Filling Defects of the Left Atrial Appendage on Multidetector Computed Tomography: Their Disappearance following Catheter Ablation of Atrial Fibrillation and the Detection of LAA thrombi by MDCT

Short title: LAA Filling Defects on MDCT

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Abstract

Background: Filling defects of the left atrial appendage (LAA) on multidetector computed tomography (MDCT) are known to occur, not only due to LAA thrombi formation, but also due to the disturbance of blood flow in the LAA of patients with atrial fibrillation (AF). The purpose of this study was to evaluate the impact of the maintenance of sinus rhythm via ablation on the incidence of LAA filling defects on MDCT in patients with AF.

Methods: A total of 459 consecutive patients were included in the present study. Prior to ablation, MDCT and transesophageal echocardiography (TEE) were performed. AF ablation was performed in patients without LAA thrombi confirmed on TEE. The LAA filling defects was evaluated on MDCT at three months after ablation.

Results: LAA filling defects were detected on MDCT in 51 patients (11.1%), among whom the absence of LAA thrombi was confirmed in 42 patients using TEE. The LAA Doppler velocity in patients with LAA filling defects was lower than that of patients without filling defects (0.61±0.19 vs. 0.47±0.21m/sec; $P<0.0001$). The sensitivity, specificity and negative predictive value of MDCT in the detection of thrombi were 100%, 91% and 100%, respectively. No LAA filling defects were observed on MDCT at three months after ablation in any of the patients, including the patients in whom filling defects were noted prior to the procedure.

Conclusions: MDCT is useful for evaluating the presence of LAA thrombi and the blood flow of the LAA. The catheter ablation of AF not only suppresses AF, but also eliminates LAA filling defect on MDCT suggesting the improvement of LAA blood flow.

Key words: Atrial fibrillation; Computed tomography; Transesophageal echocardiography; Catheter ablation

Introduction

Although radiofrequency catheter ablation has been demonstrated to be an effective treatment for maintaining sinus rhythm in patients with atrial fibrillation (AF),[1, 2] the left atrial (LA) ablation procedure is associated with a risk of stroke due to air emboli and/or thrombus formation at the tip of the catheter or sheath in the LA.[3-5] Pre-existing LA thrombi, which most commonly present within the left atrial appendage (LAA), may also cause strokes during the procedure because it involves LA catheter manipulation and the possible restoration of sinus rhythm. The presence of LA thrombi must therefore be strictly excluded before the ablation procedure.

Multidetector computed tomography (MDCT) was introduced as a noninvasive imaging modality with high spatial and temporal resolution. MDCT is a useful tool for evaluating the anatomy of the atrium and pulmonary vein (PV) prior to the ablation of AF.[6, 7] In addition, pre-acquired MDCT images can be integrated with advanced 3-dimensional electroanatomic mapping systems to assist in mapping and detecting complications, such as PV stenosis. MDCT allows for excellent visualization of the LAA and it is useful for excluding the presence of LAA thrombi; however, filling defects of the LAA on MDCT are known to occur not only due to the formation of LAA thrombi, but also due to the disturbance of the LAA blood flow in patients with AF.[8, 9] Theoretically, the filling defects caused by blood flow disturbances can disappear following the improvement of blood flow in the LAA. However, it is not known whether the LAA filling defects on MDCT remain or disappear through the process of atrial reverse remodeling following sinus rhythm restoration after catheter ablation. The purpose of the present study was to evaluate the diagnostic performance of MDCT in identifying LAA thrombi in comparison to transesophageal echocardiography (TEE) and to assess the impact of catheter ablation of AF on the incidence of filling defects in the LAA on MDCT in patients with AF.

Materials and methods

Study subjects

The present study included a total of 459 consecutive patients who were referred for LA ablation due to symptomatic AF underwent complete clinical assessment and pre-ablation imaging with MDCT and TEE. All of the patients underwent an electrophysiological study and catheter ablation. The present study received institutional approval after written informed consent was obtained from each patient. Prior to the procedure, all of the patients underwent imaging of the LA with MDCT and TEE to examine the anatomy of the LA and exclude the presence of intracardiac masses or thrombi. MDCT was performed within seven days before the procedure, and TEE was performed one day prior to the procedure. Bepridil was administered before the ablation procedure to all but 2 patients with persistent AF; the two patients, who had persistent AF, received pilsicainide and flecainide.

The risk factors for stroke in patients with AF were assessed based on the CHADS2 scoring system, whereby 1 point is given for each risk factor (congestive heart failure, hypertension, >75 years of age and diabetes) and 2 points are given for a history of transient ischemic attack or prior stroke, to determine the clinical stroke risk.[10] All of the patients received effective anticoagulation therapy with warfarin (target prothrombin time-international normalized ratio [PT-INR]: 2 to 3) for at least three weeks before ablation; the therapy was discontinued one day before the ablation procedure. Heparin was continuously administered after admission for catheter ablation. The dose of heparin was controlled to keep the ACT to between 300 and 350 seconds during the ablation procedure. The warfarin therapy was resumed on the day after the procedure and was controlled to maintain an optimal PT-INR value in all patients for at least three months. MDCT was performed at three months after the procedure.

The MDCT protocol

In all patients, MDCT scans were performed using a dual-source 128-slice CT scanner (Somatom Definition FLASH, Siemens Healthcare, Forchheim, Germany) with the patient in the supine position. During acquisition, 70 mL of nonionic contrast material (Omnipaque 350

Daiichi-Sankyo, Tokyo, Japan) was intravenously injected into the antecubital vein using a 20-gauge needle and power injector. The bolus tracking technique was used to appropriately time the onset of image acquisition. Contrast-enhanced MDCT angiography was performed, with scan initiation at the level of the aortic arch and termination immediately inferior to the cardiac apex. The scan parameters were as follows: collimation = 64×0.625mm, tube voltage = 120mV and effective amplitude = 600ï 780mA. Helical scan data were obtained using retrospective ECG gating. Electrocardiographically gated datasets were reconstructed at 75% of the cardiac cycle after the QRS complex.

