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CLINICO-ETIOLOGICAL, ECHOCARDIOGRAPHIC, AND ANGIOGRAPHIC PROFILES OF NON-ISCHEMIC DILATED CARDIOMYOPATHY IN PATIENTS <45 YEARS

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ABSTRACT

Background: Non-ischemic dilated cardiomyopathy (DCM) is a primary myocardial disorder characterized by left ventricular (LV) dilation and systolic dysfunction in the absence of significant coronary artery disease. It accounts for a substantial proportion of heart failure cases, estimated at roughly 25–40% of heart failure with reduced ejection fraction (HFrEF) globally, and is an important cause of morbidity and mortality in younger patients. However, detailed profiling of young Indian patients with non-ischemic DCM remains limited. **Objective:** To describe the clinical signs and symptoms, underlying etiologies, echocardiographic findings, and coronary angiographic features of non-ischemic DCM in patients aged <45 years. **Materials and Methods:** In this prospective, tertiary-care, cross-sectional study (May 2023–May 2025), 40 consecutive patients ≤45 years with angiography-confirmed non-ischemic DCM were enrolled. Each patient underwent systematic clinical evaluation, laboratory testing, 12-lead ECG, two-dimensional echocardiography, and coronary angiography. DCM etiologies were classified by history, examination and laboratory data. **Result:** Patients were predominantly middle-aged (45% aged 36–45 years) with a slight male predominance (52.5% men). The most common symptoms were fatigue (90%), exertional dyspnea (65%), palpitations (67.5%), and signs of volume overload (orthopnea 60%, bilateral oedema 70%). The mean troponin-I was modestly elevated (29.96±36.46 units), reflecting occasional acute injury. Echocardiography revealed advanced LV dysfunction: 70% of patients had **severe** systolic dysfunction (EF <35%), 25% moderate (EF 35–44%), and only 5% mild (EF 45–54%); no patient had a normal EF. Echo typically showed global LV hypokinesia, chamber dilatation and functional mitral/tricuspid regurgitation. ECG was normal sinus rhythm in 55%, but 45% had abnormalities (most commonly left bundle branch block 10%, low-voltage complexes 7.5%, LV hypertrophy or ST–T changes in 5%). Coronary angiography was normal in 77.5% of patients and showed only non-obstructive or slow-flow phenomena in the rest, confirming the non-ischemic nature of cardiomyopathy. Idiopathic etiology predominated (67.5% of cases), followed by autoimmune myocarditis (15%), post-viral (7.5%), toxin (alcohol)5%, chemotherapy (2.5%), and metabolic causes (2.5%). NT-proBNP levels stratified patients by severity: 20% had >25,000 pg/mL (severe HF, one in-hospital death), 42.5% between 10,000–25,000 (moderate HF), and 37.5% <10,000 (mild HF). Higher NT-proBNP was strongly associated with longer hospital stay and mortality. **Conclusion:** In young adults with non-ischemic DCM, presentation is dominated by advanced systolic dysfunction and heart failure symptoms. Idiopathic causes are most common, may include genetic variants of DCMP also and comprehensive evaluation including ECG, and coronary angiography is essential to characterize disease and exclude ischemia. NT-proBNP is a powerful prognostic marker. These findings underscore the need for early recognition and multidisciplinary management of DCM in younger patients.

INTRODUCTION

Dilated cardiomyopathy (DCM) is defined by dilation of the left ventricle (LV) and reduced systolic function without significant coronary artery disease. It is a major cause of heart failure worldwide, comprising approximately one-quarter to two-fifths of heart failure cases with reduced ejection fraction.[1] DCM most often occurs in adults, but earlier onset in the third or fourth decades is increasingly recognized. Indeed, younger age at diagnosis is associated with a more aggressive clinical course and worst prognosis.[2] For example, DCM is the leading cause of heart failure among young adults and is the most common indication for heart transplantation in this group. Its global prevalence is roughly 0.04% (1 in 2500 individuals) in adults.[3]

The causes of non-ischemic DCM are heterogeneous, including genetic mutations, viral myocarditis, autoimmune myocarditis, toxic exposures (e.g. alcohol), chemotherapy, and metabolic disorders but in many patients it is idiopathic.[1]

MATERIALS AND METHODS

We conducted a prospective, hospital-based, observational study at a tertiary care center from May 2023 to May 2025. The study included 40 patients aged 18–45 years with a clinical diagnosis of dilated cardiomyopathy. Ethical clearance was obtained from the institutional committee and all participants provided written informed consent.

