Cardioscopic Detection of Left Ventricular Thrombi – With Special Reference to a Comparison With Left Ventriculography and Echocardiography –

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Background: Thrombosis occurs in the left ventricle and causes ischemic cerebral attacks. However, differences in the incidence of left ventricular thrombi (LVT) among various categories of heart diseases are not known.

Methods and Results: From April 2000 to 31 March 2008, 258 patients (104 females and 154 males; age 63 ± 6 years) with a heart disease underwent cardioscopy of the left ventricle. LVT were detected by cardioscopy in 78 of 258 patients; 12.5% of 57 patients with stable angina, 0% of 9 with unstable angina, 45.2% of 42 with acute myocardial infarction, 23.2% of 43 with old myocardial infarction, 61.9% of 21 with idiopathic acute myocarditis, 44.3% of 68 with idiopathic chronic myocarditis, 33.3% of 6 with rheumatic valvular disease, 25.7% of 31 with idiopathic dilated cardiomyopathy and in 8.0% of 12 with idiopathic hypertrophic cardiomyopathy. Nine of 78 thrombi were globular and 69 were mural. The detection rate of LVT by cardioscopy, left ventriculography, non-contrast and contrast echocardiography was 30.2%, 2.7%, 1.9% and 7.0%, respectively.

Conclusions: LVT were frequently detected by cardioscopy in patients with heart diseases. Although invasive, cardioscopy was more sensitive in detecting LVT than left ventriculography, and non-contrast and contrast echocardiography. (*Circ J* 2011; **75**: 1920–1926)

Key Words: Cardioscopy; Contrast echocardiography; Left ventricular thrombi; Non-contrast echocardiography; Ventriculography

It is well known that a fibrillating left atrium is the site of thrombus formation and acts as a major supply source of thromboemboli in cerebral ischemic attacks.^{1–4} It is also known that the left ventricle is the site of thrombus formation in a post-infarction state.⁵ There have been many case reports on left ventricular thrombi (LVT) in other categories of heart disease such as peripartur cardiomyopathy,⁶ idiopathic dilated cardiomyopathy (DCM),⁷ acute myocarditis (AM),⁸ and antiphospholipid syndrome,⁹ and they cause cerebral embolism.^{10,11} However, the exact incidence of LVT in various categories of heart disease is not well known due to the lack of systematic surveys.

Non-contrast echocardiography (UCG),⁵ contrast UCG,^{5,12} computed tomography,¹³ magnetic resonance imaging,^{5,12} and left ventriculography have been used for the detection of LVT in left atrial thrombi or pulmonary thromboemboli.^{14,15} Although large globular thrombi can be detected, small-sized mural thrombi are difficult to detect using these imaging

modalities. Furthermore, because the shadows of the targets are imaged, definite determination of thrombi is beyond these imaging modalities. Therefore, thrombus characteristics, namely whether the thrombi are fresh or organized, or red blood cell-rich or platelet-rich, which might influence the selection of the therapeutic modality, are difficult to determine with these imaging modalities.

Cardioscopy, specially fiberoptic angioscopy of the heart, is a high resolution imaging modality that enables macroscopic pathological diagnosis of heart diseases. This technique was applied for observation of the cardiac chambers and valves and abnormal intracardiac structures, including LVT.^{16–19}

In the present study, percutaneous cardioscopy was performed to examine the incidence of LVT in patients with various types of heart diseases, and to compare the detection sensitivities among cardioscopy, left ventriculography, non-contrast UCG, and contrast UCG.

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Table 1. Background of Patients With Heart Diseases

Heart diseases	n	Gender (F/M)	Age (years)	Time from onset [#]	LVG	
					EDVI (ml/m ²)	EF
SA	56	18/38	62±7	4.7±2.3 m	76±5	64±9
UA	9	3/6	64±3	1.3±0.5 d	85±10	53±11
AMI	42	14/28	63±5	1.6±0.8 d	78±8	50±8
OMI	43	21/22	63±6	8.2±7.8 m	81±11	54±7
AM	21	13/8	48±9	5.2±3 d	78±9	47±9
CM	38	26/12	52±9	4.1±3 m	91±15	51±10
RVD	8	4/2	42±4	16.9±8 m	88±12	56±6
DCM	31	14/17	46±5	4.2±2.7 m	113±15	45±8
HCM	12	3/9	52±8	8.3±4.2 m	67±15	74±7

[#]Time from onset of subjective symptoms or clinical diagnosis to cardioscopy.

n, number of patients; F, female; M, male; LVG, left ventriculography; EDVI, left ventricular endodiastolic volume index; EF, ejection fraction; SA, stable angina; m, months; UA, unstable angina; d, days; AMI, acute myocardial infarction; OMI, old myocardial infarction; AM, acute idiopathic myocarditis; CM, chronic idiopathic myocarditis; RVD, rheumatic valvular disease; DCM, idiopathic dilated cardiomyopathy; HCM, idiopathic hypertrophic cardiomyopathy.

