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A diverse spectrum of cardiomyopathies including atypical variants

KEYWORDS: Poor ejection fraction, peripartum cardiomyopathy, systolic anterior motion

Dr. Sachin A Adukia	Dr. Arundhati G Diwan
Senior Resident, MD (Medicine).	MD (Medicine), Professor and Head, Department Of Medicine.

ABSTRACT *Aim and Objectives: To document the clinical profile of cardiomyopathy including age distribution, symptoms, clinical signs, electrocardiographic and echocardiographic changes. Material and methods: A descriptive analytical study was conducted at a teaching hospital over 2 years including 50 adults (age >18 years) with echocardiographic evidence of cardiomyopathy. Patients with valvular heart disease, pericardial disease, history of myocardial infarction and congenital heart disease were excluded. The echocardiographic criteria were as per 'American Society of Echocardiography' and 'American Heart Association'. Results: Mean age of presentation was 57 years (range 22 to 80 years); more in males (52%) with peak prevalence in 7th decade (26%). Commonest type was dilated cardiomyopathy (36%) due to primary causes and secondary causes (other than ischemia). Commonest presentation was breathlessness (100%) with one-third showing dyspnoea NYHA grade 4, cough (50%) and fatiguability (42%). Basal crepitations were the commonest clinical finding (72%). Electrocardiography commonly revealed left ventricular hypertrophy (72%), ST-T segment abnormalities (56%), ventricular premature complexes (32%), left bundle branch block (26%) and first degree AV block (14%). Echocardiography showed hypokinesia (segmental or global) in 78% and mitral regurgitation (isolated or as part of multivalvular involvement) in 76%. Subjects with dilated cardiomyopathy showed a dilated left ventricular cavity and severely reduced left ventricular ejection fraction on echocardiography. Hypertrophic cardiomyopathy and restrictive cardiomyopathy were also documented alongwith atypical variants like arrhythmogenic right ventricular dysplasia, takotsubo cardiomyopathy, peripartum cardiomyopathy, hypothyroid cardiomyopathy and takayasu arteritis related cardiomyopathy.*

Introduction

Cardiomyopathies are diseases of heart muscle resulting genetic defects, cardiac myocyte injury, or infiltration of myocardial tissues, insults to both cellular elements of the heart (notably the cardiac myocyte) and processes that are external to cells (such as deposition of abnormal substances into the extracellular matrix).[1] Cardiomyopathies are traditionally categorized into dilated cardiomyopathy (DCM), restrictive cardiomyopathy (RCM) and hypertrophic cardiomyopathy (HCM) variants. Predominant remodeling of the left ventricle (LV) distinguishes these categories.

Disorders associated with systemic or certain cardiac diseases are called specific heart muscle diseases. These include ischemic cardiomyopathy (ICM), valvular, hypertensive, inflammatory, metabolic, peripartum, general systemic disease, muscular dystrophies, neuromuscular disorders and toxic and hypersensitivity reactions.[2] Morphological changes of cardiomyopathy (CM) precede clinical manifestations of heart failure by months to years. Heart failure due to congestive cardiac failure (CCF), hypertension (HTN), diabetes mellitus (DM), rheumatic heart disease and obesity in the year 2000 ranged from 1.3 million to 4.6 million. These estimates however exclude causes such as alcoholic, familial, hypertrophic, idiopathic, dilated and other cardiomyopathies.[3] A clinical profile of cardiomyopathies is therefore needed as epidemiological data on cardiomyopathy in India is lacking.

Material and Methods

Approval from the Ethics committee for material and methods to be used, was procured before commencing data collection. 50 patients (age > 18 years) with echocardiographic findings of cardiomyopathy, visiting a teaching hospital over 2 years, were included after written informed consent in this descriptive analytical study.

Inclusion Criteria

- Patients presenting with 2D-Echo showing evidence of cardiomyopathy

Exclusion Criteria

- Valvular Heart Disease
- Congenital Heart Disease
- Pericardial Disease
- History of Acute Myocardial Infarction [Patients with ischemic heart disease (IHD) were included as IHD is a recognized cause of cardiomyopathy].[4]

Echocardiographic diagnostic criteria in Dilated Cardiomyopathy included:

- a dilated, poorly contractile left ventricle
- left atrial dilatation
- mitral valve motion consistent with reduced cardiac output

Echocardiographic diagnostic criteria in Hypertrophic

Cardiomyopathy included:

- Asymmetrical septal hypertrophy (ASH):- LV thickness was considered abnormal when ≥ 15 mm, and defined asymmetrical septal hypertrophy in presence of a septal to free wall thickness ratio between 1.3 and 1.5
- Systolic anterior motion of the mitral valve (SAM):- characterized by an abrupt anterior movement of the mitral valve reaching its peak before maximum movement of the posterior wall
- a small LV cavity
- septal immobility
- premature closure of the aortic valve[4,5]

The data was compiled and analyzed using Statistical Package for Social Sciences (SPSS v/s 18) and employing descriptive statistics such as frequencies and percentages.

