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Association Between Basal Thinning of Interventricular Septum and Adverse Long-Term Clinical Outcomes in Patients With Cardiac Sarcoidosis

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Background: Basal thinning of the interventricular septum (IVS) is an important diagnostic feature of cardiac sarcoidosis (CS), but its long-term prognostic significance remains unclear.

Methods and Results: We examined 74 consecutive patients who were diagnosed with CS. Basal IVS thickness at a point located 10 mm from the aortic annulus was measured. IVS thickness at the left ventricular minor axis level (IVS) was also measured according to the recommended procedure of the American Society of Echocardiography. Patients were divided into 2 groups based on the presence or absence of basal IVS thinning, which was defined as basal IVS ≤4 mm and/or basal IVS/IVS ratio ≤0.6. Basal IVS thinning was observed in 21 patients and was associated with greater long-term adverse events during follow-up (5.1±2.5 years), although the baseline characteristics were comparable between groups (overall, P<0.01; all-cause death, P=0.53; symptomatic arrhythmias, P<0.01; heart failure admission, P=0.027). Multivariate analysis showed basal IVS thinning was an independent determinant of long-term adverse events (hazard ratio 2.86, 95% confidence interval 1.31–6.14) even after adjustment for existing prognostic variables.

Conclusions: The presence of basal IVS thinning at the time of CS diagnosis was associated with poor long-term clinical outcomes, suggesting its prognostic significance in patients with CS. (*Circ J* 2015; **79:** 1601–1608)

Key Words: Cardiomyopathy; Echocardiography; Heart failure; Sarcoidosis

Cardiac sarcoidosis (CS) is characterized by the formation of non-caseating granulomas, most commonly in the myocardium and conduction system, particularly the interventricular septum (IVS) and free wall of the left ventricle (LV).[1–5] Although common clinical manifestations of CS are impaired systolic LV function, atrioventricular block (AVB), ventricular arrhythmias, and sudden cardiac death,[6] predictors of these adverse events at the time of diagnosis have not been well determined.

The gold standard for the diagnosis of CS is pathological evidence obtained by endomyocardial biopsy. In 1993, the Japanese Ministry of Health and Welfare published a set of diagnostic criteria for CS that require direct evidence of sarcoid granulomas from endomyocardial biopsy or indirect evidence of inflammatory myocardial lesions in extra-CS patients.[7] Despite the high specificity of endomyocardial biopsy for detecting CS, the sensitivity is relatively low, ranging from 20% to 63%, because of the non-uniformity of granulomas in cardiac tissue.[8,9]

Emerging imaging modalities, particularly myocardial thallium-201 scintigraphy, 18-fluoro-deoxyglucose positron emission tomography (FDG-PET) and cardiac magnetic resonance (CMR) combined with late gadolinium enhanced (LGE) imaging (LGE-CMR) and gallium-67 (Ga) uptake, have markedly improved the sensitivity and specificity of CS diagnosis[10–14] and been shown to have prognostic value for predicting clinical outcomes in CS patients.[15–21] Nevertheless, echocardiography is less expensive and more practically useful for the initial diagnostic screening of CS patients.[22,23] It would be useful to identify a reliable echocardiographic indicator for the

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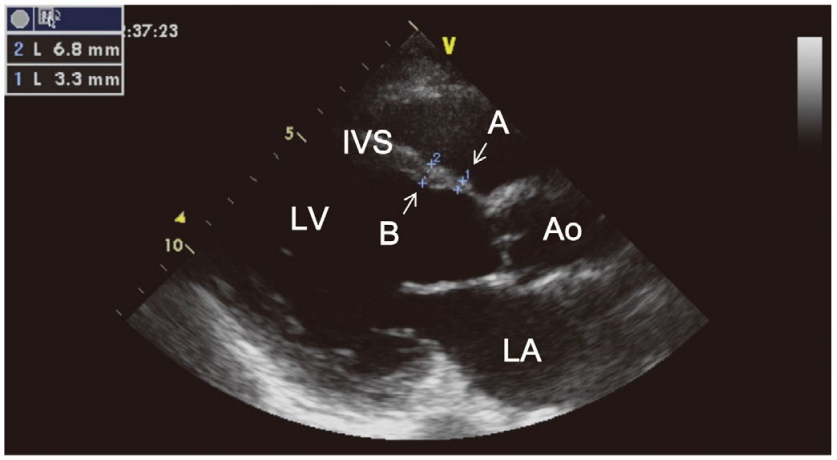

Figure 1. Representative case of evaluation of thinning of the basal interventricular septum (IVS) in a patient with cardiac sarcoidosis. End-diastolic thickness of IVS at points A and B is first measured, and the A/B ratio is then calculated. (A) 10 mm distant from the aortic annulus on the IVS (basal IVS). (B) LV minor axis level on the IVS (IVS). Ao, aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.

diagnosis of CS.

