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Comprehensive analysis of clinico-etiological features, imaging studies and patient outcomes in cardiomyopathy

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Abstract

Background: Cardiomyopathy was an anatomic and pathologic diagnosis was associated with muscle or electrical dysfunction of the heart. The American Heart Association (AHA) defines cardiomyopathy as a heterogeneous group of diseases of the myocardium, usually with inappropriate ventricular hypertrophy or dilatation. This study aimed to evaluate for etiological factors of cardiomyopathy among hospitalized patients and to correlate the clinical findings with electrocardiographic and echocardiographic findings.

Methods: A Cross-sectional study was conducted among 55 patients admitted with cardiomyopathy to the department of general medicine of the hospital for 18 months. All eligible patients had undergone relevant investigations including Echocardiogram, Doppler study, 12 lead electrocardiogram, chest radiogram and coronary angiogram whenever indicated and possible. Correlation of etiological factors with echocardiographic and electrocardiographic changes were done.

Results: Out of 55 cases 90% of the cases were presented with complaints of dyspnea, 33% of them had chest pain, 25% had fatiguability and 20% had palpitations. Half of the cases had Idiopathic Dilated Cardiomyopathy (51%). The most common etiological factor of cardiomyopathy was Non ischemia (51%). Echocardiographic findings of the cases at the time of admission are <35% of Ejection fraction are seen among 71%.Echocardiogram revealed that both Mitral regurgitation and tricuspid regurgitation mostly seen among Idiopathic dilated cardiomyopathy. Left ventricular thrombus was seen in Idiopathic DCM and Diastolic dysfunction was seen mainly in Idiopathic DCM and highest LVEF was seen among HOCM and Least LVEF was seen among Alcoholic Cardiomyopathy.

Conclusions: In the present study, the most common presenting symptoms were dyspnea, chest pain, fatiguability and palpitations. Half of the cases are of Idiopathic Dilated Cardiomyopathy followed by ischemic Dilated Cardiomyopathy. Two-dimensional echocardiography has been the most used, efficient and accessible technique for establishment of the diagnosis of cardiomyopathies.

Limitations: current study was a cross-sectional study and sample size and sampling method was purposive which was why the generalisability of results were questionable

Keywords: electrocardiography, echocardiography, clinical profile, risk factors, Tertiary care centre

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1. INTRODUCTION

Cardiomyopathy is an anatomic and pathologic diagnosis was associated with muscle or electrical dysfunction of the heart. The American Heart Association (AHA) defines cardiomyopathy as a heterogeneous group of diseases of the myocardium, usually with inappropriate ventricular hypertrophy or dilatation.[1]There are various causes of cardiomyopathy, most of which are genetic. Cardiomyopathy may be confined to the heart or may be part of a generalized systemic disorder, often leading to cardiovascular death or progressive heart failure–related disability.[1] In 2006, the AHA classified cardiomyopathies as primary (i.e., genetic, mixed, or acquired) or secondary (e.g., infiltrative, toxic, inflammatory).[1] The four major types are dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy.

Dilated cardiomyopathy, the most common form, affects five in 100,000 adults and 0.57 in 100,000 children.[2,3] It was the third leading cause of heart failure in the United States behind coronary artery disease (CAD) and hypertension.[1] Hypertrophic cardiomyopathy, the leading cause of sudden death in athletes, was an autosomal dominant disease with an incidence of one in 500 persons.[1,4] Restrictive cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy are rare, and their diagnoses require a high index of suspicion.

In adult population the prevalence of heart failure was estimated to be about 1 to 1.5%. The mortality and morbidity remain high (median survival of 3.2 years for women and 1.7 years for men).Up to 25% of all cases of CHF was caused by Dilated cardiomyopathy. The incidence and prevalence of CHF due to cardiomyopathy appears to be increasing. The incidence of DCM was reported to be 5 to 8 cases per 1,00,000 population per year. When compared to females DCM was 3 times more common in males. The frequency of occurrence was also more common in blacks[5]

With the advancement in molecular genetics and identification of underlying etiologies, dilated cardiomyopathy was being mentioned as a specific diagnosis and not by exclusion. The most common indication for cardiac transplantation in west was DCM.[6,7] Hypertrophic, dilated, and restrictive cardiomyopathy may each present with signs and symptoms that are common in heart failure with reduced ejection fraction, including peripheral edema, fatigue, orthopnea, dyspnea on exertion, paroxysmal nocturnal dyspnea, presyncope, syncope, and cardiac ischemia.[1,8,9] In certain instances, symptoms suggest one type of cardiomyopathy over another. Primary care physicians needed to recognize these symptoms and pursue appropriate diagnostic measures, beginning with electrocardiography and echocardiography.