Methods

Subjects

From 1 April 2001 to 31 March 2008, 258 patients with heart diseases [104 females and 154 males; mean age±SD, 63±6 years; 56 with stable angina (SA), 9 with unstable angina (UA), 42 with acute myocardial infarction (AMI),²⁰ 43 with old myocardial infarction (OMI; 1 month or more since the onset of AMI) complicated with or without SA,²¹ 21 with AM and 38 with chronic myocarditis diagnosed based on Japanese Circulation Society criteria,²² 6 with rheumatic valvular disease, 31 with idiopathic DCM and 12 with idiopathic hypertrophic cardiomyopathy] diagnosed by ventriculography and endomyocardial biopsy,²³ underwent cardioscopy for detection of LVT (Table 1).

The present study was performed at Toho University Medical Center, Sakura Hospital and Funabashi-Futawa Hospital and was approved by their Institutional Review Boards. All the patients provided informed consent for the procedures.

Non-Invasive Examinations Before Cardiac Catheterization

On admission, chest X-rays, electrocardiograms, blood sampling, and transthoracic non-contrast UCG followed by contrast-UCG were carried out. Contrast-UCG was not performed in hemodynamically unstable patients, so they were excluded from the analyses.

Cardioscopy System

The cardioscopy system was composed of a light source, 4.5-F fiberscope, 9-F guiding balloon catheter, intensified chilled coupled device camera, camera controller, DVD recorder and television monitor.

The fiberscope (AF 14; Olympus Corporation, Tokyo) was composed of a 4.5-F fiberscope containing 3,000 glass fibers for image guidance and 300 glass fibers for light guidance. The fiberscope could be passed through the 9-F guiding balloon catheter (Clinical Supply Co, Gifu, Japan). The balloon was inflatable with CO₂. The catheter had a Y connector at the proximal end; 1 channel for fiberscope insertion and another for saline solution flush. The fiberscope and guiding balloon catheter have been approved for clinical use by the Japanese Ministry of Health, Labor and Welfare.

Observation of the LVT by Cardioscopy

In a preliminary experimental cardioscopic study, it was confirmed that the thrombi that had formed on the endocardial surface of the infarcted portion were not detached by saline solution infusion using a power injector toward the thrombi.¹⁶

At first, the right femoral artery was cannulated and left ventriculography and coronary angiography were performed. Cardioscopy was performed in the patients based on this result. In brief, after left ventriculography, a guiding balloon catheter was introduced into the left ventricle so its distal tip was in a location that did not touch the endocardium, usually in the middle portion of the ventricle, and the balloon was inflated with CO₂ to make a dead space between the fiberscope and the target. Next, a fiberscope was introduced into the guiding catheter to place the fiberscope tip at the distal most end of the guiding catheter. Thereafter, the guiding balloon catheter was slowly advanced while infusing heparinized (10U/ml) saline solution at a rate of 5–10 ml/s for 5 s to displace the blood between the target and the fiberscope. When a thrombus was detected, the catheter was stopped so it did not touch the thrombus. The anterior, apical, lateral and inferior wall segments of the left ventricle were surveyed in each patient. When a thrombus was detected in a segment, the other segments were not surveyed. The guiding balloon catheter tip was pre-shaped into a “S”-configuration for observation of the anterior, apical and inferior wall segments, and in a “J”-configuration for the lateral wall segment observation.¹⁸

Cardioscopic measurement of thrombus size was difficult because the images obtained by cardioscopy were fish-eye images.¹⁸

Cardioscopic images were displayed on a television monitor simultaneously with the fluoroscopic images and electrocardiogram. The details of the cardioscopic procedures are reported elsewhere and are shown in Figure 1.^{17–19}

Classification of LVT

The LVT were classified by their configuration into globular (a large thrombus protruding into the cardiac chamber occupying the entire visual field of a cardioscope) and mural (a small sized thrombus attached to the left ventricular luminal surface, not protruded, and not occupying the entire visual field of the cardioscope).

