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ORIGINAL ARTICLE

Clinical Course and Outcomes in Patients with Left Ventricular Dysfunction Due to Myocardial Infarction After Kawasaki Disease

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Abstract

Long-term outcomes of patients with left ventricular dysfunction after Kawasaki disease (KD) remain unknown. The clinical course of 37 patients (29 males 8 females) with left ventricular ejection function (LVEF) less than 45% after KD since 1978 was retrospectively investigated. Cardiac events in this study were defined as admissions because of heart failure, fatal ventricular arrhythmias, heart transplantation, and death. Their ages when evaluated ranged from 1 to 70 years (median 35 years). The onset of KD ranged from 2 months to 20 years (median 21 months). All patients had bilateral coronary artery lesions, and multi-vessel occlusion occurred in 31 patients (84%). Previous myocardial infarction (MI) was found in 28 patients (76%). The interval from the onset of acute KD to the initial MI ranged from 15 days to 25 years (median 3 months). Reperfusion therapy was successful in 6 patients (30%), and coronary artery bypass grafting was performed in 23 patients (62%). Non-sustained ventricular tachycardia and fatal ventricular arrhythmias were detected in 11 patients and 22 patients, respectively. There were 15 deaths (41%). The 20-year and 50-year survival rates after KD were 84% (95% CI 67–92) and 54% (34–73), respectively ($n=37$). The 30-year cardiac event-free rate after the detection of low LVEF was 42% (95% CI 27–59). The cutoff point of the left ventricular end-diastolic dimension for cardiac events was 65 mm. Patients with low LVEF had fatal ventricular arrhythmias and a worsening of their ischemic cardiomyopathy after 30 years of age and their outcomes were poor.

Keywords Kawasaki disease · Fatal ventricular arrhythmia · Myocardial infarction · Left ventricular dysfunction · Left ventricular end-diastolic dimension

Introduction

In the 1970s and 1980s, about 20–25% of patients had coronary artery aneurysms after Kawasaki disease (KD) [1, 2]. Thrombotic occlusions in giant coronary aneurysms of the epicoronary arteries lead to severe and widespread myocardial involvement. Most patients with giant coronary aneurysms have coronary events such as myocardial infarction (MI) as children or adolescents [3]. The occurrence of MI is the most important factor in their prognosis of the patients with a history of KD [4]. Acute MI and asymptomatic occlusions of the major coronary arteries after KD affect the left ventricular ejection fraction (LVEF) [5]. A relationship between the degree of late gadolinium enhancement on magnetic resonance imaging and LVEF was found. We have reported that the outcome in patients after previous MI depends on their LVEF [6]. The clinical courses and long-term outcomes of patients with low LVEF due to KD in childhood are not always the same as that of adult patients with ischemic coronary artery disease due to atherosclerosis. The clinical courses and long-term outcomes of patients with low left ventricular function after KD in those with a longer-life span were investigated.

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Methods

The characteristics, cardiac events, management, and outcomes of patients with an LVEF of less than 45% due to coronary artery lesions after KD, who were followed up at the National Cerebral and Cardiovascular Center between 1978 and 2018, were retrospectively investigated from the medical records, selective coronary angiograms, and left ventriculograms. Their medical records included the findings on 12-lead electrocardiograms (ECGs), Holter ECGs, and two-dimensional echocardiography (2DE). The locations of MI were determined by ECG and selective coronary angiography. The LVEF was measured by Simpson's method using biplane methods with the left ventriculography. Pulmonary hypertension was confirmed in patients with mean pulmonary pressure of more than 20 mmHg by cardiac catheterization. The cardiac events in this study were defined as admission because of heart failure, fatal ventricular arrhythmias, implantable cardioverter defibrillator (ICD), left ventricular assist device (LVAD) implantation, heart transplantation, and death. The diagnosis of MI was based on the symptoms with ECG changes. The diagnoses of non-sustained ventricular tachycardia (NSVT), ventricular tachycardia (VT), and ventricular fibrillation (VF) were made by ECG or 24-h Holter ECG. Fatal ventricular arrhythmias in this study included VT and VF. The survival rate after the onset of KD, the survival rate after the detection of a low LVEF, the cardiac event-free rate, and the NSVT/VT-free survival rate were examined. Furthermore, the cutoff points for the maximum left ventricular end-diastolic dimension (LVDd), LVDd % of normal, left ventricular end-systolic dimension (LVDs), and fractional shortening (FS) on 2DE for cardiac events in 35 patients, excluding 2 patients who died at 1 year of age, were also analyzed [7]. The ethics committee of our institution approved this retrospective study (R19003-2).

