Contrast-enhanced Steady-state Free Precession in the Assessment of Hypertrophic Obstructive Cardiomyopathy after Alcohol Septal Ablation

Yasuo Amano 1*, Fumi Yamada 1, Mitsunobu Kita mur a 2, Masaki Tachi 1, Kumiko Mine 1, Shinichiro Kumita 1, and Morimasa Takayama 3

1 Department of Radiology, Nippon Medical School
2 Department of Intensive Cardiac Care Unit, Nippon Medical School
1–1–5 Sendagi, Bunkyo-ku, Tokyo 113–8603, Japan
3 Department of Cardiology, Sakakibara Heart Institute

(Received April 6, 2015; Accepted June 10, 2015; published online September 4, 2015)

Purpose: We evaluated the feasibility of contrast-enhanced steady-state free precession (ceSSFP) in the assessment of myocardial injury and obstruction of the left ventricular outflow tract (LVOT) in patients with hypertrophic obstructive cardiomyopathy (HOCM) after alcohol septal ablation (ASA).

Methods: Twelve patients with HOCM underwent 16 magnetic resonance (MR) examinations following ASA. Precontrast SSFP, ceSSFP and late gadolinium enhancement (LGE) imaging were performed with a 1.5-tesla imager. ceSSFP was performed 3 to 7 min after gadolinium injection. We visually and quantitatively evaluated the signal patterns of the myocardium after ASA on SSFP and LGE MR imaging. We observed the LVOT using ceSSFP in the 3-chamber view.

Results: We could visualize ASA-induced myocardial infarction (MI) in all 16 studies by LGE and ceSSFP but in only 6 studies (37.5%) by precontrast SSFP. Contrast was higher between MI and remote myocardium with LGE than ceSSFP \((P < 0.01)\). ASA-induced hypointense regions were well visualized by the 2 sequences after contrast in the 7 patients who underwent MR imaging within 7 weeks of ASA and in a few patients after 80 weeks from ASA. The ceSSFP allowed comparable visualization of the jet flow crossing the LVOT to that derived from echocardiographic data.

Conclusion: Contrast-enhanced steady-state free precession allows assessment of myocardial injury as well as of the left ventricular outflow tract after alcohol septal ablation in a single scan without penalty in scan time and cine imaging contrast.

Keywords: alcohol septal ablation, hypertrophic obstructive cardiomyopathy, late gadolinium enhancement, steady-state free precession

Introduction

Hypertrophic obstructive cardiomyopathy (HOCM) is characterized by myocardial hypertrophy that obstructs the left ventricular outflow tract (LVOT). In HOCM, an increase in the LVOT pressure gradient can lead to decreased cardiac output, reactive hypertrophy of the myocardium distinct from the LVOT, and the resultant decrease in coronary flow reserve. 1–3 Consequently, HOCM is associated with chest pain or syncope during exercise, and its prognosis is worse than that of hypertrophic cardiomyopathy without LVOT obstruction. 2–4 Alcohol septal ablation (ASA) is a less invasive alternative to myectomy for the treatment of HOCM 4,5; in ASA, a small volume of absolute alcohol injected via the coronary septal branch arteries leads to infarction of hypertrophied myocardium. ASA releases the LVOT obstruction, decreases reactive myocardial hypertrophy, and improves clinical symptoms in patients with HOCM.

Magnetic resonance (MR) imaging is useful for evaluating the efficiency of ASA. 6–10 Cine steady-
SSFP for Alcohol Septal Ablation

state free precession (SSFP) imaging quantifies the myocardial wall thickness and mass and permits visualization of jet flow crossing the LVOT.6,10 Late gadolinium enhancement (LGE) imaging is useful to visualize and quantify myocardial infarction (MI) after ASA.6,8,10 However, multiple breath holds and a side-by-side comparison of cine SSFP and LGE images are required to assess the relationship between LVOT obstruction and ASA-induced MI or microvascular obstruction (MVO).

SSFP may be useful for identifying MI or MVO because of its sensitivity to edema, coagulation, and hemorrhage in the tissues,11–13 but its usefulness for detecting myocardial injuries led by ASA has not been assessed. Some studies have demonstrated that contrast-enhanced SSFP (ceSSFP) permits evaluation of both myocardial wall motion and acute myocardial injury, such as infarction and myocarditis, in a single scan.14–16 ceSSFP can show MI and LVOT jet flow simultaneously in patients with HOCM after ASA, but its use has not been reported in patients. In this study, we sought to determine the feasibility of ceSSFP in the assessment of myocardial injury and LVOT jet flow in patients with HOCM after ASA.

