Myocardial Scintigraphy Using Iodine-123 15-(*p*-Iodophenyl)-3-R, S-Methylpentadecanoic Acid Predicts the Response to Beta-blocker Therapy in Patients With Dilated Cardiomyopathy but Does Not Reflect Therapeutic Effect

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Abstract

Myocardial fatty acid metabolism is disturbed in patients with idiopathic dilated cardiomyopathy.

Myocardial scintigraphy using iodine-123 15-(*p*-iodophenyl)-3-R, S-methylpentadecanoic acid(BMIPP) was used to assess the response to β-blocker therapy in 19 patients with dilated cardiomyopathy.

BMIPP myocardial scintigraphy was performed before and 6 months after initiating β-blocker therapy with metoprolol. Cardiac BMIPP uptake was assessed as the total defect score(TDS) and heart-to-mediastinum activity(H/M) ratio. Patients were classified retrospectively as responders with an improvement of at least one functional class(New York Heart Association) or an increase in ejection fraction of ≥ 0.10 at 6 months, or as nonresponders meeting neither criterion.

Responders had a significantly better pretreatment TDS($p < 0.005$) and H/M ratio($p < 0.0001$) than nonresponders. TDS exhibited no significant changes over 6 months in either group(responders: 13.2 ± 3.7 vs 12.5 ± 3.3; nonresponders: 20.8 ± 6.5 vs 20.5 ± 3.0). Responders showed no significant changes in H/M ratio(2.47 ± 0.28 vs 2.43 ± 0.42); paradoxically, nonresponders showed a significant increase from 1.82 ± 0.11 to 2.10 ± 0.19($p < 0.05$), suggesting that β-blocker therapy protected the myocardial fatty acid metabolism even in the absence of clinical improvement.

BMIPP myocardial scintigraphy provides a prediction of response to β-blocker treatment, but does not reflect the therapeutic effect in responders at 6 months.

J Cardiol 2000; 35(5): 343–351

Key Words
- Cardiomyopathies, dilated   ■ Beta-adrenergic receptor blockers
- Radionuclide imaging(^{123}I-BMIPP)

INTRODUCTION

The efficacy of β-blocker therapy for dilated cardiomyopathy(DCM)has been established[1-8].

Recently, myocardial scintigraphy has been assessed for predicting and evaluating treatment effects, but at present no radionuclide has become established in clinical use for the prediction and

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Manuscript received October 4, 1999; revised January 13, 2000; accepted January 14, 2000

evaluation of treatment effects[9-12]. Metaiodoben-zylguanidine (MIBG) myocardial scintigraphy can predict and evaluate the effect of β-blocker[9,10]. Previously, we reported that iodine-123 15-(p-iodophenyl)-3-R, S-methylpentadecanoic acid (BMIPP) myocardial scintigraphy can predict the response to β-blocker therapy in patients with DCM[12].

The present study was carried out to determine whether the therapeutic effect of β-blockers on DCM can be evaluated by BMIPP myocardial scintigraphy.

SUBJECTS AND METHODS

Patients

Nineteen patients (10 men and 9 women) with DCM aged from 29 to 79 years (mean age 58 years) were included in this study. All patients had undergone coronary angiography which had shown no significant arterial lesions.

Metoprolol administration

The day after initial BMIPP myocardial scintigraphy, administration of metoprolol tartrate was initiated at a dose of 5 mg/day, gradually increasing at 7-day intervals to the maintenance dose of 20 mg/day.

BMIPP myocardial scintigraphy

BMIPP myocardial scintigraphy was performed by single photon emission computed tomography (SPECT) before treatment and again 6 months after starting β-blocker therapy.

SPECT was begun 15 min after intravenous injection of 111 MBq of iodine-123 BMIPP (Nihon Mediphysics, purity exceeding 95%) with the patient at rest in the morning following overnight fasting. All 19 patients were at rest when they underwent BMIPP SPECT using a rotating gamma camera equipped with a low-energy, general-purpose collimator (Starcam 4000 iXR/T: General Electric). The SPECT image sequences consisted of 32 projections with a 64 × 64 matrix acquired for 30 sec over a 180° circular orbit, from the left posterior oblique (45°) to the right anterior oblique (45°) directions. The projection images were processed with a low-pass (Butterworth) filter. Standard back projection with a ramp filter was performed. No overlap occurred between the left lobe of the liver and the heart in the BMIPP SPECT images and no attenuation correction was applied.
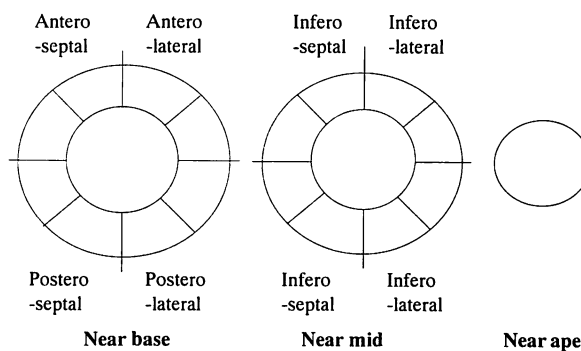