First, the entire LAA was examined in multiple axial planes without the use of multiplanar reconstruction or 3-dimensional volume rendering views. ñDelayedò imaging of the underfilled LAA was not used in this study. The LAA was visually examined for any filling defects consistent with thrombi. Filling defects were defined as areas within the LAA that had a soft tissue density of <100 Hounsfield units and that could be differentiated from the LAA endocardium or surrounding extracardiac structures. If the interpreting physician was unable to visualize the entire LAA filled with contrast, the appendage was considered to have a filling defect.[9] The LAAs were independently examined by two experienced cardiac radiologists and one cardiologist who scored them for the presence or absence of filling defects by consensus. The LAA was reported to be either ñnormalò if the entire LAA was filled with contrast and fully opacified (Fig. 1A) or ñabnormalò (a filling defect) if there was any difficulty in visualizing the LAA due to it not being fully opacified with contrast media (Fig. 1B). In all patients, MDCT was performed at three months after the ablation procedure using the same protocol.

In the present study, the morphology of the LAA was classified and divided into two types (chicken wing and non-chicken wing) based on the findings of a previous study.[11] A chicken wing LAA was defined as an LAA with an obvious bend of the dominant lobe in which it folded back on itself, with or without second lobes.[11] All other morphologies were described as non-chicken wing LAA. In addition, the term, ñsuperior LAA takeoffò was used to describe an

LAA ostium that was superior to the left superior PV (as previously described).[12]

The transesophageal echocardiogram protocol

TEE was performed according to the standard clinical procedure using a 7-MHz multiplane probe with an Aloka ProSound Alpha 10 ultrasound system (Aloka, Tokyo, Japan). Multiple planes of the LAA, including a continuous view through the LAA from 0° to 180°, were examined at the appropriate level within the esophagus. Detailed observations were made of all LAA structures, including the pectinate muscles. All of the images were interpreted by two echocardiographers who were blinded to the MDCT results. In cases in which a pulsed wave Doppler signal was obtained at the mouth of the LAA, all Doppler values were averaged and recorded. A definite thrombus was determined to be present if echo-dense material, which was acoustically separate from the endocardium was found within the LA or LAA. In each study, the LAA was graded as none, mild spontaneous echo contrast (SEC), severe SEC (very dense swirling in the LAA) or a thrombus according to a scale adapted from previous studies.[13, 14]

Ablation of AF

Catheter ablation was performed following the confirmation of the absence of LAA thrombi on TEE. Intravenous heparin was continuously administered to maintain an activated clotting time of 300 to 350 seconds; this was evaluated every 20 minutes during the procedure. In each case, all PVs were targeted to electrically disconnect them from the LA at the antrum. PV antrum isolation was performed as proximally as possible at the ostium or antrum with the guidance of a steerable circular catheter measuring 20, 25 or 30 mm in diameter (Lasso, Biosense Webster, Diamond Bar, CA, USA) and a three-dimensional mapping system.[13] The radiofrequency energy was delivered with an 8-mm ablation catheter (Fantasista, Japan Lifeline, Japan) or an open-irrigated ablation catheter with a 3.5-mm tip (Cool Path Duo, St Jude Medical St. Paul, MN, USA or EZ Steer Thermocool NAV, Biosense Webster) and a power limit of 25 to 35 watts for 30 to 60 seconds at each site. After all four PVs were electrically isolated from the LA, 20 mg of adenosine triphosphate was rapidly injected to induce dormant PV conduction

under the infusion of isoproterenol during sinus rhythm or coronary sinus pacing, as previously described. [15] If dormant PV conduction was induced, additional radiofrequency energy was applied at the earliest transient PV activation site identified on a circular mapping catheter in order to establish complete PV disconnection.

Following PV antrum isolation, electrogram-based ablation or LA linear ablation was performed in the patients with persistent AF. Electrogram-based ablation was performed targeting the sites that presented complex atrial electrograms with the endpoint of AF termination.[16] LA linear ablation consists of a roof line between the left superior PV and right superior PV and the mitral isthmus line between the left inferior PV and the mitral annulus. The endpoint of linear ablation was the presence of a bidirectional conduction block through the lines.[17]

Patient follow-up

The presence or absence of atrial tachyarrhythmia was evaluated based on symptoms and the findings of ECG recordings and 24-hour ambulatory monitoring (one month and three months after the procedure). In all patients, MDCT was performed at three months after the procedure using the same protocol. Anticoagulation therapy was continued for three months after the procedure in all patients and an anti-arrhythmic drug (150 mg of bepridil) was administered to patients with persistent AF.

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation or the median and interquartile range (IQR). Statistical significance was assessed using the unpaired Studentôs *t*-test or the Mann-Whitney test, if necessary. Categorical variables, which are expressed as numbers or percentages, were analyzed using the chi-squared test or Fisherôs exact test. All of the tests were two-tailed, and P values of <0.05 were considered to indicate statistical significance.

Results

The patient characteristics and the incidence of thrombi or severe SEC in the LAA

The baseline characteristics of the 459 patients (male: 91.2%; mean age: 55.2±10.7 years; paroxysmal AF: 241 and persistent AF: 219 patients) are listed in Table 1. The mean duration of anticoagulation therapy before ablation was 4.2±3.2 months. LAA thrombi and severe SEC were observed in five (1.1%) and four (0.9%) patients, respectively, on TEE. The patient age was higher and persistent AF was more frequently observed in the 9 patients with LAA thrombi or severe SEC on TEE than in those without (Table 2). Additionally, the patients with LAA thrombi or severe SEC on TEE were more likely to have a history of cerebral infarction and a lower LAA ostial pulsed-wave Doppler velocity (Table 2). Although the incidence of non-chicken wing LAA was similar in patients with and without LAA thrombi or severe SEC (77.8% [7/9] vs.64.7% [291/450]; P=0.64), the patients with LAA thrombi or severe SEC had a higher incidence of superior LAA take off (66.7% [6/9] vs. 8.0% [36/450]; P<0.0001) (Table 2).

Filling defects in the LAA on MDCT

The mean effective radiation doses of pre- and post-ablation MDCT were 1.4±0.3 and 1.5±04 mSv, respectively. The rate of interobserver concordance among the readers in detecting the presence or absence of thrombi was (100%). MDCT imaging of the LAA was reported to be normal without filling defects in 408 of 459 patients (88.9%), while abnormal findings due to filling defects were observed in 51 patients (11.1%). Among the 408 cases without LAA filling defects, no patients had LAA thrombi or severe SEC on TEE (Figure 2). Among the 51 patients with an LAA filling defect, MDCT was performed during sinus rhythm and ongoing AF in 17 (33.3%) and 34 (66.7%) patients, respectively. Table 4 shows the results of a comparison of the clinical variables of the patients with and without LAA filling defects on MDCT. The patients with LAA filling defects on MDCT were older and their incidence of persistent AF was higher in comparison to the patients without filling defects; no differences were observed between the two groups in the PT-INR on the day of MDCT (Table 3). Additionally, there were no significant

differences in the frequency (13.2±7.2 vs. 12.4±6.2 times/year; P=0.25) or duration (6.2±6.6 vs. 5.3±4.8 hours; P=0.32) of paroxysmal AF in the patients with and without LAA filling defects. The LA dimensions were larger and the LAA Doppler velocity was significantly lower in the patients with filling defects than in those without. Non-chicken wing LAA was more frequently observed in patients with LAA filling defects than in those without (40/51 [78.4%] vs. 251/408 [61.5%]; P=0.047). Moreover, the patients with LAA filling defects had a higher incidence of superior LAA take off (26/51 [51.0%] vs. 16/408 [3.9%]; P<0.0001) (Table 3). There was no significant difference in the duration for which anticoagulants were administered in the patients with and without LAA filling defects (Table 3).