Even though detection of basal IVS thinning on echocardiography is highly specific for CS and has been proposed as a potential diagnostic indicator, the determination of basal IVS thinning is subjective and a defined cut-off value has not been established. Recently, a report comparing mean IVS thickness between 175 CS patients and 2,130 control subjects suggested that basal IVS thinning defined as a thickness ≤4 mm and/or a basal IVS/IVS ratio ≤0.6 could be used as the criterion for diagnosing CS with 99.0% specificity and 38.9% sensitivity.[24] However, the long-term prognostic significance of basal IVS thinning assessed according to this criterion remains unclear.

The aim of the present study was to clarify the long-term prognostic significance of basal IVS thinning in CS patients.

Methods

Study Population

We examined 76 consecutive patients who were admitted between May 2003 and December 2012, and diagnosed during the hospitalization with CS based on clinical and/or pathological findings, without coronary artery disease, based on the diagnostic criteria described in the 2006 revised version of the Japanese Ministry of Health and Welfare guidelines for CS.[7,25] Patients in whom basal IVS could not be evaluated by echocardiography because of low-quality images (n=2) were excluded. The study protocol conformed with the guidelines of the institutional ethics committee (M25-047).

Study Protocol

We collected the following data: age, sex, traditional cardiovascular risk factors, cardiovascular medication, history of fatal ventricular arrhythmias, advanced AVB, congestive heart failure, catheter ablation for ventricular tachyarrhythmias, cardiac resynchronization therapy (CRT), implantation of permanent pacemaker or implantable cardioverter defibrillator (ICD) and extracardiac organ involvement of sarcoidosis, and findings of imaging modalities including Ga scintigraphy, FDG-PET, and LGE-CMR, at the time of diagnosis. Regarding the FDG-PET findings, specific "focal" or "focal on diffuse" uptake of FDG was defined as positive, according to the previous report.[26] The presence of LGE on CMR was defined as any hyperenhancement in the myocardium. In LGE-positive patients, the mass of LGE was calculated using the area of LGE as previously described.[21] The findings of Ga scintigraphy, FDG-PET and LGE-CMR were determined by the consensus of 2 experienced radiologists. Venous blood samples were serially obtained to measure plasma angiotensin-converting enzyme (ACE) activity, lysozyme, B-type natriuretic peptide (BNP), hemoglobin, serum creatinine and C-reactive protein (CRP) levels.

Echocardiographic Analysis and Definition of Basal IVS Thinning

Echocardiography was performed at the time of diagnosis of CS. LV end-diastolic and end-systolic dimensions and LV ejection fraction (LVEF), as determined by the modified Simpson's method, were evaluated.

Regarding the measurement of IVS thickness, we recorded the parasternal long-axis view and measured the IVS thickness directly from 2D images based on the recommendation of the American Society of Echocardiography (ASE) committee.[27]

According to the previously proposed definition,[24] IVS thickness at a point located 10 mm from the aortic annulus (basal IVS) was measured. IVS thickness at the LV minor axis level (IVS) was also measured, following the recommended procedure of the ASE.[27] Basal IVS thinning was defined as a thickness ≤4 mm and/or a basal IVS/IVS ratio ≤0.6 (**Figure 1**). All echocardiographic findings were interpreted and measured by 2 experienced cardiologists.

Study Endpoint and Long-Term Follow-up

The study endpoint was a composite of all-cause death, heart failure requiring admission, and symptomatic arrhythmia, which was defined as ventricular tachyarrhythmias with clinical symptoms and requiring admission, appropriate ICD treatment for termination of ventricular fibrillation or sustained ventricular tachycardia, or bradyarrhythmias requiring pacemaker implantation. Follow-up data were obtained by dedicated coordinators and investigators from the hospital records, by direct contact with patients or patients' physicians in the hospital or outpatient clinic, telephone interview of patients or, if deceased, of family members, and by mail.