Materials and Methods

Patients

We retrospectively enrolled 12 patients (2 men, 10 women; aged 51 to 85 years, mean age, 64.7 years) with HOCM who underwent contrast-enhanced MR imaging after ASA between September 2008 and August 2014 in our institution. HOCM was diagnosed as asymmetrical septal hypertrophic cardiomyopathy with an LVOT pressure gradient exceeding 30 mm Hg on echocardiography at rest.1,4–6 ASA was performed in cases with HOCM with New York Heart Association functional classification of II or III and clinical symptoms associated with an increased LVOT pressure gradient (e.g., chest oppression, dyspnea on exertion) that were refractory to beta-blocker and cibenzoline treatment.4–7,17 The patients were referred for MR study to evaluate cardiac function and MI after ASA. Our institutional review board approved the ASA and cardiac imaging studies, and all patients gave informed consent for the studies.

MR imaging

MR imaging examinations were performed using a 1.5-tesla imager with a 5-channel cardiac phased-array coil (Achieva; Philips Healthcare, Best, The Netherlands). Following localizer scans, 2-dimensional (2D) cine SSFP was acquired in the long- and short-axis plane. The typical imaging parameters of cine SSFP were: repetition time (TR), 2.8 ms; echo time (TE), 1.4 ms; flip angle, 60°; in-plane resolution, 1.9 × 1.6 mm²; and slice thickness, 10 mm. Thereafter, we manually administered gadolinium-based contrast agents at a dose of 0.15 mmol/kg and performed 2D cine ceSSFP in the 3-chamber plane 3 to 7 min after contrast administration. Ten minutes after the gadolinium injection, we performed Look-Locker imaging to determine the inversion time to null the myocardial signal.18 We acquired long- and short-axis 2D LGE imaging with inversion-recovery segmented gradient-echo imaging. The typical imaging parameters of LGE imaging were: TR, 9.3 ms; TE, 4.6 ms; flip angle, 10°; in-plane resolution, 1.6 × 1.8 mm²; and slice thickness, 8 mm, with an interslice gap of 2 mm. We performed short-axis SSFP and LGE imaging to cover the whole left ventricle and used 3-chamber ceSSFP to acquire images of the 3 to 6 slices showing the LVOT. A parallel imaging technique (i.e., sensitivity encoding) was used in each imaging sequence.

Imaging analysis

We defined ASA-induced MI on SSFP or LGE images as regions of hyperintensity that received their blood supply from the septal branch arteries.6–10,14,19 We documented a region of relative hypointensity (Hypo) located adjacent to the left or right ventricular cavity and surrounded by MI as induced by ASA.9,19 Remote myocardium was defined as septal myocardium that was localized more than one cm distant from MI.

One radiologist with 15 months’ experience in cardiac MR imaging who was blinded to the interval between the ASA and MR studies evaluated the presence of MI and Hypo. The reader interpreted SSFP before and after contrast administration (i.e., ceSSFP) and LGE images separately in each patient. Also blinded to the LVOT pressure gradient estimated by echocardiography, the reader recorded the marked jet flow crossing the LVOT on the cine ceSSFP in the 3-chamber view. Another radiologist with 17 years’ experience in cardiac MR imaging placed regions of interest (ROIs) that included at least 5 pixels on the MI or Hypo that was documented. ROIs including 16 pixels were also placed on remote myocardium. Signal intensities of the MI, Hypo, and remote myocardium were measured on the SSFP images at end-diastole and on the LGE images.

First, we evaluated the presence of MI and Hypo on precontrast SSFP, ceSSFP, or LGE images in terms of the interval between ASA and the MR
studies. Second, we compared the signal ratios between the MI and remote myocardium or Hypo between precontrast SSFP, ceSSFP, and LGE imaging. The signal ratio between the MI and remote myocardium or Hypo was defined by dividing the signal intensity of MI less that of the remote myocardium or Hypo by the signal intensity of the remote myocardium or Hypo. Third, we compared signal intensities between remote myocardium and Hypo on SSFP and LGE imaging. Analysis of variance or Student t test was used for the comparisons when appropriate, and a P value less than 0.05 was defined as significant. Fourth, we assessed the relationship between the LVOT jet flow on ceSSFP and the LVOT gradient pressure estimated by echocardiography performed within 4 weeks of the MR studies.

