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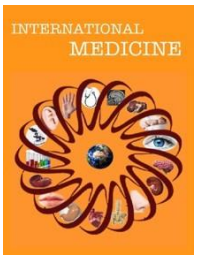

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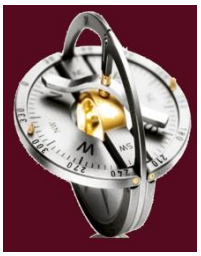

Original Research

Impact of Type 2 Diabetes Mellitus on Clinical Characteristics Myocardial Fibrosis, and Prognosis in Non-Ischemic Dilated Cardiomyopathy

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Abstract

Introduction: Non-ischemic dilated cardiomyopathy (NIDCM) is characterized by ventricular dilation and systolic dysfunction, with prognosis influenced by comorbidities such as type 2 diabetes mellitus (T2DM). T2DM may exacerbate myocardial remodelling and fibrosis, potentially worsening outcomes. ***Aims***: To evaluate the impact of T2DM on clinical features, myocardial fibrosis and long-term prognosis in patients with NIDCM. ***Materials and Methods:*** This was a 1-year prospective observational study conducted in the Department of Cardiology, Nil Ratan Sarkar Medical College and Hospital, Kolkata, West Bengal. The study included 100 adult patients diagnosed with non-ischemic dilated cardiomyopathy (NIDCM), attending the cardiology outpatient department or admitted for heart failure management. ***Results***: In our study of 100 patients with non-ischemic dilated cardiomyopathy, 40 had T2DM and 60 were non-diabetic. Compared to non-diabetics, T2DM patients were older, had higher BMI, and more frequently had hypertension (32 vs 28) and dyslipidemia (26 vs 18). They showed worse functional status with higher NYHA class III-IV (20 vs 16), lower 6-minute walk distance (310 ± 85 vs 360 ± 88 m), and higher NT-proBNP (2100 vs 1450 pg/mL). Laboratory data revealed poorer glycemic control, higher creatinine (1.5 vs 1.2 mg/dL), lower eGFR (58 vs 68 mL/min/1.73m²), and lower sodium (135 vs 137 mmol/L). Echocardiography showed lower LVEF (31 ± 7% vs 35 ± 8%) and higher E/e' ratio (16.2 ± 4.8 vs 13.8 ± 4.1), with nonsignificant trends toward more RV dysfunction and mitral regurgitation. Overall, T2DM was associated with worse clinical, functional, laboratory and echocardiographic profiles. ***Conclusions:*** T2DM in NIDCM is associated with more severe ventricular remodeling, increased myocardial fibrosis and worse clinical outcomes. Early recognition and targeted management of diabetes may improve prognosis in this population.

Keywords: *Non-ischemic dilated cardiomyopathy, type 2 diabetes mellitus, myocardial fibrosis, prognosis, heart failure.*

Introduction

Non-ischemic dilated cardiomyopathy (NIDCM) is a myocardial disorder characterized by left ventricular dilation and systolic dysfunction, in the absence of significant coronary artery disease or abnormal loading conditions [1]. It is a leading cause of heart failure and accounts for substantial morbidity and mortality worldwide [2]. The pathophysiology involves a combination of genetic predisposition, neurohormonal activation, and adverse myocardial remodelling [3].

Type 2 diabetes mellitus (T2DM) is increasingly recognized as an independent risk factor for the development and progression of cardiomyopathy, even in the absence of ischemic heart disease [4]. Hyperglycemia, insulin resistance, and metabolic dysregulation contribute to structural and functional myocardial alterations, including fibrosis, microvascular dysfunction, and impaired contractility [5,6].
Myocardial fibrosis, detectable by cardiac magnetic resonance imaging using late gadolinium enhancement, is a critical determinant of adverse outcomes in NIDCM, correlating with arrhythmias, heart failure progression and mortality [7,8]. Several studies suggest that T2DM accelerates fibrotic remodeling, exacerbating left ventricular dysfunction [9].