Statistical Analysis

Continuous data are expressed as mean±SD. Continuous

Table 1. Baseline Characteristics of Patients With CS

	Overall (n=74)	IVS thinning (n=21)	No IVS thinning (n=53)	P value
Age, years	62±8.2	63±7.7	62±8.5	0.68
Female, n (%)	48 (65)	13 (62)	35 (66)	0.75
Clinical history				
Hypertension, n (%)	19 (26)	6 (29)	13 (25)	0.73
Dyslipidemia, n (%)	19 (26)	5 (24)	14 (26)	0.82
Diabetes mellitus, n (%)	5 (7)	1 (5)	4 (8)	0.62
Smoking, n (%)	23 (31)	7 (33)	16 (30)	0.80
Cerebral infarction, n (%)	6 (8)	2 (10)	4 (8)	0.62
VT/VF, n (%)	22 (30)	4 (19)	18 (34)	0.18
CHF, n (%)	25 (34)	10 (48)	15 (28)	0.14
NYHA class, degree				0.77
I–II, n (%)	51 (69)	15 (71)	36 (68)	
III–IV, n (%)	23 (31)	6 (29)	17 (32)	
Organ involvement				
Lung, n (%)	29 (39)	7 (33)	22 (42)	0.52
Skin, n (%)	5 (7)	1 (5)	4 (8)	0.65
Eye, n (%)	21 (28)	4 (19)	17 (32)	0.24
No. of involved organs	1.8±0.9	1.6±0.8	1.8±0.9	0.23
Cardiac biopsy proven, n (%)	15 (20)	2 (10)	13 (25)	0.15

Continuous variables are presented as mean±SD. Categorical variables are presented as number of patients (%). AV, atrioventricular; CHF, congestive heart failure; CS, cardiac sarcoidosis; IVS, interventricular septum; NYHA, New York Heart Association; VF, ventricular fibrillation; VT, ventricular tachycardia.

variables were compared using unpaired t test or nonparametric means test. Categorical variables are reported as frequencies with percentages and were compared between groups using a chi-squared test. Long-term event-free survival was estimated using Kaplan-Meier curves, and a log-rank (Mantel-Cox) test was used to assess differences according to the presence or absence of basal IVS thinning at the time of CS diagnosis. Cox proportional hazard model analysis, which included determinant factors with a P value ≤0.10 in the univariate analysis, was used to assess the association of these factors with adverse events. All statistical analyses were performed using SPSS 13.0 for Windows (SPSS Inc, Chicago, IL, USA). Statistical significance was defined as $P<0.05$.

Results

Patients Characteristics

Basal IVS thinning was detected in 21 CS patients (28%). Although patients with basal IVS thinning had a higher incidence of fatal ventricular arrhythmias at the time of diagnosis than those without, no significant difference between the 2 groups was detected with respect to sex, incidence of advanced AVB, or congestive heart failure at the time of diagnosis. The prevalence of traditional cardiovascular risk factors and of extracardiac organ involvement of sarcoidosis was similar between the 2 groups (Table 1).

Echocardiographic and Other Imaging Findings

Baseline LV end-systolic dimension was larger, and basal IVS thickness and basal IVS/IVS ratio were smaller in patients with basal IVS thinning than in those without. LV end-diastolic dimension tended to be larger and LVEF was lower in patients with basal IVS thinning than in those without. The prevalence of AVB of each degree, and the rates of positive findings on Ga scintigraphy, PDG-PET and LGE-CMR imaging did not significantly differ between the 2 groups (Table 2). LGE-CMR was performed in 37 patients (50% of the study population). Patients with basal IVS thinning had significantly greater %LGE mass in the LV than those without basal IVS thinning (Table 2).

Laboratory Data and Medication

Laboratory data, including plasma ACE activity, lysozyme and BNP levels, hemoglobin, and serum creatinine and CRP levels at the time of diagnosis, were comparable between patients with and without basal IVS thinning (Table 3).

Administration of corticosteroids, other immunosuppressants, cardiovascular medication, including β-blockers, ACE inhibitors or angiotensin-receptor blockers, diuretics, statins, amiodarone and other antiarrhythmic agents, catheter ablation for ventricular tachyarrhythmias, CRT, and implantation of a permanent pacemaker or ICD were similarly performed in the 2 groups (Table 3). In patients who received corticosteroid therapy, the induction and maintenance doses of corticosteroid were comparable between the 2 groups (Table 3).

