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*Original Article*

# Comparative Analysis of Clinical Profile of Patients Admitted with Idiopathic Dilated Cardiomyopathy in a Tertiary Care Hospital

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## ABSTRACT

**Background:** The etiology of the ventricular dilation and dysfunction that occurs in idiopathic dilated cardiomyopathy (DCM) is unknown. **Aim:** The present study was aimed to study clinical characteristics of the patients admitted with idiopathic DCM and compare them with healthy controls. **Methods:** Thirty newly diagnosed patients with DCM and 30 healthy control were enrolled from Cardiology OPD, PGIMER, Chandigarh from Jan 2011 to Jun 2012. Patients with heart failure secondary to idiopathic DCM of age >18 years were included if they were willing, provide written informed consent and does not meet any of the exclusion criteria. Idiopathic DCM was diagnosed by the presence of left ventricular dilatation and systolic dysfunction (LVEF<40%) on echocardiography in the absence of coronary artery disease, hypertension or valvular disease. **Results:** Mean age of idiopathic DCM patients and control was 48.37 ± 10.82 years and 49.2 ± 9.27 (P=0.75) respectively. There were more males (66.7%) than females (33.3%) in the patient group. It was observed that the treatment with beta blockers, furosemide, spironolactone, ACE inhibitors, and ARBs significantly improved ejection fraction (EF) (P=0.000), and LVES (P=0.000). **Conclusion:** In our study, treatment with the medications significantly improved EF and LVES. However, there was no treatment-based difference in the patients on ACE inhibitors or ARBs in the improvement in EF. Our study also observed significance difference in platelets count, SGOT, SGPT, and LDL levels in idiopathic DCM patients when compared with healthy controls.

**Key words:** DCM, LVEF, LVES, NYHA Class, ACE Inhibitors, ARBs.

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## INTRODUCTION

### Background

Dilated Cardiomyopathy (DCM) refers to a group of heterogenous myocardial disorders that are characterized by ventricle dilation and depressed myocardial contractility in the absence of abnormal loading conditions (such as hypertension or valvular disease) or ischemic heart disease sufficient to cause global systolic impairment.[1,2] The term idiopathic DCM refers when etiology is not known. It is much more common than the other major forms of cardiomyopathy (hypertrophic, restrictive and arrhythmogenic right ventricular cardiomyopathy). It is a heterogeneous disease characterized by ventricular and sometimes atrial dilatation, with normal or reduced wall thickness, eventually leading to varying degrees of impaired systolic function. The clinical picture at the time of diagnosis can vary widely from patient to patient; some have no symptoms, whereas others have progressive refractory heart failure. Depending on the diagnostic criteria used, the reported annual incidence varies between 5 and 8 cases per 100,000 population.[3] Males have a 2.5-fold increase in risk, as compared with females, that is unexplained by socioeconomic factors, alcohol intake, or other variables.[4] Patients with idiopathic DCM can present a highly variable clinical course. The challenge is to identify and treat the known and treatable causes of idiopathic DCM early enough to improve symptoms and survival. The present study was aimed to study clinical characteristics of the patients admitted with idiopathic DCM and compare them with healthy controls.

### Subjects and Methods

This prospective study was conducted at Department of Cardiology, Postgraduate Institute of Medical Education & Research (PGIMER), Chandigarh from Jan 2011 to Jun 2012. Thirty newly diagnosed patients of DCM were enrolled in the study from cardiology OPD. Similar numbers of healthy controls were also enrolled. Attendants of the patients were enrolled as healthy subjects. Patients with heart failure secondary to idiopathic cardiomyopathy of age >18 years were included if they were willing, provide written informed consent and does not meet any of the exclusion criteria. A similar number of age- and sex-matched controls were included in the study. Idiopathic dilated cardiomyopathy was diagnosed by the presence of left ventricular dilatation and systolic dysfunction (LVEF<40%) on echocardiography in the absence of coronary artery disease, hypertension or valvular disease. The patients who were unwilling to participate, smokers, with coronary artery disease, diabetes mellitus, cancer, hypercholesterolemia, with concomitant infection at time of study, on sildenafil tablet, pregnancy, with peripheral vascular disease, presence of other serious co-morbid medical or surgical illness, renal failure or autoimmune disease, rheumatic heart disease, hypertrophic cardiomyopathy, hypertensive heart disease, congenital heart disease, evidence of restrictive or constrictive physiology, or alcohol intake > 60g/day were excluded from study. The study was conducted following approval from Institutional Ethics Committee.