Despite growing evidence, data on the combined impact of T2DM on clinical presentation, extent of myocardial fibrosis, and prognosis in NIDCM remain limited, particularly in diverse populations. Understanding this interplay is crucial for risk stratification and optimizing management strategies [10].

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The primary aim of this study is to evaluate the impact of type 2 diabetes mellitus (T2DM) on clinical characteristics, myocardial fibrosis, and prognosis in patients with non-ischemic dilated cardiomyopathy (NIDCM). Specifically, the study seeks to compare the demographic and clinical profiles, assess the extent of myocardial fibrosis using cardiac magnetic resonance imaging, and determine the association between T2DM and adverse cardiovascular outcomes, including heart failure progression, arrhythmias, and mortality. Additionally, the study aims to identify whether T2DM serves as an independent predictor of prognosis in NIDCM, thereby providing insights for risk stratification and targeted management in this population.

Materials and Methods

Study Type: Prospective observational study

Study Duration: 1 year, from 1st June 2024 to 31st May 2025

Study Place: Department of Cardiology, Nil Ratan Sarkar Medical College and Hospital, Kolkata, West Bengal 700014.

Study Population: Adult patients diagnosed with non-ischemic dilated cardiomyopathy attending the cardiology outpatient department or admitted for heart failure management.

Sample Size: 100 patients diagnosed with non-ischemic dilated cardiomyopathy (NIDCM)

Study Variables:

- Baseline Demographic and Clinical Characteristics
- Baseline Clinical and Hemodynamic Parameters
- Laboratory Findings
- Echocardiographic Parameters
- Cardiac MRI Findings – Myocardial Fibrosis
- Treatment and Medication Use
- Outcomes and Prognosis (Median Follow-up: 24 months)

Inclusion Criteria:

- Age ≥18 years.
- Diagnosed with non-ischemic dilated cardiomyopathy (LV dilation with systolic dysfunction, absence of significant coronary artery disease).
- Willing to provide informed consent.

Exclusion Criteria:

- Ischemic heart disease or history of myocardial infarction.
- Valvular heart disease requiring intervention.
- Hypertensive heart disease or congenital heart disease.
- Chronic kidney disease stage ≥4 or other terminal illnesses.
- Contraindications to cardiac MRI

Statistical Analysis:

Data from the study were analyzed using SPSS software, with continuous variables (e.g., age, liver enzyme levels) expressed as mean ± SD and compared using t-tests or Mann-Whitney U tests. Categorical variables were presented as frequencies and percentages, and compared using Chi-square or Fisher's exact tests. Diagnostic accuracy (sensitivity, specificity, PPV, NPV, and accuracy) was calculated for MRCP-first and EUS-first strategies, using ERCP/intraoperative findings as the reference. Kaplan-Meier analysis may be used for time-to-intervention comparisons. A p-value < 0.05 was considered significant.

Results

Table 1. Baseline Demographic and Clinical Characteristics

Variable	T2DM (n=40)	Non-DM (n=60)	Total (n=100)	p-value
Age (years, mean ± SD)	61.3 ± 9.8	56.8 ± 11.8	58.6 ± 11.2	0.04
Male sex, n (%)	28 (70%)	40 (67%)	68 (68%)	0.76
Body mass index (kg/m²)	28.5 ± 4.0	25.7 ± 3.9	26.8 ± 4.2	0.01
Hypertension, n (%)	32 (80%)	28 (47%)	60 (60%)	0.002
Dyslipidemia, n (%)	26 (65%)	18 (30%)	44 (44%)	0.001
Atrial fibrillation, n (%)	12 (30%)	10 (17%)	22 (22%)	0.12
Duration of HF (months)	28 ± 20	31 ± 23	30 ± 22	0.56