No abnormalities of the LAA were observed on TEE in 42 of the 51 patients with LAA filling defects on MDCT (Figure 3 A and B). In the remaining nine patients in whom a filling defect within the LAA was reported (Figure 4 A), a further evaluation using TEE confirmed the presence of thrombi (Figure 4 B) in five patients. With respect to the detection of LAA thrombi, the classification of filling defects on MDCT exhibited a sensitivity of 100%, a specificity of 91%, a positive predictive value of 82% and a negative predictive value of 100%.

The ablation results and clinical outcomes of AF ablation

AF ablation was performed in all 408 cases without filling defects and 42 of the cases with filling defects after confirming the absence of LAA thrombi on TEE. All PVs were isolated from the LA, and substrate modification was performed in the LA of the patients with persistent AF. The total procedure time, radiofrequency time and energy delivery were 223 ± 73 minutes, 51 ± 29 minutes and 85 ± 44 kJ, respectively. The mean fluoroscopic time for the ablation procedure was 56.2 ± 14.2 minutes in the AP view and 24.2 ± 12.2 minutes in the left anterior oblique view. Atrial ablation to modify the AF substrate was performed in addition to PV isolation in 213 (46%) patients.

All patients underwent anticoagulation therapy until the post-procedural MDCT scan. Anti-arrhythmic drugs were administered to 96.2% (201/209) of the persistent AF patients and

14.5% (35/241) of the paroxysmal AF patients. AF recurred within 90 days (prior to the post-procedural MDCT scan) was observed in 44.0% (198/450) of the patients. Among the 198 patients with recurrence, AF recurred within five days after the procedure and spontaneously disappeared in 167 patients (84.3%), while AF recurred at five to 90 days after ablation in 31 patients (15.7%). Finally, during 1410±339 days of follow-up, 368 (81.8%) and 289 (64.2%) patients remained in sinus rhythm with and without anti-arrhythmic drug treatment, respectively, after the single ablation procedure.

The MDCT and echocardiographic results after ablation

Follow-up MDCT was performed during sinus rhythm and AF in 440 and 10 patients, respectively, at three months after the procedure. No filling defects were observed at three months after the procedure in any of the 408 patients without filling defects on the pre-ablation examination. Table 4 shows the comparison of the pre- and post-ablation MDCT scans of 42 patients in whom a filling defect was observed on MDCT in the pre-ablation examination. The post-procedural MDCT scan was performed during ongoing AF in the 42 patients; this was during sinus rhythm in all but one patient. The pre-procedural MDCT scan was performed during sinus rhythm in 26 patients (97.6% vs. 61.9%; P<0.0001). Forty-one of these patients presented no LAA filling defects on MDCT at three months. No significant differences were found in the absolute INR values in the pre-procedural and post-procedural MDCT scans (1.96±0.57 vs. 1.86±0.33; P=0.43).

The echocardiography findings of the patients who underwent catheter ablation revealed that the LA dimensions were significantly smaller three months after the procedure in comparison to those observed prior to ablation (41.2±4.9 vs. 38.6±5.5mm; P<0.001). The left ventricular end-diastolic diameter before ablation was similar to that after ablation (47.4±4.6 vs. 46.8±4.2mm; P=0.43). There were no significant differences in the left ventricular ejection fraction (63.7±7.3 vs.65.5±5.7%; P=0.20) or the early diastolic filling velocity (0.75±0.18 vs. 0.69±0.19m/sec; P=0.14) before and after the ablation procedure. Echocardiography was

performed during sinus rhythm in 11 and 41 patients before and after the ablation procedure, respectively. In the 11 patients in whom the echocardiography was recorded during sinus rhythm before the ablation procedure, there was no difference in the mitral A-wave velocity before and after the ablation procedure (0.50±0.12 vs. 0.55±0.15m/sec; P=0.26).

Discussion

Main findings

The present study demonstrated the disappearance of LAA filling defects on MDCT, possibly indicating that improved LAA blood flow was achieved after catheter ablation in patients with AF. In addition, MDCT had a sensitivity of 100%, a specificity of 91% and a negative predictive value of 100% in the detection of thrombi. Atrial remodeling is achieved by eliminating AF and maintaining sinus rhythm in patients with AF. Catheter ablation involving PV isolation and left atrial linear ablation resulted in sinus rhythm maintenance in approximately 65% of the AF patients following the performance of a single ablation procedure.

The mechanisms underlying the filling defects on MDCT

Filling defects were observed in patients without LAA thrombi or severe SEC, which indicated a significant disturbance of the blood flow in the LA. Nine of the 51 patients with LAA filling defects presented with LAA thrombi and/or severe SEC. The Doppler velocity of the LAA flow of the patients with LAA filling defects was significantly lower than that of the patients without LAA filling defects. Furthermore, the patients without filling defects of the LAA on MDCT showed no abnormalities on TEE. Thus, the LAA filling defects were presumably caused not only by the presence of LAA thrombi, but also by the disturbance of the blood flow in the LAA. Figure 4 shows that although the thrombus occupied the middle part of the LAA, the filling defect on the MDCT was observed in the whole distal part of the LAA. The results show that filling defects of the LAA on MDCT can be caused both by the presence of a thrombus and by blood pooling in the tip of the LAA due to the thrombus.