Long-Term Clinical Outcomes

Patients were followed for 5.1±2.5 years. Kaplan-Meier survival curves revealed that the presence of basal IVS thinning at the time of diagnosis was associated with a higher incidence of composite adverse events (all-cause death, symptomatic arrhythmias, and heart failure requiring admission) during the observation period (Figure 2A). Although basal IVS thinning was not a significant determinant of all-cause death (Figure 2B), it was an independent determinant of symptomatic arrhythmias and heart failure requiring admission (Figures 2C,D).

Univariate Cox proportional hazards model analysis for determinants of composite adverse events revealed that the presence of basal IVS thinning, absence of corticosteroid therapy, and receiving CRT at the time of diagnosis were the

Table 2. Baseline Findings of Echocardiography, ECG and Other Imaging Modalities in Patients With CS

	Overall (n=74)	IVS thinning (n=21)	No IVS thinning (n=53)	P value
Echocardiography				
LVDd, mm	57.2±9.8	60.0±10.9	56.2±9.1	0.16
LVDs, mm	45.2±11.9	50.2±12.9	43.2±11.0	0.04
LVEF, %	34.4±12.9	32.5±12.8	35.1±13.1	0.46
LAD (mm)	39.6±7.8	40.3±8.7	41.8±7.0	0.55
Moderate-severe MR, n (%)	15 (20)	4 (19)	11 (21)	0.87
Thickness of IVS at each point				
Basal IVS	7.6±2.6	4.6±1.8	8.7±1.9	<0.01
IVS	9.7±2.4	9.3±3.1	9.9±2.2	0.48
Basal IVS/IVS ratio	0.8±0.3	0.5±0.1	0.9±0.2	<0.01
ECG				
1st degree AVB, n (%)	10 (14)	3 (14)	7 (13)	0.90
2nd degree AVB, n (%)	7 (9)	1 (5)	6 (11)	0.38
3rd degree AVB, n (%)	22 (30)	6 (29)	16 (30)	0.89
Other imaging modalities				
Ga scintigram positive, positive/n (%)	43/69 (62)	12/20 (60)	31/49 (63)	0.80
FDG-PET positive, positive/n (%)	41/64 (64)	12/18 (67)	29/46 (63)	0.79
LGE-CMR positive, positive/n (%)	36/37 (97)	9/9 (100)	27/28 (96)	0.57
%LGE mass (%)	17.0±10.0	26.7±6.7	13.6±8.6	<0.01

Continuous variables are presented as mean±SD. AVB, AV block; FDG-PET, ^{18}F-fluorodeoxy glucose-positron emission tomography; Ga, gallium; LAD, left atrial diameter; LGE-CMR, late gadolinium enhancement-cardiac magnetic resonance; LVDd, left ventricular diastolic diameter; LVDs, left ventricular systolic diameter; LVEF, left ventricular ejection fraction; MR, mitral regurgitation. Other abbreviations as in Table 1. Categorical variables are presented as number of patients (%).

Table 3. Baseline Laboratory Data and Therapies in Patients With CS

	Overall (n=74)	IVS thinning (n=21)	No IVS thinning (n=53)	P value
Laboratory data				
Hemoglobin, g/dl	13.0±1.5	12.9±1.4	13.1±1.6	0.75
Serum creatinine, mg/dl	0.8±0.2	0.8±0.3	0.8±0.2	0.48
ACE, IU/L	16.2±8.8	15.5±9.9	16.4±8.5	0.76
Lysozyme, IU/L	8.5±4.4	8.3±4.1	8.6±4.5	0.80
BNP, pg/ml	242±246	279±272	227±236	0.46
CRP, mg/dl	0.17±0.22	0.13±0.15	0.19±0.23	0.22
Medications				
Corticosteroids, n (%)	56 (76)	15 (71)	41 (77)	0.61
Corticosteroid induction dose, mg/day	30.2±2.3	30.7±2.6	30.0±2.2	0.34
Corticosteroid maintenance dose, mg/day	7.6±4.8	7.8±4.8	7.6±4.8	0.85
Other immunosuppressants, n (%)	3 (4)	0 (0)	3 (6)	0.08
ACE inhibitors, n (%)	26 (35)	10 (48)	16 (30)	0.18
ARBs, n (%)	21 (28)	7 (33)	14 (26)	0.57
β-blockers, n (%)	47 (64)	11 (52)	36 (68)	0.24
Diuretics, n (%)	34 (46)	13 (62)	21 (40)	0.09
Statins, n (%)	21 (28)	5 (24)	16 (30)	0.58
Amiodarone, n (%)	16 (22)	6 (29)	10 (19)	0.24
Other antiarrhythmic agents, n (%)	14 (19)	5 (24)	9 (17)	0.53
VT-ablation, n (%)	8 (11)	3 (14)	5 (9)	0.59
CRT, n (%)	13 (18)	5 (24)	8 (15)	0.18
Pacer/ICD, n (%)	43 (58)	11 (52)	32 (60)	0.54

