Journal of Cardiography 15: 1027-1038, 1985

Idiopathic dilated right ventricular cardiomyopathy: A report of eight cases

Shinobu MATSUI
Eiji MURAKAMI
Noboru TAKEKOSHI
Sotoyuki TSUJI

Summary

From 1970 to 1983, eight patients who had severe right ventricular dilatation with little or no left ventricular abnormality were referred to our hospital. There were two men and six women, whose ages ranged from 16 to 38 years and averaged 27. Five patients were members of two families, which had definite hereditary histories of this disease. ECGs of seven patients suggested right ventricular abnormalities. Ventricular arrhythmias were documented in seven, whose ECGs suggested that the origins of the arrhythmias were in the right or both ventricles. Cardiothoracic ratios ranged from 0.50 to 0.66 and averaged 0.59. Five patients had wide respiratory splitting of the second heart sound. Echocardiography showed a marked increase in the right ventricular dimension. Right ventricular diastolic pressure was increased in three; left ventricular diastolic pressure was increased in two. Right ventricular angiography revealed marked dilatation and low ejection fractions of the right ventricle in all, but left ventricular function was nearly normal in them, except two patients who had minimal abnormality. The average follow-up period was 6.3 years and ranged from 3 months to 11 years. During the follow-up period, only two patients had ventricular tachycardia and three developed overt heart failure. Three patients died, two suddenly, and one from an attack of ventricular tachycardia. Autopsies of these two showed extreme dilatation of the right ventricle and one of them showed concentric hypertrophy of the left ventricle. Histological abnormalities were found in both ventricles. In summary, it is suggested that idiopathic dilated right ventricular cardiomyopathy can be classified in the spectrum of dilated cardiomyopathy.

Key words

Idiopathic dilated cardiomyopathy of the right ventricle Right ventricular dilatation Dilated cardiomyopathy Ventricular tachyarrhythmia Right heart failure

Introduction

Idiopathic dilated cardiomyopathy is characterized clinically by impaired systolic pump function, leading to cardiac enlargement with congestive heart failure, arrhythmias and emboli[1]. The main feature of this disease is enlargement of the left or both ventricles. There

Division of Cardiology, Department of Internal Medicine, Kanazawa Medical University, Uchinada-machi, Kahoku-gun, Ishikawa-ken, 920-02

Received for publication February 2, 1985; accepted April 20, 1985 (Ref. No. 29-25)

are few reports of idiopathic dilated cardiomyopathy, in which cardiac dilatation is limited to the right ventricle[2,3]. In 1974, we reported five patients with severe right ventricular dilatation with no or minimal left ventricular abnormality, and we designated these cases as "idiopathic right ventricular dilatation (IRVD)"[4]. From 1970 to 1983, eight patients with IRVD were referred to Kanazawa University and Kanazawa Medical University Hospital and were followed over several years. This is a report of these eight patients with IRVD in terms of its clinical features and the relationship with idiopathic dilated cardiomyopathy.

Subjects and methods

From 1970 to 1983, eight patients with severe right ventricular dilatation and with no or only minimal left ventricular abnormality or were referred to our hospital. They had no associated abnormalities such as systemic hypertension, valvular disease, congenital malformations of the heart or vessels, or intrinsic pulmonary parenchymal or vascular disease. The diagnosis of IRVD was made by excluding any other known diseases using right ventricular angiography, echocardiography, endomyocardial biopsy and/or autopsy. Seven of the eight patients had right heart catheterization and angiography. Left ventricular size was assessed during systole and diastole, either by right heart catheterization at the levophase after injection of radiopaque medium into the right ventricle or by left ventricular angiography. Ventricular volume and ejection fraction (EF) were calculated by the area-length method[5]. Six patients underwent selective coronary angiography. Two-dimensional and M-mode echocardiograms were obtained in six patients. The right and left ventricular end-diastolic dimensions were measured on the M-mode echocardiograms. In three patients, right ventricular endomyocardial biopsies were performed by Konno's method[6]. Autopsies were obtained in two patients.

Results

1. Clinical characteristics of IRVD

1) Age, sex, familial prevalence, symptoms and outcome (**Table 1**)

Two of the eight patients were men; the male/female ratio was 1/3. There were five patients in two families, which had hereditary histories of this disease. The mean age at the time of hospitalization was 27 years with a range from 16 to 38 years. At the time of admission, chief complaints were dyspnea on exertion, palpitation and/or generalized edema. Two patients were classified as class I; 4, as class II; and 2, as class III, according to the New York Heart Association functional class. The average follow-up period was 6.3 years with a range from 3 months to 11 years. Only three patients had clinical right ventricular heart failure. Three patients died, two suddenly, and one from an attack of ventricular tachycardia.