The current study aimed to assess the risk factors, clinical features, electrocardiographic and echocardiographic correlation in patients admitted with cardiomyopathy.

Aims and Objectives

1. To evaluate for etiological factors of cardiomyopathy among hospitalized patients.
2. To correlate the clinical findings with electrocardiographic and echocardiographic findings.

2. MATERIAL AND METHODS

A Cross-sectional study was conducted among all patients admitted with cardiomyopathy to the department of general medicine of the hospital for 18 months. The sample size was calculate based on a study previously done by PK Padhi et al[6], the prevalence of 17.64%. Using the

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formula $4pq/l^2$ the sample size calculated was 55 (at 95% confidence interval and 10% absolute precision).

Inclusion Criteria: All patients with cardiomyopathy admitted to department of general medicine of the hospital

Exclusion Criteria: Patients with primary heart disease such as valvular heart diseases and other structural heart diseases. Patients with congenital heart diseases.

Tools used in the study: 12 lead ECG, ABG Analysis, Chest Radiogram, Complete Hemogram, Fasting blood sugar, Postprandial blood sugar and HBA1C, Serum urea and creatinine, Serum Electrolytes, Creatine kinase , CKMB, Troponin, Pro BNP, Liver function test, Thyroid function tests, 2D Echocardiography, Doppler Study

Procedure for data collection:

All eligible patients had underwent relevant investigations including 2D Echocardiogram, Doppler study, 12 lead electrocardiogram, chest radiogram and coronary angiogram whenever indicated and possible. Information was collected about age, gender, presenting complaints, past history, history of medications, clinical examination and laboratory investigations ,12 lead electrocardiogram. Trans-thoracic 2 dimensional echocardiogram and Doppler study were done according to standard protocol.To correlate etiological factors with echocardiographic and electrocardiographic changes.

Diagnostic criteria for RVH

- Right axis deviation of +110° or more
- Dominant R wave in V1 (>7mm tail or R/S ratio >1)
- Dominant S wave in V5 or V6 (>7mm deep or R/S <1).
- QRS duration <120ms (i.e changes not due to RBBB).

Diagnostic criteria for LVH: The most commonly used are the Sokolov- Lyon criteria: S wave depth in V1+ tallest R wave height in V5-V6 >35mm.

The study was approved by Institutional Ethics Committee of PESIMER (Re No. PESIMER/IHEC/C-6r/2022).

The data was entered into MS excel 2007 version and further analysed using SPSS version 2.0 Descriptive statistics were analyzed as follows: The categorical data was analyzed using percentages and the continuous data was analyzed using mean and standard deviation. Inferential statistics were analyzed as follows: Chi-square test, _t 'test etc. Was used. A probability value of <0.05 has been considered as statistically significant.

3. RESULTS

In the present study the cases fulfilling the inclusion criteria were 55. Out of 55 cases more than half (51%) of the cases are 41-60 years, 31% of them are elderly age(>60years)and 18% of the cases are 18-40 years. The mean and standard deviation of the age is 54.5±15.2 years.out of 55 cases more than $2/3^{rd}$ (69%) of the cases are males.

Table 1: Distribution of clinical features, past and personal history, physical examination findings, thyroid profile and outcome

Clinical features		Frequency	Percentage
Presenting clinical features	Palpitations	11	20
	Dyspnea	50	90
	Fatiguability	14	25
	Chest pain	18	33