Statistical Analysis

Statistical analysis was performed using JMP 10 software (SAS Institute Inc, Cary, NC). The measurements are expressed as medians and ranges. The cardiac event-free rate, survival rate, and NSVT/VT-free survival rate were calculated using the Kaplan–Meier method with 95% confidence intervals (CIs). The cutoff points of the maximum LVDd, LVDs, and FS on 2DE were analyzed using a receiver operating characteristic (ROC) curves. $p < 0.05$ was considered significant.

Results

Patient Population

Thirty-seven (29 males, 8 females) of 612 patients with coronary artery lesions were investigated (Table 1). Their age at present evaluation ranged from 1 to 70 years, (median 39 years). The age at onset of KD ranged from 2 months to 20 years (median 21 months). The number of patients with onset of KD per decade was as follows: 1960s, 4 patients (11%); 1970s, 13 patients (35%); 1980s, 13 patients (35%); 1990s, 3 patients (8%), and 2000s, 4 patients (11%). Using the Japanese criteria for KD, KD was diagnosed in 34 patients, with presumed KD in 3 patients. The three presumed KD patients were thought to have had acute KD in the 1960s. They had a history of persistent fever with some principal symptoms of acute KD and calcified giant aneurysms in the proximal portion of the major coronary arteries. Aspirin and intravenous immunoglobulin therapy with aspirin for acute KD were administered in 10 patients and 7 patients, respectively. Although 3 patients had massive immunoglobulin therapy in the 2000s, they were resistant to it. The treatment of 20 patients was unknown. LVEF was recognized as low and ranged from 19 to 44% (median 35%). The left ventricular end-diastolic volume (ml/m^2) was ranged from 77 to 442 (median 120), and the left ventricular end-systolic volume (ml/m^2) was ranged from 45 to 240 (median 77).

All patients had bilateral coronary artery lesions, with at least one-vessel occlusion. Bilateral giant aneurysms on coronary angiograms immediately after the acute phase and in the late period were confirmed in 35 patients (95%). Ten patients had three-vessel occlusions, and 21 patients had two-vessel occlusions. Six patients had one-vessel occlusion. The occluded coronary arteries were as follows: right coronary artery 33, left anterior descending artery 33, and left circumflex artery 12. Occlusion of the left main truncus was found in 6 patients. MI was found in 28 patients (82%). The number of patients with an initial MI per decade was as follows: 1970s, 7 patients; 1980s, 10 patients; 1990s, 7 patients; 2000s, 2 patients; and 2010s, 2 patients (Fig. 1 left). The interval from the onset of KD to the initial MI ranged from 15 days to 25 years (median 3 months) after the onset of KD (Fig. 1 right). The number of MIs was as follows: 1 in 18 patients; 2 in 8 patients; and 3 in 1 patient. The interval from the initial MI to the second MI ranged from 10 days to 30 years (median 7 years) after the onset of KD. A history of MI was unknown in 9 patients with acute KD in the 1960s and 1970s, but they had myocardial involvement with coronary artery occlusions. The locations of MIs were as follows: inferior, 7; anteroseptal 9; left main, 2; inferior and anteroseptal 7; anteroseptal and lateral, 1; inferior and

Table 1 Characteristics in patients with low left ventricular ejection fraction after Kawasaki disease

Patient	37
Male	29 (78%)
Median age at evaluation (range) years	35 (1–70)
Median age at the onset of KD (range)	21 months (2 months—20 years)
Number of the onset of acute KD per decade (patients)	
1960s, 1970s, 1980s, 1990s, 2000s	4, 13, 13, 3, 4
History of previous MI	28 (82%)
Number of the initial MI per decade (patients)	
1970s, 1980s, 1990s, 2000s	7, 10, 7, 4
Time of MI (patients)	
One	18
Repeat MI	9
Unknown	9
Location of MI	
Inferior	7
Anteroseptal	9
Left main	2
Inferior and anteroseptal	7
Others	3
Number of the initial MI per decade (patients)	
1970s, 1980s, 1980s, 1990s, 2000s	7, 10, 7, 4
Median interval from acute KD to the initial MI	3 months (15 days to 25 years)
Median interval from acute KD to time of low LVEF	4 months (15 days to 44 years)
Median LVEF (%)	35 (19–44)
Median LVEDVI (mL/m2)	120 (77–442)
Median LVESVI (mL/m2)	77 (45–240)
Coronary revascularization (patients)	
Coronary artery bypass grafting	23
Emergent	2
Elective	21
Percutaneous transluminal coronary intervention	
Percutaneous transluminal balloon angioplasty	1
Percutaneous transluminal coronary rotational ablation	2
Stent implantation	2
Ablation for VT	3
CRT-D, ICD, sICD	3, 2, 1
Death	15
Survival	22
NYHA I	20
NYHA II	2