Previous investigations have demonstrated that the morphology and characteristics of the

LAA have a significant impact on LAA flow. Di Biase et al. reported that AF patients with non-chicken wing LAA morphology were more likely to have an embolic event after controlling for the presence of comorbidities and the CHADS2 score.[11] In the present study, although the incidence of non-chicken wing LAA was similar in the patients with and without LAA thrombi or severe SEC, it was more frequently observed in patients with an LAA filling defect than in those without. Furthermore, a previous study demonstrated a significant association between a higher takeoff LAA and an increased thromboembolic risk.[12] In line with the results of the previous study, both the patients with LAA thrombus/severe SEC and LAA filling defects had a higher incidence of superior LAA take off in the present study.

The disappearance of LAA filling defects after catheter ablation

The filling defects of the LAA that were observed in 42 patients prior to the procedure disappeared in all but one patient at three months after catheter ablation. Importantly, pre-ablation MDCT demonstrated filling defects during both ongoing AF and sinus rhythm (16/42, 38.1%). While filling defects in the LAA might occur due to the presence of thrombi in the LAA, a decrease in the blood flow in the LAA may also result in filling defects. A previous study demonstrated that the LAA blood flow is reduced by stunning the LA, even during sinus rhythm, in paroxysmal AF patients.[18] In the present study, filling defects were detected on MDCT during sinus rhythm in 18 patients; the defects in all of these patients disappeared after catheter ablation, presumably due to improved LAA blood flow. However AF itself could affect the MDCT results, including the presence of the LAA filling defect.

During the first three months after the ablation procedure, the recurrence of AF was observed in 198 patients regardless of whether they were treated with anti-arrhythmic drugs. Previous studies have demonstrated that the function of the LA recovers within three months after the restoration of sinus rhythm by direct current cardioversion.[19] Takahashi et al. reported that the LA function, as evaluated by ultrasound cardiography, improved in persistent AF patients who underwent radiofrequency catheter ablation.[20] In the present study, the dimensions of the LA

were significantly reduced at three months after ablation in patients with filling defects on pre-procedural MDCT. This finding is due to the anatomical reverse remodeling induced by the maintenance of sinus rhythm with catheter ablation. The elimination of LAA filling defects after catheter ablation suggests the presence of functional reverse remodeling of the atrium as well as anatomical reverse remodeling. The reverse remodeling of the atrium, which was evaluated on echocardiograms after the catheter ablation of AF was reported in a previous study. Although the authors evaluated atrial remodeling based on the improvement of A-wave velocity[19] or the ejection fraction of the LA,[20] it could not be recorded during ongoing AF. On the other hand, LAA filling defects on MDCT can be assessed during both sinus rhythm and AF. A previous study described that the LAA mechanical reverse is responsible for the maintenance of sinus rhythm;[21] however, TEE is the gold standard technique for evaluating the blood flow of the LAA. A further study using TEE examination both before and after ablation will be required to confirm the efficacy of catheter ablation in improving the blood flow in the LAA.

The detection of thrombi on MDCT

The ability of TEE to detect thrombi in the LAA was 100%.[13, 22] However, TEE could not completely exclude the presence of thrombi in the LAA due to artifacts in some patients. Although the specificity of MDCT in the detection of thrombi in this population was 91% due to false-positives, presumably resulting from blood flow disturbances, both the sensitivity and negative predictive value were 100%. MDCT was routinely performed in some institutions prior to AF ablation because catheter ablation was performed using a three-dimensional mapping system that can be integrated with MDCT. MDCT is useful for evaluating the presence of thrombi in patients with AF. Furthermore, TEE may not be necessary in all patients without filling defects on MDCT prior to ablation. Previous studies have demonstrated that MDCT is sensitive for detecting LAA thrombi in patients with AF.[23-27] On the other hand, Dorenkamp et al.[28] reported that the sensitivity and positive predictive value of MDCT in the identification of LAA thrombi were 29% and 20%, respectively. In their study, a high CHADS2 score (Ó3) was

significantly associated with the presence of LAA thrombi. The relatively low CHADS2 scores of the patients in the present study (mean CHADS2 score: 0.6) might have resulted in this discrepancy.

Radiation exposure

MDCT was useful for revealing the cardiac anatomy and the presence of thrombi in patients with AF prior to catheter ablation. The three-dimensional image could also help to guide the ablation by integration with the mapping system during procedure. In our institution, MDCT is routinely performed after ablation in order to identify pulmonary stenosis. However the risk of complications regarding radiation exposure continues to be a cause for concern. Second generation 128-slice dual source CT scanners involve lower doses of radiation. In the previous study employing a dual-source 128-slice dual source CT scanner, the effective dose for coronary CT angiography was 1.4 mSv.[29] Furthermore, the 128-slice dual source system reduced the radiation dose to which a patient is exposed in the evaluation if the LA and PV anatomy to 1.4 mSv.[30] Although the effective dose of MDCT in the present study was similar to previous reports, the possibility of complication arising from radiation exposure should be considered. The fluoroscopic time for AF ablation in the present study was similar to that of previous reports.[31, 32] No complications related to radiation exposure, including skin injury and cancer, were observed during the follow up period in the patients of the present study .

Limitations

Although MDCT before and after catheter ablation in patients with AF showed the elimination of LAA filling defects, indicating improved LAA blood flow, no TEE data were available after the procedure. The velocity of the LAA flow, which was recorded by TEE, was significantly lower in the patients with LAA filling defects than in those without. The elimination of LAA filling defects after catheter ablation in patients with AF was evaluated by MDCT, and anti-arrhythmic drugs were administered to all patients with persistent AF for three months after the ablation procedure. The efficacy of catheter ablation alone in improving the

function of the LAA during the first three months remains unknown in patients with persistent AF. However, in the present study, persistent AF was resistant to anti-arrhythmic drug treatment prior to the ablation procedure. Even under medical treatment, catheter ablation played an important role in restoring and maintaining sinus rhythm in the patients with persistent AF. Although this study shows the efficacy of catheter ablation on eliminating MDCT filling defects in the short-term, further studies are needed to examine the long-term effects.

In order to evaluate LAA filling defects, it would be better to perform MDCT with the patient in the prone position. However, the MDCT images were to be integrated into the 3-dimensional mapping system that was used in AF ablation. MDCT was therefore performed with the patient in the supine position ï the same position as he or she would be in during the ablation procedure. In addition, MDCT images of the LAA might be influenced by the characteristics of the LAA and the patientôs heart rate during the MDCT scan. Lastly, although both MDCT and TEE were performed before the ablation procedure, they were not performed on the same day. Thus, a difference in heart rhythm might have affected the results of the present study.

Conclusion

MDCT is useful for evaluating LAA thrombi with a high level of sensitivity and negative prediction value. The maintenance of sinus rhythm that is achieved by catheter ablation results in the disappearance of LAA filling defects on MDCT, which suggests that the flow of the LAA is improved after the procedure.