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Results

50 cases were studied in all. DCM (other than ischemic etiology) was the commonest cardiomyopathy (36%). Causes of DCM included idiopathic, alcohol and diabetes. ICM was the second commonest cardiomyopathy (34%). Commonest age group to be affected was 7th decade (13 patients, 26%) followed closely by the 4th and 5th decade (24% each). Maximal occurrence for DCM was between 40 to 70 yrs of age (20 patients out of 27, 74%), with a similar occurrence for ICM (14 out of 17, 82%). Atypical cardiomyopathies like takotsubo cardiomyopathy, hypothyroidism related cardiomyopathy, Parkinson associated cardiomyopathy, peripartum cardiomyopathy and Takayasu arteritis related cardiomyopathy were included as part of DCM because their eventual phenotype was similar to DCM. Table 1 shows various types of atypical cardiomyopathies documented by us.

Table 1: Types of Atypical of Cardiomyopathy in the present study

Types of atypical CM*	Parkinson's CM	Takotsubo CM	Peripartum CM	Hypothyroid CM	Takayasu Arteritis realated	Total
No. of Cases	1	2	4	2	1	10

*CM- Cardiomyopathy

Females were commonly affected by DCM (15 out of 27, 56%), while males were more affected by ICM (11 out if 17; 65%). In HCM (obstructive and non-obstructive) males were predominantly affected (3 out of 4; 75%). Overall males were marginally more affected in the entire study than females (52% versus 48%).

Dyspnoea was found in all 50 patients (100%). Cough was seen 50% (more in patients of ICM- 11 of 17 patients; 65%). Fatiguability (42%), swelling of feet (40%), chest pain (38%), palpitations (20%) and syncope (10%) were other notable presenting features. New York Heart Asoociation (NYHA) Grade IV dyspnoea was overall seen in 17 patients (34%) on initial history recording with an equal number in ICM patients and DCM (8 patients each). On examination, signs of cardiac failure were more common in DCM and ICM group. Basal crepitations (72%), pedal edema (48%), heart murmur (44%), raised jugular venous pressure (JVP; 36%) and hepatomegaly (18%) were frequently appreciated.

On ECG analysis left ventricular hypertrophy (LVH) was the commonest finding (72%). ST-T changes like ST segment elevation, ST segment flattening and ST segment depression was seen in all types of cardiomyopathy (28 patients, 56%). T wave changes like T wave inversion (32%), left bundle branch block (LBBB; 26%) and 1o AV block (14%) were also seen. Other arrhythmias including atrial fibrillation (AF; 12%), supraventricular tachycardia (SVT; 12%), atrial flutter (2%) and ventricular fibrillation (VF; 2%) were recorded. Tachyarrhythmias more frequent than bradyarrhytmias.

In the DCM group (including atypical cardiomyopathy) and ICM group, chest x-ray suggestive of cardiomegaly was seen in 33 of 44 patients (75%), while pulmonary congestion is seen in 24 of these 44 patients (55%). HCM group showed no significant x-ray findings.

Echocardiography study revealed that dilated LV cavity, poor LV contractility, decreased left ventricular ejection fraction (LVEF), hypokinesia, diastolic dysfunction and valvular regurgitation were common findings in DCM and ICM group. Mitral valve (alone and isolated) was most commonly affected. Valvular regurgitation was seen in 26 (96.29%) of patients of DCM. There was isolated MR in 4 (14.81%) of patients, while MR associated with TR in 11 (40.74%), MR with AR in 2 (7.40%) of patients. There was isolated TR in 3 (11.11%) of patients. In patients of ICM valvular regurgitation was seen in 17 (100%) patients. There was isolated MR in 2 patients (11.76%), while associated MR with TR in 4 (23.52%) and isolated AR in 2 patients (11.76%). All our 4 patients of HCM showed increased thickness of inter-ventricular septum (IVS). The average septal thickness of 20.33 ± 4.04 mm in obstructive HCM was significantly higher than 17 mm recorded in our only case of non obstructive HCM. Asymmetrical septal hypertrophy (ASH) and systolic anterior motion of mitral valve (SAM) was observed in obstructive group. MR and LV outflow tract (LVOT) gradient was also observed in obstructive group. LVOT gradient was found in obstructive HCM and was more than 20mm Hg. SAM was seen in 2 of 3 patients of obstructive HCM only. Ratio of IVS: LVPW (LV posterior wall; LVPW) in obstructive group was 1.32:1 while in non obstructive HCM it is 1.7:1. LA enlargement was found significantly more in patients with obstructive HCM, while normal in non obstructive HCM. It was taken as indicator of presence of dynamic obstruction. Ejection fraction (EF) was normal in both groups, obstructive and non-obstructive.

