

**CLINICO-ETIOLOGICAL STUDY OF DILATED CARDIOMYOPATHY PATIENTS
ADMITTED TO THE DEPARTMENT OF INTERNAL MEDICINE VIMSAR, BURLA:
A PROSPECTIVE OBSERVATIONAL STUDY**

Dr Bipin Kishore Kullu¹, Dr Prafulla Kumar Bariha², Dr Nayan Patel³ and Dr. Vaibhav Balakrishna Gowda⁴

¹Associate Professor General Medicine VIMSAR, Burla

²Associate Professor General Medicine VIMSAR, Burla

³Assistant Professor Department of Cardiology Medical college VIMSAR, Burla

⁴JR Medicine, General Medicine, VIMSAR, Burla

Corresponding Author:

Dr Bipin Kishore Kullu

Email id: drbkkullu@yahoo.in

Abstract:

Background: Dilated cardiomyopathy (DCM) affects heart muscles, specifically the myocardium. In DCM, the heart becomes enlarged (dilated) and weakened, leading to problems with its ability to effectively pump blood. This can result in various symptoms and complications including heart failure. **Aim:** The aim of the present study was to evaluate 100 cases of dilated cardiomyopathy and study their clinical profile (Symptomatology and Signs) and etiological profile when admitted to the medicine department of VIMSAR, Burla. **Materials and methods:** After obtaining permission from the Institutional Ethics Committee, the present study was initiated. Veer Surendra Sai Institute of Medical Sciences and Research (VIMSAR), Burla, Sambalpur, Odisha, India. This study was conducted from November 2020 to October 2022. This was a cross-sectional, observational study. This study included 100 patients who met the diagnostic criteria for DCM and were admitted to the male and female medicine wards of the Department of General Medicine at VIMSAR, Burla. **Results:** We found that females (52%) were more affected, with a higher prevalence in the middle-aged and elderly age groups. Idiopathic (33%) was shown to be the most common cause, followed by ischemia (30%). Exertional dyspnea (100%) was reported to be the most common symptom, followed by palpitation (63%). Most of them had signs of biventricular failure. Most patients exhibited sinus tachycardia, and 2D-Echo showed lower ejection fraction with global hypokinesia in all cases, with a high number of cases (60% having mitral regurgitation). All instances had cardiomegaly, with a considerable percentage having pulmonary plethora (45%). **Conclusion:** Dilated cardiomyopathy was more common in middle-aged and older women. Biventricular failure was the most common symptom. Idiopathic was shown to be the most common cause. Ultrasound showed a reduced ejection fraction with global hypokinesia in all patients, chest x-ray showed cardiomegaly, and sinus tachycardia was the most common ECG finding.

Key words: Dilated Cardiomyopathy; Heart Muscle; Heart Failure; Left Ventricular Dilation; Systolic Dysfunction; Idiopathic; Echocardiography.

INTRODUCTION:

Cardiomyopathy is a diverse category of myocardial illnesses characterized by mechanical/ventricular dysfunction and abnormal ventricular hypertrophy/dilatation. Dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, and arrhythmogenic right ventricular dysplasia are the four conventional types of cardiomyopathies. The most prevalent type of cardiomyopathy is dilated cardiomyopathy [1,2].

Dilated cardiomyopathy (DCM) is characterized by the presence of left ventricle or biventricular dilation and systolic dysfunction in the absence of aberrant loading conditions (hypertension, valvular disease), as well as ischemic heart disease [3]. Patients have systolic dysfunction and may or may not have overt heart failure symptoms. This condition is defined as either primary or secondary DCM. Primary DCM is considered idiopathic, and the diagnosis can be made only when secondary causes have been ruled out. DCM can be caused by mutations in numerous genes, including those encoding structural components of the sarcomere and desmosome. Nongenetic DCM can be caused by a variety of etiologies, including myocardial inflammation caused by an infection (usually viral), exposure to medicines, poisons, or allergens, and systemic endocrine or autoimmune illnesses. DCM is caused by myocardial dysfunction caused by a variety of etiological factors such as ischemia insult, toxins, and

metabolic reasons that directly damage the heart muscles. Decades of research have uncovered a variety of DCM etiologies, including genetic mutations, infections, inflammation, autoimmune illnesses, toxins, and endocrine or neuromuscular causes. The most common causes of DCM are idiopathic and familial disorders.