2) Electrocardiography (**Table 2**)

The most frequent abnormalities on the resting electrocardiograms were T wave inversions and premature ventricular complexes. All patients had sinus rhythm. One patient had first degree atrioventricular block. Four had right atrial overload. Seven had patterns of incomplete right bundle branch block. The QRS axis was normal (from $+50°$ to $+110°$) in all except one case who had right axis deviation. Ventricular arrhythmias were documented in seven patients whose electrocardiograms suggested the origin of the arrhythmias to be in the right or both ventricles. Only two patients had ventricular tachycardia during the follow-up period. During spontaneous ventricular tachycardia, the electrocardiograms showed a left bundle branch block pattern in one patient and a right bundle branch block pattern in another. The rates of during ventricular tachycardias were 167 and 176 bpm, respectively.

3) Chest radiography (**Table 3**)

The cardiothoracic ratio ranged from 0.50 to 0.66 with an average of 0.59. Generally, the configuration of the heart was globular. No patient had pulmonary congestion.

Table 1. Clinical profiles

Case	Sex	Age (yrs.) at Onset of symptoms	Age (yrs.) at Time of admission	Age (yrs.) at Follow-up	Symptoms at the time of admission	RV failure	Outcome	Comments
1.	F	13	16	6	Dyspnea on exertion NYHA II	Yes	Sudden death (22 yrs.)	
2.	F	35	35	9	Dyspnea on exertion NYHA II	No	Died of VF (44 yrs.)	Mother of case 1
3.	M	29	34	1 Mo	Dyspnea, palpitation NYHA III	No	Sudden death (34 yrs.)	Uncle of case 1
4.	F	22	22	11	Easy fatigability NYHA II	No	Alive	
5.	F		23	8	None NYHA I	No	Alive	Sister of case 4
6.	M	21	21	11	Easy fatigability NYHA II	No	Alive	
7.	F		25	5	None NYHA I	No	Alive	
8.	F	36	38	3 Mo	Dyspnea on exertion, edema NYHA III	Yes	Alive	

Abbreviations: M=male; F=female; yrs.=years; Mo=months; NYHA=New York Heart Association functional class; RV=right ventricular; VF=ventricular fibrillation.

Table 2. Electrocardiographic findings

Case	RAE	PR prolongation	QRS Axis	QRS Form	Inverted T in precordial leads		PVC Form		VT Age at onset	VT QRS axis	VT QRS form	VT Rate (beats/min)
1.	+	−	+110	V_1: rr′ V_{2-6}: broad s	V_{1-5}	+	LBBB	?				
2.	−	−	+63	V_1: rs (r=s) V_{1-6}: broad s	V_{1-6}	+	LBBB RBBB	+	40 yrs.	−63	LBBB	167
3.	+	−	+160	II, III, aVF: q V_1: rr′ V_6: rSr′s′	—	+	Undefined	?				
4.	−	−	+50	V_1: rs (r=s) V_{1-5}: broad s	V_{1-5}	+	LBBB RBBB	+	25 yrs.	+117	RBBB	176
5.	−	−	+65	WNL	V_{1-4}	+	LBBB	−				
6.	−	−	+100	V_1: Rs V_{1-6}: broad s	V_{1-4}	+	Undefined	−				
7.	+	+ (0.32 sec)	+90	V_1: rsr′s′	V_{1-4}	−		−				
8.	+	−	+75	V_1: rr′	V_{1-6}	+	LBBB RBBB	−				

Abbreviations: RAE=right atrial enlargement; PVC=premature ventricular contraction; VT=ventricular tachycardia; yrs.=years, LBBB=left bundle branch block; RBBB=right bundle branch block; WNL=within normal limits.

Table 3. Chest radiographic, phonocardiographic and echocardiographic findings

Case	X-ray CTR	Heart sounds and murmurs	Echo		
			RVDd (mm)	LVDd (mm)	RVDd/LVDd
1.	0.56	S2 : NFS S3	64	34	1.88
2.	0.61	S2 : NFS EM	30	40	0.75
3.	0.63	S2 : NFS S4	—	—	—
4.	0.55	S2 : NFS S4	30	36	0.83
5.	0.50	WNL	24	38	0.63
6.	0.60	S3	—	—	—
7.	0.59	S2 : NFS EM	34	32	1.06
8.	0.66	S3	62	36	1.72

Abbreviations: CTR=cardiothoracic ratio; NFS=non-fixed splitting; EM=ejection murmur; WNL=within normal limits; RVDd=right ventricular end-diastolic dimension; LVDd=left ventricular end-diastolic dimension.