KD Kawasaki disease, *MI* myocardial infarction, *LVEF* left ventricular ejection fraction, *LVEDVI* left ventricular end-diastolic volume index, *LVESVI* left ventricular end-systolic volume index, *CRT-D* cardiac resynchronization therapy with cardioverter defibrillator, *ICD* implantable cardioverter defibrillator, *sICD* sub-cutaneous implantable cardioverter defibrillator

lateral 1; and unknown 1. Coumadin and aspirin were prescribed in 7 patients before the initial MI. As reperfusion therapy for acute MI, thrombolysis was performed in 16 patients, percutaneous coronary artery balloon angioplasty in addition to thrombolysis was performed in 1 adult patient, and emergency coronary artery bypass grafting (CABG) was performed in 2 patients. The reperfusion therapy was successful in 6 patients (30%). During adulthood, the numbers of patients with atherosclerotic risk factors were as follows: history of smoking, 4 patients; obesity, 3 patients; and hypercholesterolemia, 4 patients.

CABG was performed in 23 patients (62%). Emergency CABG and elective CABG were performed in 2 and 21 patients, respectively. The number of grafts was as follows:

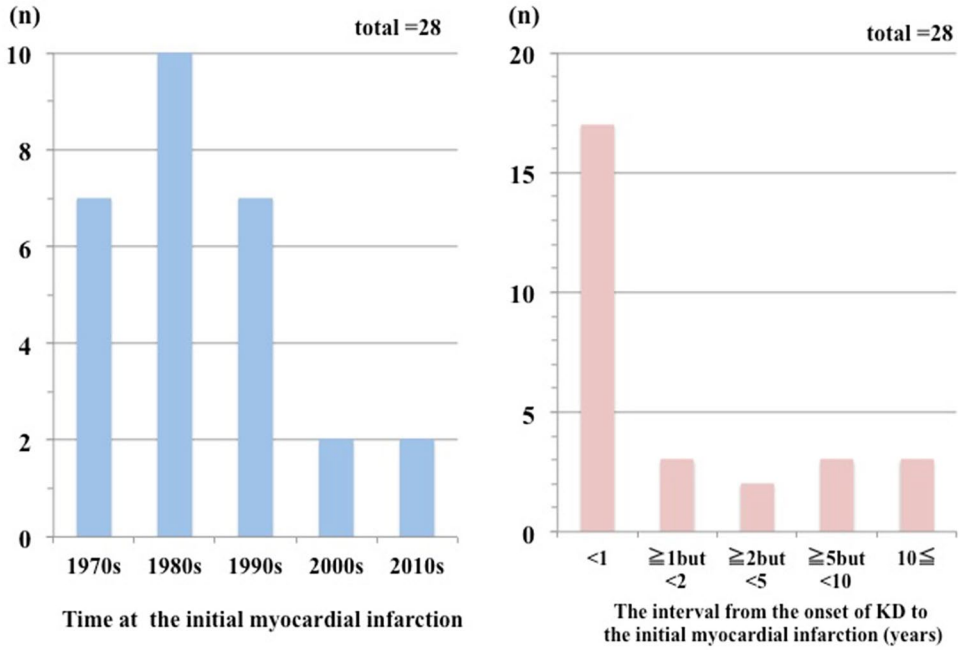

Fig. 1 Time at the initial MI and the interval from the onset of KD to the initial MI

1 vessel, 10 patients, 2 vessels, 9 patients, and 3 vessels, 4 patients. During the CABG, mitral valve replacement and mitral valve repair were performed in 2 patients and 1 patient, respectively. The age at operation ranged from 1 year 11 months to 57 years (median 11 years). The interval from the onset of acute KD to the operation ranged from 53 days to 43 years, (median 6 years; $n = 23$). The interval from the initial MI to the operation ranged from 0 day to 16 years (median 1 year; $n = 16$). Elective percutaneous transluminal coronary rotational atherectomy (PTCRA) was performed in 2 patients at 14 years of age. A stent was implanted for a complete total occlusion in 2 patients.

