CLASSIFICATION AND DISTRIBUTION OF MYOCARDIAL FASCICLE AND FIBER DISARRAY IN 14 HEARTS WITH HYPERTROPHIC CARDIOMYOPATHY IN 25 μ THICK SECTIONS

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In the usual thin sections (4–7 μ), myocardial fiber disarray in cases of hypertrophic cardiomyopathy (HCM) usually takes the form of marked cellular disarray. Its diffuse extent is specific for HCM, but its presence alone is not pathognomonic for HCM. In our previous report, we stated that thick sections (20–35 μ) was quite adequate for the study of structures of disorganized myocardial fibers. In the present paper, classification and an assessment of the distribution of myocardial fiber disarray were studied in hearts with HCM in 14 Japanese patients and in 45 hearts (25 normal hearts, 10 hearts with hypertension and 9 hearts with myocardial infarction) as the control, in the thick sections.

In these thick sections, abnormal myocardial fiber disarray in HCM was classified into poorly-differentiated and well-differentiated fascicular patterns. Abnormal myocardial fiber disarray in most hearts with HCM comprised the former pattern and its extent was over 30% in all septums of hearts with HCM in comparison with less than 10% in the controls. The diffuse disarray was mostly noted in the middle three fifths in the septum, and in the outer and middle thirds in the free wall, where disarray was rare in the control hearts. These findings support the idea that diffuse disarray in the specific portions in HCM may be congenital and specific for HCM.

The major focus of the disarray in HCM was the septum with asymmetric hypertrophy. The disarray comprising two fascicular patterns involved a large number of fibers running in a vectorically perpendicular direction to the left ventricular cavity. Theoretically, shortening of such fibers would make the wall thinner and the cavity larger, and lengthening would make the wall thick and the cavity small. Thus, a wall with a considerable number of such fibers would be hypofunctional. We conclude that diffuse disarray in the middle three fifths of the ventricular septum and in the middle and outer thirds of

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the ventricular free wall is fascicular, and that it is congenital, specific and one of the pathogenic factors for HCM.

SINCE the first report by Teare¹ in 1958, myocardial fiber disarray has been described as a histologic feature of asymmetrically hypertrophied septum in hypertrophic cardiomyopathy (HCM), with or without obstruction.¹⁻⁸ However, disarrangement of muscle cells has also been detected, albeit to a lesser extent, in numerous patients with or without heart disease.⁹⁻¹⁴

We found that so-called marked fiber disarray in the usual thin sections (4–7 μ) was seen mostly as fascicular disarray in the thick sections (20–35 μ) in 26 normal hearts, 10 hearts with hypertension, 9 with myocardial infarction and 4 with HCM.¹⁴ It was stated that fascicular disarray was

probably congenital, and that our approach using thick sections was quite adequate for the study of structures of disorganized fibers.¹⁴

Recently, the histologic classification and distribution of disorganized myocardial cells in the septum of the hearts with HCM were reported in the usual thin sections.¹³ But these have not been studied in Japanese cases with HCM.

The present study was undertaken to classify abnormal disarray in HCM in the thick sections, to determine the extent of the abnormal disarray in the septum and the free wall of the left ventricle, and to elucidate whether or not abnormal disarray is a pathogenic factor in HCM.

TABLE I SUMMARY OF CASES

Autopsy Data on 14 Patients with Hypertrophic Cardiomyopathy

No.	Age/Sex	Obstruction*	H.W. (g)	ASH	VS/PW (cm)	VS/PW ratio
1.	4 Y/m	+	180	+	1.5/0.9	1.7
2.	20 Y/m	+	415	+	2.3/1.1	2.1
3.	25 Y/f	+	970	+	2.7/1.5	1.8
4.	25 Y/f	?	450	+	2.0/1.3	1.5
5.	26 Y/m	+	560	+	1.7/1.2	1.4
6.	28 Y/m	+	600	+	2.2/1.1	2.0
7.	36 Y/m	+	770	+	2.8/1.4	2.0
8.	37 Y/m	?	680	+	2.9/1.4	2.0
9.	61 Y/m	+	903	+	3.0/1.5	2.0
10.	65 Y/m	+	450	+	1.8/1.1	1.6
11.	66 Y/m	—	700	+	2.4/1.5	1.6
12.	66 Y/m	?	325	+	1.7/1.1	1.5
13.	67 Y/f	—	350	+	1.5/1.1	1.4
14.	76 Y/m	+	430	+	1.9/1.1	1.7