Table 4. Cardiac catheterization and angiographic data

Case	Pressure (mmHg)					Volume and EF			Coronary angiogram
	PCW	PA	RV	RA	LV	RVEDV (m*l*)	RVEF (%)	LVEF (%)	
1.	—	17/4	16/6	4	100/7	486	17	48	Normal
2.	8	25/8	24/5	3	117/8	351	13	74	Normal
3.	—	25/—	24/—	8	—	—	—	—	—
4.	9	26/11	25/11	5	—	281	22	65	Normal
5.	—	—	—	—	—	—	—	—	—
6.	9	23/10	25/8	9	—	454	21	67	Normal
7.	6	26/13	29/11	6	140/16	422	47	70	Normal
8.	20	23/15	20/15	12	111/13	380	28	45	Normal

Abbreviations: PCW=pulmonary capillary wedge; PA=pulmonary artery; RV=right ventricle; RA=right atrium; LV=left ventricle; EF=ejection fraction; EDV=end-diastolic volume.

4) Phonocardiography (**Table 3**)

Five patients showed respiratory splitting of the second heart sound. The third or fourth heart sound was recorded in five patients. A grade I~II/VI ejection murmur was recorded in two patients.

5) Echocardiography (**Table 3**)

M-mode echocardiography was performed in six patients and showed increases in right ventricular end-diastolic dimensions in all patients. Left ventricular dimension was within normal range in all patients. The mean RVDd/LVDd ratio was 1.15 (range 0.63–1.88), where RVDd was right ventricular end-diastolic dimension and LVDd was left ventricular end-diastolic dimension. One patient (case 8) had mild hypokinesis of the left ventricular posterior wall.

6) Cardiac catheterization (**Table 4**)

Cardiac catheterization was performed in seven patients. Right atrial and ventricular diastolic

Table 5. Endomyocardial biopsy and autopsy findings

Case	RV endomyocardial biopsy	Autopsy	
		RV	LV
1.		Macroscopic: Marked dilatation Abundant subepicardial and myocardial fatty metamorphosis Marked decrease in myocardial muscle cells Microscopic: Thickened endocardium Subepicardial fibrosis Degeneration of myocytes with dystrophic and pyknotic nuclei	Macroscopic: Hypertrophy of the wall Patchy fibrosis Abundant subepicardial fat Microscopic: Degeneration of myocytes Interstitial fibrosis of myocardium
2.	(at age 35 years) Mild endocardial edema Mild degeneration of myocytes Increase in interstitial connective tissue of myocardium	(at age 44 years) Macroscopic: Marked dilatation Abundant subepicardial and myocardial fatty metamorphosis Microscopic: Thickened endocardium Interstitial fibrosis of myocardium Degeneration of myocytes with dystrophic and pyknotic nuclei	Macroscopic: Marked hypertrophy of the wall Abundant subepicardial fat Microscopic: Thickened endocardium Pyknotic nuclei in myocytes Interstitial fibrosis of myocardium
4.	Normal myocardium		
6.	Normal myocardium		

pressures were elevated in three patients. Pulmonary arterial pressure was normal in all patients. Left ventricular end-diastolic pressure was slightly elevated in two patients.

7) Angiography (**Table 4**)

Angiography was performed in six patients. Right ventricular enlargement and a decrease in ejection fractions were evident in all patients. Left ventricular ejection fraction was slightly decreased in two of six patients. No patients had significant stenosis ($\geqq 50\%$) of their coronary arteries.

8) Endomyocardial biopsy and autopsy findings (**Table 5, Fig. 1**)

Right ventricular endomyocardial biopsy was performed in three patients. Two of them showed normal myocardium, and one patient showed mild endocardial edema, mild degeneration of myocytes and an increase in interstitial connective tissues.

Autopsy was obtained in two patients. The right ventricular wall was covered with abundant subepicardial fat. Macroscopically, the right ventricular cavity was markedly enlarged. The endocardium appeared white and thickened. The macroscopic findings in the left ventricle revealed an increase in the wall thickness and fatty infiltration. Microscopic examination of the right ventricular myocardium showed fatty infiltration in the interstitial tissue and degeneration of the few remaining myocytes with dystrophic and piknotic nuclei. There was a marked decrease in myocardial muscle cells and the myocardium was replaced by fatty tissue. Micro-