Conflict of interest

The authors declare no conflicts of interest in association with the present study.

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Table 1. The patient characteristics of the overall population

	Entire group (n=459)
Gender (male/female)	419/40
Age (years)	55.2±10.7
AF type (paroxysmal / persistent)	241/219
AF history (years)	5.2±4.8
PT-INR	1.9±0.7
Heart failure	16
Hypertension	176
Age >75 years	10
Diabetes mellitus	53
CHADS2 score	0.6±0.8
Structural heart disease	43
History of cerebral infarction	15
Left atrial dimension (mm)	40.0±5.9
Ejection fraction (%)	64.2±7.1
LAA Doppler velocity (m/s)	0.59±0.20
Chicken wing LAA	161
Superior LAA-takeoff	42
Duration of anticoagulation before ablation (months)	4.2±3.2

AF, atrial fibrillation; PT-INR, prothrombin time-international normalized ratio; LAA, left atrial appendage.

Table 2. The clinical variables of the patients with and without LAA thrombi or severe SEC

	No LAA thrombus or severe SEC (n=450)	LAA thrombus or severe SEC (n=9)	P value
Gender (male/female)	411/39	6/3	0.05
Age (years)	55.1±10.8	62.7±5.5	0.03
AF type (paroxysmal /persistent)	241/209	0/9	<0.01
AF history (years)	5.2±4.8	4.0±2.7	0.45
PT-INR	1.9±0.7	1.8±0.4	0.58
Heart failure	16	0	0.73
Hypertension	173	3	0.97
Age >75 years	7	3	0.32
Diabetes mellitus	52	1	0.63
CHADS2 score	0.6±0.8	0.8±1.3	0.54
Structural heart disease	41	2	0.45
History of cerebral infarction	13	2	0.022
Left atrial dimension (mm)	40.0±5.9	43.2±5.0	0.09
Ejection fraction (%)	64.2±7.1	64.4±9.7	0.94
LAA Doppler velocity (m/s)	0.60±0.19	0.29±0.13	<0.0001
Chicken Wing LAA	159	2	0.64
Superior LAA-takeoff	36	6	<0.0001
Duration of anticoagulation before ablation (months)	4.2±3.3	4.5±6.4	0.13

LAA, left atrial appendage; SEC, spontaneous echo contrast; AF, atrial fibrillation; PT-INR, prothrombin time-international normalized ratio.

Table 3. The clinical variables of the patients with and without LAA filling defects

	No LAA filling defect (n=408)	LAA filling defect (n=51)	P value
Gender (male/female)	373/35	44/7	0.35
Age (years)	54.8±10.8	58.5±9.5	0.02
AF type (paroxysmal/persistent)	226/182	15/36	<0.001
AF history (years)	5.3±4.9	4.6±3.6	0.37
PT-INR on MDCT	1.9±0.8	1.9±0.5	0.75
Heart failure	14	2	0.82
Hypertension	154	22	0.55
Age > 75 years	9	1	0.69
Diabetes mellitus	41	12	<0.01
CHADS2 score	0.6±0.8	0.9±1.0	0.021
Structural heart disease	8	4	0.044
History of cerebral infarction	10	5	0.018
Left atrial dimension (mm)	39.6±6.0	41.9±4.8	0.01
Ejection fraction (%)	64.2±7.0	64.0±8.1	0.83
LAA Doppler velocity (m/s)	0.61±0.19	0.48±0.20	<0.0001
Chicken wing LAA	150	11	0.047
Superior LAA-takeoff	16	26	<0.0001
Duration of anticoagulation before ablation (months)	4.2±3.4	4.3±5.2	0.32

LAA, left atrial appendage; AF, atrial fibrillation; PT-INR, prothrombin time-international normalized ratio; MDCT, multidetector computed tomography.

Table 4. MDCT at pre- and post-ablation in patients with a filling defect on pre-ablation MDCT

	Pre-ablation (n=42)	Post-ablation (n=42)	P value
Sinus Rhythm/AF	16/26	41/1	<0.0001
LAA filling defect	42 (100%)	1 (2.4%)	<0.0001
PT-INR	1.9±0.7	1.8±0.4	0.58

AF, atrial fibrillation; LAA, left atrial appendage; PT-INR, prothrombin time-international normalized ratio.

Figure legends

Figure 1. Multidetector computed tomography of the left atrium.

(A) The left atrial appendage was filled with contrast medium on multidetector computed tomography. (B) Multidetector computed tomography demonstrates a filling defect in the left atrial appendage (dashed circle). Ao, aorta; LAA, left atrial appendage; LA, left atrium.

Figure 2. The morphology of the left atrial appendage.

The morphology of the left atrial appendage was classified as previously described. (A) Chicken wing. (B) Non-chicken wing.

Figure 3. Patients without filling defects or thrombi in the left atrial appendage.

(A) The left atrial appendage was filled with contrast medium on multidetector computed tomography. (B) The absence of thrombi was confirmed using transesophageal echocardiography. The abbreviations are the same as those in Figure 1.

Figure 4. Patients with filling defects and the absence of thrombi in the left atrial appendage.

Although a filling defect in the left atrial appendage (dashed circle) was detected on multidetector computed tomography (A), no evidence of left atrial appendage thrombi or spontaneous echocardiographic contrast was found on transesophageal echocardiography in the same patient (B). The abbreviations are the same as those in Figure 1.

Figure 5. Patients with filling defects and the presence of thrombi in the left atrial appendage.

A filling defect in the left atrial appendage (circle) was observed on multidetector computed tomography (A), and a thrombus (dashed circle) was confirmed on transesophageal echocardiography (B). The abbreviations are the same as those in Figure 1.

Figure 6. The disappearance of filling defects following catheter ablation.

A filling defect (circle), which was observed on multidetector computed tomography disappeared (dashed circle) at three months after the ablation procedure. The abbreviations are the same as those in Figure 1.