Table 2. Baseline Clinical and Hemodynamic Parameters

Variable	T2DM	Non-DM	Total	p-value
Systolic BP (mmHg)	122 ± 16	115 ± 19	118 ± 18	0.05
Heart rate (beats/min)	90 ± 17	87 ± 15	88 ± 16	0.39
NYHA class III-IV, n (%)	20 (50%)	16 (27%)	36 (36%)	0.03
6-minute walk distance (m)	310 ± 85	360 ± 88	340 ± 90	0.02
NT-proBNP (pg/mL, median [IQR])	2100 [1200-3400]	1450 [800-2500]	1650 [900-2800]	0.04

Table 3. Laboratory Findings

Variable	T2DM	Non-DM	Total	p-value
Fasting glucose (mg/dL)	160 ± 38	98 ± 22	122 ± 40	<0.001
HbA1c (%)	7.6 ± 0.9	5.7 ± 0.4	6.4 ± 1.2	<0.001
Serum creatinine (mg/dL)	1.5 ± 0.5	1.2 ± 0.3	1.3 ± 0.4	0.01
eGFR (mL/min/1.73m²)	58 ± 16	68 ± 17	64 ± 18	0.02
Sodium (mmol/L)	135 ± 4	137 ± 3	136 ± 4	0.03
LDL cholesterol (mg/dL)	112 ± 26	100 ± 27	105 ± 28	0.09

Table 4. Echocardiographic Parameters

Variable	T2DM	Non-DM	Total	p-value
LVEF (%)	31 ± 7	35 ± 8	33 ± 8	0.02
LVEDD (mm)	63 ± 8	61 ± 6	62 ± 7	0.19
LVESD (mm)	52 ± 9	50 ± 8	51 ± 8	0.32
E/e' ratio	16.2 ± 4.8	13.8 ± 4.1	14.8 ± 4.5	0.01
RV dysfunction, n (%)	14 (35%)	11 (18%)	25 (25%)	0.06
Moderate-severe MR, n (%)	16 (40%)	14 (23%)	30 (30%)	0.07

Table 5. Cardiac MRI Findings - Myocardial Fibrosis

Variable	T2DM	Non-DM	Total	p-value
Presence of LGE, n (%)	30 (75%)	28 (47%)	58 (58%)	0.007
LGE extent (% of LV mass)	11.5 ± 5.8	7.8 ± 4.9	9.2 ± 5.6	0.01
Native T1 time (ms)	995 ± 35	1012 ± 37	1005 ± 38	0.03
ECV fraction (%)	32.1 ± 5.1	28.3 ± 4.8	29.8 ± 5.2	0.004

Table 6. Treatment and Medication Use

Variable	T2DM	Non-DM	Total	p-value
ACEI/ARB use, n (%)	32 (80%)	50 (83%)	82 (82%)	0.68
Beta-blocker use, n (%)	36 (90%)	54 (90%)	90 (90%)	0.99
MRA use, n (%)	22 (55%)	40 (67%)	62 (62%)	0.21
SGLT2 inhibitor use, n (%)	18 (45%)	2 (3%)	20 (20%)	<0.001
Loop diuretic use, n (%)	30 (75%)	28 (47%)	58 (58%)	0.006

Table 7. Outcomes and Prognosis (Median Follow-up: 24 months)

Outcome	T2DM	Non-DM	Total	p-value
All-cause mortality, n (%)	12 (30%)	6 (10%)	18 (18%)	0.01
HF hospitalization, n (%)	16 (40%)	12 (20%)	28 (28%)	0.03
Combined endpoint (death or HF admission), n (%)	20 (50%)	14 (23%)	34 (34%)	0.005
Annual mortality rate (%)	10.8	4.3	7.5	0.04