Continuous variables are presented as mean±SD. Categorical variables are presented as number of patients (%). ACE, angiotensin-converting enzyme; ARB, angiotensin-receptor blocker; BNP, B-type natriuretic peptide; CRP, C-reactive protein; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter defibrillator. Other abbreviations as in Table 1.

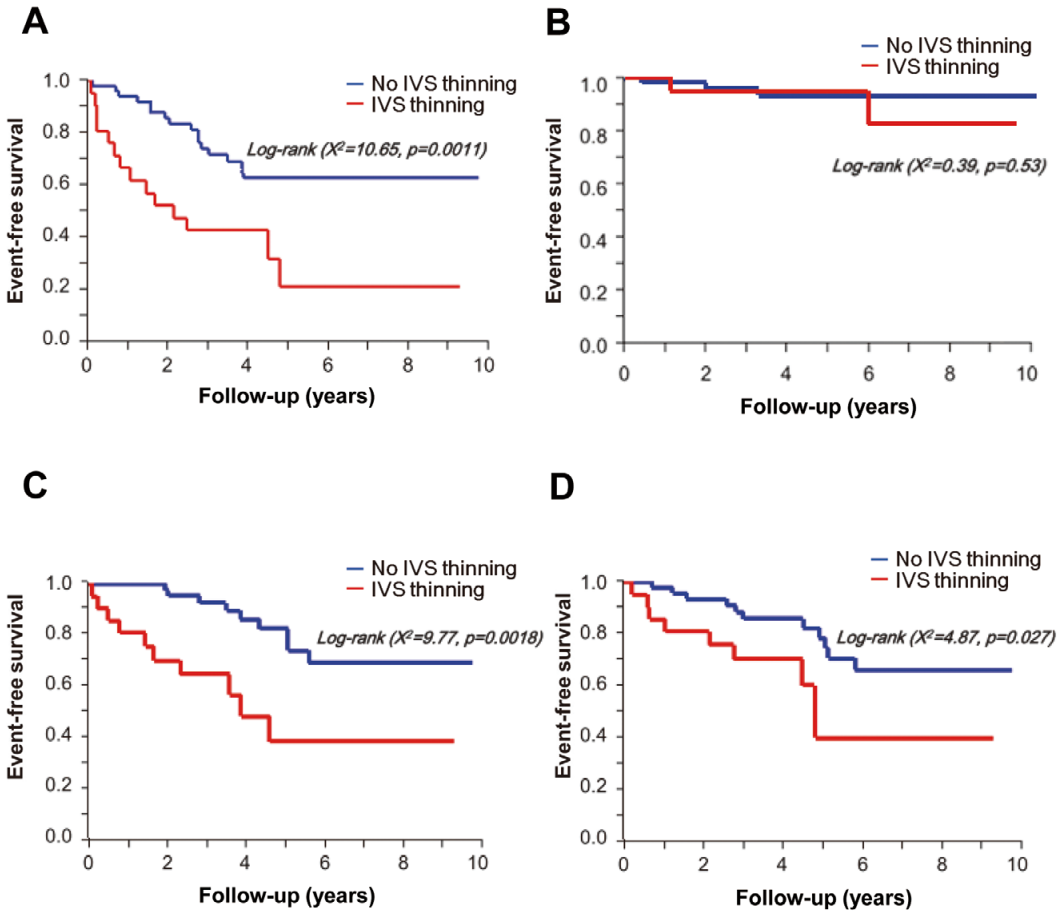

Figure 2. Kaplan-Meier analyses of long-term clinical outcomes of patients with cardiac sarcoidosis with or without basal IVS thinning. (**A**) Composite of all-cause death, symptomatic arrhythmias, and heart failure requiring admission; (**B**) all-cause death; (**C**) symptomatic arrhythmias; (**D**) heart failure requiring admission. IVS, interventricular septum.