The color of the thrombi was classified into red, cotton

Figure 1. Cardioscope and its manipulation. (A) Lateral view of a cardioscope that is composed of a fiberscope (a) and a guiding balloon catheter (b). (B) Schematic representation of the manipulation. The balloon (b) of the guiding balloon catheter was gently pushed against the endocardial surface to create a dead space. Saline solution was injected into the dead space to displace the blood, and the endocardial surface was observed by the fiberscope (a). (C) Left ventriculogram at diastole. (D) Injection of a contrast material to determine the location of the angioscope tip (arrow). Arrowhead, a pacemaker electrode.

Figure 2. A globular left ventricular thrombus (LVT). A 57-year-old male with old myocardial infarction (40 days after the onset of acute myocardial infarction). (A) Left ventriculogram at left anterior oblique projection. A globular defect in the akinetic apical segment, suggesting a thrombus (arrow). (B) Cardioscopic image. A globular LVT with a red-and-white in a mosaic pattern (arrow) that corresponded to the defect in (A).

candy-like white, red-and-white in a mosaic pattern, yellow, and red-and-yellow in a mosaic pattern. Intraobserver and interobserver agreements on red, cotton candy-like white, red-and-white in a mosaic pattern, yellow, and red-and-yellow in a mosaic pattern LVT were 96%, 88%, 81%, 100%, and 100%, respectively, and 91%, 88%, 81%, 97%, and 100%, respectively.

Statistical Analysis

The data obtained were tested by Fischer's exact test. A $P < 0.05$ was considered significant.

Results

Representative Examples

Figure 2 shows a globular thrombus that was red-and-white in a mosaic pattern and was attached to the luminal surface of the infarcted apical wall segment of the left ventricle in a patient with OMI.

Figure 3 shows demonstrable examples of mural LVT with different colors, namely red, white, yellow, and red-and-yellow in a mosaic pattern.

Figure 3. Color of the left ventricular thrombi (LVT). (A) red LVT (arrow) in a 66-year-old male with OMI. (B) White LVT (arrow) in a 47-year-old female with CM. (C) Yellow LVT (arrow) in a 50-year-old male with OMI. (D) Red-and-yellow in a mosaic pattern LVT in a 71-year-old male with OMI. Arrow, yellow LVT. Arrowhead, red LVT. OMI, old myocardial infarction; CM, chronic idiopathic myocarditis.

Table 2. LVT Detected by Cardioscopy, Left Ventriculography and UCG in Patients With Heart Diseases

Heart diseases	n	Cardioscopy		LVG	UCG	
		Thrombus present (%)	Thrombus globular/mural		Non-cont	Cont
SA	56	7 (12.5)	0/7	0	0	0
UA	9	0 (0.0)	0/0	0	0	0
AMI	42	20 (45.2)**	5/15	4	5 (2#)	6 (1#)
OMI	43	10 (23.2)	2/8	2	2	4
AM	21	13 (61.9)†	2/11	2	3 (1#)	5
CM	38	17 (44.3)	0/17	0	0	3
RVD	6	2 (33.3)	0/2	0	0	0
DCM	31	8 (25.7)	0/8	0	0	0
HCM	12	1 (8.0)	0/1	0	0	0
Total	258	78 (30.2)††	9/69	8 (2.7)	10 (1.9)	18 (7.0)

LVT, left ventricular thrombi; UCG, echocardiography; Non-cont, non-contrast UCG; Cont, contrast UCG. Other abbreviations are as per Table 1.

*P<0.01 vs. SA. †P<0.05 vs. CM. ††P<0.0001 vs. LVG Non-cont and Cont. #Thrombus suspected but inconclusive.

Incidence of LVT by Cardioscopy

The incidence of LVT in AMI was 45.2%, which was higher than in the SA group, but was not different to that of the OMI group. The incidence of LVT in the AM group was 61.9% and it was higher than that of the CM group. Although lower in incidence, LVT were observed in all other categories of heart disease except UA (Table 2).

Morphology and Color of LVT

Globular and mural thrombi were detected in 9 and 69 patients, respectively. Globular thrombi were observed in patients with AMI, OMI and AM. All globular LVT were

located in the apical portion. LVT were mural in the other types of heart diseases (Table 2). The relationship between regional wall motion and presence or absence of LVT was not examined because left ventriculograms were obtained only in 2 directions and therefore to quantitatively measure regional wall motion at the site of LVT was difficult.