Figure 1: Treatment and Medication Use

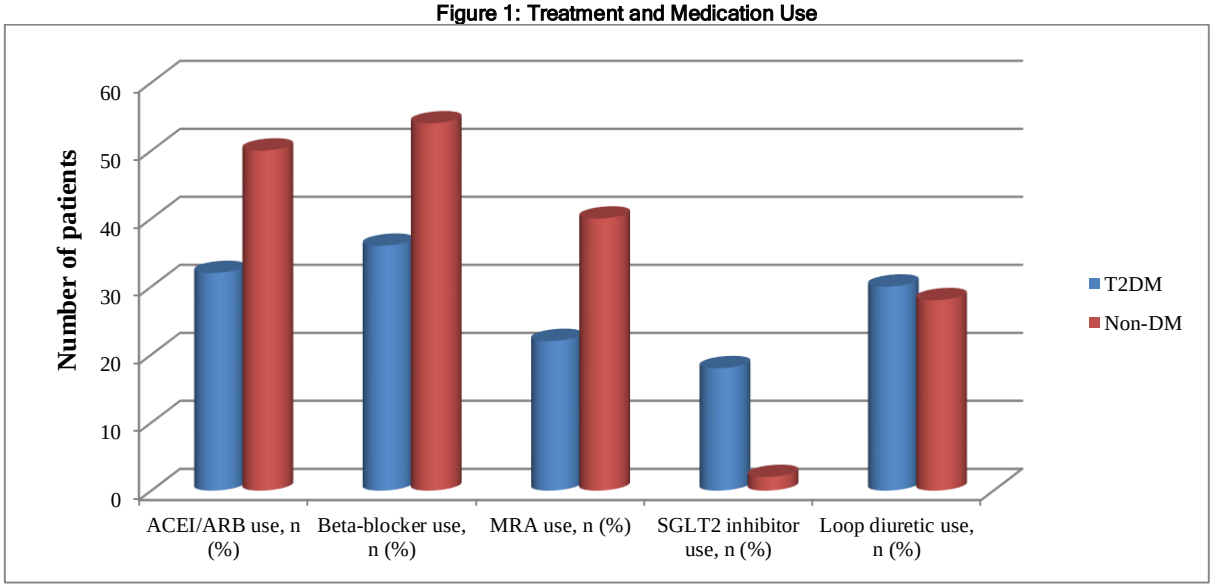
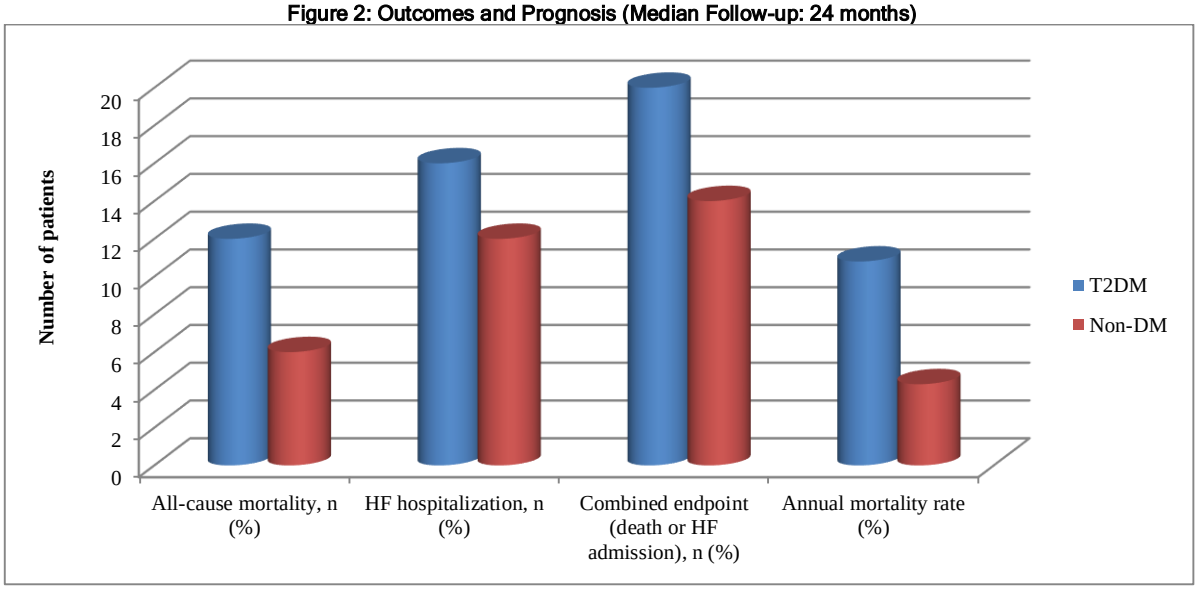