Antiplatelets and coumadin were administered to 33 patients and 20 patients, respectively. Beta-blockers, angiotensin-converting enzyme inhibitors, and amiodarone were prescribed in 26 patients, 26 patients, and 14 patients, respectively. LVAD implantation and heart transplantation were performed in 2 patients and 2 patients, respectively. NSVT and fatal ventricular arrhythmias were detected in 11 patients and 22 patients, respectively. Catheter ablation of VT was performed in 3 patients. Cardiac resynchronization therapy with cardioverter defibrillator (CRT-D) and ICD were performed in 3 and 2 patients, respectively, and sub-cutaneous ICD was implanted in 1 patient.

Worsening of ischemic cardiomyopathy and its management

Ten patients had been admitted for more than 2 months for heart failure immediately after acute MI. In the late period after a previous MI, 7 patients had a repeat admission for heart failure. They had intractable heart failure, and because of ischemic cardiomyopathy, their BNP increased to more than 200 pg/ml. Pulmonary hypertension due to low LVEF was found in 6 patients, and their left ventricular end-diastolic dimension (LVDd) on 2DE was greater than 70 mm in all 6 patients (Table 2). A 35-year-old man suddenly collapsed while working due to the severe re-stenosis of the left circumflex after PTCRA and he underwent an LVAD implantation after cardiopulmonary resuscitation (Fig. 2). Three patients underwent a CRT-D implantation and their LVEF improved. The maximum LVDd was greater than 80 mm in 3 patients with severe mitral regurgitation: one underwent mitral valve replacement, one underwent mitral valve repair, and one underwent heart transplantation.

Outcomes

Twenty-two patients (59%) survived, but two patients required cardiopulmonary resuscitation. The number of NYHA class I and II cases were 20 and 2, respectively. Fifteen patients (41%; 11 male, 4 female patients) died. Their ages at death ranged from 1 year 6 months to 70 years (median 22 years) (Fig. 3). The numbers of patients with onset of acute KD per decade were as follows: 1960s, 2 patients; 1970s, 6 patients; and 1980s, 6 patients. Their ages at death per decade were as follows: 1980s, 2 patients; 1990s, 5 patients; 2000s, 4 patients; and 2010s, 4 patients. The causes of death were as follows: sudden death, 8 patients; heart failure, 4 patients; death after heart transplantation, 2 patients: and subarachnoid hemorrhage, 1 patient (Fig. 3). The cause of the subarachnoid

Table 2 Characteristics and management of patients with worse of ischemic cardiomyopathy

Patient	1	2	3	4	5	6	7
Gender	Male	Male	Male	Male	Male	Female	Male
Age (years)	70	67	45	55	23	26	37
Birth on year	1947	1955	1968	1968	1976	1976	1982
Onset of KD (years)	14	20	5	0.3	4	1.6	3
Revascularization for coronary artery lesions							
Right coronary artery	Grafting	Grafting	Grafting	Grafting	Occlusion	Occlusion	SS
Left anterior descending artery	Grafting	Grafting*	Grafting	Grafting	Grafting	Grafting	Grafting
Left circumflex	Grafting			Stenting	SS		Stenting
Age (years) at detection of LVEF<45%	24	57	44	38	10	14	34
LVEDVI (ml/m2)	231	108	442	77	224	149	183
LVESVI (ml/m2)	183	65	240	54	147	101	138
Age (years)							
Coronary artery bypass grafting	26, 41	57	45 (with MVR)	38	10	16 (with MVP), 20	3
Percutaneous coronary intervention				46			14,15, 34
Detection time of PH	65	57	44	48	14	none	34
Device implantation	CRT-D, 65	ICD, 58		CRT-D,48			LVAD, 35
History of NYHA IV	65	58	44	48	17	14 (III)	35
Co-exits		Sarcoidosis	Atrial fibrillation		HT 19		
Outcome	Death	Survive	Death	Survive	Death	Death	Survive
Cause of death	Heart failure		Sudden death		Heart failure	Sudden death	

*The graft was occlusion, *KD* Kawasaki disease, *LVEDVI* left ventricular end-diastolic volume index, *LVESVI* left ventricular end-systolic volume index, *MVR* mitral valve replacement, *MVP* mitral valvuloplasty, *PH* pulmonary hypertension, *HT* heart transplantation, LVAD, left ventricular assist device

hemorrhage was unknown; he had had no known cerebral aneurysms before and had been taking aspirin and coumadin.

