



A-JMRHS

ECHOCARDIOGRAPHIC EVALUATION AND CLINICAL PROFILE OF DILATED CARDIOMYOPATHY: A CROSS-SECTIONAL OBSERVATIONAL STUDY IN A TERTIARY CARE TEACHING HOSPITAL

Dr. Mookambika. R.V¹, Dr. Vikash.M^{2*}

¹Professor, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu, India.

^{2*}Junior Resident, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu, India.

Corresponding Author: Dr.Vikash.M

Junior Resident, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu, India.

ABSTRACT

Background: Dilated cardiomyopathy (DCM) is a major cause of heart failure, arrhythmias, and sudden cardiac death worldwide. It is characterized by ventricular dilatation and impaired systolic function, leading to progressive cardiac dysfunction and significant morbidity and mortality. Echocardiography plays a vital role in the diagnosis and evaluation of DCM. The present study aimed to assess the echocardiographic findings and clinical profile of patients with dilated cardiomyopathy in a tertiary care teaching hospital.

Methodology: A cross-sectional observational study was conducted in the Department of General Medicine at Sree Mookambika Institute of Medical Sciences from December 2025 to May 2026. A total of 69 patients diagnosed with DCM based on clinical and echocardiographic criteria were included. Detailed history, physical examination, laboratory investigations, ECG, chest X-ray, and echocardiography were performed. Echocardiographic parameters including ejection fraction, left ventricular dimensions, and chamber dilatation were assessed.

Results: The majority of patients were males (71%) and belonged to the 51–60 years age group (33.3%). Dyspnea was the most common symptom (76.8%), while raised jugular venous pressure was the most common clinical sign (72.5%). Ischemic heart disease was the leading etiology (30.4%), and hypertension was the most prevalent risk factor (58%).

Conclusion: Dilated cardiomyopathy predominantly affects middle-aged males and commonly presents with features of congestive heart failure. Echocardiography remains an essential diagnostic tool for early identification and assessment of cardiac dysfunction in DCM.

Keywords: Dilated Cardiomyopathy, Echocardiography, Heart Failure, Left Ventricular Dysfunction, Cardiomyopathy, Clinical Profile.

INTRODUCTION

Dilated cardiomyopathy (DCM) is one of the most common forms of cardiomyopathy and represents a major cause of heart failure, arrhythmias, sudden cardiac death, and cardiac transplantation worldwide.

[1] It is characterized by dilation of the left ventricle or both ventricles accompanied by impaired systolic function in the absence of abnormal loading conditions such as hypertension, valvular heart disease, or significant coronary artery disease.[2] According to the European Society of Cardiology (ESC), DCM is defined by the presence of ventricular dilatation and reduced myocardial contractility resulting in left ventricular systolic dysfunction.[3] DCM predominantly affects younger and middle-aged individuals and remains one of the leading indications for cardiac transplantation in this population.[4]



www.ajmrhs.com
eISSN: 2583-7761

Date of Received: 23-05-2026
Date Acceptance: 31-05-2026
Date of Publication: 30-06-2026

The etiology of DCM is heterogeneous and includes both genetic and acquired causes. Genetic mutations involving cytoskeletal, sarcomeric, nuclear envelope, and mitochondrial proteins contribute significantly to familial DCM.[5] Non-genetic causes include viral myocarditis, alcohol abuse, nutritional deficiencies, autoimmune disorders, metabolic diseases, peripartum cardiomyopathy, and exposure to cardiotoxic drugs.[6] Despite advances in diagnostic techniques, a considerable proportion of cases remain idiopathic in origin. Irrespective of the etiology, the final common pathway involves ventricular remodeling, chamber dilatation, and progressive systolic dysfunction.[7]

DCM is clinically characterized by impaired myocardial contractility with left ventricular ejection fraction (LVEF) typically less than 40%.[8] Patients commonly present with symptoms of congestive heart failure such as dyspnea, orthopnea, fatigue, pedal edema, and reduced exercise tolerance. Some patients may present with arrhythmias, thromboembolic events, or sudden cardiac death, while others remain asymptomatic during the early stages due to compensatory mechanisms.[9] Continued ventricular enlargement eventually leads to worsening myocardial dysfunction, elevated intracardiac pressures, and advanced heart failure.[10]