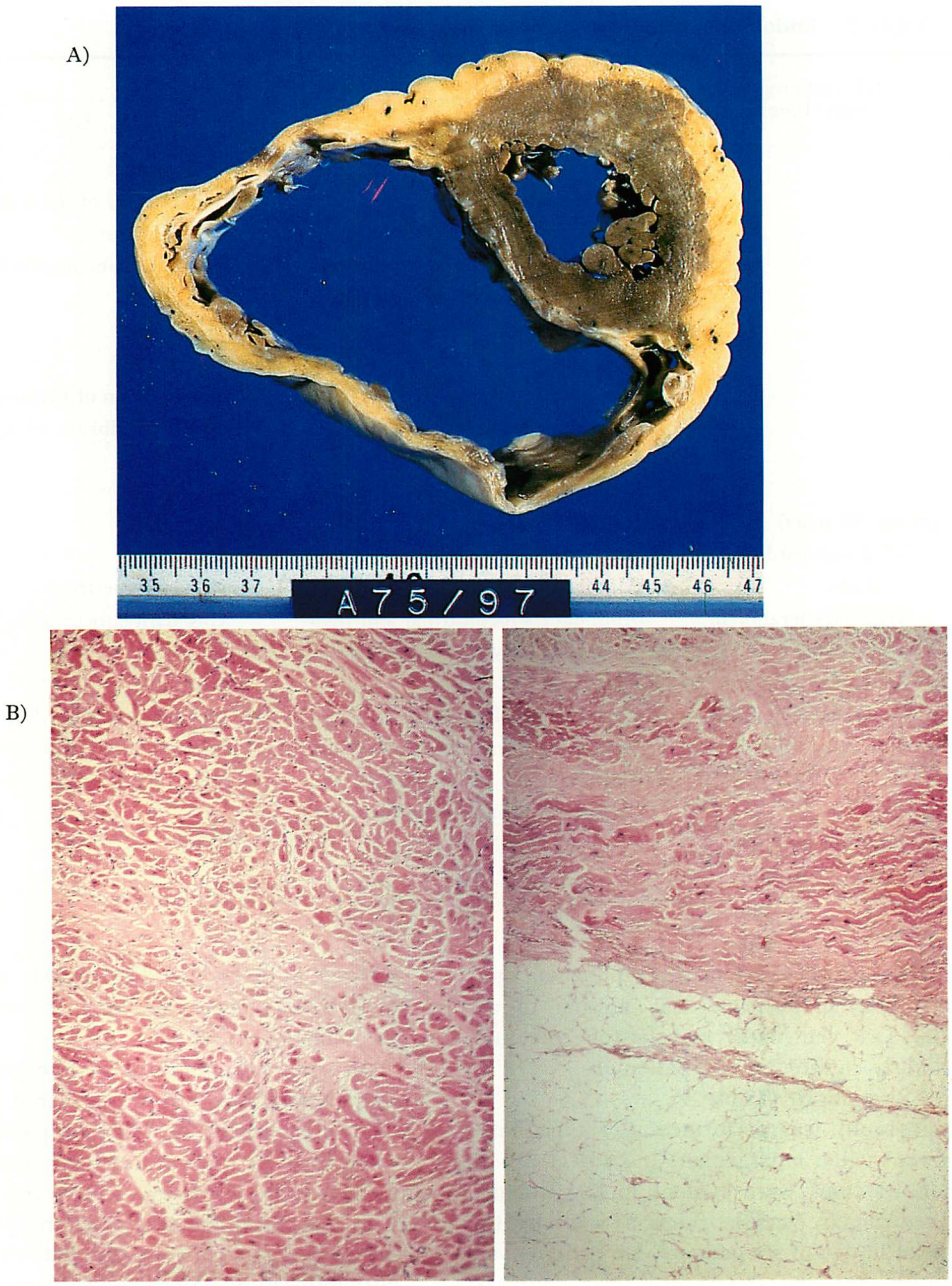


Fig. 1. Autopsy findings (Case 1).

A: Transverse section of the heart shows enlarged cavity and thin wall with fatty metamorphosis in the right ventricle. In the left ventricle, a hypertrophic ventricular wall, patchy fibrosis and abundant subepicardial fat are seen.

B: Histological section of the right ventricular myocardium (left) shows degeneration of myocytes, interstitial fibrosis and fatty infiltration, and that of the left ventricular myocardium (right) shows degeneration of myocytes and interstitial fibrosis. H & E stains.

scopically, degeneration of myocytes and interstitial fibrosis were observed in the left ventricular wall.

2. Case presentation

A 21-year-old woman (Case 1) was referred to Kanazawa Medical University Hospital for evaluation and treatment of pretibial edema and dyspnea. She had noted exertional dyspnea since 13 years of age. At the age of 16, she was first noted to have marked cardiomegaly. Soon thereafter, she underwent cardiac catheterization which revealed possibile IRVD. Since that time, she had several episodes of right heart failure. She was referred to Kanazawa Medical University Hospital for the first time on September 22, 1972 for reevaluation and treatment of congestive heart failure. Her mother died of ventricular tachycardia at 44 years of age (Case 2) and one of her uncles died suddenly in 1965 at the age of 34 (Case 3).

