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Significant reduction of left atrial volume concomitant with clinical improvement after percutaneous transluminal septal myocardial ablation for drug-refractory hypertrophic obstructive cardiomyopathy, and its precise detection with multidetector CT

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To cite: Maekawa Y, Akita K, Tsuruta H, *et al*. Significant reduction of left atrial volume concomitant with clinical improvement after percutaneous transluminal septal myocardial ablation for drug-refractory hypertrophic obstructive cardiomyopathy, and its precise detection with multidetector CT. *Open Heart* 2016;**3**:e000359. doi:10.1136/openhrt-2015-000359

Received 4 November 2015
Revised 19 January 2016
Accepted 14 February 2016

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ABSTRACT

Objective: In patients with hypertrophic obstructive cardiomyopathy (HOCM), left atrial (LA) volume measurement is very important to provide prognostic information. Recent studies demonstrated that multidetector CT (MDCT) is useful to assess the changes in LA volume. Our aim was to examine the utility of a follow-up cardiac MDCT for long-term evaluation of the effect of percutaneous transluminal septal myocardial ablation (PTSMA) on LA volume.

Methods: We studied a consecutive cohort of 20 patients with drug-refractory symptomatic HOCM after PTSMA. We evaluated LA volume analyses with cardiac MDCT on patients who underwent PTSMA as compared to echocardiography.

Results: Before PTSMA, 75% of all patients had heart failure-associated symptoms in the New York Heart Association functional class III/IV. All patients experienced relief from heart failure-associated symptoms after PTSMA. Cardiac MDCT showed significant reduction in the index of maximum LA volume during follow-up compared to before PTSMA in the same way as in echocardiography (93.6±34.1 mL/m^2 vs 82.6±35.3 mL/m^2, p=0.035). A Bland-Altman plot showed small mean differences and limits of agreement in the measurements of the index of maximum LA volume before and after PTSMA between echocardiography and MDCT.

Conclusions: The follow-up cardiac MDCT was a useful tool to evaluate the effectiveness of PTSMA on reduction of LA volume. Cardiac MDCT might provide comparable measurements of the LA volume in patients with drug-refractory symptomatic HOCM before and after PTSMA compared to echocardiography.

KEY QUESTIONS

What is already known about this subject?

- Cardiac multidetector CT (MDCT) examination before percutaneous transluminal septal myocardial ablation (PTSMA) clearly identifies the septal branch and its location in relation to the coronary artery and myocardium. Cardiac MDCT is a clinically important modality to not only determine the culprit septal branches, but also to avoid complications before PTSMA.

What does this study add?

- The follow-up cardiac MDCT is a useful tool to evaluate the effectiveness of PTSMA on reduction of LA volume. Cardiac MDCT might provide comparable measurements of the LA volume in patients with drug-refractory symptomatic hypertrophic obstructive cardiomyopathy before and after PTSMA compared to echocardiography.

How might this impact on clinical practice?

- In MDCT-guided PTSMA, MDCT is always performed before PTSMA, and this study might provide the information on the utility of follow-up MDCT after PTSMA.

INTRODUCTION

Percutaneous transluminal septal myocardial ablation (PTSMA) is one of the septal reduction therapies, and relieves symptoms related to heart failure in patients with drug-refractory hypertrophic obstructive cardiomyopathy (HOCM).[1–3] Myocardial contrast transthoracic echocardiography (MCE) is an important tool to precisely identify the target area of

the basal myocardial septum, and makes it safe and feasible to perform PTSMA.[4] Furthermore, this approach improves the clinical and haemodynamic results, and prevents misplacement of ethanol-induced necrosis as a source of potentially fatal complications. Cardiac multidetector CT (MDCT) examination before PTSMA clearly identifies the septal branch, and its location in relation to the coronary artery and myocardium. In contrast, other tools, such as invasive coronary angiography or echocardiography, cannot detect either the coronary artery or the myocardium. Cardiac MDCT has been shown to be an accurate and reproducible method of measuring cardiac chamber volumes and has been validated for ventricular chambers with cast models.[5] Cardiac MDCT plays an important role in not only the identification of the target vessel of the septal branch before PTSMA, but also the evaluation of infarct size and chamber volumes after PTSMA.[6–8] As cardiac MDCT provides full three-dimensional volume data sets with high spatial resolution, a highly flexible image procession approach is possible, thus potentially providing simultaneous accurate assessment of cardiac volumes.[9] The aim of this study was to show the utility of follow-up cardiac MDCT for a long-term evaluation of the effect of PTSMA on left atrial (LA) volume. Patients with HOCM often have an enlarged left atrium resulting from mitral valve regurgitation and diastolic dysfunction, and the size is closely associated with occurrence of atrial fibrillation and thromboembolic risk.[10] Therefore, LA volume measurement is very important as prognostic information. Although echocardiography is an established tool to analyse LA volume in patients treated with PTSMA, it is not known whether cardiac MDCT is also useful for LA volume analyses or not. In MDCT-guided PTSMA, MDCT is always performed before PTSMA and this study might provide the information on the utility of follow-up MDCT after PTSMA.