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Past history	Diabetes Mellitus Hypertension Ischemic Heart Disease	21 26 3		38 47 5	
Personal history	Alcohol Smoking	16 24		29 44	
Physical examination findings	Icterus Anemia Pedal edema	2 13 21		3 24 38	
Raised JVP	Yes No	19 36		35 65	
Heart sounds		S1	S2	S1	S2
	Loud	1	4	1	7
	Muffled	8	8	15	15
	Variable	6	1	10	1
	Soft	1	0	1	0
	Normal	39	42	71	76
Murmurs	Normal ESM PSM	30 18 7		55 32 13	
Thyroid Function test	Hyperthyroidism Hypothyroidism TSH mildly elevated Normal	1 1 1 52		2 2 2 94	
Outcome	Death Discharged	4 51		7.3 92.7	

In the present study out of 55 cases 90% of the cases are presenting with complaint of dyspnea, 33% of them have chest pain, 25% have fatiguability and 20% have palpitations.47% had previous history of hypertension, 38% were diabetic and 5% of the cases had Ischemic heart disease.44% of them had habit of smoking and 29% of them were alcoholic. On physical examination 38% of the cases had pedal edema, 24% had anemia and 3% have icterus.

Out of 55 cases increased JVP was seen among 35% of the cases. Out of 55 cases 15% of the cases have muffled, 10% of the cases have variable and 1% of the cases have loud and soft S1 sounds. The S2 Heart sounds are 15% muffled 7% loud and 1% variable in distribution. 71% of the cases have normal S1 and 76% of the cases have normal S2 heart sounds.

More than Half (55%) of the cases had normal heart sounds ,no murmurs were heard.32% of them have Ejection systolic murmur and 13% of them had Pan systolic murmur.Only 6% of the cases shown altered thyroid hormone levels like Hypothyroidism (2%), Hyperthyroidism (2%), TSH mildly elevated (2%), and Remaining 94% of them had normal thyroid hormone levels. Outcome of the cases after treatment and follow up 92.7% of them are discharged, 7.3% of them were died. (Table 1)

Table 2: Distribution of types of Cardiomyopathy

Types of Cardiomyopathy	Frequency	Percentage
Idiopathic Dilated Cardiomyopathy	28	51
Ischemic Dilated Cardiomyopathy	13	24

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Peripartum Cardiomyopathy	2	4
Viral Myocarditis	4	7
Alcoholic Cardiomyopathy	2	4
Hypertrophic cardiomyopathy	3	5
Cirrhotic cardiomyopathy	0	0
Hypothyroidism induced cardiomyopathy	1	2
Inflammatory Cardiomyopathy	1	2
VPC Induced Cardiomyopathy	1	2

Half of the cases are of Idiopathic Dilated Cardiomyopathy (51%)followed by Ischemic Dilated Cardiomyopathy (22%) Viral myocarditis (7%) then Hypertrophic cardiomyopathy (5%), Peripartum cardiomyopathy and Alcoholic cardiomyopathy (4%) and least of them 2% are Hypothyroidism induced cardiomyopathy, Inflammatory Cardiomyopathy, VPC Induced Cardiomyopathy. (Table2)

Table 3: Etiological factors of Cardiomyopathy

Etiology factors of Cardiomyopathy	Frequency	Percentage
Ischemic	13	24
Hypertrophic	3	5
Alcohol	2	4
Peripartum	2	4
Hypothyroidism	1	2
Idiopathic	28	51
Viral	4	7
Diabetic	0	0
VPC induced	1	2
inflammatory	1	2

The most common etiological factor of cardiomyopathy is Idiopathic (51%), Ischemic cause is seen among 24% followed by viral (8%),hypertrophic (5%), alcohol and peripartum (4%) each and VPC induced, hypothyroidism & inflammatory (2%)each are the etiological factors seen in our present study. (Table 3)

Table 4. Echocardiographic findings in patients with cardiomyopathy

Echo findings	Idiopathic DCM No. (%)	ID CM No. (%)	PP CM No. (%)	Viral No. (%)	Alcoholic No. (%)	HO CM No. (%)	Hypothyroidism No. (%)	Inflammatory No. (%)	VPC induced No. (%)	Total
LVEF <35 %	29%	30%	30%	28 %	23%	47%	33%	35%	25%	30 %

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

LVEF >35 %	9%	6%	7%	6%	4%	10%	0	0	0	9%
DD	4 (36)	3 (28)	1 (9)	1 (9)	1 (9)	1 (9)	0	0	0	11(20)
CLOT	1 (100)	0	0	0	0	0	0	0	0	1 (2)
MR	18 (49)	10 (27)	2 (5.5)	2 (5.5)	2 (5.5)	1 (3)	1 (3)	0	1 (3)	37(67)
TR	21 (48)	21 (48)	2 (4.5)	4 (9)	2 (4.5)	1 (2)	1 (2)	1 (2)	0	44 (80)