Physical examination revealed a slightly obese woman with an irregular heart rate of 71 bpm and blood pressure of 110/80 mmHg. Jugular venous pressure was elevated. Her chest was clear. The apical impulse was diffuse without heaves or thrills. The second heart sound was split and appeared to vary with respiration. No systolic murmur was audible. A third heart sound was audible. The liver was palpable 3-fingers-breadths below the right costal margin. There was pretibial edema.

The 12-lead ECG revealed sinus rhythm, right axis deviation, rsr' pattern in V_1, low voltage and inverted T waves in V_1–V_5 (**Fig. 2a**). Post-stress ECG showed frequent PVCs. Chest radiography showed a markedly enlarged globular shaped heart (**Fig. 2b**). She underwent cardiac catheterization and angiography. Pressure measurements in both ventricles were normal. The right ventricle was enlarged and contracted poorly (RVEF=17%). LVEF was slightly decreased (**Table 4, Fig. 2c**). The coronary arteries were normal. She died suddenly on December 16, 1975. A postmorten examination was performed eight hours after death. The results of autopsy are shown in **Table 5** and illustrated in **Fig. 1**.

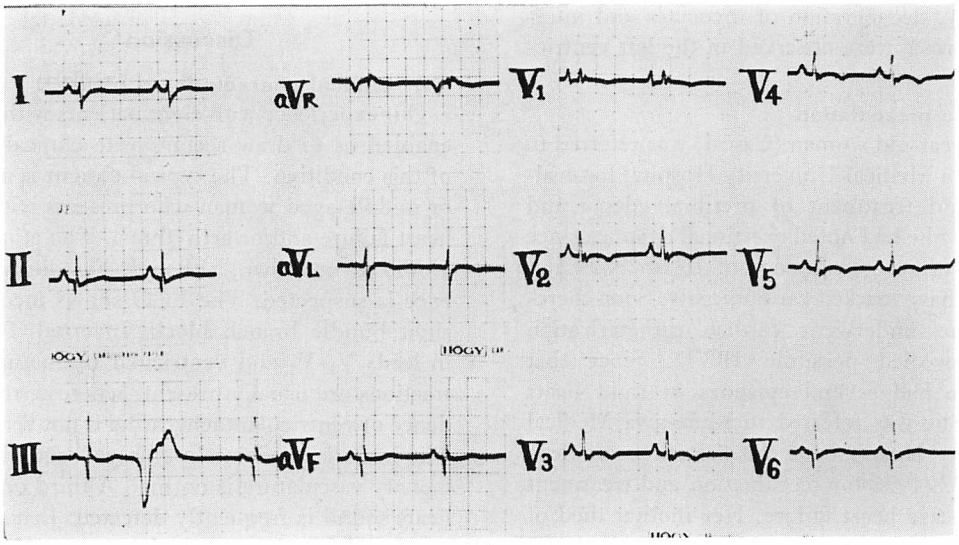
Discussion

1. Clinical characteristics of IRVD

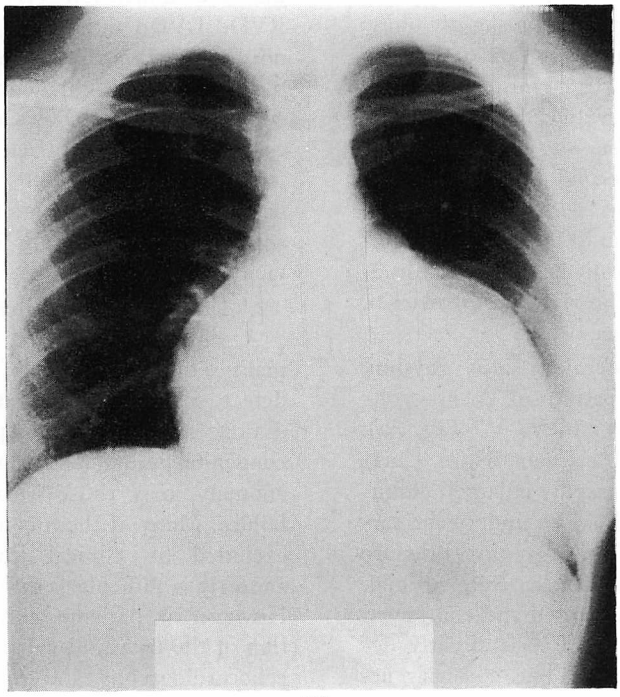
Our experience with eight patients with IRVD enabled us to draw a composite clinical profile of this condition. The typical patient is a young or middle-aged woman who presents with right heart failure and/or arrhythmia. The etiology of IRVD is unknown, although hereditary influence is suspected. The ECG shows incomplete right bundle branch block. Inverted T waves in leads V_1–V_4 and ventricular premature contractions are usually present; whereas, the incidence of ventricular tachycardia is not very high. The heart is moderately enlarged and the pulmonary vasculature is normal. A third or fourth heart sound is frequently detected. Echocardiography shows increased right ventricular diastolic dimension (RVDd). The left ventricular diastolic dimension (LVDd) is normal and RVDd/LVDd ratio is markedly increased over normal. Intracardiac pressures are nearly normal except for right ventricular diastolic pressure. Angiography shows an enlarged and poorly contracting right ventricle. There is only minimal abnormality of left ventricular contraction or none. The prognosis of this disease is grave and the cause of death is usually ventricular tachyarrhythmia.