METHODS

Ethics statement

This study was approved by IRB committee at Keio University and conducted in accordance with the guidelines of the Declaration of Helsinki. All participants provided their written informed consent to participate in this study.

Study population

We consecutively included 20 patients with HOCM treated with PTSMA between April 2012 and March 2014 in Keio University Hospital. Patients were excluded if MDCT before PTSMA and/or after PTSMA were not available (figure 1). The procedure was offered to patients referred to our HOCM clinic with refractory symptoms and who despite being on maximally tolerated medical therapy (β-blockers, calcium-channel blockers, and cibenzoline or disopyramide) were not candidates for surgical myectomy, due to either comorbidities or

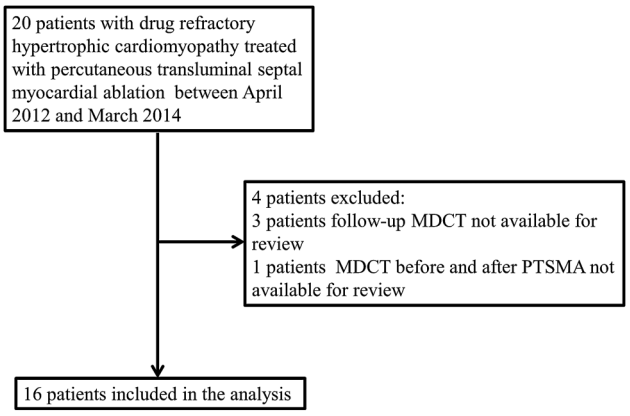

Figure 1 A consort of the patients included and excluded from the study. MDCT, multidetector CT; PTSMA, percutaneous transluminal septal myocardial ablation.

patient's preference. All patients underwent comprehensive clinical, echocardiographic and coronary angiographic evaluation at Keio University Hospital. All patients were treated using MCE-guided PTSMA. HOCM was diagnosed in accordance with American Heart Association/American College of Cardiology guidelines for the diagnosis and treatment of hypertrophic cardiomyopathy.[11] The indication for PTSMA was the presence of severe drug-refractory symptoms (New York Heart Association (NYHA) class III/IV, Canadian Cardiovascular Society class III/IV or NYHA class II symptoms with recurrent exercise-induced syncope) in combination with LV outflow tract (LVOT) pressure gradients ≥30 mm Hg at rest or ≥50 mm Hg during provocation by Valsalva manoeuver or exercise. Cardiac CT and echocardiography were performed before PTSMA, and at least 6 months after PTSMA.

PTSMA procedure

PTSMA was performed according to a previously reported protocol.[6–8 12] In brief, all patients without a previously implanted permanent pacemaker had a prophylactic temporary pacemaker inserted before the procedure. A 6Fr femoral arterial sheath was inserted for the guide catheter system, and a 5Fr radial sheath was inserted for pigtail catheter placement into the LV cavity. Continuous invasive peak-to-peak gradients were measured across the LVOT by comparing the peak LV and aortic pressures. A septal perforating artery supplying the obstructing part of the septum was identified on the coronary angiogram and cardiac MDCT, and chosen as the target vessel. After occlusion of the septal target branch by an over-the-wire balloon, the perfused septal area was visualised by echocardiography after injection of 1–2 mL of echocardiography contrast medium. That there was no back flow into the left anterior descending artery was ascertained by injection of radiographic contrast into the target vessel. After all patients received morphine chloride intravenously for pain control,