Fig. 1 Diagram of segments used for measurement of regional iodine-123 BMIPP uptake
BMIPP = 15-(p-iodophenyl)-3-R, S-methylpentade-canoic acid.

Total defect score

The myocardial SPECT images were divided into 17 segments (**Fig. 1**). The short-axis image was divided into 8 segments near the base and near the midventricular level. Regional tracer uptake was scored semiquantitatively with a 4-point scoring system (0: normal uptake, 1: mildly reduced uptake, 2: moderately reduced uptake, 3: severely reduced uptake). The total defect score (TDS) was calculated as the sum of the scores for all 17 segments.

Heart-to-mediastinum ratio

One projection of the anterior planar image derived from 32 projections of the SPECT images was selected. Regions of interest were chosen on this planar image to include all areas of the left ventricular myocardium. Another region of interest (3 × 3 pixels in size) was placed over the upper mediastinal area. The heart-to-mediastinum (H/M) ratio was calculated from the iodine-123 BMIPP uptake as a fraction of the mean counts per pixel in the myocardium divided by those in the mediastinum.

Echocardiography

All 19 patients underwent M-mode and two-dimensional echocardiography with an ultrasono-scope (SS-H160A, SS-H260A, Toshiba). The left ventricular end-diastolic dimension (LVDd) and left ventricular end-systolic dimension (LVDs) were calculated from the echocardiographic data. Fractional shortening (%) and ejection fraction (%) were used as indicators of the severity of cardiac dysfunction. Each of these parameters was calculat-

ed according to standards established by the American Society of Echocardiography.

Evaluation of treatment response

1) Clinical cardiac function was evaluated according to the New York Heart Association (NYHA) functional classification based on the fractional shortening, ejection fraction, LVDd and LVDs.

2) These assessments were made before and 6 months after initiation of β-blocker therapy.

3) Patients were classified into 2 groups : Responders who showed improvement of at least one functional class or an increase in the ejection fraction of $\geq 0.10^{6,12)}$, and the nonresponders showing neither of these improvements after β-blocker therapy.

Statistical analysis

A two-factor repeated measures analysis of variance was used for paired data, specifically the comparison of TDS and H/M ratio, echocardiographic data, heart rate, systolic blood pressure and cardiothoracic ratio before and 6 months after the start of β-blocker therapy. The H/M ratio, fractional shortening, ejection fraction, LVDd, LVDs, heart rate, systolic blood pressure and cardiothoracic ratio value for the 2 patient groups were compared using an unpaired t-test. Probability (p) values of < 0.05 were considered significant.

RESULTS

Eight of the 19 patients with DCM showed both symptomatic and objective improvement with treatment. One patient showed symptomatic deterioration and 5 were essentially unchanged. The remaining 5 patients showed improvement in NYHA functional class without an increase in the ejection fraction ≥ 0.10. Accordingly, 13 of the 19 patients were classified as responders to β-blocker therapy and the remaining 6 patients as nonresponders (**Table 1, Fig. 2**).

The responders showed no significant changes in the TDS (13.2 $\pm$ 3.7 vs 12.5 $\pm$ 3.3) or the H/M ratio (2.47 $\pm$ 0.28 vs 2.43 $\pm$ 0.42) during 6 months of treatment (**Figs. 3, 4**). ^{123}I-BMIPP myocardial SPECT bull's-eye images in a typical patient with response (Responder 2) is shown in **Fig. 3** and the planar image is shown in **Fig. 4**. Fractional shortening increased from 15.8 $\pm$ 4.4% to 22.9 $\pm$ 5.6% $(p < 0.0001)$ and ejection fraction was increased

from 32.4 $\pm$ 8.4% to 44.7 $\pm$ 9.7% $(p < 0.0001$; **Fig. 5**). LVDd values improved from 62.2 $\pm$ 6.5 to 55.2 $\pm$ 6.2 mm $(p < 0.01)$, and LVDs values were reduced from 52.5 $\pm$ 7.4 to 43.5 $\pm$ 6.7 mm $(p < 0.001)$. The cardiothoracic ratio was reduced from 57.4 $\pm$ 8.4% to 50.4 $\pm$ 6.8% $(p < 0.001)$.

