



## MULTISYSTEMIC DYNAMICS AND CLINICAL COURSE OF CARDIOMYOPATHIC REMODELING IN TYPE 2 DIABETIC PATIENTS EXPOSED TO HIGH THERMAL BURDEN: STRATEGIC PERSPECTIVES ON INDIVIDUALIZED MANAGEMENT

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### KEY WORDS

*Type 2 diabetes mellitus, cardiomyopathy, hot climate, diastolic dysfunction, myocardial remodeling, environmental stress*

### ABSTRACT

*This reformulated title underscores the integrated pathophysiological mechanisms and evolving clinical patterns of diabetic cardiomyopathy (DCM) in individuals with type 2 diabetes mellitus (T2DM) inhabiting hot climatic environments. The review delves into heat-related metabolic stressors, cardiovascular maladaptations, and proposes patient-centered, climate-conscious therapeutic models tailored for sustained cardiometabolic resilience.*

T2DM is widely acknowledged as a multifactorial disease exerting substantial cardiovascular impact, with diabetic cardiomyopathy (DCM) emerging as a distinct and progressive myocardial condition. Independent of ischemic and hypertensive origins, DCM is increasingly recognized for its insidious onset and structurally degenerative course. In environmentally extreme regions such as Central Asia and the Middle East, climatic variables may significantly alter the pathophysiology and clinical manifestation of cardiac disease in diabetic individuals.

Heat stress imposes additional burdens through mechanisms such as volume depletion, electrolyte shifts, heightened sympathetic activity, and impaired circadian metabolic regulation. These factors collectively compromise cardiac homeostasis and hinder conventional management approaches. Given the paucity of focused regional data, this investigation aims to define the pathoclinical features of DCM progression under chronic heat exposure, facilitating informed intervention strategies.

### Materials and Methods

An observational cohort of 120 patients (age 40–65) diagnosed with T2DM and early-stage cardiomyopathic indicators was enrolled in Bukhara. Exclusion criteria included known coronary artery disease, severe hypertension, and structural congenital anomalies. The study spanned all seasonal phases across one year.

### Assessed variables included:

- Glycated hemoglobin (HbA1c), fasting plasma glucose, lipid indices
- Biomarkers: NT-proBNP, hs-CRP
- Echocardiography: LVEF, E/A ratio, LVMI, GLS
- 24-hour HRV and Holter monitoring
- Environmental recordings: ambient temperature, humidity

Patients were stratified into two groups based on ambient thermal exposure:

- Group A: summer peak ( $\geq 38^{\circ}\text{C}$ )
- Group B: temperate months ( $< 30^{\circ}\text{C}$ )

#### Results

Participants in Group A exhibited statistically significant reductions in diastolic performance (mean E/A:  $0.84 \pm 0.12$  vs.  $1.01 \pm 0.10$ ;  $p < 0.01$ ), elevated LVMI ( $128.6 \pm 12.4$  g/m<sup>2</sup>), and higher NT-proBNP levels. HRV analysis highlighted attenuated autonomic responsiveness, particularly in SDNN and RMSSD indices. Nocturnal hypertension and subclinical arrhythmias were markedly more prevalent during heat-exposure periods. Clinical symptoms—fatigue, palpitations, and exertional breathlessness—intensified in Group A during thermal surges. Multivariate modeling identified environmental heat, neurohormonal stress markers, and HRV metrics as independent predictors of myocardial decline.

Table 1. Comparative Functional and Laboratory Data

| Parameter         | Group A (High Temp) | Group B (Moderate Temp) | p-value  |
|-------------------|---------------------|-------------------------|----------|
| E/A Ratio         | $0.84 \pm 0.12$     | $1.01 \pm 0.10$         | $< 0.01$ |
| LV Mass Index     | $128.6 \pm 12.4$    | $114.3 \pm 10.8$        | $< 0.05$ |
| NT-proBNP (pg/mL) | $245 \pm 38$        | $180 \pm 30$            | $< 0.01$ |
| SDNN (ms)         | $112 \pm 15$        | $135 \pm 17$            | $< 0.05$ |

#### Discussion

This study elucidates the potentiating effects of elevated environmental temperature on cardiometabolic compromise in diabetic populations. The synergism between heat-induced hemodynamic stress, neurohumoral dysregulation, and structural myocardial remodeling contributes to accelerated disease progression. These findings support implementation of seasonally-adjusted care pathways, incorporating tailored pharmacologic regimens, hydration vigilance, and proactive cardiovascular monitoring.

#### Conclusion

Diabetic cardiomyopathy exhibits distinctive patterns of clinical deterioration in high-temperature settings. Climatic context should therefore be a critical consideration in cardiovascular risk assessment and management in T2DM care. Regional health systems must evolve toward climate-integrated models of chronic disease oversight, prioritizing early detection and intervention.

#### References:

1. Maisch B, Alter P, Pankuweit S. Diabetic cardiomyopathy—fact or fiction? *Herz*. 2011;36(2):102–115.
2. 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.
3. Boudina S, Abel ED. Diabetic cardiomyopathy revisited. *Circulation*. 2007;115(25):3213–3223.
4. Movahed MR, Hashemzadeh M, Jamal MM. Diabetes mellitus is a strong independent predictor of diastolic dysfunction. *Int J Cardiol*. 2007;122(1):137–138.
5. Lorenzo-Almorós A, Pello AM, Marín-Royo G, et al. Diagnostic approaches for diabetic

cardiomyopathy. Cardiovasc Diabetol. 2017;16:28.

6. Kovacic JC, Moreno P, Nabel EG, Hachinski V, Fuster V. Cellular senescence, vascular disease, and aging. Part 1 of a 2-Part Review. Circulation. 2011;123(15):1650–1660.

7. Jayasinghe S, Jayawardena R, Hills AP. Physical activity in South Asia: A narrative review. Indian J Endocrinol Metab. 2016;20(5):588–595.

8. Andersen CA, Andersson C, Berger JS, et al. Association between temperature and risk of myocardial infarction in the United States. JAMA Cardiol. 2020;5(1):1–9.