The true prevalence of DCM is difficult to estimate because many patients remain asymptomatic and undiagnosed. The estimated prevalence is approximately 36 cases per 100,000 population, with a higher incidence among males compared to females.[11] In the United States, DCM accounts for nearly 10,000 deaths and approximately 46,000 hospitalizations annually.[12] The Global Burden of Disease study estimated that the worldwide prevalence of cardiomyopathy reached nearly 2.5 million cases in 2015, showing a substantial increase over the previous decade.[13] Geographic, ethnic, and socioeconomic variations further influence disease prevalence and clinical presentation.[14]

Echocardiography plays a central role in the diagnosis and evaluation of DCM. It provides important information regarding chamber dimensions, ventricular systolic and diastolic function, wall motion abnormalities, valvular regurgitation, pulmonary artery pressure, and intracardiac thrombi.[15] Echocardiography is widely available, non-invasive, cost-effective, and

remains the cornerstone imaging modality for assessing the severity and prognosis of DCM.[16] Early echocardiographic identification of ventricular dysfunction can help initiate timely medical therapy and improve clinical outcomes.

Understanding the clinical profile and echocardiographic characteristics of patients with DCM is essential for early diagnosis, risk stratification, and appropriate management. Therefore, the present study was undertaken to evaluate the echocardiographic findings and clinical profile of patients diagnosed with dilated cardiomyopathy in a tertiary care teaching hospital.

Aim

To evaluate the echocardiographic findings and clinical profile of patients diagnosed with dilated cardiomyopathy in a tertiary care teaching hospital.

Objectives

1. To study the demographic characteristics of patients with dilated cardiomyopathy.
2. To assess the common clinical presentations and associated co-morbidities among patients with dilated cardiomyopathy.

METHODOLOGY

This cross-sectional observational study was conducted in the Department of General Medicine at Sree Mookambika Institute of Medical Sciences over a study period from December 2025 to May 2026. The study was carried out to evaluate the echocardiographic findings and clinical profile of patients diagnosed with dilated cardiomyopathy (DCM). Patients admitted to or attending the General Medicine department with clinical suspicion of heart failure and fulfilling the diagnostic criteria for DCM were included in the study after obtaining informed consent.

The inclusion criteria comprised patients presenting with symptoms and signs suggestive of heart failure along with echocardiographic evidence of dilated cardiomyopathy. Echocardiographic criteria included left ventricular ejection fraction (LVEF) less than 45%, global hypokinesia of the left ventricle, dilatation of all cardiac chambers, and left ventricular end-diastolic dimension (LVEDD) greater than 3 cm/body surface area. Patients with pericardial disease, cor pulmonale with congestive heart failure, hypertrophic cardiomyopathy, restrictive cardiomyopathy, congenital heart disease,

and coronary artery disease were excluded from the study.

All enrolled participants underwent detailed clinical evaluation, including history taking and comprehensive physical examination. Demographic details, presenting symptoms, associated comorbidities, and clinical findings were recorded using a structured proforma. Laboratory investigations performed included complete blood count (CBC), blood urea, serum creatinine, serum electrolytes including sodium, potassium, and magnesium, liver function tests (LFT), fasting blood sugar (FBS), postprandial blood sugar (PPBS), glycated hemoglobin (HbA1c), and thyroid profile. Additional investigations such as electrocardiography (ECG), chest X-ray, and transthoracic echocardiography were carried out in all patients.

Echocardiographic evaluation was performed to assess cardiac chamber dimensions, left ventricular

ejection fraction (EF), left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), global hypokinesia, valvular abnormalities, and other structural cardiac changes associated with dilated cardiomyopathy. Clinical and echocardiographic findings were analyzed to determine the pattern and severity of cardiac dysfunction among the study population.

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) software version 25.0. Continuous variables were expressed as mean \pm standard deviation (SD), while categorical variables were presented as frequencies and percentages. Chi-square test was used to assess associations between categorical variables, and Student's t-test was used for comparison of continuous variables wherever applicable. A p-value less than 0.05 was considered statistically significant.