Table 4. Cox Proportional Hazards Model for Determinants of Long-Term Adverse Events in Patients With CS

	Univariate			Multivariate		
	HR	95% CI	P value	HR	95% CI	P value
Basal IVS thinning	3.12	1.50–6.43	0.01	2.95	1.40–6.19	0.005
Corticosteroid therapy at the time of diagnosis	0.42	0.19–0.99	0.047	0.49	0.21–1.21	0.12
CRT	2.59	1.23–5.40	0.01	1.87	0.83–4.14	0.13
Age ≥64 years	1.38	0.67–2.96	0.38	–	–	–
Female	1.36	0.64–2.80	0.35	–	–	–
Organ involvement ≥2 organs	1.66	0.80–3.61	0.17	–	–	–
LVEF ≤35%	1.10	0.53–2.27	0.79	–	–	–
Ga scintigram positive	1.18	0.57–2.56	0.94	–	–	–
FDG-PET positive	0.79	0.38–1.67	0.52	–	–	–
Hemoglobin ≤13 g/dl	1.47	0.35–4.17	0.55	–	–	–
Serum creatinine ≥0.7 mg/dl	1.11	0.51–2.66	0.98	–	–	–
ACE ≥15 IU/L	1.07	0.51–2.20	0.85	–	–	–
Lysozyme ≥9 μg/L	1.56	0.83–3.00	0.17	–	–	–
BNP ≥150 pg/ml	1.81	0.88–3.81	0.11	–	–	–

CI, confidence interval; HR, hazard ratio. Other abbreviations as in Tables 1–3.

Table 5. Long-Term Adverse Events in Patients With CS

	Overall (n=74)	IVS thinning (n=21)	No IVS thinning (n=53)	P value
Cause of death				
All, n (%)	9 (12)	5 (24)	4 (8)	0.11
Cardiac, n (%)	5 (7)	2 (10)	3 (6)	0.62
Noncardiac, n (%)	4 (5)	3 (14)	1 (2)	0.07
Symptomatic arrhythmias				
All, n (%)	17 (23)	9 (43)	8 (15)	0.02
VT/VF	15 (20)	8 (38)	7 (13)	0.04
AVB	2 (3)	1 (5)	1 (19)	0.49
Heart failure requiring admission, n (%)	20 (27)	9 (43)	11 (21)	0.05

Categorical variables are presented as number of patients (%). Abbreviations as in Tables 1,2.

only variables with P values ≤0.10. The other examined variables, including sex, extracardiac involvement, LVEF ≤35% (median), positive Ga and/or FDG-PET findings, hemoglobin ≤13 g/dl (median), serum creatinine ≥0.7 mg/dl (median), ACE ≥15 IU/L (median), lysozyme ≥9 μg/L (median), and BNP ≥150 pg/ml (median), were not significant. In the multivariate Cox proportional hazards model analysis, the presence of basal IVS thinning at the time of CS diagnosis was confirmed to be an independent determinant of composite adverse events (Table 4).

The type and prevalence of long-term adverse events are shown in Table 5. The cause of death in nearly 50% of patients with basal IVS thinning was of cardiac origin, despite no significant differences between the 2 groups. The majority of arrhythmic events were ventricular tachyarrhythmias, which were more frequently observed in patients with basal IVS thinning than in those without.

Discussion

In the present study population of 74 CS patients, basal IVS thinning was present at the time of diagnosis in 28% of patients, and was independently associated with a higher incidence of long-term adverse events, especially symptomatic ventricular tachyarrhythmias and heart failure requiring admission. These findings suggest that the criterion for basal IVS thinning used in the present study might be useful for identifying high-risk CS patients.

Basal IVS Thinning as Indicator of Long-Term Clinical Outcomes

In 1987, Valantine et al first reported that cardiac lesions frequently develop in the basal IVS, which often appeared thin and akinetic on cross-sectional echocardiography of CS patients.[5] It was also reported that the basal IVS was compromised in over 90% of hearts with CS at autopsy.[3] Although the precise pathophysiology of CS is unclear, it is characterized by the formation of inflammatory granulomas in myocardial tissue. Macrophages in these localized nodules of inflammation release several types of cytokines, inducing the recruitment of interferon-gamma-secreting T helper type 1 cells that then activate macrophages.[28] Severe inflammation in the basal IVS region is associated with thickening of the tissue as a result of inflammatory edema during the early phase of CS;[29–31] however, during the late phase, persistent inflammation may cause progressive loss of myocardial cells and severe interstitial fibrosis leading to thinning of the basal IVS,[32] which therefore might be an indicator of an advanced state of CS. Furthermore, myocardial fibrosis is based on permanent myocardial damage that is usually difficult to ameliorate, even with immunosuppressants. Thus, basal IVS thinning may reflect advanced and irreversible myocardial damage.