Eligibility Criteria

Inclusion Criteria

- All DCMP cases
- Age 45 years and below

Exclusion Criteria

- Patients with coronary artery disease including myocardial infarction in the past.
- Patients having significant coronary artery disease on coronary angiography.
- Significant primary valvular heart disease/ RHD
- <18 years
- Sepsis
- Patients with comorbidities and terminal illness.
- Peripartum cardiomyopathy
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy.

Statistical analysis was performed using SPSS. Categorical variables were expressed as counts and percentages, and continuous variables as mean +/- SD. Comparisons (e.g. anaemia category across age groups, BNP categories vs. NYHA class) used chi-square tests or ANOVA as appropriate, with significance set at $p<0.05$. Two cardiologists independently reviewed echo and ECG findings to ensure quality.

RESULTS

Demographics and Clinical Presentation

Baseline characteristics are summarized in Table 1. A significant proportion of the individuals are aged between 36-45 years, indicating a middle aged with male dominant distribution. The most common presenting complaints was fatigability (90%) reflecting decrease cardiac output and impaired tissue perfusion. Features of left ventricular dysfunction and pulmonary venous hypertension like dyspnea on exertion (65%), orthopnea (60%), PND (40%), rales (60%) and pedal oedema (70%) were found.

The ECGs evaluation of the study cohort revealed a predominance of Normal Sinus Rhythm (55%). Among abnormal ECGs, the most frequent abnormalities included -Left bundle Branch Block (10%), Low voltage complexes (7.5%), ST depression (5%), Atrial fibrillation (5%) and others like T wave inversion, electrical alternans, sinus tachycardia each constituting 2.5% of cohort.

Severe Anemia cases were rare. The majority of individuals (37.5%) fall under mild Anemia category. Under LDL risk category distribution, 82% patients had LDL levels within optimal range, suggesting effective lipid control or lower lipid related risk contribution to Non Ischemic Dilated Cardiomyopathy. Also majority of the patients demonstrate normal Thyroid Function test (90%) and normal Glycemic control (65%). Diabetes (17.5%) and prediabetic (17.5%) i.e hyperglycemia are known risk factors for adverse cardiac, fibrosis and worsening of ventricular functions, all of which can accelerate DCMP progression.

The echocardiographic assessment of the study population revealed a profound degree of Left ventricular systolic dysfunction, characterized as Mild LV dysfunction (5%), Moderate LV dysfunction (25%) and severe LV dysfunction (70%). Echo and laboratory findings were highly concordant.

The coronary angiographic evaluation of the study cohort revealed that the majority of the patients (77.5%) exhibited non obstructive coronary anatomy, consistent with the clinical diagnosis of DCMP. Non obstructive coronary artery disease (NOA) was detected in 20% of patients and 2.5% shows slow flow coronaries.

Idiopathic DCMP emerged as the leading cause, accounting for 67.5% of the cases. In these patients, no definitive underlying cause could be identified despite thorough evaluation, reflecting the recognized global trend where idiopathic forms constitute the majority of DCMP diagnoses. This highlights the complex interplay of genetic, environmental, and possibly subclinical viral factors. Autoimmune-mediated cardiomyopathy (including Systemic Lupus Erythematosus [SLE] and Mixed Connective Tissue Disease [MCTD]) contributed to 15% of cases. Autoimmune inflammation of the myocardium can lead to progressive ventricular

