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Assessment of Diastolic Function of Heart Failure Patients Due to Systemic Hypertension

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Abstract **Original Research Article**

***Introduction*:** Diastolic function as is the current conventional tradition, in order to gain deeper understanding it is more useful to view diastolic function as a problem to be solved! Once the basic problem of normal physiology of diastole has been mastered, it is nearly self-evident as to what the issues are in clinical heart failure and which physiologic mechanism and its hard-wired compensatory pathways one is confronted with. ***Objective*:** To evaluate diastolic function of heart failure patients with systemic hypertension. ***Methods*:** The present prospective observational study was undertaken in Cardiology Department, Dhaka Medical College hospital, Dhaka, Bangladesh from January to July, 2019. Fifty-four (54) patients with systemic hypertension who recently experienced heart failure with normal ejection fraction (≥50%) and no clinical history of ischaemic cardiomyopathy were studied. The patients were divided into two groups according to the degree of echocardiographic hypertrophy: Group-I (28 patients) with a ventricular mass/volume ratio >1.8 and group-II (26 patients) with a ratio <1.8. ***Results*:** Total 54 patients with diastolic dysfunction with hypertension were included in the study. Shows the age and sex distribution with highest frequency in the age group 61-70 years, comprising 31.5 percent and 51-60 years 25.9 percent, 41-50 year 24.1 percent, 71 + years 12.9 percent and 30-40 years 5.6 percent in descending order. Group I patients had a higher ejection fraction (67.62±3.14 vs 55.33 ±4.13, P<0.001), smaller ventricular diameter (28.88±2.46 vs 34.38±4.37, P<0.001), higher LV mass (154.42±6.80 vs 123.38±5.58, P<0.001), lower ETT positivity (14%vs73%, P<0.001). Clinically, group I had more frequent audible fourth heart sound (57% vs 21%, P< 0.001), low incidence of audible third heart sound (17% vs 69%, P<0.001), ECG evident LVH (96% vs 15%, P<0.001), cardiomegaly (25% vs 73%, P<0.001). ***Conclusion*:** There were no significant differences between groups for NYHA class, age, sex, heart rates and systolic blood pressure but significantly associated with Diastolic BP, ECG (LVH) and Cardiomegaly.
Keywords: Diastolic Dysfunction, Congestive Heart Failure, Systematic Hypertension.

INTRODUCTION

Diastolic function as is the current conventional tradition, in order to gain deeper understanding it is more useful to view diastolic function as a problem to be solved! Once the basic problem of normal physiology of diastole has been mastered, it is nearly self-evident as to what the issues are in clinical heart failure and which physiologic mechanism and its hard-wired compensatory pathways one is confronted with. Extensive literature exists on the therapeutic options to be employed in every conceivable clinical setting where heart failure has been diagnosed; therefore, the focus here will be on physiology and pathophysiology, and, more importantly, on what diastolic function actually is, what it is not, and how to measure it. Cardiac cycle consists of both systole and diastole, and the classical syndrome of congestive heart failure, may be either due to systolic dysfunction or diastolic dysfunction or both. Congestive heart failure (CHF) with normal left ventricular systolic function and abnormal diastolic function is a common clinical entity. Typically, signs and symptoms are indistinguishable from those of heart failure related to systolic dysfunction [1]. Coronary artery disease, systemic hypertension and aging are all associated with diastolic congestive heart failure (DCHF) [2]. Several factors predispose to increased diastolic stiffness in a left ventricle with normal systolic performance. These include fibrosis, pericardial constriction and myocardial restriction [3]. Heart failure (HF), often called congestive heart failure (CHF), is commonly defined as a clinical diagnosis in a setting of impaired pump function when the ejection fraction has been reduced and the heart muscle has weakened and is unable to pump sufficient volume of blood at the required pressure to maintain normal physiologic function. Diastolic dysfunction is caused by at least two distinct, yet interrelated. The most common condition in which these factors conspire to elevate filling pressure is hypertension. Loss of elastic properties in hypertension leading to abnormal diastolic function results from the effect of hypertrophy and increase in collagen network of the myocardium. The diastolic disorder must be distinguished from systolic abnormalities because treatment is significantly different. A history of myocardial infarction, cardiomegaly, Q-wave on the electrocardiogram and an S, gallop favours systolic dysfunction. However, an S4 gallop, left ventricular hypertrophy and normal cardiac size favour diastolic dysfunction. However, there is overlap since the disease may produce either or both' Clinical assessment without cardiac imaging is, therefore, incomplete and inaccurate in many cases [4]. To accurately distinguish between systolic and diastolic dysfunction, left ventricular function must be assessed. This can be accomplished with echocardiography, radionuclide angiography or radiographic ventriculography. Echocardiography appears great adventurous because of its portability and lack of radiation as well as its ability to evaluate valvular function, pericardial status, wall motion and chamber hypertrophy. The most commonly used Doppler parameters of Diastolic dysfunction are derived from left ventricular inflow and pulmonary venous inflow. Because the pathophysiologic mechanism in diastolic heart failure includes a high diastolic pressure-volume relationship, the therapy has been directed at reducing filling pressure by decreasing end-diastolic volume, reducing heart rate and treating hypertension with drugs that also cause regression of left ventricular hypertrophy [1]. The American College of Cardiology/American Heart Association (ACA/AHA) task force divides pharmacologic treatment into three classifications for the management of diastolic heart failure. Although prognosis is better with diastolic dysfunction than with systolic dysfunction, morbidity and mortality continue to be high in elderly patients. The reported annual mortality rate for patients with heart failure and preserved left ventricular systolic failure varies from 3 to 25 percent [5]. The syndrome of diastolic heart failure is common but neglected event in the evaluation of hypertensive patients. The signs and symptoms may be similar to those in patients with systolic heart failure.