After written informed consent, a detailed history was taken and thorough physical examination was carried out especially with reference to the risk factors for coronary artery disease. Blood investigations in the form of hemogram, liver function tests, renal function tests and lipid profile were done.

## Echocardiography

Echocardiography of all the patients and controls was done and ejection fraction was calculated using Modified Simpson's method in apical 4 chamber view. Patients with ejection fraction less than 40% were enrolled in the study. The normal value for EF was taken as 55-70%, end-diastolic volume as 65-240 ml, end systolic volume as 16-143 ml. Follow up echo was done after 3-month duration to know about improvement after optimal drug therapy in the form of diuretics, beta blockers, and ACE-I/ARBs. Treatment was decided by the treating physician after considering overall profile of the patient.

## Statistical Analysis

Data were presented as frequency, percentages, range or mean ± standard deviation wherever applicable. Mean difference in continuous variables was analyzed using student T-test. For paired data, paired T-test was used. P values less than 0.05 was considered as significant. Results were analyzed using SPSS trial version 21.

# RESULTS

A total of 60 subjects (30 idiopathic DCM and 30 healthy subjects) were included in the study. Mean age of idiopathic DCM patients was 48.37 ± 10.82 years and controls was 49.2 ± 9.27 (P=0.75). The patient group had 20 males (66.7%) and 10 (33.3%) females whereas the control group had 16 (53.3%) males and 14 (46.7%) females. There was a significant difference in platelets count (P=0.01), SGOT (P=0.01), SGPT (P=0.01), and LDL levels (P=0.01) in idiopathic DCM patients in comparison with healthy controls. A detailed presentation has been shown in table 1. Our study found that there were 15 patients (50%) in NHYA class I, 14 patients in NHYA class II, and one patient in NHYA class III. There was no patient in NHYA class IV. Gender-based distribution of NHYA class has been shown in table 2.

**Table 1: Subjects' characteristics**

| | Patients (n=30) | Healthy Controls (n=30) | P-value |
|---|---|---|---|
| Mean age( ± SD) | 48.37 ± 10.82 | 49.2 ± 9.27 | 0.75 |
| M:F | 20:10 | 16:14 | 0.29 |
| BMI (kg/m2) | 21.36 ± 1.46 | 21.39 ± 1.48 | 0.94 |
| Mean PR (b/m) | 74.3 ± 3.98 | 75.2 ± 4.3 | 0.4 |
| Mean SBP (mmHg) | 125.7 ± 8.27 | 127.57 ± 7.52 | 0.36 |
| Mean DBP(mmHg) | 75.83 ± 4.19 | 76.13 ± 3.71 | 0.77 |
| Hemoglobin(g%) | 12.56 ± 1.35 | 12.39 ± 1.28 | 0.63 |
| TLC (×106/L) | 7286.13 ± 1851 | 6605 ± 2890 | 0.28 |
| Platelets (×109/L) | 185.23 ± 49.43 | 311.67 ± 141.75 | 0.01 |
| Bilirubin (mg%) | 0.6 ± 0.28 | 0.72 ± 0.38 | 0.17 |
| SGOT (U/L) | 24.37 ± 7.85 | 39.1 ± 19.38 | 0.01 |
| SGPT (U/L) | 23.27 ± 7.59 | 38.63 ± 20.64 | 0.01 |
| ALP (U/L) | 58.33 ± 18.12 | 61.6 ± 26.89 | 0.58 |
| Albumin (g%) | 4.04 ± 0.38 | 4.05 ± 0.78 | 0.96 |
| Urea (mg%) | 25.03 ± 9.47 | 20.9 ± 9.61 | 0.099 |
| Creatinine (mg%) | 0.72 ± 0.25 | 0.72 ± 0.23 | 0.95 |
| TC ( mg/dl) | 173.93 ± 13.47 | 174.2 ± 13.3 | 0.94 |
| TG ( mg/dl) | 106.36 ± 9.12 | 108.23 ± 9.22 | 0.43 |
| LDL( mg/dl) | 83.8 ± 13.26 | 97.6 ± 18.4 | 0.01 |
| HDL( mg/dl) | 45.23 ± 5.33 | 44.9 ± 5.11 | 0.81 |

BMI: Body Mass Index; SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure; ALP: Alkaline Phosphatase, TC: Total Cholesterol; TG: Triglycerides

**Table 2: Distribution of patients according to NHYA class and gender**

| | Male | Female | Total |
|---|---|---|---|
| NHYA Class I | 10 | 5 | 15 (50%) |
| NHYA Class II | 9 | 5 | 14 (46.66%) |
| NHYA Class III | 1 | 0 | 1 (3.34%) |