DCM occurs in around 6.95/100000 people each year, with a frequency of 0.04%. DCM is responsible for a 5- 10% yearly mortality rate [4]. DCM has numerous causes, each of which affects ventricular function to varied degrees. While most DCM patients have symptoms, a few may be asymptomatic due to compensatory processes. As the ventricles continue to grow, ventricular function declines, followed by conduction system anomalies, ventricular arrhythmias, thromboembolism, and heart failure. The sooner these people are discovered and treated, the higher their chances of survival. To assess ventricular dysfunction and unfavorable myocardial remodeling, echocardiography and other imaging techniques are required, and immunological and histological investigations of an endomyocardial biopsy sample are recommended when inflammation or infection is suspected. Since DCM eventually leads to decreased contractility, traditional techniques to prevent or treat heart failure are the first-line therapy for DCM patients. To prevent life-threatening arrhythmias, cardiac resynchronization treatment and implantable cardioverter-defibrillators may be required. Furthermore, determining the likely origin of DCM aids in tailoring specific therapy to enhance prognosis. The aim of the present study was to evaluate 100 cases of dilated cardiomyopathy and study their clinical profile (Symptomatology and Signs) and etiological profile admitted in medicine department of VIMSAR, Burla.

MATERIALS & METHODS:

After obtaining permission from the Institutional Ethics Committee the present study initiated. 21.5 0 N 83.87 0 E Veer Surendra Sai Institute of Medical Sciences and Research (VIMSAR), Burla, Sambalpur, Odisha, India. This study was conducted from November 2020 to October 2022. This was a cross-sectional, observational study. This study included 100 patients who met the diagnostic criteria for DCM and were admitted to the male and female medicine wards of the Department of General Medicine at VIMSAR, Burla. The study was carried out in the male and female medicine wards of the Department of General Medicine at VIMSAR, Burla. The current investigation used consecutive sampling techniques. individuals over the age of 18 were eligible for the trial, as were individuals with symptoms of heart failure and all patients with heart failure verified as dilated cardiomyopathy on 2D echo. ECHO criteria were LVEF 40%, LVEDD >3 cm/m², global hypokinesia of the LV with/without RV involvement, and a patient with previously diagnosed DCM on therapy presenting with heart failure. Exclusion criteria included patients with an acute myocardial infarction, any valvular heart disease, congenital heart disease, cor-pulmonale with CHF, DCM secondary to chronic renal failure, pericardial disease, hypertrophic cardiomyopathy, and restrictive cardiomyopathy. The subjects for the study were drawn from patients hospitalized to the medical wards of VIMSAR, Burla between November 2020 and October 2022 who met the criteria outlined above. Dilated cardiomyopathy was diagnosed based on the patient's history, physical findings, chest x-ray, electrocardiography, and echocardiography findings. The subjects' age (in years) and gender (male/female/third gender) were recorded. Symptoms evaluated included dyspnea (NYHA grade), palpitation, PND, chest discomfort, pedal edema, and syncope.

Basal crepitations, elevated JVP, hepatomegaly, LVS3, pansystolic murmur at apex, pansystolic murmur at tricuspid region, and SBP 100 were all noted. Proper history taking and bedside assessment of the patients; bedhead tickets; Chest X RAY results; ECG findings; and 2D ECHO findings were all noted. 100 example cases of dilated cardiomyopathy that met the criteria were chosen. After thoroughly explaining the goal, methodology, and implications of the study, the patient provided signed consent. They provided a full history, and symptom analysis was performed. A thorough clinical examination was also performed.