Autopsy Data on Controls

	No. of cases	Sex m/f	Age (years)		H.W. (g)	
			Range	Mean	Range	Mean
Normal adult hearts	18	13/5	21–80	53	200–350	276
Normal infant hearts	8	5/3	4Mo.–2	1	30–60	40
Hypertensive hearts weighing over 400g	10	7/3	45–80	62	400–600	473
Myocardial infarction without hypertension	9	7/2	45–82	68	210–520	348

*: obstruction of outflow tract, H.W.: heart weight, ASH: asymmetric septal hypertrophy of over 1.3, VS: ventricular septal thickness, PW: posterior wall thickness, Y: years, m: male, f: female. The measurement of ASH was done by the method of Maron et al.¹²

Fig.1. Various histologic pictures of poorly-differentiated fascicular disarray in hypertrophic cardiomyopathy. Fern-like pattern is shown in A, B and C, whirl-like pattern in D and E, and bush-like pattern in F. Note that the areas, where fascicular structures are clear and unclear, are mixed, and bizarre shaped cells are abundantly seen. Note the lack of a clear cut border between areas with or without abnormal disarray. (A, B and C: $\times 100$; D, E and F: $\times 50$; hematoxylin-eosin stain)

SUBJECTS AND METHODS

Fourteen hearts were obtained at autopsy from Japanese patients with hypertrophic cardiomyopathy (HCM). Twelve of these patients had been diagnosed clinically and pathologically as cases of HCM. In the remaining patients echocardiogram and cardiac catheterization were not done and the diagnosis was made on the pathology alone.

Age, sex, obstruction of the outflow tract, heart weight, asymmetric septal hypertrophy (over 1.3 at autopsy), ventricular septal thickening (VS)/posterior wall thickening (PW) and VS/PW ratio at autopsy are summarized in Table I. The anatomical criteria for HCM are described in the literatures¹⁻⁸. In our cases, each major coronary artery showed luminal narrowing less than 50% and there was no significant valvular, congenital or infectious heart diseases.

Fig.2. Moderate and marked fiber disarray in poorly-differentiated fascicular disarray, and comparison between these and mild fiber disarray. A: moderate fiber disarray with bizarre shaped cells. B: marked fiber disarray with bizarre shaped cells. C_{1,2}: various mild fiber disarray, where bizarre shaped cells are rare. (A: $\times 100$; B: $\times 200$; C_{1,2}: $\times 50$; hematoxylin-eosin stain)

There was a mild hypertension only in 2 cases (Cases 13 and 14).

The heart was incised longitudinally in the center of the septum from base to apex, then serially and transversely at about 2-cm intervals, and observed macroscopically. All 14 hearts with HCM had asymmetric hypertrophy of over 1.3 (Table I).

A large transverse section of the left ventricle with maximal asymmetric septal hypertrophy was longitudinally separated into 5 or 6 blocks. All tissues were embedded in paraffin. Each block obtained from the septum and ventricular free wall was cut serially by microtome, in 5μ (thin sections) and 25μ thicknesses (thick sections). These 2 sections were stained with hematoxylin and eosin and used to compare the histologic pictures in thin and thick sections.

Eighteen normal adult hearts, 8 normal infantile hearts, 10 hearts with a weight of over 400 g (hypertension) and 9 hearts with old and/or fresh myocardial infarction were used as the controls (Table I). A large transverse section of the upper third of the left ventricle was separated into 4–6 blocks and was used for histologic study.

Disarray in the trabeculations, and the triangular portions of the anterior and posterior septum were excluded from this study, because these areas often show disarray, even in normal and diseased hearts other than HCM^{11,13,14}. Waving fibers postulated as artifact were not considered to be disarray. The distribution of disarray was studied only in the transverse sections, because disarray was detected both more frequently and to a greater extent in these sections as compared to the longitudinal plane.