**Table 3: Gender-based patients' clinical characteristics**

| | Male (n=20) | Female (n=10) | P Value |
|---|---|---|---|
| Age (year) | 49.15 ± 10.37 | 46.80 ± 12.07 | 0.584 |
| Pulse rate (/min) | 73.70 ± 4.74 | 74.60 ± 3.65 | 0.569 |
| Systolic BP | 125.90 ± 8.40 | 125.30 ± 8.43 | 0.855 |
| Diastolic BP | 75.7 ± 4.77 | 76.1 ± 2.92 | 0.810 |
| BMI | 21.22 ± 1.54 | 21.65 ± 1.31 | 0.457 |
| Hemoglobin | 12.66 ± 1.44 | 12.35 ± 1.19 | 0.556 |
| TLC | 7263.1 ± 2020.19 | 7332.2 ± 1557.76 | 0.925 |
| Platelets | 185.2 ± 56.98 | 185.3 ± 31.92 | 0.996 |
| Bilirubin | 0.655 ± 0.27 | 0.51 ± 0.28 | 0.19 |
| SGOT | 25.65 ± 8.1 | 21.8 ± 7.02 | 0.211 |
| SGPT | 24.05 ± 8.29 | 21.7 ± 6.04 | 0.434 |
| Albumin | 4.02 ± 0.42 | 4.08 ± 0.31 | 0.715 |
| Globulin | 2.91 ± 0.41 | 2.94 ± 0.36 | 0.848 |
| Alkaline Phosphatase | 56.7 ± 20.84 | 61.6 ± 11.07 | 0.495 |
| Urea | 25.7 ± 9.73 | 23.7 ± 9.3 | 0.594 |
| Creatinine | 0.75 ± 0.23 | 0.64 ± 0.29 | 0.256 |
| Cholesterol | 175.5 ± 12.25 | 170.8 ± 15.86 | 0.377 |
| Triglycerides | 105.3 ± 6.59 | 108.5 ± 12.98 | 0.374 |
| LDL | 83.65 ± 11.29 | 84.1 ± 17.25 | 0.932 |
| HDL | 45.35 ± 5.06 | 45 ± 6.13 | 0.869 |

**Table 4: Medications started in patient group**

| Drug | Number of patients |
|---|---|
| Beta blockers | 30 |
| ACE inhibitors | 22 |
| ARBs | 8 |
| Furosemide | 30 |
| Spironolactone | 30 |

ACE: Angiotensin converting enzyme; ARB: angiotensin-receptor blocker

Blood investigations showed that there was no gender-based significant difference in pulse rate, systolic and diastolic blood pressure, BMI, hemoglobin, TLC, platelets, bilirubin, SGOT, SGPT, alkaline phosphatase, albumin, globulin, urea, creatinine, total cholesterol, triglycerides, LDL, and HDL levels (Table 3).

Our study found that there was a significant difference in baseline ejection fraction (30.47 ± 8.90 vs. 63.03 ± 2.697; P=0.000), left ventricle end-systolic diameter (LVES) (35.23 ± 2.176 vs. 24.13 ± 2.674; P=0.000), and left ventricle end-diastolic diameter (LVED) (43.03 ± 2.059 vs. 36.33 ± 2.339; P=0.000) between patients and healthy control.

All the patients were on beta blockers, furosemide, and spironolactone while 22 and 8 patients were receiving ACE (angiotensin converting enzyme) inhibitors and ARBs (angiotensin-receptor blocker) respectively (table 4).

**Table 5: Echocardiography findings in patients after 3 months of medications**

| | Baseline | After 3 Months | P Value |
|---|---|---|---|
| EF (%) | 30.47 ± 8.90 | 37.77 ± 8.88 | 0.000 |
| LVED (mm) | 43.03 ± 2.059 | 42.47 ± 2.431 | 0.162 |
| LVES (mm) | 35.23 ± 2.176 | 32.23 ± 2.812 | 0.000 |
| EDV (ml) | 191.37 ± 75.20 | 190.67 ± 76.323 | 0.402 |
| ESV (ml) | 141.4 ± 54.131 | 123.43 ± 46.49 | 0.000 |

It was observed that there was significant improvement in EF (P=0.000) following 3 months medications. The medications also significantly changed LVES (P=0.000), and end systolic volume (ESV) (P=0.000). LVED and EDV were not significantly affected by medications (Table 5).