MATERIALS AND METHODS

The present prospective observational study was undertaken in Cardiology Department, Dhaka Medical College hospital, Dhaka, Bangladesh from January to July, 2019. Fifty-four (54) patients with systemic hypertension who recently experienced heart failure with normal ejection fraction (≥50%) and no clinical history of ischaemic cardiomyopathy were studied. The patients were divided into two groups according to the degree of echocardiographic hypertrophy: Group-I (28 patients) with a ventricular mass/volume ratio >1.8 and group-II (26 patients) with a ratio <1.8. Detailed history and physical examination findings were recorded in study report sheet. ECG, chest X-ray two- dimensional and M-mode echocardiography reports were recorded on the same sheet. Doppler echocardiography was done by a cardiologist and evaluation involved pulse-wave Doppler sampling of mitral inflow (tips of leaflets) and pulmonary vein inflow (right upper vein 1-2 cm deep). Data have been expressed in frequency, percentage and mean ±SD as applicable. Comparison between groups was done by Chi- square test, Student's test and Fisher exact test, as applicable. Data were analyzed by computer-based statistical software. P value <0.05 was taken as significant.

Inclusion Criteria:

a) Patients with history of hypertension for >5 years.
b) Patients with dyspnoea of cardiac origin (NYHA class II, III and IV).
c) Pulmonary oedema verified by CXR-P/A view.
d) Echocardiographically determined ejection fraction ≥50%.
e) Sinus rhythm.

Exclusion Criteria:

a) Secondary hypertension.
b) History of angina or myocardial infarction.
c) History of diabetes mellitus, chronic renal failure and hypertrophic.
d) History of rheumatic fever and rheumatic heart disease, congested heart disease (CHD) and any systemic diseases that causes left ventricular hypertrophy.
e) Any endocardial, myocardial or pericardial disease.
f) Poor echo-windows.