Figure 1

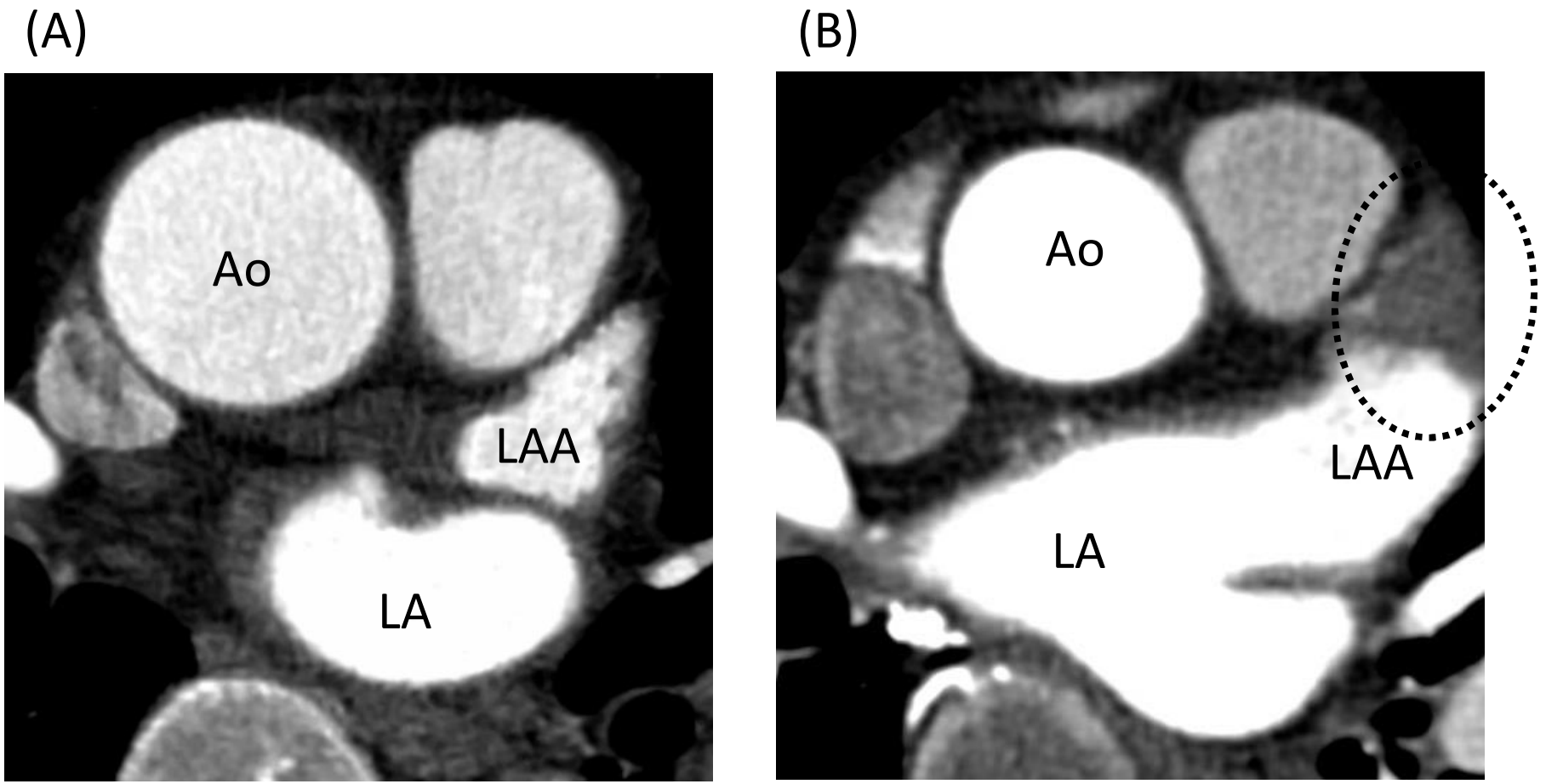
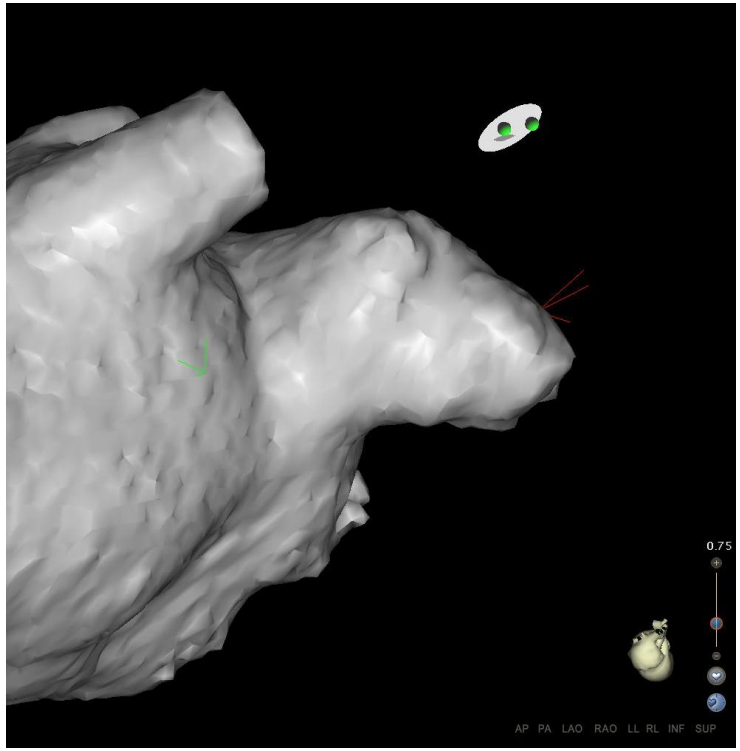


Figure 2

(A)