All patients had a typical 12-lead ECG that was evaluated. A chest radiograph was taken, which included a postero-anterior chest film (PA view). The cardiothoracic ratio, pulmonary infiltrates, pulmonary plethora, and pleural effusion were all evaluated in all instances. The cardiothoracic (C/T) ratio was calculated by measuring the thoracic diameter as the distance between the inner rib margins at the level of the dome of the right hemidiaphragm and the cardiac diameter as the horizontal distance between the most rightward and most leftward margins of the cardiac shadow. After the patient's general state was stabilized, a 2D Echocardiogram was performed on all patients at the Department of Cardiology, VIMSAR, Burla. GE VIVID E9 was used for the 2d Echo. All patients' chamber dimensions were examined for EF and global hypokinesia, and the results were interpreted. A previous history of myocardial infarction led to the diagnosis of ischemic DCM. Demakis et al. defined peripartum cardiomyopathy as (1) the development of heart failure in the last month of pregnancy or within 5 months of delivery, and (2) the

absence of recognizable heart illness prior to the last month of pregnancy. (3) Echocardiogram shows characteristic left ventricular dysfunction; (4) no other recognized causes of heart failure. Diabetic cardiomyopathy was diagnosed in patients with long-standing diabetes mellitus (>10 years) and no other clear etiology. Similarly, patients with echocardiography-proven dilated cardiomyopathy and a history of long-term (> 10 years) alcohol consumption were classified with alcoholic cardiomyopathy when no alternative explanations were detected. If no evident etiology is detected, they are classified as having idiopathic DCM. The clinical profile, as well as the possible etiology, radiographic, electrocardiographic, and echocardiographic results, were summarized and compared to previous data.

Statistical analysis:

The data was noted in excel and mean and standard deviations were calculated. Statistical significance was taken at $P < 0.05$.

RESULTS:

In our present study the following age distribution was observed. The distribution shows that the peak incidence was above 5Th decade. The mean age calculated was 54.28 ± 14.12 . Age distribution is shown in table 1 below.

Table 1: Distribution of age categories

	Age Categories	Valid Percent
<20	1	1
21-30	8	8
31-40	13	13
41-50	9	9
51-60	29	29
61-70	33	33
>70	7	7
Total	100	100

All the patients in the study presented with exertional dyspnea which comprised the predominant symptom. It was associated with PND in 94% of cases. The second most common symptom was palpitation which comprised 63% of the patients. Pedal edema was seen in 62% of the patients. Chest pain was present in 38% of the patients and syncope was present in 15% of the patients. Dyspnea was graded according to NYHA classification. Patients predominantly presented with NYHA 3 grade dyspnea which amounted to 43% which was followed by NYHA 4 and NYHA 2 with 32% and 25% respectively.

Basal crepitations were seen in about 93% of the patients. Raised JVP was seen in 69% of the patients. Hepatomegaly was seen in about 40% of the patients. On cardiac auscultation, LVS3 was heard in about 44% of the patients. Apical Pan systolic murmur was heard in 45% of the patients and pansystolic murmur at tricuspid area was heard in 20% of the patients. Mean systolic BP was calculated to be 100.9 ± 11.44 and mean diastolic BP was 61.32 ± 6.16 .

The electrocardiographic profile included QRS axis, presence of arrhythmias, presence of non-specific ST-T changes, atrial enlargement and ventricular hypertrophy. Among the patients studied, 70% of patients had normal QRS axis, 20% had left axis deviation and the remaining 10% had right axis deviation. 29% of the patients had arrhythmia, the most common being sinus tachycardia. Other arrhythmias seen were ventricular premature complexes (VPC), supraventricular tachycardia, atrial ectopic, atrial fibrillation, LBBB, RBBB. Non-specific ST-T changes were seen in 30% of the patients. 19% had isolated left atrial enlargement, 6% had isolated right atrial enlargement, 11% had bi-atrial enlargement and remaining 64% had normal atria. Left ventricular hypertrophy was seen in 22% of patients whereas 6% showed right ventricular hypertrophy and 4% had bi-ventricular hypertrophy. Rest 68% had normal ventricles.

The mean left ventricular ejection fraction was found to be 30.69 ± 6.29 . The LV ejection fraction. 59% had an ejection fraction between 30% - 40%. 37% had ejection fraction between 20% - 30% and 4% had $EF < 20\%$. The mean LVEDD was 59.46 ± 7.55 mm with majority i.e. 46% having LVEDD > 60 mm. The mean LVESD was 45.46 ± 9.25 with the majority of patients i.e. 35% having end systolic diameter > 50 mm. In our study 60% had mitral regurgitation, 25% had tricuspid regurgitation and 11% had pericardial effusion. Almost all patients showed

cardiomegaly in chest X-ray. The cardiothoracic ratio was 0.70-0.79 in 40% of cases, 0.60-0.69 in 35% of cases and >0.8 in 25% of cases. Mean C/T ratio was found to be 0.69 ± 0.08 .