2. Differential diagnosis of IRVD

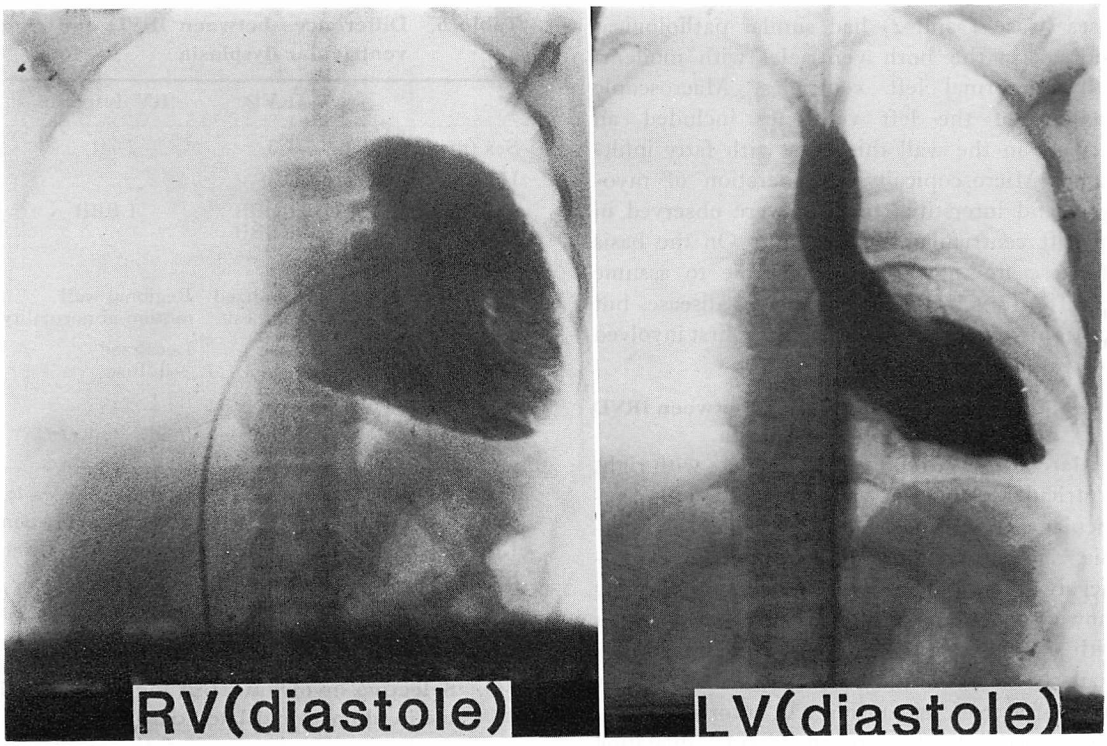
A variety of acquired or congenital abnormalities of the right heart, including atrial septal defect, partial anomalous pulmonary venous drainage, isolated tricuspid chordal rupture, congenital pulmonary regurgitation, and Ebstein's anomaly, may cause isolated right ventricular failure. Many of these conditions can be readily excluded by clinical investigation. Ebstein's anomaly is difficult to differentiate from IRVD. However, in patients with IRVD normal insertion of the tricuspid valve can be indentified by echocardiography and right ventricular angiography; furthermore, intracardiac ECG does not demonstrate an atrialized portion of the right ventricle. In Uhl's anomaly, the predominant feature is a very thin-walled, enlarged right ventricle[7]. Therefore, IRVD can be differentiated



(A)



(B)



(C)

Fig. 2. Exemplary IRVD patient (Case 1).

A: Electrocardiogram shows right axis deviation, rsr' pattern in V_1, low voltage and inverted T waves in V_1–V_5 (1 cm=1 mV).

B: Chest radiograph shows the markedly enlarged heart.