Fig.3. Various histologic pictures of well-differentiated fascicular disarray in hypertrophic cardiomyopathy. Note the clear fascicular pattern and rare cells with bizarre shape. There is a lack of a clear cut border between areas with or without the disarray. (right: $\times 30$; left: $\times 50$; hematoxylin-eosin stain)

sections in the thick sections, as detailed in the usual thin sections by Maron and Roberts.¹³ To examine percent areas of disarray, outlines of the stained tissue sections and areas of disarray were sketched on the tracing paper at a magnification of 10 times, using a stereoscopic microscope with drawing tube (Nikon: 10×0.66 to 10×4). The percent areas of disarray were calculated by a planimeter.

RESULTS

Classification of Myocardial Fascicle and Fiber Disarray in Hypertrophic Cardiomyopathy (HCM) in the Thick Sections

Disarray in HCM was divided into a poorly-differentiated fascicular pattern (Type I: Fig. 1) and a well-differentiated fascicular pattern (Type II: Fig. 3).

Poorly-differentiated fascicular disarray was characterized by the mixture of areas, where

structures as fascicular disarray were clear and unclear, and abundant bizarre shaped cells were present. Each bundle in fascicle disarray was relatively small, and various histologic pictures, such as fern-like, whirl-like and bush-like patterns, were seen (Fig. 1). In the transverse sections to the long axis of the heart, a fern-like pattern was frequently noted in the central band of the septum, and a whirl-like pattern on either side of the center in the hearts with HCM. In the longitudinal sections, a whirl-like pattern was frequently seen in the central band of the septum. An assembly with analogous three dimensional structures could be seen in the form of various pictures, depending on how the tissue had been cut. Moderate fiber disarray with bizarre shaped myocardial cells was noted in the areas with an unclear fascicular disarray (Fig. 2). Marked fiber disarray was seen mostly in the converging areas of fascicles in poorly-differentiated fascicular disarray (Fig. 2).

Fig.4. Comparison between thick (25μ) and thin (5μ) sections in disarray with 2 patterns. A_1 : poorly-differentiated fascicular disarray in the thick section. A_2 : the thin section of A_1 . Note marked cellular disarray. B_1 : well-differentiated fascicular disarray in the thick section. B_2 : the thin section of B_1 . Note analogous fascicular pattern and cellular disarray in the converging areas of fascicles. (A_1 and A_2 : $\times 100$; B_1 and B_2 : $\times 50$; hematoxylin-eosin stain)

Fig.5. Distribution of abnormal disarray in the left ventricle of the hearts with hypertrophic cardiomyopathy. Disarray distributed diffusely in the septum in Case 7 (left), in the septum and anterior wall in Case 3 (middle), and in the septum and whole of the free wall in Case 6 (right). These patients had obstruction of the outflow tract, as observed both clinically and pathologically. ⊗: abnormal disarray comprising poorly-differentiated and well-differentiated fascicular patterns. RV: right ventricular cavity, LV: left ventricular cavity, A: anterior wall and P: posterior wall.

Fig.6. Gross fiber orientation in abnormal disarray (Type I: poorly-differentiated fascicular disarray; Type II: well-differentiated fascicular disarray) in hypertrophic cardiomyopathy. Note the considerable number of fibers running in a vectorically perpendicular direction to the left ventricular cavity. $\updownarrow$: direction of fibers running perpendicularly to the left ventricular cavity. $\leftrightarrow$: direction of fibers running circularly to the left ventricular cavity. $\cdots$: direction of fibers running longitudinally to the left ventricular cavity. RV: right ventricular cavity, LV: left ventricular cavity, A: anterior wall and P: posterior wall.

Well-differentiated fascicular disarray comprised a relatively large bundle with clear fascicular structure and rare bizarre shaped cells, and each fiber in the bundles was arranged in a normal pattern (Fig. 3). Moderate and marked fiber disarray with bizarre shaped cells were rare, even in the converging areas of fascicles.

Mild fiber disarray (Y shaped) with rare bizarre shaped myocardial cells was not counted as abnormal, because it was seen at all areas in the septum and the free wall of the hearts in both HCM and the controls (Fig. 2). In 14 hearts from patients with HCM, diffuse moderate or marked fiber disarray was noted only in the areas with fascicle disarray.

In the usual thin sections ($4-7\mu$), poorly-differentiated fascicular disarray observed in the thick sections was seen as mostly marked or moderate cellular disarray, and partially as fascicle disarray (Fig. 4-A). Disarray with a well-differentiated fascicular pattern in the thick sections was seen as fascicle disarray with mostly

analogous and sometimes non-analogous pattern in the usual thin sections (Fig. 4-B). In the latter, moderate or marked cellular disarray in the thin sections was detected in the converging areas of fascicles and in the areas where the fascicle bent at an acute angle in the thick sections.