RESULT

Table No. 1. Demographic profile

Age groups (Years)	No. of patients (N)	Percentage (%)
≤ 40	9	13.1
41-50	11	15.9
51-60	23	33.3
61-70	11	15.9
71-80	13	18.9
> 80	2	2.9
Gender		
Male	49	71
Female	20	29

The total number of patients in the study was 69, with a mean age of 58.4 years (SD = 12.6). Most of

the patients were male (71%) and in the 51-60 age group (33.3%).

Table No. 2. Distribution Based on Symptoms and Signs of Dilated Cardiomyopathy

Symptoms	No. of patients (N)	Percentage (%)
Easily fatigued	41	59.4
Chest pain	37	53.6
Syncope	9	13.1
Dyspnea	53	76.8
Orthopnoea	41	59.4
Palpitation	40	58
PND	43	62.3
Pedal edema	42	60.9
Cough	41	59.4

Abdominal pain	38	55.1
Signs		
Tachycardia	40	58
Bradycardia	4	5.8
Drop beats	27	39.1
Raised JVP	50	72.5
Hepatomegaly	45	65.2
Basal crepitation	41	59.4
Murmur	11	15.9

The most common symptom was dyspnea, which affected 53 patients (76.8%), followed by PND, pedal edema, and hepatomegaly, which each affected more than 60% of the patients. The least common symptom was syncope, which only

affected 9 patients (13.1%). Among the signs, the most prevalent was raised JVP, which was observed in 50 patients (72.5%), while the least prevalent was bradycardia, which was observed in only 4 patients (5.8%).

Table 3: Distribution Based on Signs of Dilated Cardiomyopathy

Signs of DCM	No. of patients (N)	Percentage (%)
Tachycardia	40	58
Bradycardia	4	5.8
Drop beats	27	39.1
Raised JVP	50	72.5
Hepatomegaly	45	65.2
Basal crepitation	41	59.4
Murmur	11	15.9

Table 4: Distribution of Etiology and Risk Factors of Dilated Cardiomyopathy

Etiology of DCM	No. of patients (N)	Percentage (%)
Diabetic	14	20.3
Peripartum	8	11.6
Ischemic	21	30.4
Alcoholic	12	17.4
Idiopathic	14	20.3
Total	69	100
Risk factors		
Smoking	18	26.1
Alcoholism	26	37.7
Hypertension	40	58
Diabetes mellitus	22	31.9
Dyslipidaemia	19	27.5
Previous MI	9	13.1

Table 5: Distribution Based On Nyha Grades

NYHA grades	No. of patients (N)	Percentage (%)
I	0	0
II	11	15.9
III	14	20.3
IV	28	40.6
None	16	23.2

Total	69	100
-------	----	-----

The table shows how 69 patients with heart failure were classified according to the New York Heart Association (NYHA) Functional Classification, which measures the impact of the disease on their daily activities. The table reveals that none of the patients belonged to the first category, which means that they all experienced some degree of limitation in their physical activity. Most of the patients (40.6%) were in the fourth and most severe category, indicating that they had symptoms even at rest and could not perform any physical activity without

discomfort. The second most common category was the third one, with 20.3% of the patients, followed by the second one, with 15.9% of the patients. These categories meant that the patients had marked or slight limitations in their physical activity, respectively. The table also shows that 16 patients (23.2%) did not have any NYHA grade assigned, which could imply that they were either not assessed or had other conditions that affected their functional status.

Table 6: Distribution of ECG Changes Observed

ECG changes	No. of patients (N)	Percentage (%)
QRS axis		
Normal	52	75.4
Left axis deviation	11	15.9
Right axis deviation	6	8.7
ST-T changes	20	29
Atrial enlargement		
Left atrial enlargement	8	11.6
Right atrial enlargement	5	7.2
Ventricular hypertrophy		
Left ventricular hypertrophy	12	17.4
Right ventricular hypertrophy	7	10.1
Both LVH+RVH	24	34.78
Atrial fibrillation	12	17.39
Intraventricular conduction delay	20	28.98
Q-wave	8	11.59
Heart Block	8	11.59
VPC & VT	12	17.39
PSVT	4	5.79

The table also shows that some ECG changes were more prevalent than others, such as ventricular hypertrophy, atrial fibrillation, and intraventricular conduction delay. Ventricular hypertrophy, which means enlargement of the lower chambers of the heart, was observed in 43 patients (62.32%), either

in the left, right, or both ventricles. Atrial fibrillation, which means irregular and fast heart rhythm, was observed in 12 patients (17.39%). Intraventricular conduction delay, which means delayed electrical impulses in the lower chambers of the heart, was observed in 20 patients (28.98%).