The 20-year, 40-year, and 50-year survival rates after KD were 84% (95% CI 67–92), 68% (53–84), and 54% (34–73), respectively ($n=37$) (Fig. 4, left). The 10-year and 30-year survival rates from the detection of a low LVEF were 70% (53–82) and 57% (38–74), respectively (Fig. 4, right). The 10-year, 20-year, and 30-year cardiac event-free rates after the detection of a low LVEF were 66% (95% CI 50–80), 51% (35–67), and 42% (27–59), respectively ($n=37$) (Fig. 5, left). The 10-year, 20-year, and 30-year-NSVT/VT-free survival rates from the initial MI were 79% (60–90), 32% (17–52), and 10% (3–31), respectively ($n=28$) (Fig. 5, right).

Cutoff Points of the Left Ventricular Diameters on 2DE for Cardiac Events

The cutoff point of the LVDd for NSVT was 58 mm (AUC, 0.980, $p<0.0001$, $n=35$) (Table 3) and that of LVDd % of normal was 123% (AUC, 0.976, $p<0.0001$). The cutoff points of the LVDs and FS for NSVT were 44.5 mm (AUC, 0.988, $p<0.0001$) and 24.1% (AUC, 0.986, $p<0.0008$), respectively. The cutoff point of the LVDd for cardiac events was 65 mm (AUC, 0.745, $p<0.01$, $n=35$) and that of LVDd % of normal was 142% (AUC, 0.736, $p=0.008$). The cutoff points of the LVDs and FS for cardiac events were 52 mm (AUC, 0.780, $p=0.0077$) and 23.6% (AUC, 0.750, $p=0.0383$), respectively. The cutoff point of the LVDd for pulmonary hypertension was 70 mm (AUC, 0.850, $p=0.0008$, $n=35$). The cutoff point of the LVDd % of normal was 147% (AUC, 0.842, $p=0.0026$). The cutoff points of the LVDs and FS for pulmonary hypertension were 56 mm (AUC, 0.924, $p=0.0005$) and 17% (AUC, 0.878, $p=0.0006$), respectively.

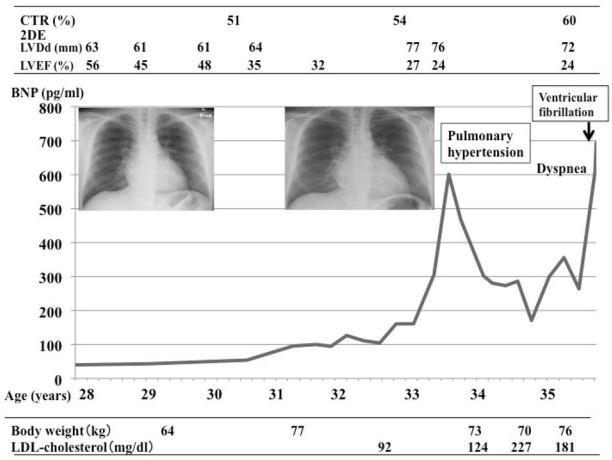

Fig. 2 Worsening of heart failure in patient 7. The patient had an anteroseptal myocardial infarction at the age of 3 years and he underwent coronary artery bypass grafting to the left anterior descending artery. He had an asymptomatic occlusion of the right coronary artery. He underwent percutaneous transluminal coronary rotational atherectomy (PTCRA) due to the severe stenosis of the left circumflex (LCX) at the ages of 14 years and 15 years. He had the coronary risk factors, including obesity and hypercholesterolemia. His obesity worsened his left ventricular function after 30 years of age. Pulmonary hypertension was suspected based on the peak velocity of tricuspid regurgitation on 2DE. He suddenly fell while working at the age of 35 years. His emergency coronary angiogram showed severe re-stenosis of the LCX. He had re-PTCRA and stent implantation for dissection of the LCX. However, his LVEF did not improve, and a left ventricular + assist device was implanted while he awaited heart transplantation (33)