Red, cotton candy-like white, red-and-white in a mosaic pattern, yellow, and red-and-yellow in a mosaic pattern LVT were observed in 24, 8, 11, 27 and 2 patients, respectively. The incidence of red thrombi was higher than cotton candy-like white and red-and-white in a mosaic pattern LVT. The incidence of yellow thrombi was higher (P<0.05) than cot-

Table 3. Color of LVT

Heart diseases	Red	White	Red-and-white	Yellow	Red-and-yellow
SA	0	1	1	5	0
UA	0	0	0	0	0
AMI	11	3	6	0	0
OMI	2	1	1	6	0
AM	8*	3	2	0	0
CM	2	0	1	12 ^{††}	2
RVD	1	0	0	1	0
DCM	3	0	2	3	0
HCM	0	1	0	0	0
Total	24 [‡]	8	11	27 [§]	2

Abbreviations as per Tables 1,2.

*P<0.05 vs. CM. ^{††}P<0.01 vs. AM. [‡]P<0.05 vs. White, Red-and-white and Red-and-yellow. [§]P<0.05 vs. White, Red-and-white, and Red-and-yellow.

ton candy-like white and red-and-white in a mosaic pattern LVT. Yellow LVT was more frequently observed in CM than in AM (Table 3).

Comparison of Sensitivity in Detecting LVT Among Cardioscopy, Non-Contrast UCG and Contrast UCG

LVT was detected by cardioscopy, left ventriculography, non-contrast UCG and contrast-UCG in 30.2%, 2.7%, 1.9%, and 7.0%, respectively, of the patients. The detection rate for cardioscopy was higher (P<0.0001) than that for the other 3 imaging modalities. Although globular LVT were detected, mural LVT were only slightly detectable by left ventriculography, non-contrast UCG and contrast UCG (Table 2). The detection rate of contrast UCG was higher (P<0.01) than that of non-contrast UCG (Table 2).

Complications

No serious complications such as cerebral or systemic embolism were noted during or after cardioscopy.

Treatments

Anticoagulant therapy with warfarin was performed in patients with globular thrombus until the thrombus had disappeared on contrast UCG. Antiplatelet therapy with aspirin and/or ticlopidine was performed in all patients with AMI and OMI to prevent coronary thrombosis, irrespective of the presence or absence of LVT. These treatments were not carried out in patients with other types of heart disease having mural LVT.

Follow up for 1 Year

Twenty-one days after cardioscopy, cerebral ischemic attack occurred despite the use of warfarin in a patient with OMI in whom a globular thrombus was observed. A 1-year follow-up study was completed with 189 patients. In addition to the above mentioned patient, cerebral ischemic attacks were observed in 3 patients with mural thrombus and in 1 patient without mural thrombus. Atrial fibrillation was not observed in any of these patients. Whether the thrombi observed by cardioscopy were the sources of cerebral ischemic attacks was not determined.

Discussion

Incidence of LVT

In the present study, LVT were observed by cardioscopy, on average, in approximately 30% of patients with heart diseases, suggesting that LVT was prevalent in patients with heart diseases.

LVT was observed in AMI and AM more frequently than in other heart diseases. It is known that LVT are formed more frequently in a left ventricle with contraction disturbance or deformation, which causes stagnation and turbulence of blood flow. The left ventricular contraction (ejection fraction) was reduced in patients with AMI and AM. This might be the reason why LVT was frequent in these 2 diseases. Although left ventricular contraction was also decreased in patients with DCM, the incidence of LVT was less than that in AMI and AM. Loss of antiplatelet and/or anticoagulant activity of the endocardial cells due to ischemia or inflammation might have also contributed to the higher incidence of LVT in AMI and AM. Occurrence of LVT in patients with SA might have also been due to ischemia-induced endocardial cell damages.¹⁸

Fibrillating atrium is a major supplying source of thromboemboli in patients with ischemic cerebral attacks. In the present follow-up study, ischemic cerebral attacks occurred in 4 patients without atrial fibrillation. Although definite evidence is lacking, there is a possibility that LVT was the mother thrombus.