RESULTS

Total 54 patients with diastolic dysfunction with hypertensive were included in the study. Shows the age and sex distribution with highest frequency in the age group 61-70 years, comprising 31.5 percent and 51-60 years 25.9 percent, 41-50 year 24.1 percent, 71 + years 12.9 percent and 30-40 years 5.6 percent in descending order. The patients were classified into two groups according to the degree of echocardiographic hypertrophy in group I (28 patients with a mass/volume ration > 1.8) and group II (26 patients with M/V ration < 1.8). Table shows age <50 were 24.1 percent of which 9 from group I (32.1%) and 4 from group II (15.4%); age >50 were 75.9 percent of which 19 from group I (67.9%) and 22 from group II (84.6%). Diastolic dysfunction of group I and group II patients according to sex, presented male were 34 (63%) of which 21 from group I (75%) and 13 from group II (50%), female were 20 (37%) of which 7 from group I and 13 from group II (50%). Table shows types of diastolic dysfunction, with sample volume position at mitral inflow with tips of leaflets. There are 3 types of diastolic dysfunction - impaired relaxation (IR), pseudo normalization (PN) and restricted filling (RF). By E/A ratio, cases of various types are 22 (IR), 10 (PN) and 22 (EF); by deceleration time (tDec), 21 (IR), 10 (PN) and 23 (RF), by IVRT are 21 (IR), 11 (PN) and 22 (RF). Table also shows types of diastolic dysfunction with sample volume position at right upper pulmonary vein inflow- impaired relaxation are easily diagnosed by S/D ratio >1, but the differentiation between pseudo normalization and estricted filling is difficult unless compared with mitral flow PWD, especially tDec and IVRT. By S/D ratio, cases of various types are 22 (IR), 10 (PN) and 22 (RF); by AR velocity are 21 (IR), 10 (PN), 2 (RF) and by Ad/ARd are 21 (IR), 11 (PN) and 22 (RF). Cardiac catheterization has been done in 24 cases out of 54 cases of diastolic dysfunction. Among 24 cases, diastolic dysfunction was found in 19 cases (LVEDP > 15 mmHg) and left ventricular end-diastolic pressure was normal in 5 cases, all of them were of impaired relaxation variety. Therefore, Doppler echocardiographic estimation is comparable (significant) with cardiac catheterization estimation. ETT were positive in 5 patients in group I and 19 patients of group II, whereas ETT were negative in 24 patients in group I and 19 patients of group II. So, group I show more negativity than group II. Among 24 ETT positive patients, group I consists of 5 patients, of which 2 are CAG positive and 3 are negative. Group II consists of 19 patients of which 17 are positive and 2 are negative. According to degree of hypertrophy, NYHA class II dyspnoea in 28 cases of group I and in 26 cases of group II. Mean age is 53.65 years with SD 11.28 in group I and 57.29 with SD 9.44 in group II. Males were 34 and female 20. Mean heart rate ±SD in group I is 80.92±8.03 and 78.75±6.18 in group II. S3 were mainly found in group II, whereas S4 group I. Both systolic and diastolic blood pressure is higher in group II, LVH were more positive in group I, cardiomegaly in group II, ejection fraction more in group I and duration of hypertension were almost equal.

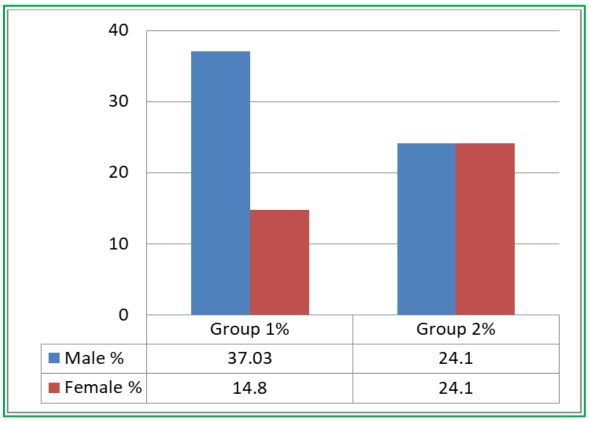

Figure 1: Sex distribution of the study patients (n=54)

Table 1: Age distribution of the study patients (n=54)

Age group (Years)	Male	Female	Total
30-40	2 (6.1)	1(4.8)	3(5.6)
41-50	8 (24.2)	5(23.8)	13(24.1)
51-60	8 (24.2)	6(28.6)	14(25.9)
61-70	11 (33.3)	6(28.6)	17(31.5)
70+	4 (12.1)	3(14.2)	7(12.9)
Total	**33 (61.1)**	**21 (38.9)**	**54(100)**

Table 2: Distribution of diastolic dysfunction of group 1 (M/V >1.8) and group 11(M/V >1.8) patients according to age (n=54)

Sex	Group 1 (n=28) MV>1.8 Range (1.9-2.9) N(%)	Group 11 (n=26) MV>1.8 Range (1.2-1.7) N(%)	Total N(%)
<50	9 (32.1)	4 (15.4)	13 (24.1)
>50	19 (67.9)	22 (84.6)	41 (75.9)
Total	**28 (51.9)**	**26 (48.1)**	**54 (100)**

p>0.05(not significant)