C: Angiogram shows the enlarged right ventricular cavity (left) and normal left ventricular cavity (right) in end-diastole.

from this condition. Uhl's anomaly results in cyanosis and right heart failure during infancy. The ECG usually shows a lack of right ventricular force. Pathologically, there is complete absence of the right ventricular myocardium, but sections from the left side of the heart show no special features. Adult cases of Uhl's anomaly complicated by ventricular tachycardia have rarely been reported. In our series of IRVD, symptoms appeared in adolescence or adulthood, and the right ventricular wall thickness measured by right ventricular angiography ranged from 1.5 to 4 mm. ECGs showed right ventricular hypertrophy due to diastolic overload. Right ventricular endomyocardial biopsy (Case 2, 4 and 6) was normal or showed only minimal changes of the right ventricular myocardium. Autopsy (Case 1 and 2) showed abundant subepicardial and myocardial metamorphosis, decreased myocardial muscle cells in the right ventricular wall, and pathologic left ventricular involvement with thickened wall, fatty infiltration and degeneration of myocardium. These findings made Uhl's anomaly unlikely in the present cases.

3. Left ventricular involvement in IRVD

Most patients reported in the present study had severe right ventricular dilatation with relatively good left ventricular function. However, two patients (Case 1 and 8) had mildly depressed left ventricular function, and two autopsied

cases (Case 1 and 2) had similar pathological findings in the both ventricles with moderatedly abnormal left ventricles. Macroscopic findings of the left ventricles included an increase in the wall thickness with fatty infiltration. Microscopically, degeneration of myocytes and interstitial fibrosis were observed in the left ventricular myocardium. On the basis of these findings, it is reasonable to assume that IRVD is a diffuse myocardial disease, but the right ventricular myocardium is first involved for reasons not yet clear.

4. Similarities and differences between IRVD and right ventricular dysplasia

Marcus et al[9]. reported 24 patients with right ventricular dysplasia who presented with ventricular tachycardia, supraventricular arrhythmia, right heart failure or asymptomatic cardiomegaly. The right ventricular angiogram usually showed an enlarged right ventricular chamber with segmental wall motion abnormalities, particularly in the infundibulum, which may show areas of systolic expansion. At autopsy or surgery apparently discrete aneurysmal dilatation was noted in the infundibulum, apex and inferior wall of the right ventricle.

Microscopic examination of the tissue from the area of involvement showed infiltration of the interstitial tissue by fat and hypertrophy or degeneration of the few remaining myocytes[10]. Right ventricular dysplasia is similar to IRVD in clinical profile, but there are notable differences between these conditions, such as sex predominance, hereditary influence, QRS configurations of premature ventricular contractions, ventricular tachyarrhythmias, right ventricular wall motion abnormalities, and right and left ventricular involvement (**Table 6**)[11]. Furthermore, the term " dysplasia " indicates abnormality of development; pathologically, alteration in size, shape and organization of adult cells. However, " cardiomyopathy " indicates a disorder that directly affects the myocardium of one or both ventricles in a diffuse or multifocal fashion. Based on its angiography, endomyocardial biopsy and autopsy findings, it is thought that IRVD can be classified as idiopathic dilated cardiomyopathy.

Table 6. Differences between IRVD and right ventricular dysplasia

	IRVD	RV dysplasia
Sex (male/female)	1/3	2.7/1
Hereditary influence	++	±
Configuration of PVCs	LBBB RBBB	LBBB
Incidence of VT	+	++
RV wall motion	Generalized hypokinesis	Regional wall motion abnormality
RV involvement (pathological)	Diffuse	Localized ~diffuse
LV involvement (pathological and functional)	+	±

Abbreviations: RV=right ventricular; LV=left ventricular; PVC=premature ventricular contraction; LBBB=left bundle branch block; RBBB=right bundle branch block; VT=ventricular tachycardia.

5. Selective involvement of the right ventricle in idiopathic dilated cardiomyopathy

The selective involvement of the right ventricle in the patients with idopathic dilated cardiomyopathy is rare, but has been reported by several authors[2,3,11,12]. Viola et al[11]. first described a case of idiopathic myocardopathy with failure of right ventricular contractility. However, their case showed elevated left ventricular end-diastolic pressure (=14 mmHg) and pathologic changes in the left ventricular myocardium. Kreulaen et al.[12] reported two patients with primary myocardial disease who had clinical evidence of right heart failure. In one case there was no pulmonary hypertension, but this patient also showed severe left ventricular dysfunction and asynergy. Hatle et al[2]. reported four patients with chronic myocardial disease who had predominantly right ventricular involvement. Signs of left ventricular disease could not be detected in one of these cases. Recently, Fitchett et al[3]. reported 14 patients with predominantly right-sided dilated cardiomyopathy. The clinical and pathological data of their patients are very similar to those in the present study except for the sex predominance

and an inflammatory cellular infiltration which was not found in our series.