Determination of Composition and Age of LVT by Color

It is well known that a thrombus (in any vessel or cardiac chamber) formed exhibits a red color within a few days; a dark-red color at around 1 week; and a yellow color due to deposition of β -carotene and organization around 1 month. When red blood cells are washed out from the surface of a recently formed thrombus, the thrombus appears soft and exhibits a white color due to exposed platelets and fibrin.²⁴ Therefore, the red, cotton candy-like white, red-and-white in a mosaic pattern, yellow, and red-and-yellow in a mosaic pattern LVT were considered to be fresh, fresh, fresh, old, and fresh-and-old in mixture, respectively, and, red, white and cotton-candy-like white, red-and-white in a mosaic pattern were considered to be red blood-rich, fibrin and/or platelet-rich, and both red blood cell- and fibrin and/or platelet-rich, respectively.

LVT exhibiting red, cotton candy-like white, red-and-white in a mosaic pattern, and red-and-yellow in a mosaic pattern were observed in chronic diseases, suggesting repeated formation of fresh thrombi even in chronic diseases. In addition to the anatomical and functional properties of the left ventricle, the loss of anti-thrombotic action of the endocardial cells and changes in humoral mechanisms such as the thrombotic, thrombolytic and fibrinolytic actions of the blood might have contributed to the repeated LVT formation.

Comparison With Other Imaging Modalities

The rate of detection of LVT by cardioscopy was significantly higher than that by non-contrast and contrast UCG in the present study, indicating that cardioscopy is a more sensitive method for detecting LVT.

Siebenlink observed LVT by contrast UCG in 9.0% (conclusive) of 156 patients with post-infarction.⁵ Weinsaft observed LVT by non-contrast UCG in 6% and by contrast UCG in 12% of 121 patients with post-infarction.¹² In the present study, the patients with myocardial infarction were observed by non-contrast UCG in 5.8% and by contrast UCG

in 11.6%. The detection rate by non-contrast and contrast UCG were not different among these 3 studies.

Weinsaft observed LVT by delayed-enhanced magnetic resonance in 16.3% of 121 patients with myocardial infarction.¹² LVT were observed in 35.1% of 85 patients with myocardial infarction in the present study. Although a comparison was not performed in the same patient population, the sensitivity of cardioscopy for detecting LVT seems higher ($P<0.05$) than that of magnetic resonance imaging.

Magnetic resonance imaging can survey the entire left ventricle whereas cardioscopic observation is limited to at most 4–5 sections due to the limited volume of saline solution useable for blood displacement. Which of these 2 imaging modalities is more sensitive at detecting LVT remains to be elucidated.

Magnetic resonance imaging shows only the shadow of the thrombus, whereas cardioscopy can observe the thrombus directly. Therefore, it is evident that the specificity of cardioscopy is superior to that of magnetic resonance imaging and any other clinically available imaging modality with respect to detecting LVT.

Source of Cerebral Embolism

In addition to the left atrium, the carotid arterial trees,²⁵ subclavian artery,²⁶ aortic arch,²⁷ ascending and descending aorta,²⁸ and venous thrombi through a patent foramen ovale²⁹ are not infrequently the source of emboli in cerebral ischemic stroke. However, the source of the emboli is not routinely determined in 25–50 % of patients.³⁰

The left ventricle has not received attention as a source of emboli supply. The results from the present study strongly suggest that the left ventricle is an important source of emboli in cerebral ischemic attacks.

Complications and Clinical Usefulness of Cardioscopy

If it is demonstrated that cerebral ischemic attacks occur in patients with small-sized mural LVT, cardioscopy, although invasive, can be used for detection of the LVT because they are only slightly detectable by other imaging modalities. This technique is easy for clinicians who are skillful at cardiac catheterization. However, it is essential that the LVT not be touched by the tip of the guiding catheter or fiberoptic in order to avoid LVT detachment and the resultant embolism.

Study Limitations

Cardioscopic observation of the left ventricular endocardial surface was limited up to 4 wall segments due to the limited volume (up to 500ml to prevent the occurrence of acute congestive heart failure) of the saline solution necessary for displacement of the blood. It was therefore unclear whether thrombi existed in other segments.

Conclusions

LVT were detected by percutaneous cardioscopy in 78 of 258 patients with various types of heart disease. The incidence of LVT was higher in patients with AMI and AM than in other categories of heart diseases. The LVT were globular in 9 and mural in 69. The overall detection rate was 30.2%, 2.7%, 1.9%, and 7.0% by cardioscopy, ventriculography, non-contrast UCG and contrast UCG, respectively, indicating that cardioscopy is more sensitive than the other 3 imaging modalities for detecting LVT.

Disclosures

There are no conflicts of interest to disclose. Relationships With Industry: No relationships. Funds: This study was performed without financial support.

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