Several analytical modalities other than echocardiography have been evaluated for use in risk stratification of CS patients.[15–21] FDG-PET findings in CS patients are reported to be predictive of severe adverse events, including death,[16] and ventricular arrhythmias.[18] LGE-CMR imaging analysis was reported as able to detect myocardial scarring, which was found to be the best independent predictor of poor outcomes in 175 patients with CS.[19] Furthermore, the extent of LGE evaluated by quantitative CMR analysis was also shown to be significantly associated with adverse events, even in CS patients who had received steroid treatment.[20,21]

Relationship Between Basal IVS Thinning and Future Arrhythmic Events

In the present study, CS patients with IVS thinning had a higher incidence of adverse events, particularly symptomatic arrhythmias, over the mean follow-up period of 5.1 years. Because the AV node is located near the basal IVS, thinning of this area is speculated to be complicated by AVB. However, the development of ventricular tachyarrhythmias, rather than AVB, was more common in CS patients with basal IVS thinning compared with those without. Uemura et al also reported that basal IVS thinning and the development of AVB were not necessarily correlated.[33]

Significance of Basal IVS Thinning on Echocardiography for Clinical Management of CS

Echocardiography is the first-line modality for evaluation of patients with CS. Regional wall motion abnormalities, ventricular aneurysms, pericardial effusions, LV dysfunction, and basal IVS thinning are commonly observed in CS patients, although the sensitivity of these findings varies widely.[34–37] Basal IVS thinning was reported to be indicative of CS; nevertheless, echocardiographic evaluation alone is typically not sufficient for a definitive diagnosis of CS. For this reason, a multimodality approach should provide higher sensitivity and specificity for diagnosing CS.

Despite the observation of basal IVS thinning in patients with CS, basal IVS thinning has not been clearly defined. We used the criterion previously proposed by Morimoto et al.[24] Basal IVS thinning evaluated by echocardiography using this criterion was a strong prognostic determinant of long-term

adverse events in CS patients, indicating that evaluation of basal IVS thinning based on this criterion may be useful for detecting high-risk CS patients at the time of diagnosis. Such patients should be closely monitored and considered for intensive treatment to prevent future adverse cardiac events.

Study Limitations

First, although our CS cohort was relatively large, the sample size was still small, thereby limiting the ability to generalize the findings and the statistical power for detecting differences in negative data. Second, because patients without a pacemaker, ICD, or CRT were not always monitored by 24-h electrocardiography, it is possible that the incidence of several tachy- and bradyarrhythmias might have been underestimated. For this reason, the study endpoints regarding arrhythmic events were defined as either symptomatic ventricular arrhythmias requiring admission, ICD treatment for termination of ventricular fibrillation or sustained ventricular tachycardia or bradyarrhythmias requiring permanent pacemaker implantation. Third, LGE-CMR was performed for only half of the CS patients; therefore, the comparison between the 2 groups in terms of the extent of LGE would be underpowered. Fourth, although LVEF is one of the most important prognostic indicators in various heart diseases, it was not associated with clinical outcome in this study. The reason could be explained by the fact that the average LVEF in our population was 34.4%, which was lower than in a recent large-scale nationwide CS study (44.9%) showing the prognostic effect of decreased LVEF (<35%) at diagnosis on long-term clinical outcome.[38] This might be one of the reasons for the nonsignificant effect of LVEF on clinical outcomes in the current study. Finally, because of the retrospective nature of this study, there was a potential for sampling bias and incomplete data, and further prospective studies in larger populations are warranted.

Conclusions

The presence of basal IVS thinning at the time of CS diagnosis was associated with poor long-term clinical outcomes, particularly with respect to future ventricular tachyarrhythmias and heart failure requiring admission, suggesting that this characteristic has considerable prognostic significance in patients with CS.

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Disclosure

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