The nonresponders showed no significant changes in the TDS (20.8 $\pm$ 6.5 vs 20.5 $\pm$ 3.0; **Fig. 6**). The H/M ratio was significantly increased from 1.82 $\pm$ 0.11 to 2.10 $\pm$ 0.19 after the 6-month period $(p < 0.05$; **Fig. 7**). ^{123}I-BMIPP myocardial SPECT bull's-eye images in a typical patient with nonresponse (Nonresponder 4) are shown in **Fig. 6** and the planar image is shown in **Fig. 7**. No significant changes occurred in fractional shortening (15.5 $\pm$ 2.4% vs 13.0 $\pm$ 3.5% ; **Fig. 8**), ejection fraction (32.3 $\pm$ 4.9% vs 27.8 $\pm$ 5.3%), LVDd (66.1 $\pm$ 5.2 vs 66.3 $\pm$ 9.2 mm) or LVDs (57.6 $\pm$ 5.7 vs 59.2 $\pm$ 5.6 mm). The cardiothoracic ratio did not change significantly (56.7 $\pm$ 3.1% vs 55.7 $\pm$ 1.5%).

One patient among the nonresponders (Nonresponder 2) suffered progressive heart failure that could not be controlled medically and was treated with partial left ventriculectomy (Batista operation).

On entry to the study, the responders had significantly better TDS and H/M ratio than the nonresponders (TDS : 13.2 $\pm$ 3.7 vs 20.8 $\pm$ 6.5, $p <$ 0.005 ; H/M ratio : 2.47 $\pm$ 0.28 vs 1.82 $\pm$ 0.11, $p <$ 0.0001 ; **Table 1, Fig. 9**). No significant differences were seen between responders and nonresponders in heart rate, systolic blood pressure, cardiothoracic ratio, fractional shortening, ejection fraction, LVDd, or LVDs.

Responders had significantly lower TDS than nonresponders (12.5 $\pm$ 3.3 vs 20.5 $\pm$ 3.0, $p <$ 0.001). Responders had significantly higher H/M ratio than nonresponders (2.43 $\pm$ 0.42 vs 2.10 $\pm$ 0.19, $p < 0.05$) at 6 months after the initiation of medication. Significant differences were seen between responders and nonresponders 6 months after the start of β-blocker therapy in systolic blood pressure (124.0 $\pm$ 11.2 vs 104.3 $\pm$ 13.9 mmHg, $p <$ 0.005), cardiothoracic ratio (50.4 $\pm$ 6.8% vs 55.7 $\pm$ 1.5%, $p < 0.05$), fractional shortening (22.9 $\pm$ 5.6% vs 13.0 $\pm$ 3.5%, $p < 0.01$), ejection fraction (44.7 $\pm$ 9.7% vs 27.8 $\pm$ 5.3%, $p < 0.01$), LVDd (55.2 $\pm$ 6.2 vs 66.3 $\pm$ 9.2 mm, $p < 0.01$) and LVDs (43.5 $\pm$ 6.7 vs 59.2 $\pm$ 5.6 mm, $p < 0.001$; **Table 1**).

Serum glucose, triglyceride and total cholesterol concentrations, which may influence fatty acid