Table 3: Distribution of diastolic dysfunction of group 1 and group 11 patients according to sex (n=54)

Sex	Group1 (n=28) N(%)	Group 11 (n=26) N(%)	Total N(%)
Male	21 (75)	13 (50.0)	34 (63.0)
Female	7 (25)	13 (50.0)	20 (37.0)
Total	**28 (51.9)**	**26 (48.1)**	**54 (100)**

p>0.05(not significant)

Table 4: Classification of patients according to left ventricular hypertrophy (echocardiography findings) (n=54)

Variables	Group1 (n=28) Mean± SD	Group 2 (n=26) Mean± SD	p-Value
LVPWT mm	15.04±0.82	11.50±1.10	<0.001** *
IVST mm	15.15±0.92	11.50±0.98	<0.001** *
LVIDd	48.65±2.95	51.25±3.67	<0.01**
LVIDs	28.88±2.46	34.38±4.37	<0.001** *
LV mass	154.42±6.80	123.38±5.58	<0.001** *
LVVd	66.7±73.37	83.17±6.37	<0.001** *
EF	67.62±3.19	55.334±.13	<0.001***

* * =significant, * * * = highly significant

Table 5: Patients age of hypertension and diastolic dysfunction (n=54)

Age of patients (years)	Group 1 n=28 N(%)	Group 2 n=26 N(%)	Total N(%)
30-40	7 (25.0)	4 (15.4)	11 (20.4)
41-50	7 (25.0)	4 (15.4)	11 (20.4)
51-60	6 (21.4)	10 (38.5)	16 (29.6)
61-70	6 (21.4)	3 (11.5)	09(16.6)
70+	2 (7.1)	5 (19.2)	07(13.0)
Total **Mean± SD**	**28 (51.9)** **11.31±5.76**	**26 (48.1)** **12.96±5.49**	**54 (100)**

p>0.05(Not significant)

Table 6: Distribution of types of diastolic dysfunction by Doppler test (mitral inflow) (n=54)

Sample volume position Mitral flow	Impaired relaxation value Cases	Pseudo normalization value Cases	Restricted filing value Cases
E/A Ratio	<1 (22)	1.0-1.5 (10)	>1.5 (22)
tdec (m/sec)	>240 (21)	180-220 (10)	<180 (23)
IVRT(m/sec)	>110 (21)	80-100 (11)	<70 (22)

Table 7: Distribution of types of diastolic dysfunction by Doppler test (pulmonary venous inflow) (n=54)

Sample volume position Pulmonary venous flow	Impaired relaxation value Cases	Pseudo normalization value Cases	Restricted filing Value Cases
S/D Ratio	<1 (22) (1.0-2.0)	1.0-1.5 (10) (.3- 1.0)	>1.5 (22) (0.1-0.5)
AT velocity (cm/sec)	>25 (21)	>25 (10)	>35 (23)
Ad/ARd (m/sec)	>1 (21)	<1 (11)	<1 (22)

Table 8: Correlation between Doppler echocardiographic findings and cardiac catheterization (n=54)

Total number =54	Diastolic dysfunction	
	Present N (%)	Absent N(%)
Doppler echocardiography (n=54)	54 (100)	0(0.0)
Cardiac catheterization (n=24) done only in ETT positive patients	19 (79.2)	5 (20.8)

P<0.01(highly significant)

Table 9: ETT findings among groups of the study participants (n=54)

Total number =54	ETT positive	ETT Negative
	No (%)	No (%)
Group 1 (n=28)	4 (14.3)	24 (85.7)
Group 2 (n=26)	19 (73.1)	7 (26.9)

Table 10: CAG findings of ETT positive patients among study participants (n=24)

Total number =24	CAG positive	CAG Negative
	N(%)	N(%)
Group 1 (n=5)	2 (40.0)	3(60.0)
Group 2 (n=19)	17 (89.5)	2 (10.5)

Table 11: Clinical findings according to degree of hypertrophy (n=54)