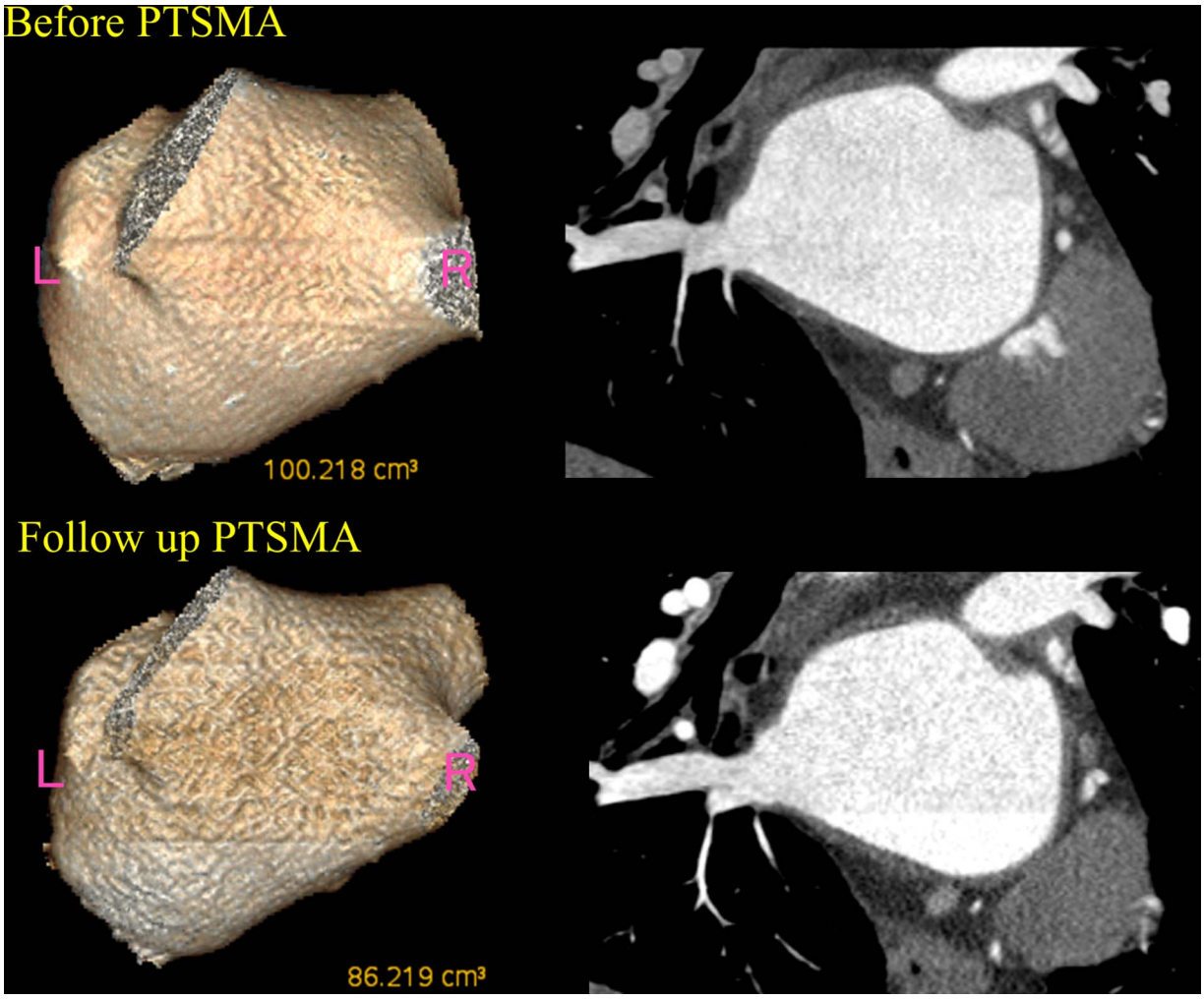

Figure 2 Upper panel, representative volume rendering and multiplanar reconstruction image of MDCT showing left atrium before PTSMA. LA volume is shown. Lower panel, Volume rendering image and multiplanar reconstruction image of MDCT showing left atrium after PTSMA during follow-up of identical case. LA volume is shown. LA, left atrial; MDCT, multidetector CT; PTSMA, percutaneous transluminal septal myocardial ablation.

absolute ethanol was slowly injected into the target vessel through the balloon catheter. The balloon was deflated and removed after 10 min. The occlusion of the target vessel was verified angiographically at the end of the procedure. All patients were monitored in the coronary care unit after the procedure for at least 48 hours. Creatine phosphokinase was monitored at 8 hour intervals over the first 24 hours and daily thereafter for 2 days. All patients were followed up for least every 2 months after the PTSMA procedure at our clinic.

