular treatments. However, existing studies have only evaluated the relationship between the presence of vulnerable plaque and the incidence of thromboembolic complications. Another advantage of the IR-SSFP sequence is that the differentiation of lipid-rich plaque and an intraplaque hemorrhage can be evaluated by a combination of IR-SSFP images and conventional T1-weighted black blood images.

In our patient’s case, the plaque depicted as low signal on the conventional T1-weighted image and high signal on the IR-SSFP image was consistent with a lipid-rich necrotic core. The OCT findings also supported this result, and the yellowish plaque obtained from the aspirated blood was probably derived from this plaque because the other arteries were healthy and did not contain a major thrombus on gross appearance.

This study has several limitations. The specimen was obtained from the aspirated blood, and it was thus difficult to confirm that the specimen was truly derived from the plaque that was suspected to be the lipid-rich necrotic core on MRI. In addition, a correlation between the imaging and histopathologic findings is lacking. Further studies are necessary to determine this correlation using other diseases, including carotid artery stenosis.

In conclusion, MR plaque imaging using the IR-SSFP sequence may be useful to detect both lipid-rich plaque and intraplaque hemorrhages in peripheral artery disease.

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