Our only patient with RCM was 45 years female presenting with cough, breathlessness, fatigability and swelling of feet. On physical examination pulse rate was 70/min, irregular, low volume pulse. Blood pressure was 110/70 mmHg, minimal pedal edema and elevated JVP. Cardio-respiratory examination revealed apical systolic murmur with bilateral scattered crepitations. The ECG showed an axis of +10o with AF and T-inversion in V3- V6. Chest x-ray revealed mild cardiomegaly with bilateral pulmonary congestion. Echocardiography showed extraneous echodensities in LV apex with evidence of decreased EF (50%), diastolic dysfunction, LA enlargement (47 mm).

Data on peripartum cardiomyopathy showed that it affected multigravida and primigravida equally. Patients presented with higher grades of dyspnoea (NYHA grade III or IV). Symptoms began within 1 month after delivery (vaginal or Caesarean).Chest X-ray showed moderate cardiomegaly and pulmonary congestion. Echocardiography showed features of DCM with poor EF. However, the number of cases was too small (only 4 cases) to make accurate conclusions. Table 2 shows echocardiographic findings of our study. None of our patients of any group had pericardial effusion or intra-cavitatory clots.

Table 2: Echocardiographic features in Cardiomyopathy in present study

Radiological findings	TYPES OF CARDIOMYOPATHY	Total					
	DCM	ICM	HOCM	HCM	RCM	ARVD**	
LVID(d)* mm	57.29 ± 3.82	58.11 ± 3.85	42 ± 9.64	57	47	48	-
LVID (S) † mm	42.03 ± 4.68	42.7 ± 5.27	26 ± 10.39	38	33	30	-

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IVS (mm)	10.48 ± 2.22	10.41 ± 1.62	20.33 ± 4.04	17	16	9	-
LVPW ‡ (mm)	9.96± 1.74	10.79 ± 1.30	15.33 ± 3.51	10	16	9	-
LA	33.85 ± 3.76	38.29 ± 4.90	45.33 ± 10.11	18	47	24	-
Ejection Fraction (%)	29.81 ± 8.26	27.35 ± 9.20	61.66 ± 2.88	75	50	60	-
Hypokinesia	24	16	0	0	0	0	40
ASH §	0	0	3	0	0	0	3
SAM ||	0	0	2	0	0	0	2
DD ¶	11	4	2	1	2	0	20
LVOT Obstruction	0	0	3	0	0	0	3
Valvular regurgitation							
AR	3	2	0	0	0	0	5
MR	4	2	2	0	0	0	8
MR,AR	2	2	0	0	0	0	4
MR,TR	11	4	0	0	1	0	16
TR	3	1	0	0	0	0	4
MR (combined + isolated)	20	14	2	0	1	0	37

*LVID (d)- LV diameter in diastole, † LVID (s)- LV diameter in systole , ‡LVPW- LV posterior wall thickness, § ASH- Asymmetric septal hypertrophy, || SAM- Systolic anterior motion of mitral valve,¶ DD- Diastolic dysfunction, ** ARVD- Arrhythmogenic right ventricular dysplasia.

Discussion

Hollister R.M and Goodwin J.F[6] (1963) studied 52 patients with cardiomyopathy, 25 patients had DCM, 18 patients had HCM and 9 patients had RCM. This was concordant with our study. In our study, age range of HCM (obstructive and non-obstructive) was between 31-70 years with an overall mean age of 60.75 yrs and male predominance. Braunwald et al[7] (1964) found that average age of presentation for HCM among Western population was 26 years with a strong male preponderance; similar to that recorded in the apical variant of HCM by Panja[8] (1988). In our study, no patient of HCM had a positive family history. However, Marron et al [9] (1984) while studying 70 families of HCM patients, found that other members showed evidence of HCM in 39 families. Of these, 30 families had autosomal dominant transmission.