Discussion

During the 1980's, some infants with bilateral giant aneurysms died within a year of the initial episode of KD because of heart failure after a previous MI. In the late 1990's and the 2000's, some unexpected sudden deaths occurred in patients with left ventricular dysfunction, more than 20 years after KD. Prior to their sudden deaths, they had no cardiac events for many years after a previous MI. Multifocal premature ventricular contractions and NSVT are probable risk factors in such patients [6, 8]. The extent score on ^{99m}Tc myocardial perfusion imaging, which indicates myocardial ischemia, and the interval from the onset of KD were the independent risk factors for the appearance of NSVT in this population [9]. In this study, 3 patients who had been diagnosed with presumed KD in adulthood had acute KD in the 1960s. The acute KD and coronary artery lesions were unknown in those days, the coronary artery lesions caused by KD and myocardial involvement persisted long into adulthood, and the disease was recognized by a low LVEF [10]. They were asymptomatic in adolescence, even with a low LVEF caused by coronary artery occlusion. Most patients with low LVEF were in NYHA class I. However, they became symptomatic in their 30 s and 40 s, because of worsening of ischemic cardiomyopathy and the appearance of fatal ventricular arrhythmias [11]. Unexpected sudden death in patients with a low LVEF can occur from their 20 s with aging [6]. It seems that the interval from a previous MI to the cardiac event such as fatal ventricular arrhythmia and worsening of ischemic cardiomyopathy in this population is longer than in older adult patients after MI due to atherosclerosis [5, 6, 9]. They may depend on the degree of myocardial involvement and preserved myocardial function except for the infarct lesion. Indeed, the function of preserved myocardium with aging would have an effect.

Until about the 1990s, antithrombotic therapy with coumadin and antiplatelet therapy were not necessarily administered to patients with giant aneurysms [4, 12]. Therefore, many patients with giant aneurysms often had acute MI immediately after KD, and they had a low LVEF, which greatly affected their outcomes. Although coumadin and aspirin therapy are very useful as an antithrombotic therapy, careful monitoring for major hemorrhagic complications must be performed in infants and older adult patients. Furthermore, the progressive stenosis also leads to coronary artery occlusion. Involvement of the coronary arterial wall has continued since the appearance of coronary artery aneurysms due to acute KD vasculitis [13–16]. Myocardial ischemia due to progressive coronary artery stenosis can cause a further decrease in the low LVEF with aging. Therefore, CABG has been performed as a useful coronary revascularization procedure to prevent myocardial involvement due to coronary artery occlusions [17]. Acute MI in the late period can occur not only in childhood but also in adulthood [18–20]. Atherosclerotic factors affect the coronary arteries after KD. Various coronary risk factors are added with aging [21, 22]. Ischemic cardiomyopathy can deteriorate more and more due to various coronary risk factors with aging. Pulmonary artery hypertension can occur gradually after many years, because of worsening of low LVEF, because patients with a very low LVEF and pulmonary artery hypertension had intractable heart failure, and they required repeated admissions for heart failure. Ultimately, fatal ventricular arrhythmias such as ventricular fibrillation can occur suddenly during the end stage. The cutoff values of LVDd for cardiac events on 2DE that were found in the present study are useful. If the LVDd is less than 60 mm, most patients are asymptomatic and stable. However, if the LVDd gradually increases beyond 65 mm, careful management is needed. To improve the low LVEF and prevent sudden death, a CRT-D can be considered.

The possibility of fatal ventricular arrhythmias leading to sudden death in patients with low LVEF is considered to be high. Reentry tachycardia between non-viable myocardium at the infarct area and normal myocardium can induce fatal ventricular arrhythmias in the late period