Table 1   Patient profiles and measured variables

Pt No.	Age (yr)	Gender	H/M ratio Base	6 mo	TDS Base	6 mo	HR(beats/min) Base	6 mo	SBP(mmHg) Base	6 mo	CTR(%) Base	6 mo	NYHA Base	6 mo	FS(%) Base	6 mo	EF(%) Base	6 mo	LVDd(mm) Base	6 mo	LVDs(mm) Base	6 mo
Responders (n=13)																						
1	67	F	2.36	2.13	7	11	90	88	110	120	72	64	3	2	12	21	25	42	60	56	53	45
2	67	M	2.24	2.02	11	10	100	78	148	130	47	40	2	1	18	25	36	48	57	60	47	45
3	77	F	2.03	1.84	11	9	64	68	130	132	60	61	2	1	22	25	45	49	53	52	41	39
4	36	M	2.77	2.71	13	11	100	88	110	130	46	41	3	2	13	23	27	46	62	46	54	36
5	41	M	2.87	1.94	11	10	93	84	90	108	60	46	4	2	12	28	26	53	62	55	54	40
6	68	M	2.32	2.18	14	11	72	68	130	130	48	49	2	1	20	28	40	53	64	51	51	37
7	49	F	2.31	2.79	9	15	80	72	130	134	68	54	3	1	15	23	32	48	57	48	48	45
8	33	F	2.42	2.62	12	13	78	85	120	126	65	51	3	2	19	25	39	43	67	64	54	48
9	42	F	2.90	2.87	13	10	70	65	130	130	54	52	2	1	22	33	44	62	55	51	43	34
10	29	M	2.39	2.57	14	13	64	72	126	131	56	46	2	1	10	19	21	39	77	62	69	50
11	63	F	2.85	3.28	20	10	94	76	147	109	50	49	3	2	12	15	25	30	63	62	55	53
12	75	F	2.39	2.41	18	12	72	72	120	132	56	49	2	1	19	21	38	43	62	49	51	38
13	74	F	2.24	2.30	18	18	80	70	110	100	64	53	3	2	11	12	23	25	70	62	62	55
Mean			2.47	2.43	13.2	12.5	81.3	75.8	123.2	124.0	57.4	50.4			15.8	22.9	32.4	44.7	62.2	55.2	52.5	43.5
±SD			0.28	0.42	3.7	3.3	12.8	8.0	15.9	11.2	8.4	6.8			4.4	5.6	8.4	9.7	6.5	6.2	7.4	6.7
p value*			NS		NS		NS		NS		<0.001				<0.0001		<0.0001		<0.01		<0.001	
Nonresponders (n=6)																						
1	48	F	1.70	2.22	25	26	70	90	100	110	55	57	2	3	17	12	35	25	57	60	47	53
2	55	M	1.75	2.21	21	21	111	80	112	90	60	58	4	4	16	11	36	23	70	78	57	67
3	75	M	1.89	2.24	11	19	60	64	90	90	56	55	3	3	12	8	25	23	68	66	60	61
4	65	M	1.82	1.80	17	17	60	64	110	110	61	55	2	2	13	13	27	27	69	72	60	63
5	58	M	2.00	2.29	21	21	64	60	120	126	54	54	2	2	18	17	36	34	70	70	58	58
6	79	M	1.81	1.95	30	19	60	55	120	100	54	55	2	2	17	17	35	35	63	52	64	53
Mean			1.82	2.10	20.8	20.5	70.8	68.8	108.6	104.3	56.7	55.7			15.5	13.0	32.3	27.8	66.1	66.3	57.6	59.2
±SD			0.11	0.19	6.5	3.0	20.0	13.3	11.8	13.9	3.1	1.5			2.4	3.5	4.9	5.3	5.2	9.2	5.7	5.6
p value**			<0.05		NS		NS		NS		NS				NS		NS		NS		NS	
p value***			<0.0001	<0.05	<0.005	<0.001	NS	NS	NS	<0.005	NS	<0.05			NS	<0.01	NS	<0.01	NS	<0.01	NS	<0.001

*Differences between baseline and 6 months for responders, **Differences between baseline and 6 months for nonresponders, ***Differences between the groups before and after 6 months of β-blocker administration.

Pt=Patient; H/M=heart-to-mediastinum; TDS=total defect score; HR=heart rate; SBP=systolic blood pressure; CTR=cardiothoracic ratio; NYHA=New York Heart Association functional classification; FS=fractional shortening; EF=ejection fraction; LVDd(s)=left ventricular end-diastolic (end-systolic) dimension; base=baseline; mo=months; F=female; M=male.

uptake in the myocardium, were similar in responders and nonresponders before β-blocker treatment (glucose: 92.5 ± 11.0 vs 102.3 ± 27.9 mg/dl; triglyceride: 100.1 ± 60.8 vs 91.6 ± 8.9 mg/dl; total cholesterol: 184.3 ± 32.1 vs 204.5 ± 29.8 mg/dl, respectively).

No significant differences were seen between the 2 patient groups in any of the medications or doses.