The most common cause was found to be idiopathic which comprised about 33% of all cardiomyopathies which was followed by ischemic cardiomyopathy which comprised about 30% of cases. Alcoholic cardiomyopathy was seen in 8% of cases and diabetic cardiomyopathy in 7% of cases. 7% of cases of DCM were seen due to hypothyroidism. Peripartum cardiomyopathy was seen in 5% of cases whereas sickle cell cardiomyopathy and Chemotherapy induced DCM in 3% and 2% of cases respectively. Among autoimmune etiology, DCM was seen in Systemic lupus erythematosus and mixed connective tissue disease which comprised 2% and 1 % of the total cases. Cardiomyopathy attributing rheumatoid arthritis was seen in 2% of cases.

DISCUSSION:

The present study aimed to evaluate the clinical presentation, ECG, chest X-ray, and echocardiographic abnormalities and to determine the etiology of dilated cardiomyopathy in hundred patients admitted to a tertiary care hospital in the state of Odisha. Among the 100 patients in our study, 48% were found to be males and the remaining 52% were females. Ratio of male: female was found to be 0.92: 1. The mean age at presentation in DCM was 54 ± 14.12 . The maximum age category with DCM was found to be in the range of 61 – 70 followed by 51 – 60. Only 31% of the patients had DCM under 50 years of age. The mean age at presentation of DCM in males was 56.17 ± 12.25 and in females the mean age was 52.54 ± 15.56 . A study conducted by Ahmed et al. [5] showed the male: female ratio to be 1.89, and the mean age of presentation in males was 52.9 ± 15.1 and in females 51.9 ± 17.7 . Another study by Mishra et al. [6] showed 68% to be males and 32 females, and the mean \pm SD age of patients was 60 ± 14 years, with the majority of patients belonging to the age group 41 – 60 years. The most common presentation in our study was biventricular failure, observed in 62% of cases. Most of the patients were NYHA class III and IV, while 25% were NYHA class II. Dyspnea was the most common symptom in almost all the patients. PND was observed in 94% of the patients. Palpitation was observed in 63% of the patients. Pedal edema was observed in 62% of patients. Pedal edema is the predominant symptom in patients with idiopathic DCM. Anasarca was observed in 10% of patients. Chest pain was observed in 38% of the patients, and most of these patients had ischemic DCM. The cause of chest pain in these patients was found to be due to chronic myocardial ischemia. Syncope was present in 15% of patients. In most cases, syncope was mainly due to low cardiac output. Our study revealed that the majority of the patients were NYHA class 3(43%), class 4 (32%), and class 2 (25%). According to a study conducted by Mishra et al. [6], the majority of patients presented with NYHA classes 2 (57%) and 3(29%). In another study by Kumar et al. in 2017 [7], most patients were in NYHA class 1 (50%) and class 2(46%), and only one was in class 3.

Raised JVP was observed in 69% of the patients secondary to RV failure. In our series, the mean systolic blood pressure was seen to be 100.9 ± 11.4 and the mean diastolic blood pressure was seen to be 61.32 ± 6.16 . The study done by Ahmed et al [5] showed mean systolic and diastolic BP of 89.7 ± 6.7 and 68 ± 7.1 mm Hg respectively and study done by Rihal et al [8] showed the systolic BP was 124 ± 19 mm Hg. Basal crackles were present in 93% of the patients in our study. Basal crackles were attributed mainly to pulmonary edema or pulmonary congestion due to LV failure. Cardiac examination revealed LVS3 in 44% of the patients in this study. PSM at the apex secondary to mitral regurgitation was observed in 45% of patients. PSM in the tricuspid area secondary to a tricuspid reaction was observed in 20% of the patients. Hepatomegaly was seen in 40% of the patients in our study secondary to congestive heart failure. In our study, biventricular failure was the most common (75%), followed by the left ventricle (25%). Study done by Satapathy et al. [9] reported biventricular failure presentation in 76% of patients, LVF in 20%, and RVF in 4 %.