Results

Twelve patients with HOCM underwent 16 MR imaging examinations without complication; two underwent MR studies twice and one, 3 times. Imaging analysis was performed successfully in all studies. The interval between ASA and MR studies varied from 1.5 weeks to 335 weeks (median, 16.0 weeks), depending on clinical symptoms, temporal pacemaker implantation, and need for follow-up. The dose of injected alcohol ranged from 1.3 to 5.4 mL (mean ± standard deviation [SD], 3.18 ± 1.26 mL). The interval between MR studies and the interval between ASA and MR studies varied from 1.2 to 78.0 mm Hg. The LVOT gradient pressure was below 30 mm Hg (range, 30.1 to 78.0 mm Hg) in the 3 studies (Fig. 3). The remaining patient had an LVOT gradient pressure of 19.7 mm Hg. The LVOT gradient pressure was below 30 mm Hg (14.6 ± 3.9 mm Hg; range, 10.2 to 20.0 mm Hg) in 5 of the 6 studies showing normal LVOT jet flow on cine ceSSFP and 40.1 mm Hg in the remaining patient.

Discussion

We could visualize ASA-induced MI with high contrast on LGE MR imaging, which is considered the standard technique for its assessment. Precontrast SSFP was not feasible for assessing myocardial injuries. We could identify MI in all patients with ceSSFP despite its lower signal ratio than LGE imaging. Hypo was observed in all patients with HOCM who underwent MR studies within 7 weeks after ASA and in 2 patients who underwent MR studies later than 80 weeks after ASA using LGE and ceSSFP imaging. Compared with echocardiography, cine ceSSFP in the 3-chamber view might be feasible for evaluating jet flow crossing the LVOT. Therefore, ceSSFP allows for simultaneous assessment of ASA-induced myocardial injuries and LVOT obstruction in patients with HOCM.

SSFP is sensitive to coagulation and hemorrhage, which have been identified in ablated myocardium by histological study. The ceSSFP imaging acquired 3 to 7 min after gadolinium injection may reflect an expanded interstitial space or hyperemia in addition to tissue edema. These factors may have contributed to the feasibility of ceSSFP for visualizing both MI and MVO following ASA. On the other hand, we could visualize MI

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Fig. 1. A 51-year-old man with hypertrophic obstructive cardiomyopathy after alcohol septal ablation (ASA). A magnetic resonance (MR) imaging study was performed 2.5 weeks after ASA. (A) Late gadolinium enhancement imaging shows myocardial infarction (arrowhead) and microvascular obstruction (MVO, arrow) adjacent to the left ventricle with high imaging contrast. (B) Precontrast steady-state free precession (SSFP) imaging shows myocardial infarction (arrowhead) with lower contrast. (C) Contrast-enhanced SSFP allows clear visualization of myocardial infarction (arrowhead) and MVO (arrow).

Fig. 2. A 66-year-old woman with hypertrophic obstructive cardiomyopathy after alcohol septal ablation (ASA). A magnetic resonance (MR) imaging study was performed 80 weeks after ASA. (A) Late gadolinium enhancement imaging shows myocardial infarction. (B) Contrast-enhanced steady-state free precession (SSFP) imaging allows visualization of myocardial infarction (arrowhead) and a relatively hypointense region (arrow) as well as the left ventricular outflow tract.
in limited cases by precontrast SSFP. Signal intensities on SSFP are affected by T1 and magnetization transfer as well as by T2.11,12 We used no preparation pulse, such as T2-preparation or black-blood pulse,11,12 to improve visualization of myocardial edema on routine cine SSFP.

Hypo surrounded by MI may be divided into 2 types based on the interval between ASA and acquisition of MR studies–MVO observed early after ASA and dense scarring observed later. MVO is the core of MI induced by ASA.8,13,19 On LGE imaging, the signal intensity of MVO was higher than that of remote myocardium. This might be explained by the T1-shortening effect of some hemoglobin contents or by partial volume effect.23 On ceSSFP imaging, MVO was hypointense to remote myocardium, possibly because of the severe hypoperfusion and coagulation of MVO.11–14 The measurement of the signal ratio on LGE or ceSSFP imaging could be useful for differentiating between MVO and the area not successfully ablated.