Echocardiogram revealed that both Mitral regurgitation and tricuspid regurgitation mostly seen among Idiopathic dilated cardiomyopathy. Clot was seen in Idiopathic DCM and Diastolic dysfunction was seen mainly in Idiopathic DCM and highest LVEF was seen among HOCM and Least was seen among Alcoholic Cardiomyopathy. (Table 4)

Table 5. Electro cardiogram findings in patients with cardiomyopathy

Echo findings	Idiopathic DCM	IDCM	PPCM	Viral	Alcoholic	HOCM	Hypothyroidism	Inflammatory	VPC induced	Total
VPCs	2	0	1	2	0	0	0	0	1	6
LBBB	2	2	0	0	0	0	0	0	0	4
RBBB	0	1	0	0	0	0	0	0	0	1
LVC and PRP	1	0	0	1	0	0	1	0	0	3
ST depression	0	1	0	0	0	0	0	0	0	1
LAHB	0	0	0	0	0	0	0	0	0	0
Heart blocks	0	0	0		0	0	0		0	0
LVH with strain	1	0	0	0	0	1	0	0	0	2
T wave invesrion	2	1	0	1	1	1	0	0	0	6
Atrial flutter/Fibrillation	3	3	0	0	0	0	0	0	0	6

Most common ECG abnormality was VPCs (Ventricular premature beats), T wave inversions and Atrial flutter/fibrillation observed in11% of patients, followed by LBBB(7%),Lowvoltage complexes and Poor R wave progression.(LVC&PRP) observed in 5% of the patients. (Table 5)

Table. 6 Outcome in the various cardiomyopathy cases

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

Types of Cardiomyopathies	Outcome	
	Discharges No. (%)	Death No. (%)
Idiopathic Dilated Cardiomyopathy	26 (51)	2 (50)
Ischemic Dilated Cardiomyopathy	12 (24)	1 (25)
Peripartum Cardiomyopathy	2 (4)	0
Viral Myocarditis	3 (6)	1 (25)
Alcoholic Cardiomyopathy	2 (4)	0
Hypertrophic cardiomyopathy	3 (6)	0
Hypothyroidism induced cardiomyopathy	1 (2)	0
Inflammatory Cardiomyopathy	1 (2)	0
VPC Induced Cardiomyopathy	1 (2)	0

Outcome of the cases after treatment and follow up were 51 cases were discharged. Out of 51, 26 (51%) are NIDCM type and 12(24%) are IDCM type. Death was seen among NIDCM, IDCM, Viral types of cardiomyopathies. (Table 6)

4. DISCUSSION

A cross-sectional study was conducted in the department of general medicine of tertiary care hospital in Andhra pradesh to assess risk factors, clinical features, electrocardiographic and echocardiographic correlation in patients admitted with cardiomyopathy. In the present study the cases fulfilling the inclusion criteria are 55. Out of 55 cases more than half (51%) of the cases are 41-60 years, 31% of them are elderly age(>60years) and 18% of the cases are 18-40 years. The mean and standard deviation of the age was 54.5±15.2 years.it was observed that the Out of 50 cases who fulfilled clinical and echocardiographic criteria for DCM, 28 (56%) were male and 22 (44%) were female, with a male: female ratio of 1.27:1.

In the present study out of 55 cases 90% of the cases had presented with complaints of dyspnea, 33% of them had chest pain, 25% had fatiguability and 20% had palpitations. In the study by Behera et al[10]it was observed that Dyspnea was the most common presenting clinical feature (n=43, 86%), followed by palpitation (n=14, 28%), peripheral edema (n=10, 20%), and syncope (n=8, 16%). Two cases were presented with left hemiparesis (embolism). Out of 43 cases presenting with dyspnea, 17 cases (39.53%) belonged to NYHA Class-IV, 15 cases to NYHA Class-II (34.88%), and 11 cases to NYHA Class-III (25.58%).