Echocardiography

Echocardiograms were performed at Keio University Hospital. Conventional echocardiographic analysis, including two-dimensional and Doppler, were performed by technicians blinded to clinical information. The LA volumes were calculated by the modified Simpson's method using the apical four−chamber and two-chamber views.[13] Maximum LA volume (LAV_{max}) was defined as the largest LA volume just before mitral valve opening; minimum LA volume (LAV_{min}) was defined as the smallest LA volume in the ventricular end diastole. LA ejection fraction was calculated by the following formula:

$$((LAV_{max}-LAV_{min})/LAV_{max})\times 100.$$

The LA volume was indexed to the body surface area to derive LA volume index. All studies were reviewed by two expert readers blinded to clinical and MDCT data.

Cardiac MDCT imaging protocol

MDCT examinations were performed on a Discovery CT750 HD scanner (GE Healthcare, Waukesha, Wisconsin, USA) or Aquilion ONE ViSION edition (Toshiba Medical Systems, Tochigi, Japan). All patients were scanned craniocaudally while they lay in the supine position. β-blockade was administered intravenously before the examination if heart rate was higher than 65 bpm to obtain optimal image quality for MDCT coronary angiography. The scanning

parameters for MDCT coronary angiography were as follows: tube voltage, 100 or 120 kVp; tube current, 150–750 mA (determined based on a prespecified body mass index protocol) using prospective or retrospective electrocardiogram-gating with detector collimation of 0.625×64 mm or 0.5×320 mm and rotation speed of 350 ms. A double-channel injection system (Dual Shot; Nemoto, Tokyo, Japan) and right antecubital venous access were used. First, the individual circulation time was estimated based on injection of a 20 mL test bolus of contrast material (Iopamiron 370 mg/mL iodine concentration; Bayer, Osaka, Japan) followed by 20 mL of saline. Repetitive scans of the ascending aorta at the level of the left main coronary artery were acquired during a breath hold. The time of peak attenuation was then used as the delay time. Next, a dual-phase contrast protocol was used: weight×0.84 mL (at a rate of weight×0.07 mL/s) of contrast material followed by weight×0.56 mL (at a rate of weight×0.07 mL/s) of a mixture of contrast material and saline solution.[14] Contiguous 0.625 mm-thick or 0.5 mm-thick coronary CT angiography images were then generated.

LA volume measurements by cardiac MDCT

LA volume was automatically calculated by an independent workstation (Advantage workstation 4.6; GE Healthcare, Waukesha, Wisconsin, USA) using Simpson's methods with manual tracing of the endocardial borders on successive slices. Long axis two-chamber and four-chamber multiplanar reconstruction images (1.25 mm thick) were used for the measurement of LA volume (figure 2). The ostia of the pulmonary veins and the LA appendage were excluded from the LA measurements, as described previously.[15] LA volume measurements were performed before PTSMA and during the follow-up period. Images were analysed and LA measurements were performed by two expert readers blinded to clinical and echocardiographic data.

Clinical demographic data

Patient demographic data included prevalence of concomitant diseases, history of invasive therapies, and procedural data. We investigated all-cause mortality as well as new admission for heart failure, new requirement for invasive therapies (including myectomy), repeated-PTSMA, permanent pacemaker implantation, stroke and cardiac death (including sudden death during the short term and long term).