DISCUSSION

Scintigraphic prediction and evaluation of therapeutic effects of β-blocker therapy in dilated cardiomyopathy

Suwa et al.[9] have reported that MIBG imaging can predict the effect of β-blocker therapy before the start of treatment, whereas Fukuoka et al.[10] have concluded that MIBG imaging cannot predict therapeutic effect before the start of treatment but can predict prognostic improvement when performed at one month. Two reports have stated that MIBG imaging can evaluate the effectiveness of β-blocker therapy[10,11]. Treatment response has not yet been predicted based on thallium findings, and no study has used this method to evaluate β-blocker therapy for DCM[13], although one study has reported that thallium can evaluate the prognosis in DCM.
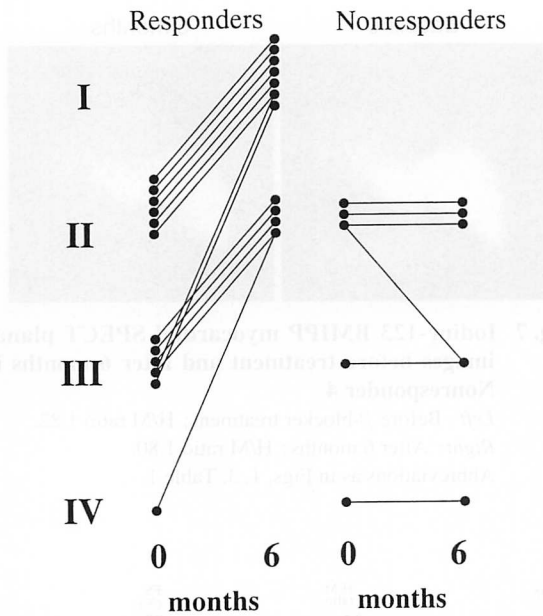
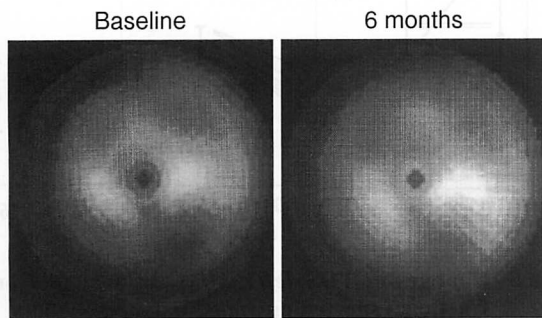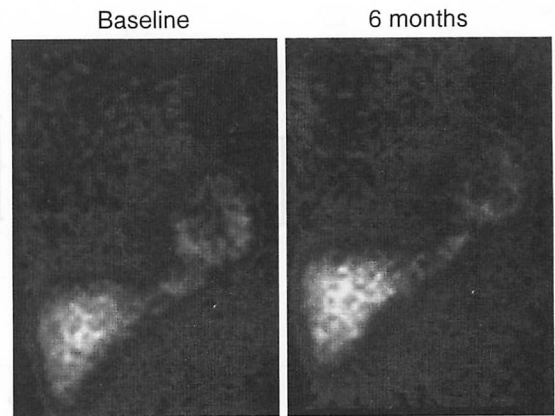Fig. 2 Changes in New York Heart Association classification

Fig. 4 Iodine-123 BMIPP myocardial SPECT planar images before and 6 months after treatment in Responder 2
Left: Before β-blocker treatment; H/M ratio 2.24.
Right: Six months after treatment; H/M ratio 2.02.
Abbreviations as in Figs. 1, 3, Table 1.

Fig. 3 Iodine-123 BMIPP myocardial SPECT bull's-eye images before and 6 months after treatment in Responder 2
Left: Before β-blocker treatment; TDS 11.
Right: Six months after treatment; TDS 10.
SPECT = single photon emission computed tomography. Other abbreviations as in Fig. 1, Table 1.

We previously reported that BMIPP myocardial scintigraphy can predict the response to β-blocker therapy in patients with DCM[12]. On the other hand, whether BMIPP can reflect therapeutic effects is not clear. In this study, BMIPP myocardial scintigraphy after 6 months did not reflect the therapeutic effects of β-blocker administration.

Action mechanism of β-blocker therapy for dilated cardiomyopathy

The exact mechanism by which β-blockers act to improve the status of patients with DCM is not known. However, a rapid heart rate and dilation of the ventricles with high wall stress increase the metabolic rate in the failing myocardium. To satisfy these increased energy requirements, anaerobic glycosis increases. Therefore, slow heart rate therapy should decrease anaerobic glucose metabolism, shifting energy production back toward the normal, more efficient metabolism using free fatty acids. This reduction in metabolic stress could result in a more favorable myocardial energy balance that enhances recovery of the failing myocardium[7,14-16].