Out of 55 cases 47% had previous history of hypertension, 38% were diabetic and 5% of the cases had ischemic heart disease. In the study by M. Haghjoo et al[11] Of the HCM patients investigated in this study, 9% had a family history of premature SCD and 5% exhibiting a malignant‘family history (presence of at least two affected first-degree relatives).

Half of the cases are of Idiopathic Dilated Cardiomyopathy (51%)followed by ischemic Dilated Cardiomyopathy (22%) Viral myocarditis (7%) then Hypertrophiccardiomyopathy (5%), Peripartum cardiomyopathy and Alcoholic cardiomyopathy (4%) and least of them 2% are Hypothyroidism induced cardiomyopathy, Inflammatory Cardiomyopathy, VPC Induced Cardiomyopathy. In Jain et al[12] study ischemic cardiomyopathy comprised 37% of cases followed by idiopathic dilated cardiomyopathy seen in 30% of patients, 14.5% were alcoholic cardiomyopathy, 30% were idiopathic, 7.8% diabetic cardiomyopathy, 7% peripartum cardiomyopathy.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

The most common etiological factor of cardiomyopathy was Idiopathic (51%), ischemic cause was seen among 24% followed by viral (8%),hypertrophic (5%), alcohol and peripartum (4%) each and VPC-induced, hypothyroidism & inflammatory (2%)each are the etiological factors seen in our present study. In the study by Behera et al[10] it was observed that Out of 50 cases, 40 cases were idiopathic for which an etiology could not be found out by history or investigations. Ten cases belonged to non-familial/secondary type of DCM of which two were peripartum cardiomyopathy. Pacheco OE et al[13]studied 91 new patients with the diagnosis of dilated cardiomyopathy. ischemic cardiomyopathy was identified as the most common cause of dilated cardiomyopathy, representing 37% of the patient and an idiopathic etiology was found in 22% of the patients. In the present study, 21.8% patients had ischemic cardiomyopathy.

Echocardiogram revealed that both Mitral regurgitation and tricuspid regurgitation mostly seen among Idiopathic dilated cardiomyopathy. Clot was seen in Idiopathic DCM and Diastolic dysfunction was seen mainly in Idiopathic DCM and highest LVEF was seen among HOCM and Least was seen among Alcoholic Cardiomyopathy.

In the study by Behera et al[10]it was observed that out of 50 cases, eight cases were presented with syncope out of which seven were associated with severe LV dysfunction. On applying the Fisher exact test to test the association of syncope with severity of LV function, with probability of significance (0.05). Hence, the association of syncope and severity of LV function was statistically significant and so syncope can be considered as poor prognostic factor

In the Genetic studies among large families demonstrated that morphological LV hypertrophy reaches a plateau at the third decade of life in b-myosin heavy chain and in a-tropomyosin mutations, whereas it increases continuously thought life in cardiac myosin binding protein C mutations.[14] The changes in cardiac morphology observed in some adults with HCM occur in the context of development of systolic dysfunction (defined as LV ejection fraction.[15] This phase defined end stage (ES) HCM was a cause of progressive heart failure and was characterized by substantial cardiac remodeling and gradual evolution from the typical hypertrophied, non-dilated, and hyper-dynamic state to one of systolic dysfunction. ES diagnosis was primarily dependent on ejection fraction <50%, and ES commonly does not present as a dilated cardiomyopathy, with only almost 50% of patients showing associated LV cavity enlargement or regression in wall thickness; a small proportion of ES patients even demonstrate persistent marked hypertrophy with non-dilated left ventricle. [16]

Using these criterions McKenna et al[17] reported right ventricular hypertrophy in 44% of 73 patients with HCM. More recently, Maron at al found[18] in 46 HCM patients studied by cardiac MRI, that right ventricular mass was increased in the majority of them. To date, the clinical and prognostic significance of right ventricular hypertrophy was not known. The hallmark of HCM, left ventricular hypertrophy (LVH), can appear at virtually any age and increases or decreases dynamically throughout life.[19,20] Studies suggest that extreme LVH occurs more frequently in the young, i.e. in puberty or early adulthood.[21,22] A modestly decreasing magnitude of LVH with increasing age has also been found, however, this inverse relation was largely gender-specific for women.[23] These studies suggest that LVH was a dynamic feature of HCM, although all studies were cross-sectional in design.