Table 1 Baseline characteristics

	Ablation patients (n=16)
Age, years	70±15
Women, n (%)	12 (75)
Body mass index, kg/m^2	23±4
NYHA class III/IV, n (%)	12 (75)
Syncope, n (%)	5 (31)
Atrial fibrillation, n (%)	2 (13)
Previous stroke, n (%)	2 (13)
Hypertension, n (%)	8 (50)
Diabetes mellitus, n (%)	2 (13)
Dyslipidaemia, n (%)	8 (50)
Current smoker, n (%)	2 (13)
Chronic obstructive pulmonary disease, n (%)	0 (0)
Coronary artery disease, n (%)	1 (6)
Previous myectomy, n (%)	0 (0)
Previous septal ablation, n (%)	1 (6)
Family history of HCM, n (%)	2 (13)
Intraventricular septum thickness, mm	15±2
End-diastolic diameter, mm	42±5
End-systolic diameter, mm	23±3
Resting LVOT gradient, mm Hg	107±45
LVEF, %	78±6
ICD, n (%)	5 (31)
Permanent pacemaker, n (%)	0 (0)
β-blocker, n (%)	16 (100)
Calcium-channel blocker, n (%)	6 (38)
Na channel antagonist, n (%)	8 (50)
Amiodarone, n (%)	1 (6)
Diuretics, n (%)	3 (19)
Warfarin, n (%)	3 (19)

Data are expressed as mean±SD or n (%).
HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; NYHA, New York Heart Association.

Table 2 Procedure details and outcomes

	Ablation patients (n=16)
Number of septal arteries injected	2.4±1.4
Amount of ethanol injected, mL	3.8±2.3
Residual LVOT gradient at rest, mm Hg	17±16
Peak CK, IU/L	1180±591
Procedural and in-hospital complications	
Pacemaker dependency, n (%)	0 (0)
Cardiac tamponade, n (%)	0 (0)
Sustained ventricular tachycardia, n (%)	0 (0)
Cardiac surgery, n (%)	0 (0)
Resuscitated sudden cardiac arrest, n (%)	0 (0)
Stroke, n (%)	0 (0)
Death, n (%)	0 (0)
Heart failure, n (%)	0 (0)
Clinical events during follow-up period	
Pacemaker implantation, n (%)	0 (0)
New ICD, n (%)	0 (0)
Repeat-PTSMA, n (%)	1 (6)
Death, n (%)	0 (0)
Heart failure admission, n (%)	0 (0)

Data are expressed as mean±SD or n (%).
CK, creatinine phosphokinase; ICD, implantable cardioverter-defibrillator; LVOT, left ventricular outflow tract; PTSMA, percutaneous transluminal septal myocardial ablation.

Table 3 CT and echocardiographic analyses before and after PTSMA

	Before PTSMA	Follow-up	p Value
CT analyses	(n=16)	(n=16)	
LAV_{max}, mL	137.7±47.9	121.0±49.0	0.034
$LAVI_{max}$, mL/m^2	93.6±34.1	82.6±35.3	0.035
Echocardiographic analyses	(n=16)	(n=16)	
LAV_{max} (MOD), mL	116.4±34.1	99.1±37.2	0.002
LAV_{min} (MOD), mL	77.1±37.0	62.8±32.8	0.011
$LAVI_{max}$ (MOD), mL/m^2	78.5±27.7	66.1±26.3	0.002
$LAVI_{min}$ (MOD), mL/m^2	51.8±26.7	41.8±22.7	0.010
LAEF (MOD), %	35.8±14.1	38.7±12.5	0.414

Data are expressed as mean±SD.
CT; LAEF, left atrial ejection fraction; $LAVI_{max}$, the index of maximum left atrial volume; $LAVI_{min}$, the index of minimum left atrial volume; LAV_{max}, maximum left atrial volume; LAV_{min}, minimum left atrial volume; MOD, modified Simpson's rule; PTSMA, percutaneous transluminal septal myocardial ablation.

Statistical analyses

Continuous variables are expressed as mean±SD unless otherwise specified. Categorical variables are expressed as absolute values and percentages. A paired t-test was used to evaluate the LA volume before PTSMA by cardiac MDCT and echocardiography, and compared with that after PTSMA. Correlations were determined between the echocardiographic and MDCT measurements using Pearson correlation coefficient, and agreement was expressed according to the Bland-Altman method. All p values are two-sided. Results were considered statistically significant at a p value <0.05. JMP V.10 software (SAS institute Japan, Tokyo, Japan) was used for all analyses.

RESULTS

Baseline characteristics

A total of 20 patients who underwent PTSMA were identified. Of these patients, four were excluded because they did not have 1 of the 2 cardiac MDCT available for review. We evaluated LA volume analyses by cardiac MDCT in 16 patients who underwent PTSMA, compared to echocardiography. The mean follow-up period was 18 months (range 8–31 months). Baseline characteristics are listed in table 1. Before PTSMA, 75% of all patients had heart failure associated symptoms in NYHA functional class III/IV, and 31% had previously experienced syncope. The resting LVOT gradient was 107±45 mm Hg and the LV ejection fraction was 78±6%. All patients received β-blocker treatment, and 50% of the patients had taken Na-channel antagonists, either disopyramide or cibenzoline.