Figure 2: Outcomes and Prognosis (Median Follow-up: 24 months)



In our study of 100 patients with non-ischemic dilated cardiomyopathy (NICM), 40 patients had type 2 diabetes mellitus (T2DM) and 60 were non-diabetic. The mean age of patients in the T2DM group was 61.3 ± 9.8 years, which was significantly higher compared to 56.8 ± 11.8 years

in the non-diabetic group (p = 0.04). The majority of patients were male in both groups, with 28/40 (70%) in the T2DM group and 40/60 (67%) in the non-diabetic group, which was not statistically significant (p = 0.76). Body mass index (BMI) was significantly higher in diabetic patients, with a mean of 28.5 ± 4.0 kg/m² compared to 25.7 ± 3.9 kg/m² in non-diabetic patients (p = 0.01). Hypertension was more prevalent among diabetic patients, affecting 32/40 (80%) versus 28/60 (47%) of non-diabetic patients (p = 0.002), and dyslipidemia was also significantly higher in the T2DM group, observed in 26/40 (65%) versus 18/60 (30%) of non-diabetic patients (p = 0.001). Atrial fibrillation was present in 12/40 (30%) of diabetic patients and 10/60 (17%) of non-diabetic patients, which was not statistically significant (p = 0.12). The mean duration of heart failure was comparable between groups, 28 ± 20 months in T2DM versus 31 ± 23 months in non-diabetics (p = 0.56) which was not statistically significant.

In our study, hemodynamic and functional parameters were compared between patients with T2DM (n=40) and non-diabetic patients (n=60). The mean systolic blood pressure was higher in the T2DM group (122 ± 16 mmHg) compared to non-diabetics (115 ± 19 mmHg), which was of borderline statistical significance (p = 0.05). Heart rate was similar between the groups, with 90 ± 17 beats/min in T2DM and 87 ± 15 beats/min in non-diabetics (p = 0.39) which was not statistically significant. Functional status, assessed by NYHA class III-IV, was worse in diabetic patients, with 20/40 (50%) compared to 16/60 (27%) in non-diabetics (p = 0.03) which was statistically significant. The 6-minute walk distance was significantly lower in the T2DM group (310 ± 85 m) versus non-diabetics (360 ± 88 m, p = 0.02) which was statistically significant, indicating reduced exercise capacity. NT-proBNP levels were also significantly higher in diabetic patients, with a median of 2100 pg/mL [IQR 1200-3400] compared to 1450 pg/mL [800-2500] in non-diabetics (p = 0.04) which was statistically significant.

In our study, Fasting blood glucose was significantly higher in the T2DM group, with a mean of 160 ± 38 mg/dL versus 98 ± 22 mg/dL in non-diabetics (p < 0.001) this difference was statistically significant, and HbA1c was also elevated in diabetics (7.6 ± 0.9% vs 5.7 ± 0.4%, p < 0.001). this difference was statistically significant Renal function markers showed higher serum creatinine in T2DM patients (1.5 ± 0.5 mg/dL) compared to non-diabetics (1.2 ± 0.3 mg/dL, p = 0.01) this difference was statistically significant, with a corresponding lower estimated glomerular filtration rate (eGFR) in diabetics (58 ± 16 vs 68 ± 17 mL/min/1.73m², p = 0.02). This difference was statistically significant Serum sodium was slightly lower in T2DM patients (135 ± 4 mmol/L) versus non-diabetics (137 ± 3 mmol/L, p = 0.03) this difference was statistically significant. LDL cholesterol was higher in diabetics (112 ± 26 mg/dL) compared to non-diabetics (100 ± 27 mg/dL), although this difference was not statistically significant (p = 0.09).