BMIPP myocardial scintigraphy

BMIPP has been proposed as a probe for myocardial fatty acid utilization[17]. Animal experiments have shown that myocardial uptake of iodine-123 BMIPP is reduced to the extent that myocardial mitochondrial function is impaired[18,19]. These studies reflect acute myocardial damage, but no animal experimental study of this agent has investigated ongoing myocardial damage. Myocardial uptake of iodine-123 BMIPP has been found to be reduced in patients with DCM[20].

Various abnormalities in myocardial energy metabolism have been reported in patients with DCM, including mitochondrial dysfunction, reduced adenosine 5′-triphosphate production, disorders of the cell membrane, and disturbance of fatty acid-binding proteins[21,22]. Abnormalities in fatty acid metabolism also have been demonstrated in some cases[23,24]. Accordingly, we hypothesized that fatty acid utilization might be improved by β-
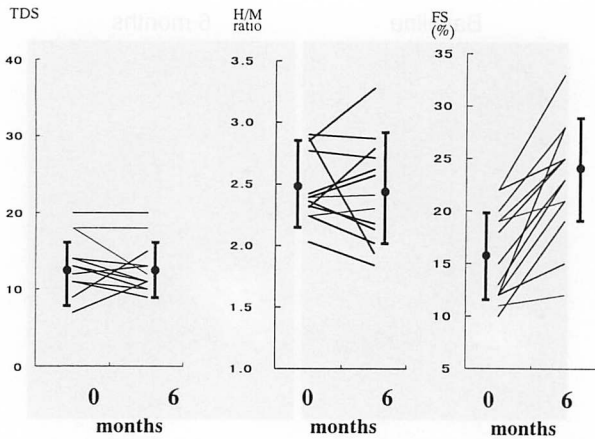
Fig. 5   Changes in TDS (*left*), H/M ratio (*middle*) and fractional shortening (*right*) in responders
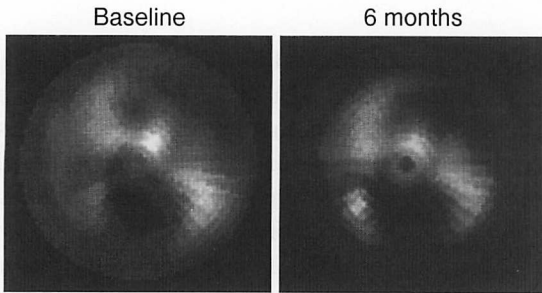Abbreviations as in Table 1.
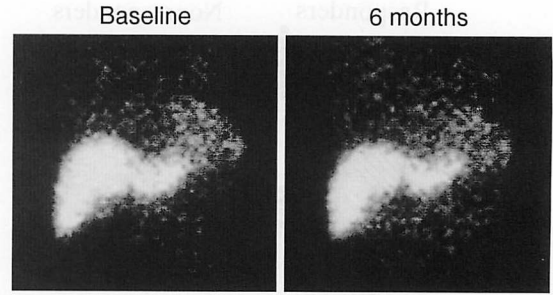

Baseline   6 months

Fig. 7   Iodine-123 BMIPP myocardial SPECT planar images before treatment and after 6 months in Nonresponder 4
Left: Before β-blocker treatment; H/M ratio 1.82.
Right: After 6 months; H/M ratio 1.80.
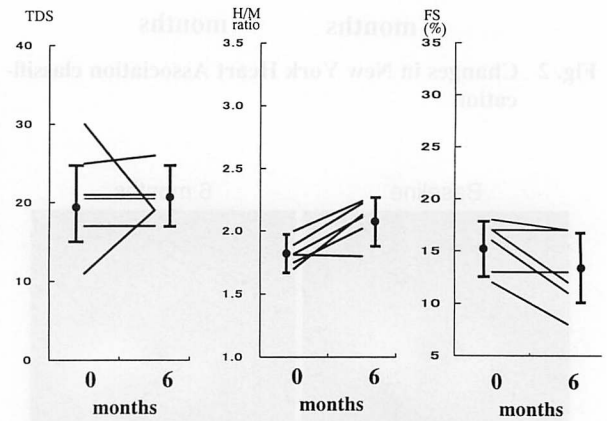Abbreviations as in Figs. 1, 3, Table 1.

Baseline   6 months

Fig. 6   Iodine-123 BMIPP myocardial SPECT bull's-eye images before treatment and after 6 months in Nonresponder 4
Left: Before β-blocker treatment; TDS 17.
Right: After 6 months; TDS 17.
Abbreviations as in Figs. 1, 3, Table 1.