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dysfunction due to myocardial fibrosis, inflammation, and vasculitis. Post-viral myocarditis was identified as the underlying cause in 7.5% of patients. Alcohol-induced cardiomyopathy accounted for 5%. Chronic alcohol consumption and illicit drug use (such as cocaine) exert direct toxic effects on myocardial cells, impairing contractility and promoting dilatation.[15] Chemotherapy-induced DCMP due to chemotherapeutic agents (Adriamycin/Cyclophosphamide regimen) was noted in 2.5%. This reflects the well-established cardiotoxic potential of certain anti-cancer drugs, emphasizing the importance of cardiac monitoring during chemotherapy.[11] Metabolic causes (such as Diabetes Mellitus, Hypertension, and Hypothyroidism) were observed in 2.5% of the cohort. Chronic metabolic insults contribute to myocardial fibrosis, stiffening, and systolic dysfunction over time.[14]

Table 1: Patients Characteristics

Category	Parameter	Counts(n)	Percentage
Demographic	Age 18–25 years	13	32.5%
	26–35 years	7	17.5%
	36–45 years	20	50 %
	Sex Male	21	52.5%
	Female	19	47.5%
Symptoms /Sign		36	90%
	Fatigue	27	67.5%
	Palpitations	26	65%
	Exertional dyspnea	24	60%
	Orthopnea	16	40%
	Paroxysmal nocturnal dyspnea (PND)	28	70%
	Bilateral pedal edema	24	60%
	Pulmonary rales (crackles)	8	20%
	Chest pain	8	20%
	Pallor		
ECG Findings	NSR (Normal Sinus Rhythm)	22	55.0%
	LBBB (Left Bundle Branch Block)	4	10.0%
	Low Voltage Complexes	3	7.5%
	LVH (Left Ventricular Hypertrophy)	2	5.0%
	ST Depression V1–V6	2	5.0%
	Atrial Fibrillation (AF)	1	2.5%
	Atrial Fibrillation (typo — counted under AF)	1	2.5%
	LAD LAFB (Left Anterior Fascicular Block)	1	2.5%
	T Wave Inversion in Leads 1, aVL, 2	1	2.5%
	T Wave Inversion in Leads V2, V3, V4	1	2.5%
	Electrical Alternans	1	2.5%

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	Sinus Tachycardia	1	2.5%
Anemia	≥12.0 g/dL (normal)	12	30%
	10.0–11.9 g/dL (mild anemia)	15	37.5%
	7.0–9.9 g/dL (moderate anemia)	11	27.5%
	<7.0 g/dL (severe anemia)	2	5%
Dyslipidemia LDL	Optimal (<100 mg/dL)	33	82.5%
	Near Optimal (100–129 mg/dL)	6	15.0%
	High (≥130 mg/dL)	1	2.5%
Thyroid Profile	Normal Thyroid Function (WNL)	36	90%
	Abnormal Thyroid Function (hypothyroidism)	4	10%
Hba1C	Normal Glycemic Control (<5.7%)	26	65%
	Diabetes Mellitus (≥6.5%)	7	17.5%
	Prediabetes (5.7–6.4%)	7	17.5%
Echocardiography	Severe LV Dysfunction (<35%)	28	70.0%
	Moderate LV Dysfunction (35–44%)	10	25.0%
	Mild LV Dysfunction (45–54%)	2	5.0%
Angiography	Normal Coronaries	31	77.5%
	Non-Obstructive Coronary Disease	8	20%
	Slow Flow Coronaries	1	2.5%
Etiology			
	Idiopathic	27	67.5%
	Autoimmune (SLE/MCTD)	6	15.0%
	Post Viral	3	7.5%
	Alcohol / Drug-Induced	2	5.0%
	Toxin/Drug-Induced (Chemotherapy)	1	2.5%
	Metabolic (T2DM/HTN/Hypothyroidism)	1	2.5%