Most common ECG abnormality was VPCs (Ventricular premature beats), T wave inversions and Atrial flutter/fibrillation observedin11% of patients, followed by LBBB (7%), Low voltage complexes and Poor R wave progression. (LVC & PRP) observed in 5% of the patients. Repolarization abnormalities are frequently reported. In Steffel et al.,[24] they were the most common ECG abnormality (72%). They occurred secondary to LBBB and ventricular

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

hypertrophy but were also isolated. In our series, 29.1% had an isolated repolarization abnormality; we unlikely observed no correlation between segmental involvement and localization of repolarization abnormalities. Steffel et al.,[25] report that sustained monomorphic VT was rarely induced, even with isoproterenol infusion, and no specific electrocardiographic or echocardiographic finding was predictive of VT inducibility. Zakhary et al.,[26] found that left ventricular noncompaction was associated with a higher incidence of ventricular arrhythmias, even in patients with preserved ejection fraction. Similarly, our patients with VT had a normal ejection fraction without fibrosis. Their coronary angiographies were also normal.

Outcome of the cases after treatment and follow up are 48 cases are discharged. Out of 51, 26 are Id DCM type and 12 are IDCM type. Death was seen among Id DCM, IDCM, Viral types of cardiomyopathies. In the studies done in highly selected patient populations of severely affected patients treated in tertiary referral centres. These populations underrepresented clinically stable and asymptomatic patients.[27,28] Recent reports with less referral bias indicate overall annual mortality rates from HCM of 1 to 2% (SCD and heart failure related death). Annual mortality from SCD alone in HCM patients was 0.4 to 1%.117.

In the present study most common presenting symptoms were dyspnea, chest pain, fatiguability and palpitations. Half of the cases are of Idiopathic Dilated Cardiomyopathy followed by ischemic Dilated Cardiomyopathy.Echo- cardiographic findings of the cases at the time of admission are <35% of EF are were common, >35% EF. TR, MR seen among most cases. Most common ECG abnormality was VPCs (Ventricular premature beats), T wave inversions and Atrial flutter/fibrillation followed by LBBB(7 %), Low voltage complexes and Poor R wave progression. Death was seen among NIDCM, IDCM, Viral types of cardiomyopathies.

5. REFERENCES:

1. Maron BJ, Towbin JA, Thiene G, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. Circulation. 2006;113(14):1807–1816.
2. Dec GW, Fuster V. Idiopathic dilated cardiomyopathy. N Engl J Med. 1994;331(23):1564–1575.
3. Towbin JA, Lowe AM, Colan SD, et al. Incidence, causes, and outcomes of dilated cardiomyopathy in children. JAMA. 2006;296(15):1867–1876.
4. Rosamond W, Flegal K, Friday G, et al. Heart disease and stroke statistics—2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee
5. Anderson KM, Kannel WB. Prevalence of congestive heart failure in Framingham Heart study subjects. Circulation 1994;13: S107-S112.
6. Kothari S, Aajesh A, Saxena A, Juneja R. Dilated cardiomyopathy in Indian children. Indian Heart J 2003; 55 : 147.
7. WHO / ISFC. Task force on cardiomyopathies. Report of the WHO / ISFC. Taskforce on the definition and classification of cardiomyopathies. Br. Heart journal1980 ; 44 : 672-673.
8. Luk A, Ahn E, Soor GS, Butany J. Dilated cardiomyopathy: a review. J ClinPathol. 2009;62(3):219-225.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