## DISCUSSION

The present study included 30 idiopathic DCM patients. All patients in our study were adults with mean age of 48.37 years. Ushasree *et al*[5] studied the epidemiology of 107 DCM patients and observed that 71.96% patients were adult. In the present study, 86.6% patients were aged more than 40 years. Idiopathic DCM have also been reported in the pediatric population. Our study found male predominance over female (M:F::2:1) which is in comparison with previously reported studies.[5,6] It has been previously suggested that probably male hormones make susceptible to factors, which alter membrane integrity and permeability[7], and cardioprotective nature of estrogens has already been established.[8]

Our study observed that there was a significant difference in platelet count, SGOT, SGPT, and LDL levels in idiopathic DCM patients in comparison with healthy controls. Sezgin *et al* have shown that there was no difference in LDL levels in idiopathic DCM and controls.[9] Elevated serum cytokines may be associated with decreased lipoproteins. Sesh *et al* demonstrated that platelet counts were significantly lower in DCM canines when compared with healthy canines.[10]

Most of the patients were in NYHA class I (50%) and class II (46.7%) and only 1 (3.3%) was in class III and none in class IV. Sitges *et al* observed fourteen (61%) patients with idiopathic DCM were on NYHA functional class II and 9 (39%) in class III.[11] Shah *et al* found that the IHD group had 4, 4, and 3 patients in NYHA Class I, II, and III, respectively, as compared with 4, 6, and 2 patients respectively in the DCM group.[12]

In our study, medical treatment of idiopathic DCM was considered optimal when the maximum tolerated dose of beta blockers and ACE inhibitors was administered. To achieve this condition, low doses of beta blockers were tested and slowly up-titrated to the highest dosage tolerated within 2 months. ACE inhibitors were introduced and the dosages increased before the second evaluation. In the patients with side effects with ACE inhibitors (n=8), ARBs were introduced. Diuretics were given in form of furosemide and spironolactone. The patients were evaluated after 3 months and echocardiography was repeated. The patient group had baseline ejection fraction of 31.7 ± 10.2 (range 15 to 48 %) which improved to 39.27 ± 10.4 (range 21 to 55 %) which was taken after 3 months after starting treatment in the form of ACE inhibitors/ARBs, beta blockers, and diuretics. Control group had normal ejection fraction. This significant improvement in EF can lead to lesser number of ICD and BIV implantations and heart transplantations. In patients with impaired LV function and heart failure symptoms, the ICD can significantly reduce mortality, but economical issues, the risk of complications, and the inappropriate shock rate should also be considered. Many patients with severe LV dysfunction and heart failure symptoms can improve their clinical condition after the optimization of medical treatment, especially with beta blockers.[13] Treasure *et al* found that there was a significant difference in left ventricular ejection fraction between DCM patients (by ventriculography) and control patients (by ventriculography in four and echocardiography in three.[14] Vischer *et al* showed that under optimal medical therapy, symptoms, and EF improved in DCM patients.[15] Our study observed that mean LVED was 43.03 mm which was not significantly changed after medications. On the other hand, LVES and ejection fraction were significantly decreased and increased respectively in the patients post medications. When the change in EF (7.27 ± 1.45 vs. 7.37 ± 1.77; P=0.873; ACE inhibitors vs. ARBs) was compared in the patients who received ACE inhibitors or ARBs, no statistically significance difference in outcome was observed. The study also did not observe sex-based difference in improvement in EF (7.5 ± 1.39 vs. 6.9 ± 1.73; P=0.314; male vs. female).

## CONCLUSION

In our study, treatment with beta blockers, ACE inhibitors, ARBs, spironolactone/ furosemide significantly improved ejection fraction and LVES. However, there was no treatment-based difference in the patients on ACE inhibitors or ARBs in the improvement in ejection fraction. Our study also observed significance difference in platelets count, SGOT, SGPT, and LDL levels in idiopathic DCM patients when compared with healthy controls. However, the sample size in our study was small, and large volume studies are required.

## ACKNOWLEDGEMENT

None

## CONFLICT OF INTEREST

Nil

## ABBREVIATIONS USED

DCM: Dilated Cardiomyopathy; NYHA: New York Heart Association; ACE Inhibitors: Angiotensin Converting Enzyme Inhibitors; ARB: Angiotensin Receptor Blocker; LVEF: Left Ventricular Ejection Fraction; LVES: Left Ventricle End-Systolic Diameter; LVED: Left Ventricle End-Diastolic Diameter; ICD: Implantable Cardioverter-Defibrillator.

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