Fuster et al [10] (1981) found that symptoms of CCF were present in 73% patients of DCM and symptoms of systemic emboli were present in only 4% of patients. Schoeller et al [11] (1993) found dyspnoea NYHA grade II-40%, grade III-36% and grade IV-24%. Rihal et al[12] (1994) have found that out of 102 patients 30 patients had NYHA dyspnoea grade 1, 28 grade II, 35 grade III and 9 grade IV. Anderson et al [13] (1995) studied 83 patients of idiopathic DCM and found higher NYHA grade of dyspnoea (III or IV). In India, Parale et al [14] (2001) have reported breathlessness in 100% patients; consistent with our study. However, we recorded an overall higher NYHA grade of dyspnoea in majority patients with DCM or ICM (30 patients out of 44; 68%) compared to other studies. In the present study 5 patients and 3 patients of DCM and ICM group respectively, had syncope. Viable comparative data for syncope was not available.

Breathlessness was invariable and present in all 4 patients (100%) of HCM. 2 patients had NYHA grade 1 dyspnoea, while grade II dyspnoea and grade III dyspnoea was seen in 1 patient each. History of syncope was present in all 3 patients of HCM – obstructive type. Braunwald et al [7] (1964) have found that out of 64 patients of HCM (obstructive and non-obstructive type), breathlessness was present in 83% cases, chest pain 39%, palpitation 17% giddiness 34% and syncope in 26% cases. Godwin et al [15] (1964) found that out of 29 patients of HCM, breathlessness was present in 69%, chest pain 38%, palpitation 13.8% and syncope in 41% cases. Shapiro et al [16] (1983) studied 39 patients of HCM and reported breathlessness in 59%, chest pain 47%, palpitation 31% and syncope in 23% cases.

In the present study, signs of right heart failure like raised JVP, congestive hepatomegaly, ascites and pedal oedema were observed in less than half the patients of DCM and ICM both. On cardio-respiratory examination in patients of DCM, systolic murmur was appreciated in 13 (48%), gallop rhythm in 3 (11%), precordial thrill in 2 (7.40%) and 19 (70%) patients had respiratory crepitations. Likoff et al [17] (1987) found that 86% patients had S3 and 73% had evidence of right sided failure. Dec et al [1] (1994) found right heart failure in fewer than 50% of patients. Parale et al [14] (2001) observed similar findings as ours.

In our study, systolic murmur and precordial thrill was present in 1 patient of obstructive HCM while the only patient of non-obstructive HCM had no physical findings.

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Godwin et al [15] (1964) found that out of 29 cases of HCM, 26 (90%) had murmur, 23 (79%) had LV apex, 10 (34%) had thrill and 20 (69%) had third or fourth heart sound. Braunwald et al [7] (1964) reported that out of 64 cases of HCM, all had murmur (100%), 33(51%) had LV apex, 45(73%) had thrill and 38(59%) had third or fourth heart sound.

ECG profiling of DCM patients by Wilensky et al [18] (1988) revealed a range of QRS axis from -130o to + 130o; mean QRS axis was -21o. Parale et al [14] (2001) reported left axis deviation (LAD) in 59% patients of DCM. Techuan Chou mentions LAD in 42% patients of DCM. Findings in the present study were comparable with above studies. Out of 27 patients of DCM in our study, normal axis was seen in 19 (70%), left axis deviation 8 (27%). None of our DCM patients had RAD. Wilensky et al [18] (1988) found mean PR interval 0.20 sec. Schoeller et al [11] (1993) found first degree AV block in 18% and second degree AV block in 11% patients. These compared with our recordings of 1st degree AV block in 2 (7%) of DCM and 5 (29%) in ICM. We recorded LVH without strain in 44% (12/27) of DCM and 47% (8/17) of ICM patients. Momiyama et al (19) (1994) reported 69% LVH on ECG.