Fig. 8   Changes in TDS (*left*), H/M ratio (*middle*) and fractional shortening (*right*) in nonresponders
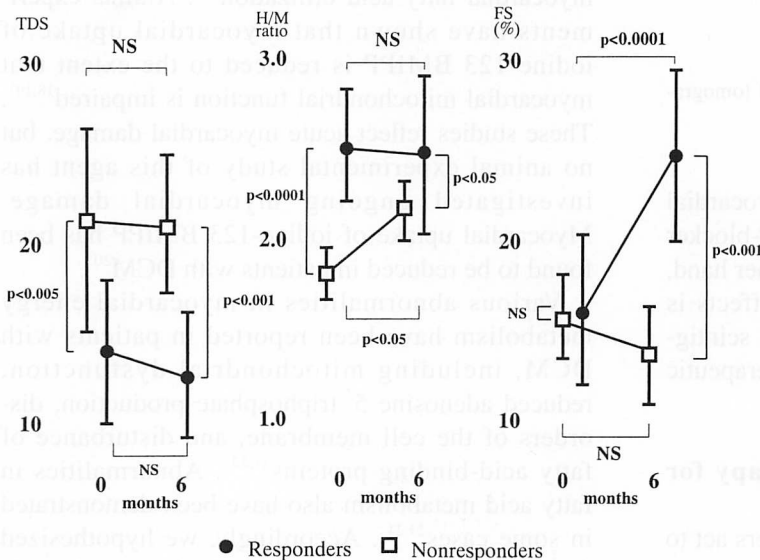Abbreviations as in Table 1.

● Responders   □ Nonresponders

Fig. 9   Changes in mean TDS (*left*), H/M ratio (*middle*) and fractional shortening (*right*) before and 6 months after β-blocker treatment
Abbreviations as in Table 1.

blocker therapy in DCM.

Echocardiographic values for ejection fraction and fractional shortening improved significantly in responders. Excessive cardiothoracic ratio also decreased significantly. In contrast, no significant changes were observed scintigraphically in the TDS or H/M ratio. Discrepancies between perfusion abnormalities and fatty acid metabolism have been noted frequently in cases of ischemic heart disease. Accumulation of technetium-99 m [^{99m}Tc-methoxy-isobutyl isonitrile (MIBI), tetrofosmin] or thallium-201 (^{201}Tl) in excess of that of BMIPP after thrombolysis and/or percutaneous transluminal coronary angioplasty has been observed. Tamaki et al.[25] have reported frequent mismatches in distribution between BMIPP and ^{201}Tl uptake during the 4 weeks following onset of myocardial infarction. Such discordance becomes less frequent after 4 weeks, suggesting that recovery of fatty acid metabolism is time-dependent[26,27]. Recovery of myocardial metabolism presumably requires a long time in cases of DCM. Takeuchi et al.[28] have reported H/M ratios of 2.63 ± 0.11 in early images in normal subjects. The H/M ratio in our responders before drug administration was close to this normal range, so fatty acid metabolism may largely have been maintained before treatment in this group.

No significant changes were evident in ejection fraction and fractional shortening in nonresponders. One patient deteriorated in NYHA functional class, but other nonresponders did not. TDS showed little improvement with therapy. The H/M ratio improved significantly in nonresponders, unlike other variables. Possible reasons are outlined below.

1) The 6 nonresponders showed no improvement in various variables, but no significant worsening in ejection fraction, fractional shortening, or cardiothoracic ratio. One patient had reduction in NYHA function, but no deaths occurred. These results suggest that β-blocker therapy may have accomplished clinical stabilization even in these patients. Kim et al.[20] found in a study of coenzyme Q 10 administration that BMIPP scintigraphy may detect therapeutic effect in the absence of improvement in other variables. Thus, β-blocker therapy might have resulted in slight subclinical improvements.

2) The discordance between accumulation of BMIPP and myocardial blood flow tracers (^{201}Tl or ^{99m}Tc-MIBI, tetrofosmin) raises questions concern-

ing maintenance of viable myocardium. However, any defect shown by both BMIPP and blood flow tracers would indicate that damage in this area had progressed to myocardial fibrosis and could not be ameliorated by medical therapy. A few nonresponders with an improved H/M ratio may have had viable myocardium within a focal defect shown by BMIPP.

3) The H/M ratio in nonresponders before treatment was 1.82 ± 0.11, which is considerably lower than normal (2.63 ± 0.11)[28]. Therefore, nonresponders may have more potential for improvement with therapy.