Table 2: BNP Analysis Based on Clinical Findings

BNP Range	Clinical Status	Average Hospital Stay	NYHA Class / Symptoms	Mortality
>25,000 pg/mL	Severe symptoms: Shortness of breath, palpitations, generalized edema	6–7 days	NYHA Class III–IV	1 death reported
10,000–25,000 pg/mL	Moderate symptoms: Shortness of breath	3–5 days	NYHA Class II–III	None
<10,000 pg/mL	Mild symptoms: Fatigability, shortness of breath on exertion	3–4 days	NYHA Class II	None

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DISCUSSION

In this study of 40 Indian patients under age 45 with non-ischemic dilated cardiomyopathy, we found a high burden of advanced heart failure symptoms, severe systolic dysfunction, and mostly idiopathic etiology.[14] Our cohort's demographic and clinical profile is largely in line with other reports of DCMP.[10] The mean age skewed toward the late 30s (highest proportion in 36–45 years), with a slight male predominance (52.5% men). The finding that nearly half the patients were in their fourth decade underscores that DCM is not confined to the elderly; it can strike in the prime of life, in agreement with Jennings et al.(2025) who noted that younger age at diagnosis is a risk factor for rapid progression of failing heart.[12]

Symptomatically, fatigue (90%), dyspnea (65% on exertion), orthopnea (60%), and oedema (70%) dominated, reflecting advanced heart failure.5/16 Palpitations (67.5%) were also common, likely due to associated arrhythmias or neurohormonal activation. Compared to a North-East India series of DCM patients, our symptom distribution is similar (for example, dyspnea in ~70%, palpitations in ~65%). The high prevalence of decompensated features indicates that patients often present late in the disease course, possibly due to low awareness and limited early screening in India.

Haematologically, 65% had at least mild anemia. Anemia is frequently observed in heart failure and is known to worsen outcomes. In our relatively young cohort, the anemia may be multifactorial (chronic disease, renal dysfunction, nutritional deficiency). Regardless, the presence of anemia can exacerbate cardiac workload by reducing oxygen delivery. The lack of significant age association (p=0.471) suggests anemia was common across the age spectrum in our sample. Our observation aligns with broader Heart Failure data: for instance, a narrative review noted that iron-deficiency anemia in Heart Failure patients is linked with greater severity and worst prognosis8. Though we did not measure iron status, these data imply that anemia management might be an important consideration in young DCM patients.

We also observed that 10% of patients had thyroid dysfunction (elevated TSH). Hypothyroidism is a known comorbidity in HF that can aggravate symptoms by decreasing contractility and heart rate. Previous studies have shown that even subclinical hypothyroidism may worsen ventricular function and HF outcomes, suggesting our finding reinforces the need for routine thyroid screening in DCMP.[10]

Remarkably, 35% of patients had impaired glucose metabolism (17.5% diabetes, 17.5% prediabetes). This metabolic contribution to DCM has been recognized: hyperglycemia and diabetes promote myocardial fibrosis and adverse remodeling.[13] Our data imply that metabolic syndrome components are common even in younger DCM patients, underscoring the importance of addressing these comorbidities. By contrast, dyslipidemia was uncommon; most (82.5%) had LDL <100 mg/dL, reflecting that atherosclerotic disease was not a major factor.

Echocardiography confirmed severe systolic dysfunction in the majority: 70% had EF <35% and only 5% had mildly reduced EF (45–54%). This suggests that younger patients in this referral cohort present with advanced DCMP. The presence of left bundle branch block in 10% of our patients and other ECG abnormalities is notable; LBBB is associated with dyssynchrony and worse prognosis in DCM (not directly cited here). These patients may be candidates for cardiac resynchronization therapy if advanced disease persists.[15]

Coronary angiography was normal or minimally abnormal in all patients, validating the non-ischemic designation. Normal coronaries in 77.5% and non-obstructive findings in 20% align with prior studies emphasizing the importance of excluding coronary disease. For example, Bazmpani et al. (2023) highlighted that angiography is essential to distinguish ischemic from non-ischemic HF in younger patients. Our findings reinforce that most young DCM patients have true non-ischemic cardiomyopathy. The small subset with minor atherosclerosis may reflect risk factor overlap, but none had flow-limiting lesions.