Variables	Group 1 (Mean± SD)	Group II (Mean± SD)	p-Value
Dyspnoea (NYHA II-IV)	28(100%)	26(100%)	NS
Age (years) (Mean± SD)	55.65±11.28	57.29±9.44	>0.10 NS
Sex			
Male	21 (75.0)	13 (50.0)	
Female	7 (25.0)	13 (50.0)	
Heart Rate(Mean± SD)	80.92±8.03	78.75±6.18	>0.10NS
Heart Sound			
S3	9(32.1%)	18(73.1%)	
S4	19(67.9%)	6(26.9%)	
Systolic BP (Mean± SD) (mmHg)	169.23±24.81	176.67±23.34	>0.10NS
Diastolic BP (Mean± SD) (mmHg)	93.85±8.87	98.96±9.09	<0.05*
ECG(LVH)			
Positive	25(92. 9%)	4(15.4%)	<0.01**
Negative	2(7.1%)	22(84.6%)	
Cardiomegaly			
Positive	7(25.0%)	7(26.9%)	<0.01**
Negative	21(75.0%)	19(73.1%)	

* =significant, * * =highly significant, NS= Not significant

DISCUSSION

Heart failure (HF) is a clinical syndrome whose symptoms and signs are due to increased extravascular water and decreased tissue / organ perfusion. The present prospective observational study was undertaken to evaluate left ventricular diastolic dysfunction in congestive heart failure resulting from systemic hypertension. As the sample size was not very large, they may not be representative of all hypertensive heart diseases with congestive heart failure in the community. In the present study, age of the study population ranged from 30 to 75 years, with incidence in 61-70 years comprising 31.5 percent, followed by 51-60 years 25.9 percent, 41-50 years 24.1 percent, 71 + years 12.9 percent and 30-40 years 5.6 percent. Paul and Gheorghiade et al., [1] also found diastolic dysfunction more in elderly (50 to 60 years) group. Although by standard echocardiographic criteria is reversal of E/A ratio favours the diagnosis of diastolic dysfunction, Marantz et al., [6] have shown that this inversion may be normal in older subjects. So, it is important to use the other parameters hi elderly subjects. In the series of 54 hypertensive patients with LV failure with normal ejection fraction, it is possible to divide into two groups: group I with a mass/volume ratio > 1.8 whose dominant character is the high degree of reactive hypertrophy more in the elderly; and group II with a mass/volume ration <1.8 whose dominant character is high rate of ischaemia, also more in elderly group. Left ventricular mass (LVM) is estimated using M-mode at end-diastole, using the measurement of IV septal thickness (IVST), posterior wall thickness (PWT), left ventricular diameter (LVID) as follows: LVM = 0.8 (1.04 f (ST+ PWT + LVID)]3 - LVID3} + 0.6 g where, 1.04 is the specific gravity of myocardium; 0.8 is the correction factor added since anatomic weight would otherwise be overestimated by approximately 20 percent [7]. LV volumes were calculated on the basis of end-systole and end-diastole diameter. To evaluate the Influence of the ventricular mass and rate of regional ischemia, the cutting point is mass/volume ratio is 1.8 with maximal level of differentiation between hypertrophy and ischaemia [8]. Diastolic dysfunction was common in male (61.1%) than female (38.9%). Less number of female patients was involved in the study as small number of female patients attends the hospital for treatment. High incidence of hypertrophy were found in male patients in comparison to female (75% vs 25%) but incidence is ischaemia were equal in both male and female (50% vs 50%). The most significant findings according to echocardiographic left ventricular hypertrophy were increased left ventricular posterior wall thickness and interventricular septal thickness, decreased LV end-diastolic and end-systolic diameter, increased LV mass and higher ejection fraction in group I (hypertrophic group) than in group II (ischaemic group). These results are somewhat similar to the results described by Topol and Araill et al [9] and Miguel et al., [8]. Duration of hypertension did not correlate with the incidence of diastolic dysfunction and there was no significant difference between group I and group II. There was no published data both at home and abroad to compare the relationship between duration of hypertension and incidence of diastolic dysfunction with the present study. Doppler patterns of diastolic dysfunction include normal diastolic function, impaired relaxation, pseudonormal filling and restricted filling. These patterns evolve from one to another in a single individual with changes in disease evaluation, treatment and loading condition as described by Gerald et al., [10]. Impaired relaxation is typically manifested by E/A ration <1, an increased deceleration tune (tDec) and an increased isovolumetric relaxation time (IVRT) with sample volume position at mitral flow. Pulmonary venous flow is also abnormal characterized by increased S/D ratio, decreased atrial reversal (AR) velocity and increased ratio of atrial duration (Ad) to atrial reversal duration (ARd). At this state, patient is