Table 7: Distribution of Echocardiography Findings

Echo findings	No. of patients (N)	Percentage (%)
Ejection fraction	Mean±SD = 31.65±7.3%	Min-Max: 18-44%
40%-45%	9	13
30-39%	32	46.4
20-29%	24	34.8
<20%	4	5.8

LVEDD	Mean±SD = 5.96±0.8 cm	Min-Max: 4.5-6.9 cm
4.5-4.9 Cm	11	15.9
5.0-5.9 Cm	21	30.4
>6 Cm	37	53.6
LVESD	Mean±SD = 4.9±0.6 cm	Min-Max: 3.5-5.6 cm
3.5-4 Cm	10	14.5
4-4.9 Cm	22	31.9
>5 Cm	37	53.6
Mitral regurgitation	47	68.1
Tricuspid regurgitation	3	4.3
Atrial regurgitation	2	2.9
Pericardial effusion	5	7.2
Left ventricular clot	0	
PAH	4	5.79
Diastolic dysfunction	6	8.6
Left Atrial Enlargement	34	49.27
Right Ventricular Enlargement	24	34.78

The least common abnormalities were tricuspid regurgitation, atrial regurgitation, and left ventricular clots, which were observed in 3, 2, and 0 patients, respectively.

Indicating a reduced pumping ability of the heart, indicating an enlargement of the left lower chamber of the heart.

Table 8: Association of Echocardiographic Features with Clinical Outcomes

Variables	Severe symptoms	Mild to moderate symptoms	p-value
EF<35%	18	23	0.233
EF>35%	9	21	
LA size>40 mm	22	17	0.000*
LA size<40 mm	4	26	
RV size>26 mm	16	12	0.000*
RV size<26 mm	5	36	
LVEDD>52 mm	20	29	0.522
LVEDD<52 mm	3	7	

DISCUSSION

The present study evaluated the demographic profile, clinical presentation, etiological factors, and risk factors associated with dilated cardiomyopathy (DCM) among patients attending a tertiary care teaching hospital. The findings of the study demonstrated that DCM was more commonly observed among middle-aged and elderly individuals, with the highest prevalence noted in the 51–60 years age group (33.3%). Male predominance was observed, with males accounting for 71% of the study population. Similar findings have been reported in previous studies where DCM was more common among males due to higher prevalence of

cardiovascular risk factors such as smoking, alcoholism, hypertension, and ischemic heart disease.[17] The increased prevalence in middle-aged and older individuals may be related to prolonged exposure to metabolic and cardiovascular risk factors contributing to ventricular remodeling and myocardial dysfunction.[18]

Dyspnea was the most common presenting symptom in the present study, affecting 76.8% of patients, followed by paroxysmal nocturnal dyspnea (PND), pedal edema, orthopnea, and fatigue. These findings are consistent with the clinical manifestations of congestive heart failure resulting from impaired systolic function and ventricular dilatation.[19]

Similar observations were reported by Dec and Fuster, who identified dyspnea and exercise intolerance as the predominant presenting symptoms in patients with DCM.[20] The high prevalence of pedal edema, orthopnea, and PND in our study reflects advanced ventricular dysfunction and elevated intracardiac filling pressures. Syncope was the least common symptom, observed in only 13.1% of patients, possibly due to lower incidence of severe arrhythmias or advanced conduction abnormalities in the study population.[21]

Among the clinical signs, raised jugular venous pressure (JVP) was the most frequently observed finding, followed by hepatomegaly, basal crepitations, and tachycardia. Raised JVP and hepatomegaly indicate systemic venous congestion and right-sided heart failure secondary to progressive left ventricular dysfunction.[22] Basal crepitations reflect pulmonary venous congestion and pulmonary edema associated with left-sided heart failure. Tachycardia observed in a majority of patients may represent a compensatory mechanism to maintain cardiac output in the setting of reduced stroke volume.[23] Bradycardia was uncommon in the present study and was observed in only a few patients.