9. Nihoyannopoulos P, Dawson D. Restrictive cardiomyopathies. Eur J Echocardiogr. 2009;10(8):iii23-iii33.
10. Gersh BJ, Maron BJ, Bonow RO, Dearani JA, Fifer MA, Link MS, et al. 2011ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am CollCardiol 2011; 58: 2703-2738 [PMID: 22075468 DOI: 10.1016/j.jacc.2011.10.825].
11. Ammash NM, Seward JB, Bailey KR, Edwards WD, Tajik AJ. Clinical profile and outcome of idiopathic restrictive cardiomyopathy. Circulation 2000; 101: 2490-2496 [PMID: 10831523 DOI: 10.1161/01.CIR.101.21.2490]
12. Hershberger RE, Lindenfeld J, Mestroni L, Seidman CE, Taylor MR, Towbin JA; Heart Failure Society of America. Genetic evaluation of cardiomyopathy— a Heart Failure Society of America practice guideline. J Card Fail. 2009;15(2):83-97
13. Maron BJ, Epstein SE, Roberts WC. Causes of sudden death in competitive athletes. J Am CollCardiol. 1986;7(1):204-214.
14. Pacheco OE, Novoa JE, Cox RA. Dilated cardiomyopathy: a clinical review of patients evaluated at a tertiary care center in Puerto Rico. P R Health Sci J. 1995;14(4): 269–73.
15. Ahmad S, Rabbani M, Zaheer M, Shirazi N. Clinical ECG and Echocardiographic profile of patients with dilated cardiomyopathy. Indian J Cardiol2005 ; 8 : 25-29.
16. Singh G, ayyar SB, Bal BS, Arora P, Arora JS. Clinical profile of dilated cardiomyopathy – A study of 138 cases. JAPI 2002 ; 50 : 1556.
17. Routray SN , Behera M, Mishra TK, Pattnaik UK, Satapathy C, Nayak CR.Clinical profile and long term follow up of patients with dilated cardiomyopathy. JAPI 2002 ; 50 : 1495-1496.
18. Rihal E. Clinical profile of dilated cardiomyopathy. Indian J Cardiol2005; 8: 25-28.
19. Taliercio C P, Seward J B, Driscoll D J, Fisher L D, Gersh B J, Tajik A J. Idiopathic dilated cardiomyopathy in the young: clinical profile and natural history. J Am CollCardiol. 1985; 6(5): 1126–31
20. Niimura H, Bachinski LL, Sangwatanaroj S, Watkins H, Chudley AE, McKenna W, Kristinsson A, Roberts R, Sole M, Maron BJ, Seidman JG, Seidman CE: Mutations in the gene for cardiac myosin-binding protein C and late-onset familial hypertrophic cardiomyopathy. N Engl J Med 1998, 338:1248- 57.
21. Biagini E, Coccolo F, Ferlito M, Perugini E, Rocchi G, Bacchi-Reggiani L, Lofiego C, Boriani G, Prandstraller D, Picchio FM, Branzi A, Rapezzi C: Dilated- hypokinetic evolution of hypertrophic cardiomyopathy: prevalence, incidence, risk factors, and prognostic implications in paediatric and adult patients. J Am CollCardiol 2005, 46:1543-50.
22. Harris KM, Spirito P, Maron MS, Zenovich AG, Formisano F, Lesser JR, Mackey-Bojack S, Manning WJ, Udelson JE, Maron BJ: Prevalence, clinical profile, and significance of left ventricular remodeling in the end-stage phase of hypertrophic cardiomyopathy. Circulation 2006, 114:216-25.
23. Keren A, Popp RL. Assignment of patients into the classification of cardiomyopathies. Circulation. 1992;80:1622.
24. Elliott PM, GimenoBlanes JR, Mahon NG, et al. Relation between severity of left-ventricular hypertrophy and prognosis in patients with hypertrophic cardiomyopathy. Lancet 2001;357:420-4.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE9, 2024

25. Maron BJ, Piccininno M, Casey SA, et al. Relation of extreme left ventricular hypertrophy to age in hypertrophic cardiomyopathy. Am J Cardiol 2003;91:626- 8.
26. Maron BJ, Casey SA, Hurrell DG, et al. Relation of left ventricular thickness to age and gender in hypertrophic cardiomyopathy. Am J Cardiol 2003;91:1195-8.
27. Lazzeri C, Barletta G, Badia T, Capalbio A, Del Bene R, Franchi F et al. Multiparametric electrocardiographic evaluation of left ventricular hypertrophy in idiopathic and hypertensive cardiomyopathy. Ital Heart J 2005;6:304 –10.
28. Finocchiaro G, Haddad F, Knowles JW, Caleshu C, Pavlovic A, Homburger J, et al. Cardiopulmonary responses and prognosis in hypertrophic cardiomyopathy: a potential role for comprehensive noninvasive hemodynamic assessment. JACC Heart Fail. (2015) 3:408–18. doi: 10.1016/j.jchf.2014.11.011