Distribution of Abnormal Disarray in HCM, and Comparison of the Extent among 14 Hearts with HCM and 45 Hearts from Controls (18 Normal Adult Hearts, 8 Normal Infantile Hearts, 10 Hearts from Patients with Hypertension and a Heart Weight of over 400 g and 9 Hearts with Myocardial Infarction)

Distribution of abnormal disarray with poorly-differentiated and well-differentiated fascicular patterns was studied in the septum and the left ventricular free wall of the hearts with HCM and controls. These data are summarized in Table II.

All septums of the 14 hearts with HCM had an extent of abnormal disarray of over 30% in comparison with less than 10% in the controls (Table

II). The abnormal disarray was mostly noted in the middle three fifths of the septum (Fig. 5). In the left ventricular free wall, 10 hearts with HCM revealed a disarray of over 10% in comparison with less than 10% in the controls (Table II). The disarray was mostly detected in the outer and middle third of the free wall (Fig. 5). In the hearts of controls, disarray was rarely and focally noted in the outer third of the free wall and in the middle one fifth of the septum.

Diffuse abnormal disarray was noted only in the septum of 4 hearts, in the septum and the anterior and/or posterior wall of 5 hearts, and in the septum and the whole free wall of the left ventricle of 5 hearts (Fig. 5). In 9 hearts with obstruction of the outflow tract, diffuse abnormal disarray was noted only in the septum of 2 hearts, in the septum and in the anterior and/or posterior wall of 4 hearts, and in the septum and the whole free wall of the left ventricle of 3 hearts. In 2 hearts without obstruction, there was a diffuse disarray only in the septum of one heart, and in both the septum and the free wall of the left ventricle of another.

Diffuse abnormal disarray comprised a poorly-differentiated fascicular pattern in 12 of 14 hearts with HCM, a well differentiated fascicular pattern in one heart (Case 3) and the mixed

pattern of the two types in one heart (Case 8). In Case 8, the septum had a poorly differentiated fascicular disarray and the anterior wall of the left ventricle had a well differentiated fascicular pattern.

There was no correlation between the extent of abnormal disarray and septal thickness, the grade of asymmetric septal hypertrophy or heart weight in 14 hearts with HCM, although the most extensive diffuse disarray in each heart was noted in a septum with asymmetric hypertrophy.

Gross Fiber Orientation of Abnormal Disarray

Fiber orientation was vectorically separated into circular, longitudinal and perpendicular directions to the left ventricular cavity (Fig. 6). Generally, myocardial fibers of normal hearts run in a circular direction in the middle third of the left ventricular wall and in a longitudinal direction on either side of the central band.^{11,13}

In abnormal disarray with poorly differentiated and well differentiated fascicular patterns, numerous fibers running in a vectorically perpendicular direction to the left ventricular cavity were mixed together with fibers which ran in vectorically circular and longitudinal directions in all 14 hearts with HCM (Fig. 6). The quantity of fibers running in a vectorically perpendicular

TABLE II THE EXTENT OF ABNORMAL DISARRAY IN HEARTS WITH HYPERTROPHIC CARDIOMYOPATHY AND CONTROLS

Ventricular Septum

	<i>Hypertrophic cardiomyopathy</i>	<i>Normal adults</i>	<i>Normal infants</i>	<i>Hypertension</i>	<i>Myocardial infarction</i>
≤ 10%	0	18	8	10	9
> 10%	0	0	0	0	0
> 30%	7	0	0	0	0
> 50%	2	0	0	0	0
> 70%	5	0	0	0	0
<i>Total</i>	<i>14</i>	<i>18</i>	<i>8</i>	<i>10</i>	<i>9</i>

Left Ventricular Free Wall

	<i>Hypertrophic cardiomyopathy</i>	<i>Normal adults</i>	<i>Normal infants</i>	<i>Hypertension</i>	<i>Myocardial infarction</i>
≤ 10%	4	18	8	10	9
> 10%	5	0	0	0	0
> 30%	4	0	0	0	0
> 50%	1	0	0	0	0
> 70%	0	0	0	0	0
<i>Total</i>	<i>14</i>	<i>18</i>	<i>8</i>	<i>10</i>	<i>9</i>

direction varied from area to area and case by case. The area without abnormal disarray comprised fibers running in a relatively circular direction in the middle third of the left ventricular wall and in a longitudinal direction, bilaterally. Fibers running in a vectorically perpendicular direction were rarely seen.