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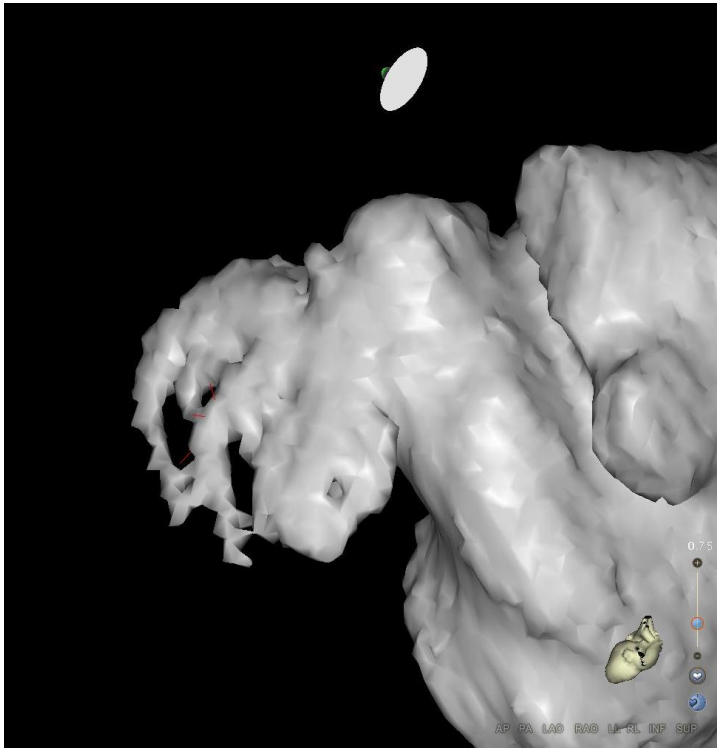
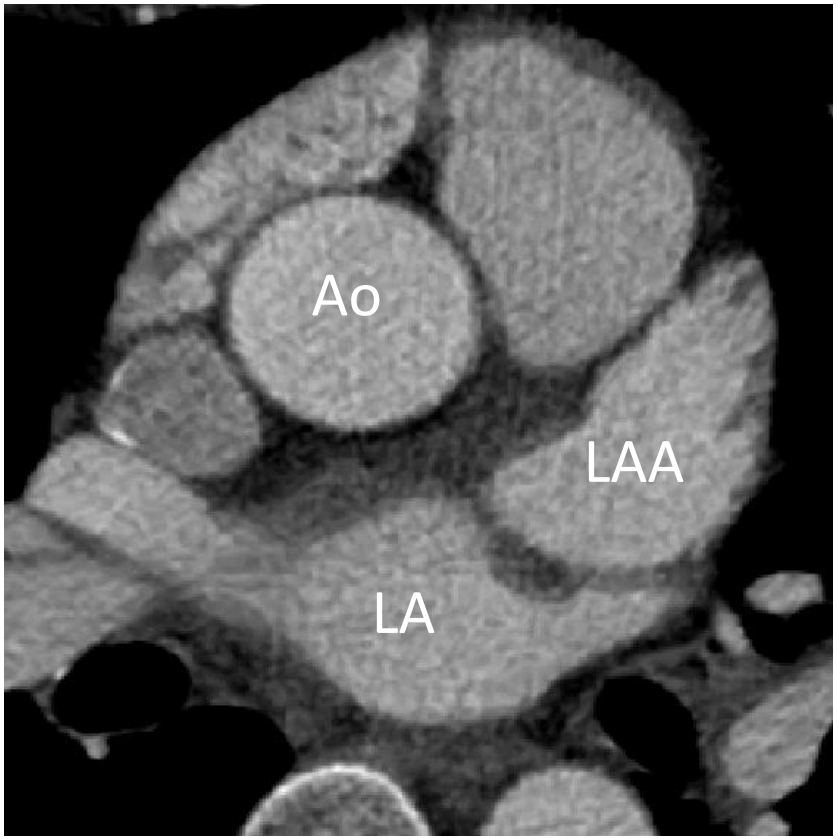


Figure 3

(A)



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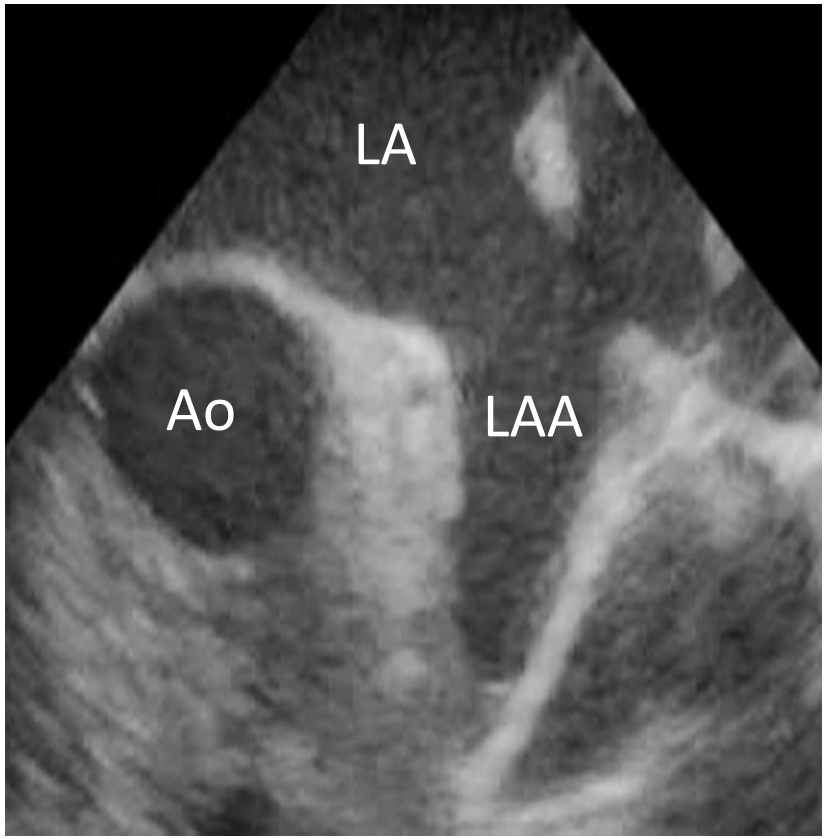