Acute procedure and long-term outcome

All patients experienced relief from heart failure associated symptoms after PTSMA (table 2). No complications occurred during the periprocedural period. The residual LVOT gradient at rest was 17±16 mm Hg and peak creatine phosphokinase was 1180±591 IU/L. One patient (6%) experienced relief from dyspnoea on exertion within 3 months after PTSMA, but had dyspnoea again 6 months after the procedure. Finally, the patient underwent a repeat PTSMA. No patient required a permanent pacemaker implantation and no patient experienced stroke, death or heart failure admission during the follow-up period (table 2).

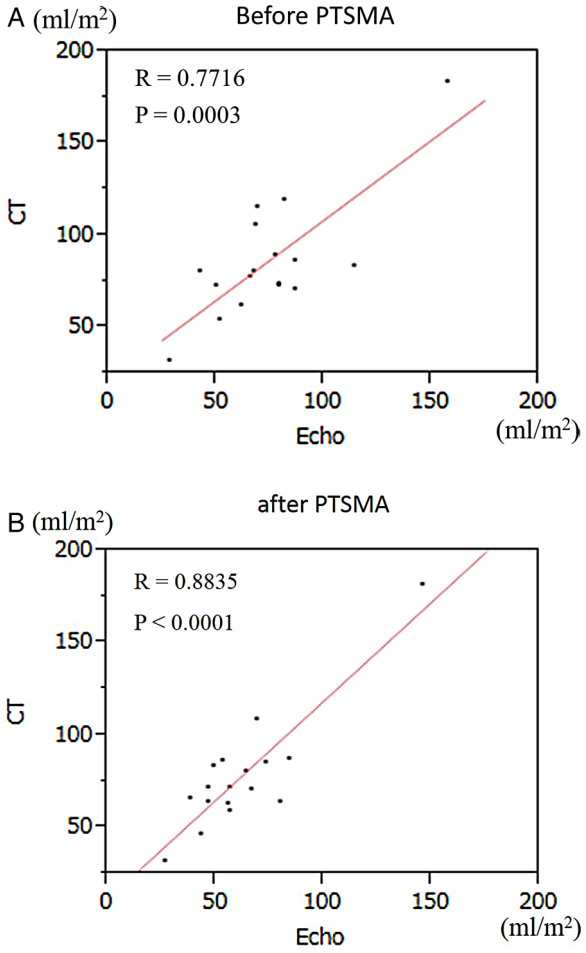

Figure 3 These plots show the linear regression analysis of cardiac MDCT-derived index of maximum LA volume before (A) and after (B) PTSMA against echocardiographic measurements. The index of maximum left atrial volume correlated strongly between cardiac MDCT and echocardiographic analyses. LA, left atrial; MDCT, multidetector CT; PTSMA, percutaneous transluminal septal myocardial ablation.