DISCUSSION

Most of the disarray in hypertrophic cardiomyopathy (HCM) was in the form of marked cellular disarray in the usual thin sections (4–7 μ).^{1,4,13} In our data in the thick sections (25 μ), abnormal disarray in HCM comprised mostly poorly-differentiated and sometimes well-differentiated fascicular disarray. The extent of disarray in the septum was over 30% in all 14 hearts with HCM in comparison with less than 10% in 45 hearts of controls (25 normal hearts, 10 hearts with hypertension and 9 hearts with myocardial infarction). The abnormal disarray in the hearts with HCM was mostly noted in the middle three fifths of the ventricular septum, and in the outer and middle thirds of the left ventricular free wall, where hearts without HCM had rare disarray. Disarray in the hearts without HCM was mostly noted in the subendocardium of the septum of the right ventricle, in the inner third of the left ventricular free wall and in the triangular portions of the anterior and posterior septum, where a similar extent of disarray was noted in the hearts with HCM. Although the left ventricular side of the septum in HCM had catenoid shape,¹⁵ disarray was rarely seen in the subendocardium of the left ventricular septum. This supports the hypothesis that the presence of disarray is not specific for HCM, but diffuse disarray in the specific portions in HCM is primary, congenital and highly specific for HCM.^{2,4,5,12–14,16}

Maron and Roberts¹³ reported that the percent area of disorganized septum ranged from 0–94% (mean $31 \pm 3\%$) in HCM, according to quantitative analysis in the usual thin sections. Our study was done on thick sections. Also the extent of disarray was semiquantitatively examined, as the strict borderline between areas with or without abnormal disarray was often unclear in the thick sections (Figs. 1–4). In our data, all septums of 14 hearts with HCM had a disarray of over 30%, and the free wall in 10 hearts a disarray of over 10%. The center of the distribution of disarray was the septum with asymmetric

hypertrophy. We are of the opinion that rare cases of HCM without diffuse abnormal disarray, as detailed by Maron and Roberts,¹³ should be histologically and etiologically differentiated from most patients of HCM with diffuse abnormal disarray, because this disarray in cases of HCM is fascicular disarray and such is congenital. Correlations among the extent of disarray in the septum, heart weight, septal thickening and the ratio of asymmetric septal hypertrophy were not detected, as also reported by Maron and Roberts.¹³ These findings do not necessarily mean that disarray is not pathogenic for HCM, but that there are various other factors (obstruction of the outflow tract, mitral regurgitation, fibrosis, etc) to determine clinical and pathological parameters in HCM.

In addition, it was evident that disarray in HCM involves a large number of myocardial fibers running in a vectorically perpendicular direction to the left ventricular cavity. Theoretically, contraction of fibers running in vectorically circular and longitudinal directions to the left ventricular cavity would make the wall thicker and the cavity smaller (systole), and relaxation of the fibers would make the wall thin and the cavity large (diastole). Conversely, shortening of fibers running in a perpendicular direction would make the wall thinner and the cavity larger, and lengthening of the fibers would make the wall thick and the cavity small. The wall with a considerable number of fibers running in the vectorically perpendicular direction would be hypofunctional. As a result of the compensatory mechanism, myocardial fibers in the walls with or without abnormal disarray would be hypercontractable and hypertrophied. Such would account for the relative lack of systolic septal thickening and hypermovement of the free wall in echocardiographic study,^{17,18} and hypertrophy of the myocardial cells with or without disarray in HCM.^{1–5,14} Thus, a diffuse abnormal disarray in the specific portions is one of the pathogenic factors for HCM.

It was reported that HCM with obstruction of the outflow tract involved a diffuse disarray in the septum, and HCM without obstruction is related to both the septum and the left ventricular free wall.⁴ Four of our 9 hearts with obstruction had a diffuse disarray of over 30% in the left ventricular free wall. In 3 of these 4, the disarray was distributed over the entire left ventricular free wall. These findings show that obstruction of the outflow tract does not only occur in

hearts in which the diffuse disarray is localized in the septum.

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