In our study, echocardiographic parameters were compared between patients with T2DM (n=40) and non-diabetic patients (n=60). Left ventricular ejection fraction (LVEF) was significantly lower in the T2DM group, with a mean of 31 ± 7% compared to 35 ± 8% in non-diabetics (p = 0.02). Left ventricular end-diastolic diameter (LVEDD) and end-systolic diameter (LVESD) were slightly higher in diabetics (63 ± 8 mm vs 61 ± 6 mm and 52 ± 9 mm vs 50 ± 8 mm, respectively), but these differences were not statistically significant (p = 0.19 and 0.32). The E/e' ratio, a marker of diastolic dysfunction, was significantly elevated in the T2DM group (16.2 ± 4.8 vs 13.8 ± 4.1, p = 0.01), showing a non significant indicating worse diastolic function. Right ventricular dysfunction was present in 14/40 (35%) of diabetic patients versus 11/60 (18%) of non-diabetics, showing a non significant trend (p = 0.06). Moderate-to-severe mitral regurgitation was observed in 16/40 (40%) of T2DM patients compared to 14/60 (23%) of non-diabetics, also not statistically significant (p = 0.07).

In our study, cardiac magnetic resonance (CMR) parameters were compared between patients with T2DM (n=40) and non-diabetic patients (n=60). Late gadolinium enhancement (LGE), indicating myocardial fibrosis, was present in 30/40 (75%) of diabetic patients compared to 28/60 (47%) of non-diabetics (p = 0.007) was not significantly. The extent of LGE, expressed as a percentage of left ventricular mass, was significantly higher in the T2DM group (11.5 ± 5.8% vs 7.8 ± 4.9%, p = 0.01) was significantly. Native T1 time was slightly lower in diabetic patients (995 ± 35 ms) compared to non-diabetics (1012 ± 37 ms, p = 0.03), was significantly. While extracellular volume (ECV) fraction was significantly higher in the T2DM group (32.1 ± 5.1% vs 28.3 ± 4.8%, p = 0.004) was significantly.

In our study, the use of guideline-directed medical therapy was compared between patients with T2DM (n=40) and non-diabetic patients (n=60). ACE inhibitors or ARBs were used in 32 patients with T2DM and 50 non-diabetic patients (p = 0.68) was not significantly. Beta-blockers were prescribed to 36 patients with T2DM and 54 non-diabetic patients (p = 0.99), was not significantly. Mineralocorticoid receptor antagonists (MRAs) were used in 22 patients with T2DM and 40 non-diabetic patients (p = 0.21), was not significantly. SGLT2 inhibitors were significantly more common in the T2DM group, used in 18 patients with T2DM compared to 2 non-diabetic patients (p < 0.001). Loop diuretics were also used more frequently in T2DM patients, in 30 patients versus 28 non-diabetic patients (p = 0.006) was not significantly.

In our study, clinical outcomes were worse in patients with T2DM (n=40) compared to non-diabetic patients (n=60). All-cause mortality occurred in 12 patients with T2DM versus 6 non-diabetic patients (p = 0.01), was significantly .Heart failure hospitalizations were more frequent in the T2DM group, with 16 patients affected compared to 12 non-diabetic patients (p = 0.03) was significantly. The combined endpoint of death or heart failure admission was observed in 20 patients with T2DM and 14 non-diabetic patients (p = 0.005) was significantly. The annual mortality rate was higher in the T2DM group at 10.8% versus 4.3% in non-diabetics (p = 0.04) was significantly. These results indicate that patients with T2DM had significantly higher mortality, increased heart failure hospitalizations, and worse overall outcomes compared to non-diabetic patients.