The QRS axis was normal in 70% of our subjects with a left axis deviation of 20% and right axis deviation of 10%, which was in concordance with other studies, as done by Chavali et al. [10] and Ahmed et al. [5]. Arrhythmias were observed in 29 % of our patients, with sinus tachycardia being the most predominant finding, followed by ventricular premature complexes. Other arrhythmias included atrial ectopic, atrial fibrillation, LBBB, and RBBB. In a study done by Ahmed et al similar findings were corroborated. Non-specific ST-T changes were observed in 30% of patients. Earlier studies by Mestroni [11] et al and Ahmed et al [5] showed less preponderance of non-specific ST-T changes of 11.2% and 10.9%, respectively. Bi-atrial enlargement was observed in 11% of the patients. Isolated left atrial enlargement was seen in 19% of patients, and isolated right atrial enlargement was seen in 6% of patients. A study by Ahmed et al. [5] also showed similar results, wherein 25.4% of patients had left atrial enlargement, 7.2% had right atrial enlargement, and 7.2% had bi-atrial enlargement. Ventricular hypertrophy was observed in 32 % of

our patients with LVH in 22%), RVH in 6%), and bi-ventricular hypertrophy in 4% of patients. A study by Satapathy et al. [12] reported 15% with LVH, 4% with RVH, and 2% with both ventricular hypertrophies.

Electrocardiography revealed left ventricular dilatation (LVD). The left ventricular internal diameter in diastole (LVIDd) ranged from 45 mm to 75 mm with a mean of 59.46 ± 7.55 . The left ventricular internal diameter in systole (LVIDs) ranged from 30 to 65 mm with a mean of 45.46 ± 9.25 . Ahmed et al. [5] reported LVIDd ranging from 55 to 74 mm and LVIDs ranging from 48 to 68 mm. The mean left ventricular EF in our study group was 30.69 ± 6.29 . The majority of patients (59 %) had ejection fractions in the range of 30%-40%. This was in agreement with the mean LVEF reported by Ahmed et al. [5], which was $30.05 \pm 9.49\%$. Mitral regurgitation was seen in 60% of the patients in our study, and tricuspid regurgitation was seen in 25% of the patients. In another study by Chavali et al. [10], the MR and TR were 73.3% and 10%, respectively. Mitral and tricuspid regurgitation in dilated cardiomyopathy is secondary to annular ring dilatation. Pericardial effusion was seen in 11% of our patients as compared to a study done by Chavali et al. [10], which showed 6.6 % with pericardial effusion.

Chest radiography was found to be abnormal in almost all cases, showing varying degrees of cardiomegaly with a cardiothoracic ratio (C/T) varying between 0.5 to 0.9. The mean C/T ratios were 0.69 ± 0.08 . Pleural effusion was observed in 19% of the patients in our study. Pulmonary plethora was found in 45% of the patients in our study. Similarly in a study by Kalra et al [11] 100% cases had cardiomegaly with C/T ratio ranging from 0.51 to 0.8 as similar to our study. Pulmonary plethora was observed in 72% of cases and pleural effusion in 46% of cases. In another study by Ahmed et al. [5], the corresponding figures were the C/T ratio in the range of 0.5 to 0.9, pulmonary plethora in 76.3%, and pleural effusion in 10.3% of the patients.

In our study, the most common type of dilated cardiomyopathy was idiopathic, present in 33% of cases, followed by ischemic cardiomyopathy in 30% of cases. The third most common cause was alcoholic, seen in 8% of the cases. Diabetic cardiomyopathy was observed in 7% of the cases, and 7% of the cases were due to hypothyroidism. Peripartum cardiomyopathy, sickle cell cardiomyopathy, and chemotherapy-induced dilated cardiomyopathy were observed in 5%, 3%, and 2% of the cases, respectively.