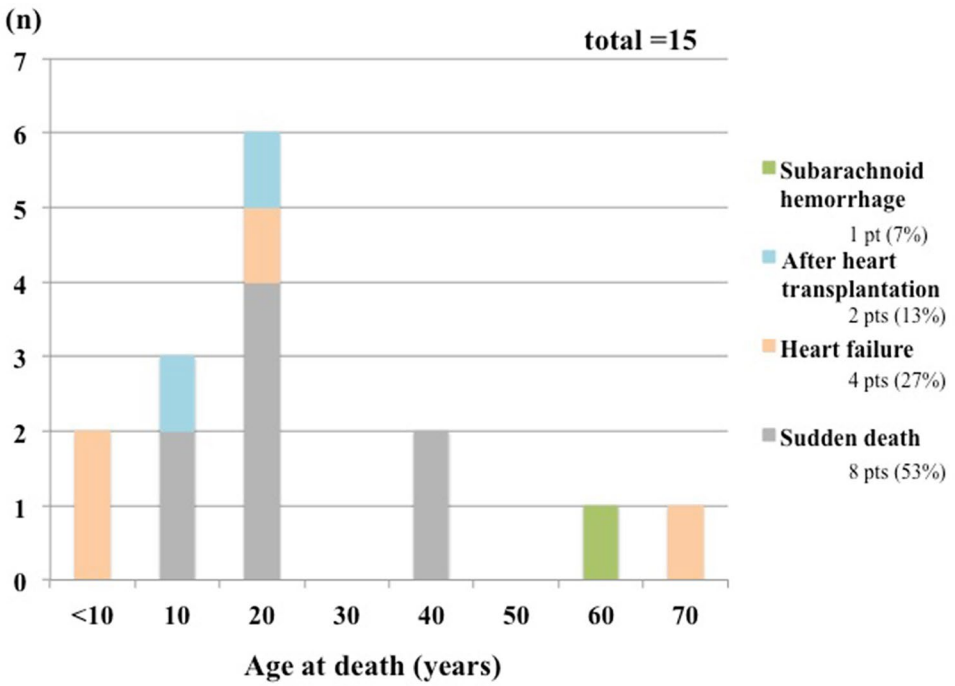

Fig. 3 Causes of death and age at death of patients with low LVEF

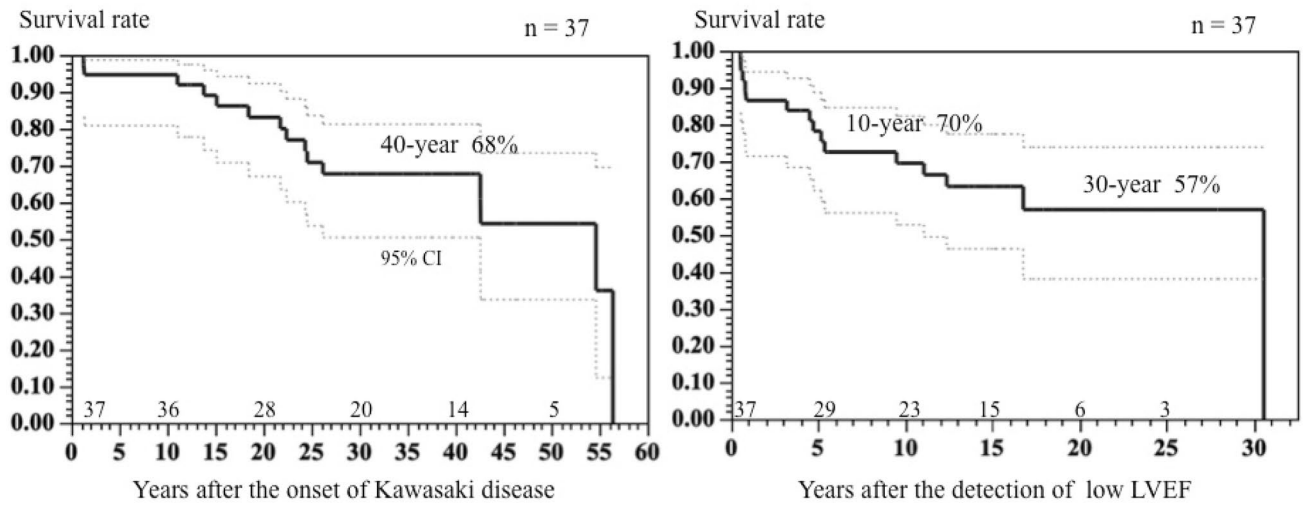

Fig. 4 Survival rate in patients with low LVEF. 95%CI, 95% confidence interval. (Left) Survival rate after the onset of KD in patients with a low LVEF. (Right) Survival rate after the detection of low LVEF in patients with a low LVEF

after a previous MI. In fact, the prevalence of fatal ventricular arrhythmias in this population remains unknown and it must be clarified in future. To prevent sudden death due to fatal ventricular arrhythmias, an ICD implantation and catheter ablation of VTs are recommended. Patients with an LVEF of less than 35% and NSVT would have an optimal indication for primary prevention [23, 24]. Furthermore, the indication for CRT in such patients should be considered. There are probably such patients, now in their 40 s and 50 s, at risk, and the cause of cardiac events in this group and their prevention are important issues. Symptoms caused by KD are rare and evidence of ischemia is often not present until the actual cardiac event occurs. Therefore, most such patients are likely to drop out from follow-up from adolescence. Patients with low LVEF should be educated about the need for careful follow-up.