We believe another type of Hypo, identified later than 80 weeks after ASA, has not been reported. However, Butz and colleagues2 reported a patient with HOCM who showed extensive MI associated with calcification 11 years after ASA. Dense scarring with few vessels or metaplasia, including fatty metamorphosis, might have replaced the core of the MI. The Hypo was clearly visualized on ceSSFP imaging, possibly because of the sensitivity of ceSSFP to marked decreases in water content and perfusion.22

ceSSFP imaging also showed the LVOT jet flow with the cine mode. Visualized using the present ceSSFP technique, the jet flow may be consistent with the LVOT pressure gradient. These results indicate that contrast enhancement does not prevent cine imaging assessment using the SSFP sequence.15 ceSSFP may allow observation of both myocardial injuries and LVOT obstruction after ASA in a single scan. Because we performed ceSSFP during the interval between gadolinium injection and Look-Locker T1 scout imaging, there was no penalty of examination time.

Our study has some limitations. First, the sample size was relatively small. Second, the interval between ASA and MR studies varied because of the clinical status of each patient. Nonetheless, our findings indicate the feasibility of ceSSFP and LGE MR imaging for the identification of MI and

Table. Signal ratios between myocardial infarction (MI) and remote myocardium or relative hypointense region on magnetic resonance imaging after alcohol septal ablation (ASA)

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<tr>
<th></th>
<th>ceSSFP</th>
<th>LGE</th>
<th>P value</th>
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<tr>
<td>MI/remote</td>
<td>0.64 ± 0.51 (0.044 to 1.93)</td>
<td>4.96 ± 2.63 (1.38 to 10.8)</td>
<td>&lt;0.01</td>
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<td></td>
<td>(n = 16)</td>
<td>(n = 16)</td>
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<tr>
<td>MI/Hypo</td>
<td>0.65 ± 0.41 (0.11 to 1.63)</td>
<td>1.09 ± 0.70 (0.089 to 2.18)</td>
<td>0.021</td>
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<tr>
<td></td>
<td>(n = 10)</td>
<td>(n = 9)</td>
<td></td>
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<tr>
<td>within 7 weeks of ASA</td>
<td>0.62 ± 0.27 (0.11 to 1.08)</td>
<td>1.23 ± 0.73 (0.089 to 2.18)</td>
<td>0.042</td>
</tr>
<tr>
<td></td>
<td>(n = 7)</td>
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ceSSFP, contrast-enhanced steady-state free precession; Hypo, region with relative hypointensity; LGE, late gadolinium enhancement; MI, myocardial infarction. LGE imaging showed MI after alcohol septal ablation for hypertrophic obstructive cardiomyopathy with a higher contrast than that with contrast-enhanced SSFP.
MVO following ASA. Third, we did not determine the clinical importance of Hypo in this study. Previous studies have demonstrated a significant relationship of the location of MVO or MI to the effectiveness of ASA. Fourth, the use of the parallel imaging technique and phased-array coil affected the estimate of the myocardial signals. The imaging planes differed between LGE and ceSSFP. In the present study, we assessed the signal ratio between MI and remote myocardium or Hypo to avoid measurement of the background signal and its SD, which are affected most by the speed-up technologies. We did not perform short-axis ceSSFP because it could not show the LVOT and because acquisition of both short-axis and 3-chamber ceSSFP might be difficult until Look-Locker imaging. Fifth, we estimated the LVOT jet flow visually using SSFP but not qualitatively. We did not perform the phase contrast imaging routinely because of its longer scan time and lower temporal resolution and difficulty in determining the appropriate velocity encoding for HOCM. Lastly, we did not compare MR imaging and histological findings. Thus, we derived interpretations of MR images about ASA-induced MI, MVO, and dense scarring from findings reported of previous studies.

In conclusion, ceSSFP can sufficiently demonstrate myocardial injuries induced by ASA despite its lower image contrast than that of LGE MR imaging. In addition, the ceSSFP technique allows for the assessment of LVOT jet flow in a single scan and can be incorporated into a routine protocol in patients with HOCM after ASA without penalty, such as the increased scan time and decreased contrast of cine SSFP.

References