In our study, LBBB was seen in 9 (33%) DCM patients; ICM subjects showed LBBB in 3 (18%) and RBBB in 1 patient (6%). Likoff et al [17] (1987) found LBBB in 22% patients, Barbosa et al [20] (1989) reported LBBB in 25% and RBBB in 4.4%, Schoeller et al [11] (1993) found LBBB in 41% and RBBB in 3.5%. This data was concordant with our data. –we observed that 'P' waves were absent in 6 patients with AF and 1 with VF. Various investigators including Likoff (1987), Barbosa (1989), Wilensky (1988), Schoeller (1993), Anderson (1995) and Parale (2001) all had AF and ventricular premature complexes (VPCs) as the commonest arrhythmias.(11,13,14,17,18,20)

Our study included 4 HCM patients, and QRS axis was normal seen in all 4 with ST- T and T wave changes in 1 patient each (25%) and LVH in 1 (25%). No conduction abnormalities were seen in any of them. Savage et al [21] (1978) found ST changes in 81%, LVH in 64%, LA enlargement in 48% and abnormal Q wave in 33% patients on ECG. Shapiro et al [16] (1983) found ST changes in 54% and LVH in 87% patients.

In our study, out of 27 patients of DCM, 16 patients (59.25%) showed evidence of cardiomegaly with cardio thoracic ratio (CT ratio) more than 0.5. Out of 17 patients of ICM, 12 patients (70.58%) had evidence of cardiomegaly. Fuster et al [10] (1981) studied 104 patients of DCM; only 24 of these survived at the end of follow up (6-20 years). They found that 86% of the patients died had a CT ratio >0.55, where only 14% of those alive had a CT ratio >0.55. Cardiomegaly and pulmonary congestion was absent in all our 4 patients of HCM. In contrast, Godwin et al [15] (1964) found cardiomegaly in chest x-ray 15/29 patients with obstructive HCM. Table 3 and table 4 compare our echocardiographic findings with 2 earlier studies. According to table 3, LV dimensions were lesser, but all patients had hypokinesia and poor LVEF which is comparable to other studies.

Table 3: Comparison of echocardiography parameters between previous studies and present study including LV diastolic and systolic dimensions, and LVEF.

Study	LVID d (mm)	LVID s (mm)	Ejection Fraction (%)
Rihal et al (1994)	69+/-9	60+/-9	23+/-9
Anderson et al (1995) [13]	68+/-3.9	43+/-4.64.6	21+1-9.1
DCM (present study)	57.29 ± 3.82	42.03 ± 4.68	29.81 ± 8.26
ICM (present study)	58.11 ± 3.85	42.7 ± 5.27	27.35 ± 9.20

As per **table 4,** compared with other studies LVPW and IVS were within normal limits. Abbasi et al [22] (1972) had concluded that posterior left ventricular wall motion was markedly reduced in dilated cardiomyopathy.

Table 4: Comparison of echocardiography parameters between previous studies and present study including LV posterior wall thickness and IV septal thickness.

Study	LVPW (mm)	IVS (mm)
Abbassi et al (1972)	8.0+/-2 (mm)	-
Anderson et al (1995)	9.0+/-1.7	-
DCM (present study)	9.96± 1.74	10.48 ± 2.22
ICM (present study)	10.79 ± 1.30	10.41 ± 1.62

In a study done by Karl et al [23] (1998) mean left atrial diameter was 47+/-7mm which is more than the mean LA dimension in this study.

Karl et al [23] (1998) found that MR was present in 89% of the patients of cardiomyopathy. Kono et al [24] (1991) concluded that increased LV sphericity was an important factor in evolution heart failure. In the present study, 11 (41%) of the DCM group and 4 (15%) of the ICM group showed diastolic dysfunction. Anderson et al [13] (1995) observed presence of diastolic dysfunction even in patients without LV dilatation.

Gilbert et al [25] reported septal thickness in HCM patients (20.7mm) which was significantly higher than normal (8.7mm). He found mean septal size (mm) in patients with various groups: obstruction at rest-24.8 mm, latent obstruction-20.2 mm and no obstruction-17.1 mm. He also noted higher incidence of ASH and higher ratio of IVS: LVPW in obstructive group as compared with non obstructive group. He found LA enlargement significantly more often in patients with obstruction. He also documented severe SAM in all patients with obstruction at rest and in no patients without obstruction. All these parameters compared to our findings.

Since we had only one patient of restrictive cardiomyopathy, we found it inadequate for comparative studies. 4 cases of peripartum cardiomyopathy were recorded by us, all of whom showed favorable outcome on serial follow up.

To conclude, commoner types of cardiomyopathies can be diagnosed by basic investigations like echocardiography and clinical suspicion index. Potential for pharmacological response and maintenance justifies further research into this entity.

Limitations of our study were that the sample was too small to generalize the results. Cardiac investigations including electrophysiological studies would have added value to the study. Also, prognosis could not be ascertained as this study was cross sectional.

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