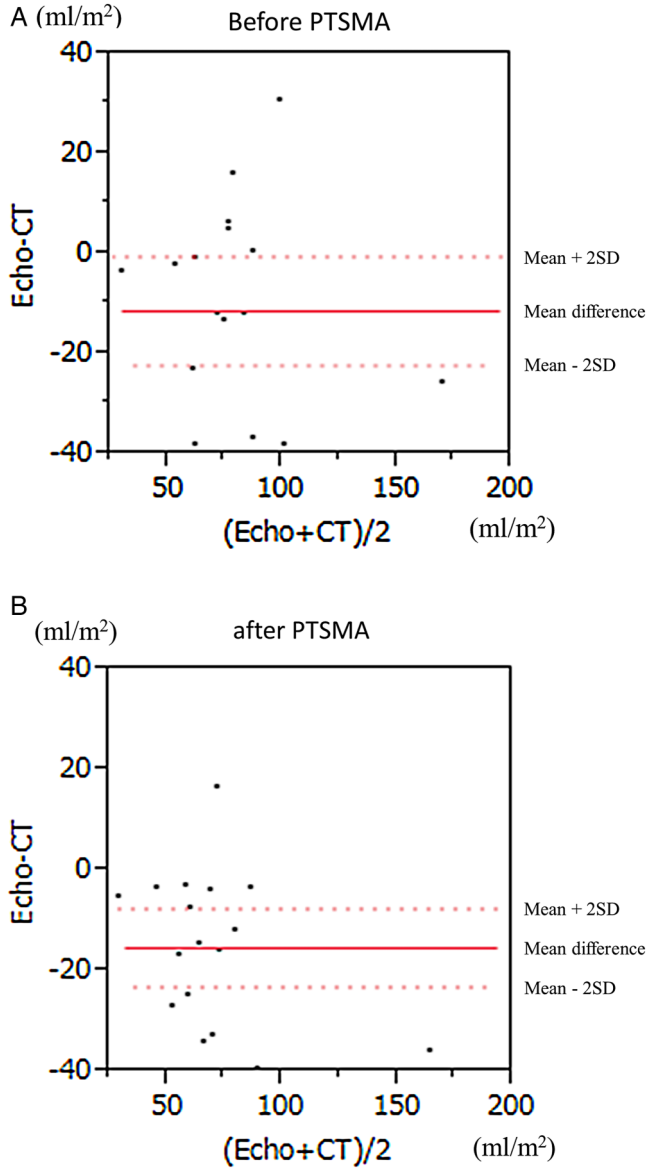

Figure 4 Bland-Altman analyses between cardiac MDCT and echocardiographic measurements of the index of maximum left atrial volume before (A) and after (B) PTSMA. Solid lines on Bland-Altman plots indicate mean differences; dashed line indicates limits of agreement (2SD of differences). MDCT, multidetector CT; PTSMA, percutaneous transluminal septal myocardial ablation.

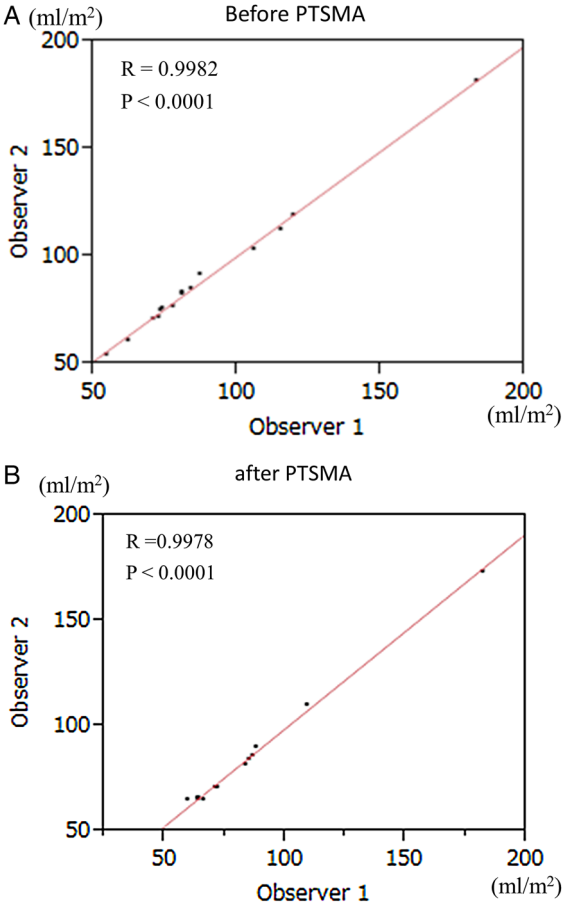

Figure 5 Interobserver variances for measurement of the index of maximum LA volume by cardiac MDCT. (A) Linear regression of the index of maximum LA volume after PTSMA measured by two independent observers. (B) Bland-Altman analysis for the index of maximum LA volume after PTSMA between two independent observers. LA, left atrial; MDCT, multidetector CT; PTSMA, percutaneous transluminal septal myocardial ablation.

Effect of PTSMA

All patients improved to lesser degree of NYHA functional class (p=0.025); 10 patients (63%) improved in the degree of mitral valve regurgitation while six patients remained in the same degree of mitral valve regurgitation although patients had not had more than mild mitral valve regurgitation before PTSMA. After PTSMA, the degree of mitral valve regurgitation decreased from 2.0±0.7 to 1.4±0.6 (p <0.001).