4) The sample size is very small in this study, especially for nonresponders. The H/M ratio may be affected considerably by the technique and this effect would be exaggerated by the low patient numbers.

5) The H/M ratio was calculated from the anterior planar image. This planar image was derived from 32 projections of the SPECT images. The SPECT image was acquired for 30 sec. Ordinary anterior planar images employ 10-minute counts. Therefore, these SPECT data may have limited the diagnostic accuracy.

Therapeutic effects of β-blockers with dilated cardiomyopathy

The TDS ($p < 0.005$) and the H/M ratio ($p < 0.0001$) in responders were significantly higher than in nonresponders at entry into the study, whereas no significant differences were noted between the 2 groups at entry for any other variables. BMIPP myocardial scintigraphy can predict the response of patients with DCM to β-blocker therapy, as previously reported. Since BMIPP myocardial scintigraphy reflects myocardial mitochondrial function and intracellular adenosine 5'-triphosphate levels[19,20], the findings from BMIPP myocardial scintigraphy before β-blocker administration may reflect the volume of the cardiac muscle capable of responding to treatment.

In patients with BMIPP accumulation maintained before β-blocker administration, the early indication of therapeutic effect can be evaluated by echocardiography, and there is little need for repeated BMIPP scintigraphy. With advances in treatment, the 5-year survival rate in patients with DCM has improved to 80%[21,29]. Early prediction of therapeutic efficacy may be useful for planning effective rehabilitation to improve the quality of

life.

Future directions

Long-term observation is necessary to determine whether cardiac function in nonresponders might improve subsequently, and whether the H/M ratio in responders might show a late increase after the interval in this study.

A limitation of this study is that the BMIPP results were not compared with a blood flow tracer. Such a comparison is necessary to determine whether adding or substituting BMIPP scintigraphy can provide more information than that obtainable with the blood flow tracer alone.

CONCLUSION

No deterioration of myocardial fatty acid metabolism was seen in either responders or nonresponders to β-blockers treatment. BMIPP myocardial scintigraphy was useful for predicting the therapeutic effects of β-blocker therapy in DCM, as reported previously. However, BMIPP myocardial scintigraphy after 6 months did not reflect the therapeutic effects in responders.

──────── 要　　約 ────────

^{123}I-BMIPP心筋シンチグラフィーは特発性拡張型心筋症における β 遮断薬の改善効果を予測しうるが，治療効果は反映しない

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秋元　正樹　　木原　浩一　　鄭　忠和

我々は心筋の脂肪酸代謝を評価する ^{123}I-BMIPP心筋シンチグラフィーが，特発性拡張型心筋症に対する β 遮断薬療法の治療効果を予測できることを報告した．本研究においてBMIPP心筋シンチグラフィーを用いて特発性拡張型心筋症に対する β 遮断薬投与前後での心筋のBMIPP集積の変化を検討した.

対象は特発性拡張型心筋症患者19例(男性10例，女性9例，平均年齢58歳)である．BMIPP心筋シンチグラフィーを β 遮断薬投与開始前および投与6ヵ月後に施行した．^{123}I-BMIPP(111MBq)を安静・空腹時に静注し，15分後に撮像した．BMIPP集積は短軸断層像から総欠損スコア(TDS)を算出し，planar像からH/M比を計測し評価した．NYHA心機能分類の改善，あるいは左室駆出率が10%以上改善した群を改善群，それ以外を非改善群とした．β 遮断薬はメトプロロールを投与し，20mg/dayを維持量とした.

β 遮断薬投与前のBMIPPのTDSおよびH/M比は，2群間に有意差(TDS: $p<0.005$, H/M比: $p<0.0001$)を認めた．いずれも改善群が有意に高値を示した．治療前と6ヵ月後のTDSは両群とも有意な変化を認めなかった(改善群: 13.2±3.7 vs 12.5±3.3，非改善群: 20.8±6.5 vs 20.5±3.0).H/M比の変化は，改善群では有意な変化を認めなかった(2.47±0.28 vs 2.43±0.42)が，非改善群では有意に増加した(1.82±0.11 vs 2.10±0.19, $p<0.05$)．この結果から β 遮断薬療法は臨床的な改善を示さなくても，心筋の脂肪酸代謝を保護することが示唆された.

BMIPP心筋シンチグラフィーは特発性拡張型心筋症患者に対する β 遮断薬の効果を治療前に予測できるが，投与6ヵ月後のBMIPP心筋摂取は改善群においては治療効果を明瞭には反映しなかった.

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