Discussion

In our prospective observational study of 100 adult patients with non-ischemic dilated cardiomyopathy (NIDCM), 40 had type 2 diabetes mellitus (T2DM) and 60 were non-diabetic. Patients with T2DM were older (61.3 ± 9.8 vs 56.8 ± 11.8 years; p = 0.04) [11] and had higher BMI (28.5 ± 4.0 vs 25.7 ± 3.9 kg/m²; p = 0.01) [12], and more frequently had hypertension (32 vs 28 patients; p = 0.002) [13] and dyslipidaemia (26 vs 18 patients; p = 0.001) [14], while the male sex distribution, atrial fibrillation (12 vs 10 patients; p = 0.12) and duration of heart failure (28 ± 20 vs 31 ± 23 months; p = 0.56) [15] were similar between groups. Functionally, T2DM patients had worse status: systolic BP was modestly higher (122 ± 16 vs 115 ± 19 mmHg; p = 0.05) , NYHA class III-IV was more common (20/40 = 50% vs 16/60 = 27%; p = 0.03) [16], the 6-minute walk distance was lower (310 ± 85 vs 360 ± 88 m; p = 0.02) [17], and NT-proBNP levels were higher (median 2100 vs 1450 pg/mL; p = 0.04) .Laboratory findings included worse glycaemic control (fasting glucose 160 ± 38 vs 98 ± 22 mg/dL; p < 0.001; HbA1c 7.6 ± 0.9% vs 5.7 ± 0.4%; p < 0.001) , higher serum creatinine (1.5 ± 0.5 vs 1.2 ± 0.3 mg/dL; p = 0.01) [18], lower eGFR (58 ± 16 vs 68 ± 17 mL/min/1.73 m²; p = 0.02) , and slightly lower sodium (135 ± 4 vs 137 ± 3 mmol/L; p = 0.03) ; LDL cholesterol was higher in diabetics (112 ± 26 vs 100 ± 27 mg/dL) but not statistically significant (p = 0.09). Echocardiographically, T2DM patients had lower LVEF (31 ± 7% vs 35 ± 8%; p = 0.02) [19] and higher E/e′ ratio (16.2 ± 4.8 vs 13.8 ± 4.1; p = 0.01) ; groups did not significantly differ in LVEDD, LVESD, or in trends toward RV dysfunction (14 vs 11 patients; p = 0.06) or moderate-to-severe mitral regurgitation (16 vs 14 patients; p = 0.07) .CMR findings showed higher prevalence of late gadolinium enhancement (LGE) in diabetics (30/40 = 75% vs 28/60 = 47%; p = 0.007) [10], greater LGE extent (11.5 ± 5.8% vs 7.8 ± 4.9%; p = 0.01) , lower native T1 time (995 ± 35 vs 1012 ± 37 ms; p = 0.03) [20], and higher extracellular volume fraction (32.1 ± 5.1% vs 28.3 ± 4.8%; p = 0.004) .

Conclusion

In our study of patients with non-ischemic dilated cardiomyopathy, those with type 2 diabetes mellitus (T2DM) were older and had higher body mass index, with a greater prevalence of hypertension and dyslipidemia compared to non-diabetic patients. Functional capacity was worse in diabetic patients, who exhibited more severe heart failure symptoms, reduced exercise tolerance, and higher levels of NT-proBNP. Glycemic and renal parameters were also poorer in the T2DM group, accompanied by subtle electrolyte abnormalities. Echocardiography revealed lower left ventricular systolic function and signs of diastolic dysfunction in diabetic patients, with trends toward greater right ventricular involvement and mitral regurgitation. Cardiac MRI demonstrated increased myocardial fibrosis and higher extracellular volume in the T2DM group. Use of guideline-directed therapies was generally similar, although SGLT2 inhibitors were more commonly prescribed in diabetics. Clinically, T2DM patients experienced worse outcomes, including higher rates of heart failure hospitalization, combined adverse events, and mortality, indicating

that diabetes is associated with more severe disease and poorer prognosis in non-ischemic dilated cardiomyopathy.

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