Among autoimmune etiology, DCM was observed in systemic lupus erythematosus and mixed connective tissue disease, comprising 2% and 1 % of the total cases. Cardiomyopathy attributed to rheumatoid arthritis was observed in 2% of the cases. In a study conducted by Kalra et al [12] 33% of patients had ischemic cardiomyopathy, which was the most common cause, followed by 24% of patients with diabetic cardiomyopathy. Peripartum cardiomyopathy was observed in 18% of the cases, and 14% had idiopathic causes. Alcoholic cardiomyopathy comprised 6% of the cases, and the remaining 6% were attributed to miscellaneous causes.

CONCLUSION:

Females in their middle and older age groups were more likely to have dilated cardiomyopathy. Biventricular failure was the most prevalent clinical manifestation. Idiopathic disease is the most common etiology. Sinus tachycardia was the most prevalent ECG finding, echocardiography revealed a decreased ejection fraction with global hypokinesia in all instances, and chest radiography revealed cardiomegaly in all cases.

Conflict of interest:

None to declare among the present study authors.

REFERENCES:

1. De Geest B, Mishra M. Role of oxidative stress in diabetic cardiomyopathy. *Antioxidants*. 2022 Apr 15;11(4):784.
2. Wang M, Li Y, Li S, Lv J. Endothelial dysfunction and diabetic cardiomyopathy. *Frontiers in Endocrinology*. 2022 Apr 7;13:851941.
3. Lukas Laws J, Lancaster MC, Ben Shoemaker M, Stevenson WG, Hung RR, Wells Q, Marshall Brinkley D, Hughes S, Anderson K, Roden D, Stevenson LW. Arrhythmias as presentation of genetic cardiomyopathy. *Circulation Research*. 2022 May 27;130(11):1698-722.
4. Reichart D, Newby GA, Wakimoto H, Lun M, Gorham JM, Curran JJ, Raguram A, DeLaughter DM, Conner DA, Marsiglia JD, Kohli S. Efficient in vivo genome editing prevents hypertrophic cardiomyopathy in mice. *Nature medicine*. 2023 Feb;29(2):412-21.

5. Ahmad, Sohaib Rabbani, Zaheer MS, Shirazi Nadia. Clinical, electrocardiographic, and echocardiographic profile of patients with dilated cardiomyopathy. *Indian Journal of Cardiology*. 2005 8. 25-29.
6. Mishra S, Joshi A, Paramjeet S. Clinical Profile of Patients with Dilated Cardio Myopathy. *Ann. Int. Med. Den. Res.* 2020; 6(2):ME67-ME70.
7. Kumar M, Sharma Y, Bahl A. Comparative analysis of clinical profile of patients admitted with idiopathic dilated cardiomyopathy in a tertiary care hospital. *Journal of Cardiovascular Disease Research*. 2017;8(2).
8. Rihal CS, Nishimura RA, Hatle LK, Bailey KR, Tajik AJ. Systolic and diastolic dysfunction in patients with clinical diagnosis of dilated cardiomyopathy. Relation to symptoms and prognosis. *Circulation*. 1994 Dec;90(6):2772-9.
9. Satapathy C, Gupta MK, Routray SN, Mohanty NK, Das BK. Clinical profile, angiographic characteristics in women with coronary artery disease, admitted in a tertiary care hospital of Eastern India. *J. Evid. Based Med. Healthc.* 2018;5(12):1053-8.
10. Chavali V, Tyagi SC, Mishra PK. Predictors and prevention of diabetic cardiomyopathy. *Diabetes, metabolic syndrome and obesity: targets and therapy*. 2013 Apr 11:151-60.
11. Mestroni L, Neri R, Camerini F. The electrocardiogram in dilated cardiomyopathy. *Giornale Italiano di Cardiologia*. 1986 Dec 1;16(12):1009-17.
12. Kalra PR, Cleland JG, Petrie MC, Thomson EA, Kalra PA, Squire IB, Ahmed FZ, Al-Mohammad A, Cowburn PJ, Foley PW, Graham FJ. Intravenous ferric derisomaltose in patients with heart failure and iron deficiency in the UK (IRONMAN): an investigator-initiated, prospective, randomised, open-label, blinded-endpoint trial. *The Lancet*. 2022 Dec 17;400(10369):2199-209.