Because cardiology was developing in the 1980's, the options for treatment and management of heart failure were

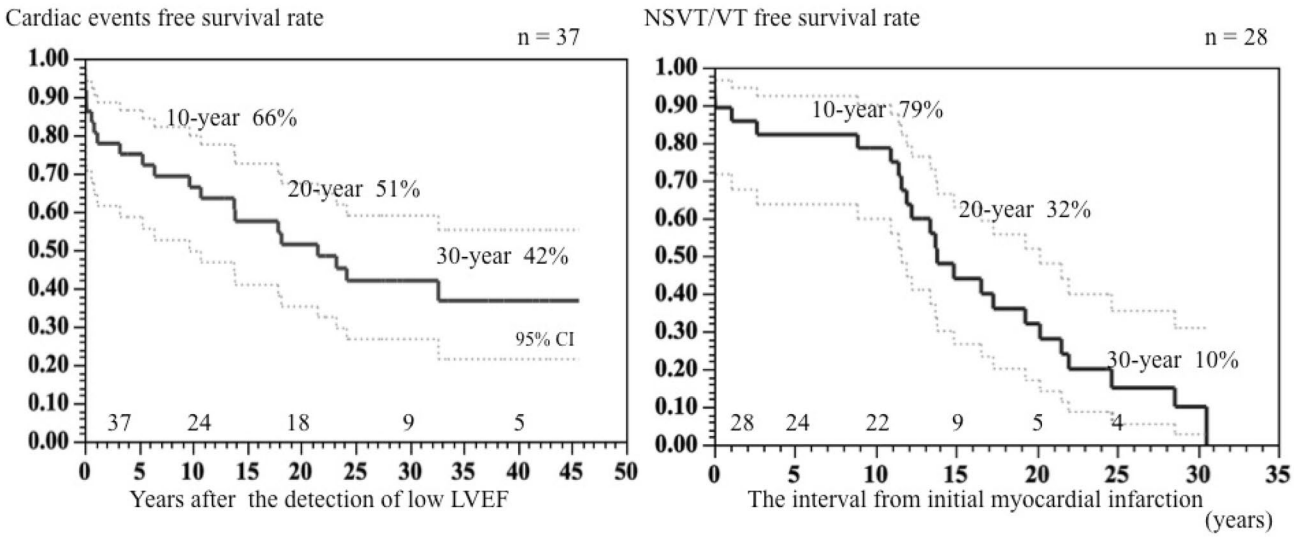

Fig. 5 (Left) Cardiac event-free rate from the detection of low LVEF and (Right) NSVT/VT-free rate after the initial myocardial infarction

Table 3 Cutoff points of the left ventricular diameters by two-dimensional echocardiography

	Cutoff value	AUC	p	Hazard ratio	95% CI	
NSVT						
Dd mm	58	0.980	< 0.0001	2.48	1.32	12.47
Dd (% of Normal)	123	0.976	< 0.0001	1.46	1.12	2.79
Ds mm	44.5	0.988	< 0.0001	2.70	1.32	55.48
FS (%)	24.1	0.944	0.0008	0.64	0.31	0.88
Cardiac events						
Dd mm	65	0.745	0.010	1.14	1.03	1.31
Dd (% of Normal)	142	0.736	0.008	1.46	1.12	2.79
Ds mm	52	0.780	0.008	1.11	1.02	1.23
FS (%)	23.6	0.750	0.038	0.90	0.80	0.995
Pulmonary hypertension						
Dd mm	70	0.850	0.0008	1.24	1.08	1.57
Dd (% of Normal)	147	0.842	0.0026	1.09	1.03	1.2
Ds mm	56	0.931	0.0001	1.27	1.1	1.58
FS (%)	17	0.878	0.0006	0.78	0.62	0.91

AUC area under the curve, *NSVT* non-sustained ventricular tachycardia, *Dd* end-diastolic dimension, *Ds* end-systolic dimension, *FS* fractional shortening

very limited. The number of patients in this study was small. The optimal treatment was not only not uniform, but it also varied in each patient depending on many factors. The treatment of coronary artery disease and ischemic cardiomyopathy has improved over the last 40 years. In particular, improvements in percutaneous coronary intervention and device treatment for fatal ventricular arrhythmias and intractable heart failure are remarkable. Coronary reperfusion and revascularization for reducing ischemia, anti-heart failure medications, and anti-arrhythmogenic therapy are mandatory. Therefore, reduction in the myocardial involvement by treatment would improve the survival rate and quality of life of patients in future.

Study Limitations

This study had a small number of patients and included patients from the 1970s up to the present. Because the treatment and the examinations for acute KD and coronary artery lesions have improved remarkably over the many years, this study had some limitations.

Conclusion

Patients with low LVEF had fatal ventricular arrhythmias and worsening of their ischemic cardiomyopathy worsened after 30 years of age, and their outcomes were poor. Therapy to treat myocardial involvement and fatal ventricular arrhythmias other than coronary artery revascularization is mandatory for patients with a longer-life span.

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Declarations

Conflict of interest The authors declare no conflict of interest.

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