LA volume evaluated by cardiac MDCT and echocardiography

Cardiac MDCT showed a significant reduction of the index of maximum LA volume during follow-up compared with that before PTSMA. Echocardiographic analyses also revealed smaller maximum LA volume index during follow-up compared with that before PTSMA (table 3). Figure 3A, B demonstrates the strong correlation between echocardiographic and MDCT measurements for the index of maximum LA volume before and after PTSMA. A Bland-Altman plot showed small mean differences and limits of agreement in the measurements of the index of maximum LA volume before and after PTSMA between echocardiography and MDCT (−11.5±5.1mL/m^2, −15.5±3.7 mL/m^2; figure 4A, B). Two independent observers blinded to the clinical and MDCT or echocardiographic data performed the same

analyses. A good correlation in the index of maximum LA volume measurements before and after PTSMA was obtained between them (figure 5A, B). The Bland-Altman plot of the differences by the two observers showed a significant mean difference and limits of agreement. The interobserver variability was -0.3 ± 0.5 mL/m^2 and -0.2 ± 0.8 mL/m^2 for the index of maximum LA volume before and after PTSMA.

DISCUSSION

This study showed that (1) cardiac MDCT is reliable and a comparable method to evaluate LA volume before and after PTSMA compared to echocardiography, and (2) cardiac MDCT revealed significant LA volume reduction in patients after PTSMA compared with that before PTSMA.

Although it is very important to perform cardiac MDCT before PTSMA not only to determine the culprit septal branches but also to facilitate septal branch wiring,[7] our study demonstrated that cardiac MDCT was also an important tool to evaluate the therapeutic effect of PTSMA. A few recent reports have demonstrated a close correlation between cardiac CT and echocardiography quantification for LA volume.[16–18] In addition, follow-up cardiac MDCT also provides information on infarct size and septal branches; echocardiography is, however, a feasible and reproducible method to evaluate LA volume. Recently, a study demonstrated that measurements of LA volumes by cardiac MR and MDCT are feasible in patients with permanent atrial fibrillation, and that the reproducibility of the two methods is superior to that of transthoracic echocardiography-derived measurements.[19] Although widely used for assessment of LA, echocardiography underestimates the true volumes and may be less suitable for detecting small volume changes. Recent studies suggest that MDCT is useful to assess the changes in LA volume.[20–22] Therefore, LA volume measurement by MDCT in our study to compare between before and after PTSMA might be a better methodology as compared to echocardiography.

In patients with HOCM, both systolic anterior motion-related mitral insufficiency and elevated LV end-diastolic pressure due to LV hypertrophy cause LA enlargement. The 2014 European Society of Cardiology guidelines indicated that the size of the left atrium provides important prognostic information on patients with HCM.[23] Our study showed significant LA volume reduction in drug-refractory patients with HOCM after PTSMA. This point is consistent with previous studies showing that patients undergoing PTSMA, performed by experienced operators, experienced favourable outcomes without an increased risk of sudden cardiac death.[3] In addition, PTSMA could aid in maintaining normal sinus rhythm by preventing LA enlargement and may improve the quality of life in drug-refractory patients with HOCM, especially aged patients.

Previous studies revealed that PTSMA improved short-term and long-term outcomes in drug-refractory patients with HOCM even if drug therapy did not comply with accepted guidelines. In our study, all patients received β-blocker therapy, which means that our patients were truly 'drug refractory'. Our strategy for drug-refractory patients faithfully followed guidelines on the diagnosis and management of HCM.[11 23] All patients referred to our hospital were observed for at least a couple of months, and we treated all patients with non-vasodilating β blockers that were titrated to maximum tolerated dose during the observational period.

Study limitation

Our study population was of limited size. A relatively few patients, however, are truly drug-refractory HOCM.

CONCLUSIONS

The follow-up cardiac MDCT was a useful tool to evaluate the effectiveness of PTSMA on reduction of LA volume. Cardiac MDCT might provide comparable measurements of the LA volume in patients with drug-refractory symptomatic HOCM before and after PTSMA compared to echocardiography.

Acknowledgements The authors thank Ms Kaori Suzuki for her technical assistance.

Contributors KA, HT, KH, SY, MM, MJ and KF conceived and designed the study. YM and YY were responsible for analysing the data and writing of the manuscript.

Competing interests None declared.

Patient consent Obtained.

Ethics approval This study was approved by IRB committee at Keio University.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement No additional data are available.

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