The etiological distribution of DCM in this study showed ischemic heart disease as the most common cause, accounting for 30.4% of cases. This finding correlates with the increasing burden of coronary artery disease and cardiovascular risk factors in the Indian population.[24] Diabetic and idiopathic cardiomyopathy each accounted for 20.3% of cases. Diabetes mellitus contributes to myocardial fibrosis, microvascular dysfunction, and metabolic derangements leading to diabetic cardiomyopathy.[25] Alcoholic cardiomyopathy constituted 17.4% of cases, highlighting the cardiotoxic effects of chronic alcohol consumption on myocardial contractility and ventricular remodeling.[26] Peripartum cardiomyopathy was the least common etiology, affecting 11.6% of patients, which is comparable with findings from earlier epidemiological studies.[27]

Hypertension was the most prevalent risk factor identified in the present study, affecting 58% of patients, followed by alcoholism, diabetes mellitus, dyslipidemia, and smoking. Hypertension contributes to chronic pressure overload, myocardial hypertrophy, and eventual ventricular dilatation and

failure.[28] Alcoholism and smoking further accelerate myocardial damage through oxidative stress, endothelial dysfunction, and direct myocardial toxicity.[29] The presence of multiple cardiovascular risk factors among patients with DCM emphasizes the importance of early identification and aggressive risk factor modification.

Overall, the findings of the present study are consistent with previous literature demonstrating that DCM predominantly affects middle-aged males and commonly presents with symptoms and signs of congestive heart failure. Ischemic heart disease and hypertension emerged as major contributors to the development of DCM, highlighting the growing burden of cardiovascular disease in developing countries.

CONCLUSION

The present study highlighted the clinical and echocardiographic profile of patients with dilated cardiomyopathy (DCM) attending a tertiary care teaching hospital. DCM was predominantly observed among middle-aged and elderly males, with the highest incidence seen in the 51–60 years age group. The majority of patients presented with classical symptoms and signs of congestive heart failure, particularly dyspnea, paroxysmal nocturnal dyspnea, pedal edema, orthopnea, raised jugular venous pressure, and hepatomegaly, indicating advanced ventricular dysfunction at the time of presentation.

Echocardiographic evaluation played a crucial role in establishing the diagnosis and assessing the severity of cardiac dysfunction. Findings such as reduced left ventricular ejection fraction, global hypokinesia, chamber dilatation, and increased ventricular dimensions were commonly observed among study participants. Ischemic heart disease emerged as the most common etiology of DCM, followed by diabetic, idiopathic, and alcoholic cardiomyopathy. Hypertension, alcoholism, diabetes mellitus, smoking, and dyslipidemia were identified as major contributing risk factors.

The study emphasizes the importance of early clinical recognition, detailed echocardiographic assessment, and aggressive management of cardiovascular risk factors for timely diagnosis and improved prognosis in patients with dilated cardiomyopathy.