usually mildly symptomatic with exertional activities with normal filling pressure and normal LA dimension. Pseudonormalization refers to normal appearance of mitral flow (E: A ratio between 1.0 and 1.5, tDec 180-220, IVRT 80-100) and pulmonary venous flow (S: D ratio <1, AR velocity>25, Ad/ARd<1). At stage II, the effects of impaired relaxation on early diastolic filling become opposed by the elevated left atrial pressure and the early diastolic transmitral pressure gradient and mitral flow velocity pattern return to normal. This phenomenon is called pseudonormalization to indicate that although left ventricular filling (appears normal, significant abnormalities of diastolic functions are present. In most cases, left atrial and left ventricular and diastolic filling pressure is elevated, the left atrium is increased in size and patients often complain of exertional dyspnea [11-13]. The restricted filling pattern is characterized by increased E: A ratio, decreased tDec, decreased IVRT on mitral flow, and decreased S:D ratio, increased AR velocity and Ad/ARd ratio is decreased on pulmonary venous flow. At stage III, it represents a severe decrease in LV chamber compliance. Diastolic filling pressures are elevated and patients are markedly symptomatic and demonstrate a severely reduced functional capacity. The left atrium is dilated and hypocontractile [11]. Cardiac catheterization were done only in 24 cases, of which 19 showed elevated LV end-diastolic pressure more than 15 mmHg and 5 showed normal LV end-diastolic pressure, all of them showed diastolic dysfunction by Doppler assessment. Thus, Doppler evaluation provides a noninvasive, safe and rapid bedside alternative to cardiac catheterization for the assessment of LV diastolic dysfunction [14]. Exercise tolerance tests were done in all cases that were physically active. Out of 54 cases, 24 were positive for provocable myocardial ischaemia. Among 24, group I showed 5 positive cases and group II showed 19 positive cases. Thus, there were two distinct subgroups: group I characterized by high degree of reactive hypertrophy and low incidence of ischaemia and group II with only moderate hypertrophy and high risk of ischaemia. These findings correlated well with the results of Iriate et al., [15]. Coronary angiogram (CAG) was performed in 24 ETT positive cases, out of which, 2 patients of group I was positive, 3 were normal. And 17 patients of group II were positive, 2 were normal. Five patients, 2 from group I and 3 from group II were found to have provocable myocardial ischaemia but CAG were normal. The ETT positive but CAG negative cases constitute patients of syndrome X and their iscahemia may have been caused by hypertensive microangiopathy [16]. There was no significant difference between groups for NYHA class, age, sex, heart rate, systolic blood pressure. A fourth sound was more common in group II, whereas the third sound was more frequent in group II. ECG evidenced LHV were more frequent in group I than group II, CXR evidenced cardiomegaly in group II than group I. The findings correlate well with the study of Iriak et al., [15]. Our findings indicate that patients with diastolic dysfunction in hypertensive heart disease with CHF with normal ejection presented in two ways: one characterized by severe hypertrophy and the other by a high rate of ischaemia. But their clinical profile was uniform and indistinguishable from CHF due to depressed LV systolic function as reported by Echeverria et al., [17] Doughtery et al., [18] Soufer et al., [19] and Kessher et al., [20]. Out study showed the possible influence of degree of hypertrophy or of regional myocardial ischaemia on the pathophysiologic or clinical characteristics of hypertensive heart disease results in CHF with normal ejection fraction. Therapy should be aimed at pathophysiologic regression of the hypertrophy in the first case and at improvement of ischaemia in the second case. Doppler echocardiography is an important tool in the case of such patients. It provides a noninvasive, safe and rapid bedside alternative to cardiac catheterization for the assessment of ventricular diastolic function.

CONCLUSION

Congestive heart failure from left ventricular diastolic dysfunction in hypertensive patients presents with two different profiles: one characterized by severe hypertrophy and low incidence of ischaemia and the other by high rate of myocardial regional ishaemia and low incidence of hypertrophy. They present a uniform clinical pattern, and they are indistinguishable from CHF due to low ejection fraction. Therapy should be aimed at pathophysiologic regression of the hypertrophy in the first case and at improvement of the ischaemia in the second.

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