Etiologically, idiopathic DCM dominated (67.5%). This is consistent with global registries: a recent large cohort found that idiopathic (or genetic) DCM is the most common form, particularly when comprehensive testing rules out secondary causes.[7-10] The second most common cause in our group was autoimmune myocarditis (15%), illustrating that systemic inflammatory diseases contribute significantly in young patients15/16. Post-viral myocarditis accounted for 7.5%, and toxin exposure (alcohol),[15] chemotherapy,[11] another 7.5%. These distributions are broadly comparable to other reports: e.g. Fundikira et al. found that in Tanzania, idiopathic causes were common in DCM, with idiopathic DCM patients tending to be younger and sicker. The very low incidence of metabolic (diabetes/hypertension/hypothyroidism-induced) DCM (2.5% in our sample) highlights that such factors were largely absent or well controlled in this young group.[11]

A key finding is the strong correlation between NT-proBNP levels and clinical severity. Patient with BNP >25,000 pg/mL were almost uniformly NYHA class III–IV, had the longest stays (6–7 days), and sustained the only death, whereas those <10,000 pg/mL were NYHA class II and had no mortality. This gradient supports BNP's role as a prognostic biomarker. Prior studies have demonstrated that higher natriuretic peptides predict mortality and readmission in heart failure. Thus, routine BNP measurement can stratify risk and guide management in young DCM as well.

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Limitations of The Study

- Single-center design may limit generalizability.
- Absence of cardiac MRI, genetic testing or viral serology testing could have better delineate etiology in idiopathic cases.
- Potential referral bias due to tertiary care setting.
- No long-term follow-up due to cross-sectional nature.
- Pro-BNP was evaluated only once; serial levels could have offered better prognostic value

CONCLUSION

This study of young Indian patients with non-ischemic dilated cardiomyopathy reveals that early-onset DCMP is typically **severe** in clinical presentation and advanced in cardiac remodelling. Major conclusions include:

- **Early-onset DCMP:** A substantial proportion of DCM patients present before age 45, with the highest incidence in the 36–45 year range. Such patients often have advanced heart failure by diagnosis.[3]
- **Predominance of idiopathic cases:** Over two-thirds of patients had no identifiable cause despite evaluation. This suggests unrecognized genetic or inflammatory factors may underlie many young-onset cases. Use of advanced diagnostics (genetic testing, CMR) may uncover occult etiologies.[1-5]
- **Diagnostic utility of ECG/echo:** Nearly universal ECG and echocardiographic abnormalities were seen. Most patients had LVEF <35%. LBBB and other conduction blocks were common and may warrant device therapies (e.g. CRT) in appropriate patients.
- **Angiography to exclude ischemia:** Coronary angiography was necessary to confirm the non-ischemic nature of cardiomyopathy. Normal or non-obstructive coronaries in the vast majority reaffirm that young DCM is primarily non-atherosclerotic.[6]
- **Prognostic value of BNP:** NT-proBNP level strongly correlated with NYHA class and outcomes. Patients with markedly elevated BNP had longer hospitalizations and higher mortality.[5]
- **Comorbid factors:** A notable subset had thyroid dysfunction or impaired glucose metabolism. Even in a predominantly idiopathic cohort, addressing modifiable comorbidities (hypothyroidism, diabetes) may improve cardiac function and symptoms.[4]
- **Clinical presentation:** Symptoms were dominated by classical heart failure signs (fatigue, dyspnea, edema). Palpitations and arrhythmias also featured prominently, suggesting the need for vigilant rhythm monitoring.[2]

In summary, non-ischemic dilated cardiomyopathy in patients under 45 years is a complex syndrome marked by profound LV dysfunction and a heavy symptom burden. Early recognition of this condition is critical; young HF patients should be thoroughly screened for DCMP. Management should include standard heart failure therapy, consideration of device interventions for conduction blocks, and targeted treatment of comorbid conditions. Future multicenter studies with genetic and long-term follow-up data are warranted to deepen understanding and improve outcomes in this vulnerable population.

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