Conclusions

In most patients with idiopathic dilated cardiomyopathy, functional and pathological abnormalities are more pronounced in the left ventricle than in the right ventricle. However, IRVD is a pathologic condition in which the right ventricular myocardium is replaced by fatty and fibrous tissue at first. Based on the pathologic and functional left ventricular involvement, it is suggested that IRVD can be classified as one in the spectrum of idiopathic dilated cardiomyopathies. Therefore, it is appropriate to designate IRVD as " idiopathic dilated right ventricular cardiomyopathy (IDRVC) ". The etiology, clinical course and treatment of IDRVC remain obscure, and further studies to clarify these problems are mandatory as more cases accumulate.

特発性拡張型右室心筋症

金沢医科大学　循環器内科
松井　忍，村上映二，竹越　襄，
辻　外幸

1974年，一次的に右室の著明な拡張を示す症例を特発性右室拡張症 (idiopathic right ventricular dilatation: IRVD) と命名し報告して以来，同様の症例を8例経験し，かつ，長期間観察し得たので，その臨床的特徴ならびに心筋生検・剖検所見より，本症の clinical entity としての位置づけを検討した．

臨床的特徴：症例は16歳から38歳までの8症例 (男性2例，女性6例) である．8例中3例と2例は，おのおの同一家系内にあった．心電図では7例に心室性期外収縮が認められた．QRS 波は一般に低電位傾向，右室拡張期負荷所見を示し，T 波は右室胸部誘導で陰転していた．肺動脈圧には異常なく，右室内腔の拡大ならびに駆出率の低下が著しかった．左室駆出率は2例で軽度の低下を示した．1～12年の経過観察中，3例に心室頻拍発作，3例に右心不全徴候を認めた．同一家系内の3例が突然死を来たした．

心筋生検ならびに剖検所見：心筋生検を行った3例では，1例に軽度の変化をみるのみであった．突然死を来たした3例中2例の剖検では，右室心筋脱落・変性ならびに線維化が著しかった．左室心筋には軽度ないし中等度の線維化が認められた．

結論：以上の成績より本症を遺伝的素因の強い，一次的に右室心筋をび慢性に侵す心筋症と考えた．また左室心筋にも軽度ないし中等度の変性が認められることから，拡張型心筋症の一亜型とも考えられる．したがって本症を特発性拡張型右室心筋症 (Idiopathic dilated right ventricular cardiomyopathy: IDRVC) と命名するのが妥当と考えた．Right ventricular dysplasia とは，臨床的特徴，剖検所見より若干異なる clinical entity である可能性が示唆された．

References

1) Johnson RA, Palucious I: Dilated cardiomyopathies of the adult. New Engl J Med **307**: 1051–1058, 1982
2) Hatle L, Stake G, Storstein O: Chronic myocardial disease. Acta Med Scand **199**: 407–411, 1976
3) Fitchett DH, Sugrue DD, MacArthar CG, Oakley CM: Right ventricular dilated cardiomyopathy. Br Heart J **51**: 25–29, 1984
4) Funazu T, Kawato M, Takekoshi N, Murakami E, Kitagawa M, Murakami M: Five cases of idiopathic right ventricular dilatation. Heart **6**: 1176–1184, 1974 (in Japanese)
5) Arcilla RA, Tsai P, Thilenius O, Ranninger K: Angiographic method for volume estimation of right and left ventricles. Chest **60**: 446–454, 1971
6) Konno S, Sakakibara S: Endo-myocardial biopsy. Dis chest **44**: 345–350, 1963
7) Uhl HSM: A previously undescribed congenital malformation of the heart: Almost total absence of the myocardium of the right ventricle. Bull Johns Hopkins Hosp **91**: 197–209, 1952
8) Vecht RJ, Carmichael DJS, Gopal R, Philip G: Uhl's anomaly. Br Heart J **41**: 676–682, 1979
9) Marcus FI, Fontaine GH, Guiraudon G, Frank R, Laurenceau JL, Malergue C, Grosgogeat Y:

Right ventricular dysplasia: A report of 24 adult cases. Circulation **65**: 384–398, 1982
10) Manyari DE, Klein G, Gulamhusein S, Boughner D, Guirauson GM, Wyse G, Mitchell LB, Kostuk W: Arrhythmogenic right ventricular dysplasia: A generalized cardiomyopathy? Circulation **68**: 12–5257, 1983
11) Viola AR, Adaro FVM, Roncoroni AJ: Idiopathic myocardiopathy resulting in failure of the right ventricle. Am J Med **48**: 235–238, 1970
12) Kreulaen TH, Gorlin R, Herman MV: Ventriculographic patterns and hemodynamics in primary myocardial disease. Circulation **47**: 299–308, 1973

訂　　正

1032頁　Fig. 1 B

図説の (left) と (right) を入れ換える

1302頁　右4行　Hb 27.0 g/al → dl

1305頁　右7行　左心室左 → 左心室圧

Correction

P. 1032 : Legend of Fig. 1 B

(left) and (right) should be switched.