REFERENCES

1. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, et al. Classification of the cardiomyopathies: A position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2008;29(2):270-6.
2. Bozkurt B, Colvin M, Cook J, Cooper LT, Deswal A, Fonarow GC, et al. Current diagnostic and treatment strategies for specific dilated cardiomyopathies. *Circulation*. 2016;134(23):e579-646.
3. Pinto YM, Elliott PM, Arbustini E, Adler Y, Anastakis A, Böhm M, et al. Proposal for a revised definition of dilated cardiomyopathy. *Eur Heart J*. 2016;37(23):1850-8.
4. Dec GW, Fuster V. Idiopathic dilated cardiomyopathy. *N Engl J Med*. 1994;331(23):1564-75.
5. Hershberger RE, Hedges DJ, Morales A. Dilated cardiomyopathy: The complexity of a diverse genetic architecture. *Nat Rev Cardiol*. 2013;10(9):531-47.
6. Jefferies JL, Towbin JA. Dilated cardiomyopathy. *Lancet*. 2010;375(9716):752-62.
7. McNally EM, Mestroni L. Dilated cardiomyopathy: Genetic determinants and mechanisms. *Circ Res*. 2017;121(7):731-48.
8. Richardson P, McKenna W, Bristow M, Maisch B, Mautner B, O'Connell J, et al. Report of the 1995 WHO/ISFC task force on the definition and classification of cardiomyopathies. *Circulation*. 1996;93(5):841-2.
9. Felker GM, Thompson RE, Hare JM, Hruban RH, Clemetson DE, Howard DL, et al. Underlying causes and long-term survival in patients with initially unexplained cardiomyopathy. *N Engl J Med*. 2000;342(15):1077-84.
10. Mann DL. Mechanisms and models in heart failure: A combinatorial approach. *Circulation*. 1999;100(9):999-1008.
11. Codd MB, Sugrue DD, Gersh BJ, Melton LJ. Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy. *Circulation*. 1989;80(3):564-72.
12. Roger VL. Epidemiology of heart failure. *Circ Res*. 2013;113(6):646-59.
13. GBD 2015 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015. *Lancet*. 2016;388(10053):1545-602.
14. Mestroni L, Taylor MR. Dilated cardiomyopathy: Genetics and precision medicine. *Annu Rev Genomics Hum Genet*. 2019;20:1-23.
15. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults. *Eur Heart J Cardiovasc Imaging*. 2015;16(3):233-70.
16. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF, Dokainish H, Edvardsen T, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. *Eur J Echocardiogr*. 2016;17(12):1321-60.
17. Codd MB, Sugrue DD, Gersh BJ, Melton LJ. Epidemiology of idiopathic dilated and hypertrophic cardiomyopathy. *Circulation*. 1989;80(3):564-72.
18. Hershberger RE, Morales A, Siegfried JD. Clinical and genetic issues in dilated cardiomyopathy. *J Am Coll Cardiol*. 2010;57(16):1641-9.
19. Mann DL, Chakinala M. Heart failure and dilated cardiomyopathy. In: Braunwald's Heart Disease. 11th ed. Philadelphia: Elsevier; 2019. P. 509-32.
20. Dec GW, Fuster V. Idiopathic dilated cardiomyopathy. *N Engl J Med*. 1994;331(23):1564-75.
21. Jefferies JL, Towbin JA. Dilated cardiomyopathy. *Lancet*. 2010;375(9716):752-62.
22. Braunwald E. Heart disease: A textbook of cardiovascular medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.
23. McMurray JJV, Pfeffer MA. Heart failure. *Lancet*. 2005;365(9474):1877-89.
24. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India. *Circulation*. 2016;133(16):1605-20.

25. Rubler S, Dlugash J, Yuceoglu YZ, Kumral T, Branwood AW, Grishman A. New type of cardiomyopathy associated with diabetic glomerulosclerosis. *Am J Cardiol.* 1972;30(6):595-602.
26. Piano MR, Phillips SA. Alcoholic cardiomyopathy: Pathophysiologic insights. *Cardiovasc Toxicol.* 2014;14(4):291-308.
27. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy. *Eur J Heart Fail.* 2010;12(8):767-78.
28. Dzau VJ, Antman EM, Black HR, Hayes DL, Manson JE, Plutzky J, et al. The cardiovascular disease continuum validated. *Circulation.* 2006;114(25):2850-70.
29. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease. *J Am Coll Cardiol.* 2004;43(10):1731-7.

How to cite this article: Dr. Mookambika. R.V, Dr. Vikash.M, ECHOCARDIOGRAPHIC EVALUATION AND CLINICAL PROFILE OF DILATED CARDIOMYOPATHY: A CROSS-SECTIONAL OBSERVATIONAL STUDY IN A TERTIARY CARE TEACHING HOSPITAL, *Asian J. Med. Res. Health Sci.*, 2026; 4 (2):1361-1369.

Source of Support: Nil, Conflicts of Interest: None declared.