Figure 4

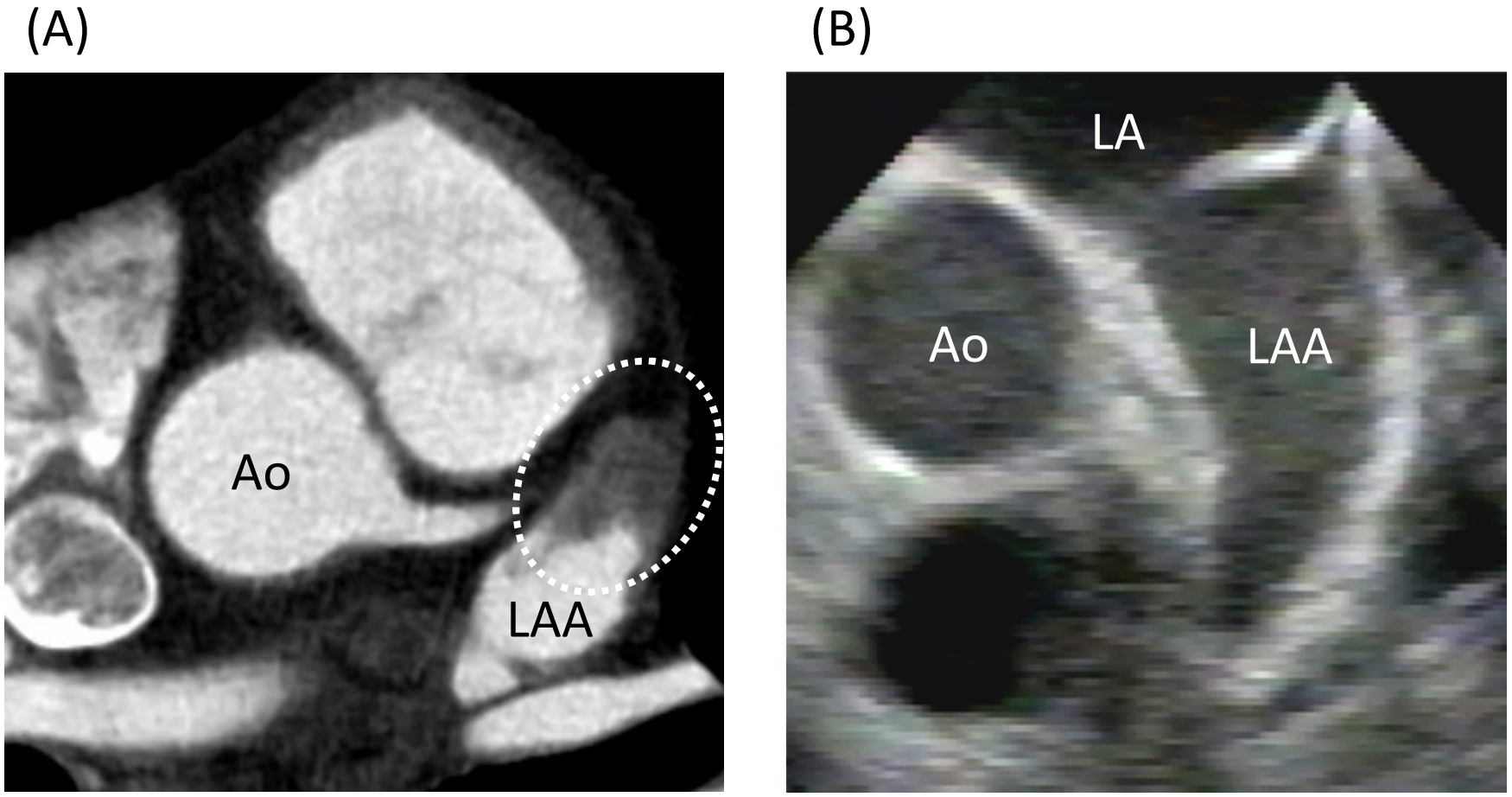

Figure 5

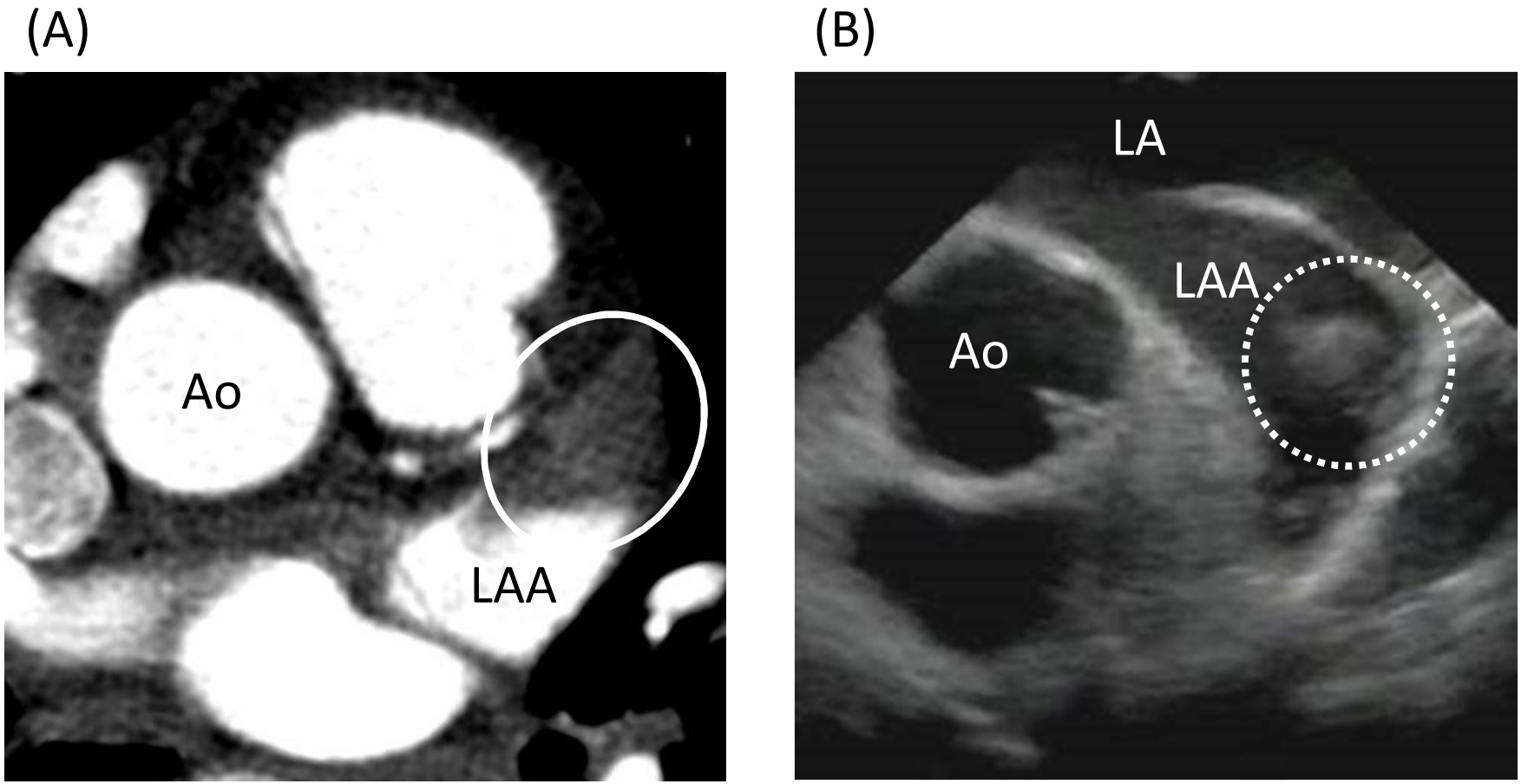

Figure 6